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The effects of three different implementation strategies for heart failure guidelines on the management of heart failure in New Zealand primary care: a cluster randomised trial

VOLUME 1

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A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy in General Practice
The University of Auckland, 2011.
Abstract

This thesis aimed to evaluate the effectiveness of three implementation strategies on change over time in the primary care management of heart failure (HF) in New Zealand. Emergence of the Internet as a tool for continuing medical education (CME) prompted the study, which compared an Internet-based CME course, small–group education sessions and a passive mail-out of national guidelines. These were “one-off” educational interventions as is common in CME.

A single-blind stratified cluster randomised controlled trial (cRCT) design was used. Sixty nine practices were randomised to one of the three implementation strategies. The two active arms participated in almost identical education sessions based on four recommendations of the 2001 New Zealand Heart Foundation HF guideline. These recommendations were selected as primary study outcomes because their performance was suboptimal. Two of the recommendations – use of echocardiography and use of high dose angiotensin converting enzyme inhibitors (ACEi) – were not new in HF management. Introduction of β-blockers to the treatment schedule illustrated a dramatic change in accepted treatment. The indication for reintroducing spironolactone for HF had changed.

Patients were identified using a HF scoring system. Twenty four practices completed the study and 359 patients participated. Retrospective data were collected from the patient cohort for up to five years.

Most echocardiograms were performed before the educational intervention. In each group, ACEi prescription decreased over time, β-blocker use increased and spironolactone use remained static. None of the three implementation strategies had a statistically significant effect on outcomes. Two patient variables that negatively predicted echocardiography referral and prescribing were older age and female sex. The increase in β-blocker prescribing was mainly attributed to initiations that occurred
in secondary care before the educational intervention. The intervention arms did not affect the dose prescribed. Older age was negatively associated with dose.

None of the implementation strategies was superior in promoting change in primary care. Changes that occurred either were negative or could be attributed to factors operating beyond the control of the study, not to education. Ongoing work is required to develop interventions that will reduce barriers to changing HF management in primary care.
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“... non confundar in aeternum.”
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<tbody>
<tr>
<td>ACC</td>
<td>American College of Cardiology</td>
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<tr>
<td>ACEi</td>
<td>Angiotensin Converting Enzyme inhibitor</td>
</tr>
<tr>
<td>AGREE</td>
<td>Appraisal of Guidelines Research and Evaluation (Instrument)</td>
</tr>
<tr>
<td>AHA</td>
<td>American Heart Association</td>
</tr>
<tr>
<td>AHCP R</td>
<td>Agency for Health Care Policy and Research – now the AHRQ</td>
</tr>
<tr>
<td>AHRQ</td>
<td>Agency for Healthcare Research and Quality – formerly the AHCP R</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin Receptor Blocker</td>
</tr>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
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<tr>
<td>BNF</td>
<td>British National Formulary</td>
</tr>
<tr>
<td>BNP</td>
<td>Brain Natriuretic Peptide (see also NT-Pro BNP)</td>
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<tr>
<td>CABG</td>
<td>Coronary Artery Bypass Graft</td>
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<tr>
<td>CFPC</td>
<td>The College of Family Physicians of Canada</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary Heart Disease</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence Interval</td>
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<tr>
<td>CME</td>
<td>Continuing Medical Education</td>
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<tr>
<td>COPD</td>
<td>Chronic Obstructive Pulmonary Disease</td>
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<tr>
<td>cRCT / cRT</td>
<td>cluster Randomised Controlled Trial / cluster Randomised Trial</td>
</tr>
<tr>
<td>CTR</td>
<td>Cardio Thoracic Ratio</td>
</tr>
<tr>
<td>CXR</td>
<td>Chest X-Ray</td>
</tr>
<tr>
<td>DHB</td>
<td>District Health Board (NZ)</td>
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<tr>
<td>DM</td>
<td>Diabetes Mellitus</td>
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<tr>
<td>DTB</td>
<td>Drugs and Therapeutics Bulletin</td>
</tr>
<tr>
<td>EBM</td>
<td>Evidence-Based Medicine</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiograph / Electrocardiography / Electrocardiogram</td>
</tr>
<tr>
<td>Echo</td>
<td>Echocardiograph / Echocardiography / Echocardiogram</td>
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<tr>
<td>EF</td>
<td>Ejection Fraction</td>
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<tr>
<td>ESC</td>
<td>European Society of Cardiology</td>
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<tr>
<td>GP</td>
<td>General Practitioner</td>
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<tr>
<td>HF</td>
<td>Heart Failure</td>
</tr>
<tr>
<td>IPA</td>
<td>Independent Practitioner Association (NZ) became PHO</td>
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<tr>
<td>JVP</td>
<td>Jugular Venous Pressure</td>
</tr>
<tr>
<td>LV</td>
<td>Left Ventricle / Ventricular</td>
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<tr>
<td>Abbreviation</td>
<td>Definition</td>
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<tr>
<td>LVD</td>
<td>Left Ventricular Dysfunction</td>
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<tr>
<td>LVEF</td>
<td>Left Ventricular Ejection Fraction</td>
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<tr>
<td>LVF</td>
<td>Left Ventricular Failure</td>
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<tr>
<td>LVFx</td>
<td>Left Ventricular Function</td>
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<tr>
<td>LVSD</td>
<td>Left Ventricular Systolic Dysfunction</td>
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<tr>
<td>LVSFx</td>
<td>Left Ventricular Systolic Function</td>
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<tr>
<td>MI</td>
<td>Myocardial Infarction</td>
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<tr>
<td>MIMS</td>
<td>Monthly Index of Medical Specialities</td>
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<tr>
<td>NICE</td>
<td>National Institute for Clinical Excellence or since April 2005 National Institute for Health and Clinical Excellence.</td>
</tr>
<tr>
<td>NPV</td>
<td>Negative Predictive Value</td>
</tr>
<tr>
<td>NT-ProBNP</td>
<td>N-Terminal-Prohormone Brain Natriuretic Peptide (see also BNP)</td>
</tr>
<tr>
<td>NYHA</td>
<td>New York Heart Association</td>
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<td>NZGG</td>
<td>New Zealand Guidelines Group</td>
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<tr>
<td>OR</td>
<td>Odds Ratio</td>
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<tr>
<td>Pharmac</td>
<td>Pharmaceutical Management Agency (NZ)</td>
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<tr>
<td>PHO</td>
<td>Primary Health Organisation (NZ) previously IPA</td>
</tr>
<tr>
<td>PPV</td>
<td>Positive Predictive Value</td>
</tr>
<tr>
<td>PTCA</td>
<td>Percutaneous Transluminal Coronary Angioplasty</td>
</tr>
<tr>
<td>RAAS</td>
<td>Renin Angiotensin Aldosterone System</td>
</tr>
<tr>
<td>RCT</td>
<td>Randomised Controlled Trial</td>
</tr>
<tr>
<td>RNZCGP</td>
<td>Royal New Zealand College of General Practitioners</td>
</tr>
<tr>
<td>SIGN</td>
<td>Scottish Intercollegiate Guidelines Network</td>
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1. Introduction

1.1 Background

**IMPLEMENTATION OF EVIDENCE:** The translation of research evidence into practice can be challenging. This process is not made any easier by the sheer volume of published research and the wide range of conditions that general practitioners (GPs) manage. The production of guidelines can assist practice as they critically appraise available scientific evidence and present it as concise graded recommendations. The next stage is to use an implementation strategy that will successfully incorporate the new evidence into everyday use. The Internet has become a credible method of delivering educational interventions. However, many early studies of the Internet have based their results on pre-test and post-test knowledge and not on comparisons of patient outcomes before and after the intervention. (1-5) The usefulness of the Internet will only be known after a head-to-head comparison with another implementation strategy that is known to work in primary care, and with a control group to monitor external influences. The study will also need to be randomised and to have a sufficiently long follow up time.

**HEART FAILURE MANAGEMENT:** The effect of any implementation strategy will be small if the educational intervention is aimed at an outcome that results from processes which are already managed at a close to optimal level. If a condition is managed below this level there is scope for improvement. The onus is on the doctors to identify the gaps in their knowledge and performance so that they know when to change how they manage heart failure (HF) in their patients. The gaps in the way GPs manage HF have been well documented as have reasons for these gaps. The gold standard diagnostic test of echocardiography is not used in all suspected HF patients. (6-9) This may be because some GPs believe that their clinical diagnostic skills are sufficient or that they think the waiting list is too long or they make the decision to ration this resource and refer younger patients. (9-12) There is a large body of evidence behind the use of ACEi and at high doses but some GPs believe that a low dose is sufficient or are (wrongly) convinced that many patients will suffer adverse effects from high doses or find the titration process too time consuming. (9-20) The introduction of β-blockers to HF management regimens, after trials have demonstrated significant benefits in
mortality and morbidity, has meant a reversal of teaching and as such is a difficult concept to come to terms with and adopt. (10, 12, 21-24) Spironolactone has returned as an option in HF treatment after a landmark trial demonstrated reductions in mortality and morbidity; however the indication has changed from potassium-sparing diuretic to an aldosterone blocker, an important biochemical rationale for its use. (25-27) A recent systematic review has proved that aldosterone blockade reduces all-cause mortality in HF and reduced hospitalisations and increased ejection fraction. (28)

The New Zealand National Heart Foundation published an updated HF guideline (29) at the end of 2001 and this guideline was used as a base for the educational intervention developed for this thesis. The four recommendations mentioned above (echocardiography, ACEi, β-blocker and spironolactone use) were chosen as they represented evidence-based medicine and there were demonstrated gaps, both local and international, between current and optimal HF management. (6-8, 30-36) This was later confirmed in the study and in an audit of South Auckland practices. (37) Only four recommendations were selected because the message of what to do would have been lost with an increased number.

**STUDY DESIGN:** This study aimed to test the effectiveness over time of small group, Internet and guidelines-only education on HF management (via the four recommendations). The unit of randomisation was the practice, not the GP, to avoid contamination of the implementation strategies within practices. A cluster randomisation was appropriate for this type of study. Practices were stratified by size of practice to avoid confounding with GP variables that were associated with practice size. The participating practices were located in North and West Auckland, New Zealand.

To avoid the varying and confusing definitions of HF that currently exist, patients were identified using a standardised HF scoring system. (38) Data collection was retrospective and encompassed up to five years of medical records.

The proportion of patients who received an echocardiogram and the proportions of patients who received prescriptions for the three drug classes were calculated. The doses prescribed over time for the medications were measured. This study directed the interventions at GPs but since many HF patients are also managed by secondary care at some stage, it is possible that drugs are initiated during a hospital admission and so any new prescription will be attributed to the provider that made it.
This study also examined the effects of a HF diagnosis listed in patients’ medical record problems list and whether the length of time of diagnosis or no listing (but documented evidence to suspect HF) had an effect on their management. Patient participants included those with high scores indicative of HF, those with echocardiographic proof but also those whom the GP believed had HF. The NZ guidelines recommend treating with medication while waiting for the results of investigations.

Letters written to GPs by specialists after hospitalisation of the GP’s HF patients are also explored given the influence of secondary care on primary care HF management.

1.2 Conceptualising the thesis

The comparison was between a one-off CME event (small-group) format, commonly used in NZ to deliver medical education, and what was then a more novel method of delivering education: the Internet. The thesis aimed to assess which implementation strategy had the most effect on patient outcomes up to three years after the education was delivered.

The study was original in that it was longitudinal, following the management of HF in patient cohorts for up to 5 years (data collected from the 18 months preceding the intervention), using a cluster randomisation, and comparing the specified implementation strategies.

Both small group and Internet interventions were based on the same educational ‘curriculum’. The active intervention arms were provided with knowledge, scenarios in which this knowledge could be applied, and examples from practice. The small group participants had added input from a specialist and an academic GP. These two doctors may have acted as opinion leaders (expert and peer). The small group format also allowed interaction with other GPs and the opportunity to exchange experiences and concerns. Small group education has been shown to significantly improve prescribing decisions, professional practice and practice outcomes (compared with no intervention control). (39, 40) The Internet participants had the freedom to spend as much (or as little) time as they wanted, when they wanted in study. At the time the educational interventions were delivered, the method used for the control group – passive dissemination of the guidelines – was believed to be relatively ineffective as a mechanism of change.
Any organisational solutions were not offered to the participants as these may not have been suitable given the individual nature of the practices. Practices involved in the study were at varying degrees of paperlessness and electronic communication. Practices also differed in whose responsibility it was to manage their paper mail. Practice structures varied as did staff responsibilities and job descriptions.

The approach taken to this thesis was pragmatic rather than theory driven. The implementation strategies that were selected were those that could be delivered by the study team, and also were methods used by the primary care community. Once the educational intervention was delivered, it was left to the GPs to implement it into their practices using strategies that would be workable in their own environments.

The unique nature of the participating GPs must also be considered. The personal characteristics of the GPs (e.g. age, sex, personality, beliefs, past experiences) and other external and internal influences modify how they practice. Thus GPs’ approach to prescribing new drugs is not consistent. An added obstacle for HF is confirming diagnosis and GPs may lack familiarity with this condition.

Each practice has a unique dynamic which stems from the interaction of the GPs, other practice staff, the way the practice functions and the composition of the patient population of the practice.

These variables lead to the question of whether the barriers to optimal practice and making change are the same for each GP and for each practice. Would the attempts to identify the barriers to change inform the GPs about what changes they needed to make thereby rendering the testing of implementation strategies redundant?

Identifying barriers to change for one problem across all GPs, and then tailoring an intervention to challenge and resolve all the barriers poses significant challenges. The complementary approach is to identify drivers to change (external and internal) and harness these as well, but again, are these the same and equally effective for all GPs?

The influence of secondary care adds a further layer of complexity to any potential implementation strategy. GPs may be reluctant to change management specified by the hospital, which suggests a requirement for simultaneous education at different levels and clear communication between levels.

Added to these challenges is the condition studied for this thesis. The complexity of HF management should not be underestimated. In HF there is an initial problem of
establishing diagnosis. Even once confirmed, it does not automatically result in proper management for patients. HF is a complex syndrome, usually found in older age and accompanied by multiple co-morbidities. The management for the co-morbidities may require reference to a number of other individual guidelines, leaving the GP to integrate the recommendations that are beneficial to the patient and the patient to concur and/or take the medication. The reasons for not prescribing or stopping a medication or information such as NYHA class, used as a rule of thumb for prescribing β-blockers and spironolactone, are usually not documented in primary care medical records (or in secondary care) so the verifiable appropriateness of drug regimens cannot be determined.

1.3 Structure of the Thesis

This thesis is divided into seven chapters. The objectives of each chapter are as follows:

Chapter 1. Introduction. To describe the background to this study.

Chapter 2. Translating evidence into practice. This chapter describes the guideline used in this study and attitudes towards guidelines and evidence-based medicine (EBM). The next section describes different sources of evidence, their credibility, and how change occurs through dissemination rather than implementation. GP and practice characteristics that make them more likely to implement change are discussed as well as the responsibility to keep knowledge up to date. Chapter 2 also describes barriers to implementation, theoretical domains that could influence implementation, and different implementation methods and their successes. The methods discussed are those that could be used to communicate recommendations on the management of HF.

Chapter 3. Heart failure. This chapter discusses patient characteristics that increase the risk of a patient developing HF, prevalence and incidence, signs, symptoms, and diagnostic tests recommended for use in HF. Emphasis is given to information about echocardiography use since this is one of the study outcomes. This chapter also examines the barriers to changing practice in primary care, including some specific to HF and then compares the characteristics of patients included in clinical trials of HF with the ‘typical’ HF patient found in primary care. The main medications used in HF and the evidence behind them are also outlined along with studies that illustrate HF
treatment in primary care, patient factors that could influence this treatment, and a brief overview of secondary care management for HF.

**Chapter 4. Methods.** This chapter describes the methodology, how patients were identified and the data that were collected from their medical records. Details of the methods of analysis used in thesis are also given.

**Chapter 5. Results.** This chapter describes the characteristics of the practices, GPs, and patients involved in the study. Chapter 5 explains the analyses of the effect of the study arm (implementation strategy) on receipt of echocardiography, prescription of ACEi, β-blocker and spironolactone, and doses of interest. This chapter then describes who was responsible for making the diagnosis, how many medications were initiated in the study time periods and who initiated the medications of interest. This chapter also presents the results from the analyses of the effect that a listed diagnosis of HF (i.e. recorded in the problem list of a patient’s medical record) had on the four study outcomes.

There is comment on samples of letters from specialists to GPs. Extracts from one patient’s medical record are then presented to demonstrate how their GP considered initiating a β-blocker. This is included as a rare example of ‘think out aloud’ reasoning which clearly documents the GP’s intent.

**Chapter 6. Discussion.** The Discussion chapter considers the findings of the study and compares them with other research. This discussion consists of comparisons of prescription rates (primary outcome), doses prescribed (secondary outcome), and the responsibility for diagnosis, initiation of medications and electrolyte testing related to spironolactone initiation (tertiary outcomes). Findings from the surveys and questionnaire are used to support the study outcomes and are compared with overseas data. The effect of listed diagnosis on HF management (post-hoc analysis) is described. More recent studies regarding the changing primary care management of HF, and the use of the Internet as an implementation strategy are also discussed, as is the effectiveness of the implementation strategies used in this thesis. Strengths and weaknesses of this study are examined. Suggestions for recommendations in practice, policy and research are proposed.

**Chapter 7. Conclusion.** The findings of this study and their implications are summarised.
2. Translating Evidence into Practice

2.1 Introduction

This chapter discusses issues relating to the transfer of research evidence, regarding the optimal management of HF, into general practice. It describes what a guideline is and how it should be used, before considering the New Zealand Heart Foundation HF guideline that was used as the base of the educational interventions of this thesis. A discussion follows of GPs’ attitudes towards evidence and guidelines (section 2).

Section 3 examines the sources of information that GPs use, how much they are influenced by them, their preferred format for receiving information, and how quickly and effectively they adopt this information without any implementation strategies. Much of the data in section 3 have been obtained from surveying respondents whose representativeness is uncertain. Their unvalidated responses may not represent true patterns of practice and behaviour. There is also a discussion of which personal and professional characteristics of GPs make them most likely to implement change, and which practice characteristics increase the likelihood of implementation of change.

This chapter’s main section (section 4) focuses on organised methods of implementing change in clinical management, using where possible Cochrane reviews to present a broad scope of carefully selected studies, but also including individual trials of interest. The Cochrane reviews identified much heterogeneity between studies so any attempts at meta-analysis may not have resulted in additional knowledge. As efforts to change practice can result in less than impressive outcomes, so it is important to consider both the enablers and barriers to changing practice in primary care. Different implementation strategies have different influences on the uptake of evidence, however there are many competing theories regarding successful implementation. Understanding is needed of both the explicit and implicit theories. (41) The difficulties lie in operationalising the constructs of these theories (41, 42) and pre-determining the effects of context and participants in an educational intervention. One method to testing implementations strategies is to use a pragmatic approach which is defined as:

“solving problems in a realistic way which suits the present conditions rather than obeying fixed theories, ideas or rules” (43)
A pragmatic approach was used to develop and conduct this study of Internet, small group and self-directed CME.

**SEARCH STRATEGIES:** Medline and EMBASE were used for search strategies that included the terms or contractions of the terms: “Internet”, “Web-based”, “Continuing Medical Education”, “Medical Education”, “General Practice”, “Primary Care”, “Heart Failure”, “Small group”, “Guidelines”, “Implementation”.

The Effective Practice and Organisation of Care (EPOC) Cochrane library was also searched for relevant implementation strategies.

English language articles only were used, as it was not feasible to pay the additional costs associated with translation. Study populations were adults only. Searches were not limited by study type.

Additional papers were suggested by the study team as well as by other members of the Department of General Practice and Primary Care, University of Auckland. Other articles were identified during retrieval of articles identified in searches, or from reference lists. Some hand searching was carried out in the British Journal of General Practice, European Heart Journal, European Journal of Heart Failure, Journal of Continuing Education in the Health Professions, the New Zealand Medical Journal, and New Zealand Family Physician (NZ journal, not indexed).

### 2.2 Guidelines and evidence-based medicine

From a definition by the Institute of Medicine, [Clinical Practice] Guidelines are:

“systematically developed statements to assist practitioner and patient decisions about appropriate healthcare for specific clinical circumstances”. (44)

The development of evidence-based guidelines began in the 1990s, as evidence-based medicine became increasingly recognised as the current theory of best clinical practice. (45) The concept of evidence-based medicine (EBM) emphasises the application of the best available research for the care of patients, (46) but research evidence alone does not warrant clinical decision-making for individual patients. (47-49) EBM now requires the use of clinical expertise to integrate research evidence along with the clinical state and circumstances and patient values and preferences. (48-51)
Guidelines need to be based on valid scientific evidence, be appealing to the intended user and offer practical routes to their application. (52, 53) A good definition of a guideline is given as:

“an attempt to distil the best available scientific information into a coherent, graspable, working knowledge base for the busy practitioner.” (54)

Guidelines set out optimum management approaches for managing a given condition. They aim to improve healthcare outcomes and health service efficiency, and reduce inappropriate practice variation between doctors. (52, 55-57) As the name suggests, they are guide lines that allow for differences between patients while helping doctors not to deviate too far from the track. However, a good guideline that is not referred to is of no use. (54) Adherence to guidelines can be expected for at most 80 to 90% of eligible patients whereas absolute adherence would suggest a blind following of recommendations via an “obedience-based medicine” (58) that could be hazardous for the patient. (44)

Guidelines have also been viewed in a negative light. There may be uncertainty around their effectiveness in bringing about change, they could be used in litigation and they pose a challenge to the nature of professionalism by representing a reduction in clinical freedom and autonomy. (52, 59-62) The “cookbook” argument is that the “recipes” (recommendations) are simple, or even reductionist, and prescriptive. They do not capture the complexity of the medications and patient characteristics which may differ from those reported in the randomised controlled trials (RCTs) comprising the evidence base for the guidelines. (54, 59, 62-64) Guidelines therefore risk a depersonalised form of medicine antithetical to ‘best patient care.’

In an editorial entitled “Clinical trials and the real world: selection bias and generalisability of trial results”, Sharpe maintains that clinical experience and judgement are still needed for appropriate interpretation and implementation of any guideline. (65) He adds that in decision areas where evidence is inadequate or lacking, the skills of clinical experience and judgement are critical. The 1997 New Zealand guideline for the management of chronic HF states how clinical management can and should vary between patients. It emphasises that it is inappropriate to view guidelines as a rigid prescription for the management of HF. (66) Surprisingly this statement was not included in the updated 2001 version of the guideline. (29) Ultimately guidelines are not the final word in management. (54, 64)
Doctors also need to recognise that published guidelines (indeed any published material) can quickly become outdated as its evidence base becomes superseded by new research. (63, 67) There is a question of balance; how many recommendations need to be invalid before a guideline is updated and can a single out-dated recommendation invalidate a whole guideline? Other questions to consider when evaluating the validity of guidelines are: 1. Have interventions been superseded or replaced? 2. Is there new evidence that changes the relation between benefits and harms? 3. Have outcomes, originally considered important been shown to be unimportant (or vice-versa)? 4. Is there evidence that the guideline is no longer needed – that disease management is optimal? (67)

The currency of 19 clinical practice guidelines whose development was facilitated by the Agency for Healthcare Research and Quality (AHRQ) was assessed (17 of which were still in use by 2000 when the study took place). The authors approached former guideline panel members and searched the literature (although this was criticised by an accompanying editorial (68) for not including some key journals). Out of the 17 guidelines assessed, only 3 were still valid, 7 needed a major update, 6 required a minor update, and no conclusion was able to be reached for one guideline. A survival analysis of the guidelines indicated that 90% of guidelines would be valid at 3.6 years [95% Confidence Interval (CI) 2.6 to 4.6 years], but only 50% of the guidelines would still be valid at 5.8 years (5.0 to 6.6). The topic of the guideline will also influence how long a guideline remains valid since rapid scientific advances render a guideline obsolete more quickly. (69) Guidelines based mainly on level I evidence (high quality), or having recommendations that are strength of evidence A (see next page for further definition), will withstand change better than those based on lower grade evidence. (68) The authors suggest revision of guidelines three years after completion (i.e. the rounded lower CI of 90% validity) although this may depend on the speed at which the topic evolves. (69)

Clinical practice guidelines tend to be developed in high volume and more costly practice areas where there is more high quality evidence to develop management recommendations (65) and change is feasible and will improve patient outcomes. An example is cardiovascular medicine, described as “relatively evidence-replete” with many RCTs in the fields of hypertension, dyslipidaemia, coronary heart disease, and HF. (65) Guidelines are less commonly produced for topics where there is uncertainty regarding the appropriateness or effectiveness of current practice. (64)
The development of evidence-based guidelines requires the objective interpretation of a systematic review of the literature. (44, 54) The development process organises and evaluates available data to demonstrate which research findings are most likely to give benefit, adding expert opinion where the research evidence is either unavailable or inconclusive. (44, 53, 54) This evaluation entails grading systems as described below.

### 2.2.1 Grading systems

The recommendations in guidelines are accompanied by strength of evidence grades. Two main systems – the American AHCPR and the Scottish Intercollegiate Guideline Network (SIGN) – were in use when the 2001 New Zealand HF guideline was written. Other guideline organisations use different systems and where evidence grading systems have been updated this is indicated.

The AHCPR rates the quality of research evidence according to the study methodology used to generate the evidence. This classification system is then condensed for use in guidelines, (29) as described in table 2.2.a.

Table 2.2.a. AHCPR evidence source, levels and grades

<table>
<thead>
<tr>
<th>Evidence</th>
<th>Source</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level I</td>
<td>Large, well-conducted randomised controlled trials (RCTs)</td>
<td>A = good evidence</td>
</tr>
<tr>
<td>level II</td>
<td>Small, well-conducted RCTs</td>
<td></td>
</tr>
<tr>
<td>level III</td>
<td>Well-conducted cohort studies</td>
<td></td>
</tr>
<tr>
<td>level IV</td>
<td>Well-conducted case-control studies</td>
<td></td>
</tr>
<tr>
<td>level V</td>
<td>Uncontrolled or poorly controlled studies</td>
<td>B = fair evidence</td>
</tr>
<tr>
<td>level VI</td>
<td>Represents conflicting evidence, but tending to favour the recommendation</td>
<td></td>
</tr>
<tr>
<td>level VII</td>
<td>Expert opinion</td>
<td>C = expert opinion</td>
</tr>
</tbody>
</table>

The levels of evidence and strength of evidence classification system was used in the 1994 AHCPR publication, HF: Evaluation and Care of Patients with Left-Ventricular Systolic Dysfunction. (70) A variation on the AHCPR grading system was used by SIGN and National Institute of Clinical Excellence (NICE) and incorporates evidence from meta-analysis. Their statements of evidence are seen in table 2.2.b. (71)
Table 2.2.b. Grading system used by SIGN and NICE

<table>
<thead>
<tr>
<th>Evidence</th>
<th>Source</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>From meta-analysis of RCTs</td>
<td>A = at least one RCT as part of a body of literature of overall good quality and consistency addressing the specific recommendation</td>
</tr>
<tr>
<td>Ib</td>
<td>From at least 1 RCT</td>
<td></td>
</tr>
<tr>
<td>IIa</td>
<td>From at least one well-designed controlled study without randomisation</td>
<td>B = availability of well conducted clinical studies but no RCTs on the topic of recommendation</td>
</tr>
<tr>
<td>IIb</td>
<td>From at least one other type of well-designed quasi-experimental study</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>From well-designed non-experimental descriptive studies (comparative studies, correlation studies or case studies)</td>
<td>C = evidence from expert committee reports or opinions and/or clinical experiences of respected authorities. There is an absence of directly applicable clinical studies of good quality.</td>
</tr>
<tr>
<td>IV</td>
<td>From expert committee reports or opinions and/or clinical experience of respected authorities.</td>
<td></td>
</tr>
</tbody>
</table>

Another level of recommendation is added in the form of a Good Practice Point which recommends best practice from the clinical experience of the guideline development group. (71)

Management of chronic heart failure in primary and secondary care guidelines (2003) from NICE have added two further levels of hierarchy and grades of evidence. These are: (72)

Table 2.2.c. Additional grades of evidence from NICE guidelines

<table>
<thead>
<tr>
<th>Grade</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>DS</td>
<td>Evidence from diagnostic studies,</td>
</tr>
<tr>
<td>NICE</td>
<td>Evidence from NICE guidelines or health technology appraisal programmes.</td>
</tr>
</tbody>
</table>

The NICE HF guidelines have since been updated (Chronic Heart Failure. National clinical guideline for diagnosis and management in primary and secondary care, 2010) and currently use the GRADE systema to evaluate the quality of evidence. (73)

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a GRADE is described at the end of this section.
In comparison the current SIGN grading system [used in the latest update of the SIGN Management of chronic heart failure, a national clinical guideline 2007 (74)] is more complex than the previous system with weightings given within the levels of evidence. The +/- rating (see table 2.2.d, next page) is determined by the number of criteria on the checklist which are fulfilled, and the likelihood of the conclusions of the study changing where the criteria are not met or explained (see table 2.2.d). This rating is then condensed into 4 grades of recommendation (table 2.2.e, next page). (75) This system was published in 2001; two years after SIGN had released their HF guidelines.

The European Society of Cardiology (ESC) HF guidelines (2001) (76) and the American College of Cardiology (ACC) / American Heart Association (AHA) guidelines for the evaluation and management of chronic HF in the adult (published in 2001) (77) use similar abbreviated forms of levels of evidence (see table 2.2.f).

The ESC HF guidelines have been updated twice [2005 (78) and 2008 (79)], and the levels of evidence as described above have also been updated. A further ESC guideline update is expected in 2011/12. (80) The ACC/AHA guidelines were updated in 2005 (81) and in 2009 (82) with similar levels of evidence definitions (see table 2.2.f).

The ESC [except 2001 guidelines (76)] and ACC/AHA assign Classes of Recommendations to the evidence. The ESC and ACC/AHA treatment/procedure recommendations range from beneficial/useful/effective to equivocal and to not useful/ineffective/potentially harmful. (77-79, 81) Or can be described as a benefit/risk continuum. (82)

The ACC/AHA guideline developers explain that the strength of evidence does not necessarily reflect the strength of recommendation. (77, 82)

The 2001 guidelines for the management of patients with chronic HF in Australia grades recommendations on levels of evidence from I through to IV. These levels are derived from an Australian publication from the National Health and Medical Research Council (NHMRC), and include the addition of Expert Opinion (EO). (83) The updated 2006 Australian HF guidelines (84) now include Grades of recommendation from A to D, similar to the ESC and ACC/AHA Levels of Evidence. (76-79, 81)

The NZ HF guideline 2009 update was published in June 2010. (85) The revised guideline utilises the same levels of evidence and the Grades of recommendation as the 2006 Australian HF guidelines. (84, 85)
Table 2.2.d. SIGN revised grading system for levels of evidence

<table>
<thead>
<tr>
<th>Evidence</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 ++</td>
<td>High quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias</td>
</tr>
<tr>
<td>1 +</td>
<td>Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias</td>
</tr>
<tr>
<td>1 –</td>
<td>Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias</td>
</tr>
<tr>
<td>2 ++</td>
<td>High quality systematic reviews of case-control or cohort studies or High quality case-control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal</td>
</tr>
<tr>
<td>2 +</td>
<td>Well conducted case-control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal</td>
</tr>
<tr>
<td>2 –</td>
<td>Case-control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal</td>
</tr>
<tr>
<td>3</td>
<td>Non-analytic studies e.g. case reports, case series</td>
</tr>
<tr>
<td>4</td>
<td>Expert opinion</td>
</tr>
</tbody>
</table>

Table 2.2.e. SIGN revised system for grades of recommendations

<table>
<thead>
<tr>
<th>Grade</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At least one meta-analysis, systematic review, or RCT rated as 1 ++ and directly applicable to the target population or A systematic review of RCTs or a body of evidence consisting principally of studies rated as 1 + directly applicable to the target population and demonstrating overall consistency of results</td>
</tr>
<tr>
<td>B</td>
<td>A body of evidence including studies rated as 2 ++ directly applicable to the target population and demonstrating overall consistency of results or Extrapolated evidence from studies rated as 1 ++ or 1 +</td>
</tr>
<tr>
<td>C</td>
<td>A body of evidence including studies rated as 2 + directly applicable to the target population and demonstrating overall consistency of results or Extrapolated evidence from studies rated as 2 ++</td>
</tr>
<tr>
<td>D</td>
<td>Evidence level 3 or 4 or Extrapolated evidence from studies rated as 2 +</td>
</tr>
</tbody>
</table>

† derived from Harbour et al. 2001 (75)
Table 2.2.f. Level of Evidence definitions from ESC and ACC/AHA guidelines over time

<table>
<thead>
<tr>
<th>Grade</th>
<th>Level of Evidence Definition</th>
<th>ESC 2001 (76)</th>
<th>ACC/AHA 2001 (77)</th>
<th>ESC 2005, 2008 (78, 79)</th>
<th>ACC/AHA 2005, 2008 (81, 82)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At least 2 randomised trials supporting recommendation</td>
<td>Data derived from multiple randomized clinical trials</td>
<td>Data derived from multiple randomised clinical trials or meta-analyses</td>
<td>Data are derived from multiple randomized clinical trials or meta-analyses. *Multiple populations evaluated.</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>One randomised trial and/or meta-analysis supporting recommendation</td>
<td>Data derived from a single randomized trial or nonrandomized studies</td>
<td>Data derived from a randomised trial or large non-randomised studies</td>
<td>Data are derived from a single randomized trial, or non-randomized studies. *Limited populations evaluated.</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Consensus statement from expert based on trials and clinical experience</td>
<td>Consensus opinion of experts was the primary source of recommendation</td>
<td>Consensus of opinion of the experts and/or small studies, retrospective studies, registries</td>
<td>Only consensus opinion of experts, case studies, or standard of care. *Very limited populations evaluated.</td>
<td></td>
</tr>
</tbody>
</table>

*Added in 2009 guideline (82).
The Advisory Council To Improve Outcomes Nationwide in Heart Failure (ACTION-HF, 2001) describes itself as a supplement to raise awareness of new issues in HF management and does not include evidence levels or strength of evidence gradings. (86)

The issue that could occur with different systems is the same study types/trials are identified as different evidence levels of evidence. The corollary of this is that the same articles will be assigned different grades of evidence. Doctors in some countries will practise with certainty (implementing recommendations with A grades) whereas in other countries the recommendations will appear less robust. While one objective of using guidelines is to standardise local management of conditions, equivalence of grading systems would assist in promoting international consistency in the management of conditions.

The problems raised by differing quality of evidence and the grade A recommendations that are used by guideline writers (87) and potential solutions have been proposed by the Grading of Recommendations Assessment, Development and Evaluation (GRADE) Working Group. (88) GRADE has proposed a sophisticated approach to guideline recommendation development. All members of the guideline development team (including patients and advocates) rank the importance of outcomes from not important to critical. (89, 90) Values and preferences are clearly acknowledged, (89, 91) which is a new development for guidelines. The quality of the evidence for each outcome is defined from High to Very Low (i.e. from robust estimate of effect which further evidence is unlikely to change to a very uncertain estimate of effect), RCTs are initially graded as High quality, observation studies as low quality evidence, but quality may be up- or downgraded. (89-92) Recommendations are either Strong or Weak/Conditional and either for or against that aspect of management. (89, 92)

**SUMMARY:** Guidelines aim to present the best possible research evidence to assist doctors in optimal patient management. A helpful aim would be for all guideline developers to use the same evidence and recommendation grading systems in order to standardise the way in which conditions are managed.
2.2.2 A description of the New Zealand Heart Failure Guideline

The 2001 New Zealand Heart Foundation Guideline for the management of HF (NZ HF guideline) is a 29 page document including evidence tables, with two pages of algorithms: one for diagnosis and one for treatment. (29) The CME course that was developed for this thesis was based on the 2001 NZ HF guideline. (29) A version of this guideline can be found in Appendix 4E.

The guidelines prior to the 2001 version were published in 1997 in the NZ Medical Journal (66), and were aimed primarily at GPs, since they manage most HF patients and see them at an early stage of the condition. In addition, specialist review was suggested when necessary, depending on the stability and severity of the condition. The objective of review was to establish the diagnosis, individualise treatment and initiate specialist investigations where needed. (66) The 1997 guideline states that echocardiography is “the single most informative diagnostic test and is an essential base measurement in heart failure” which has wide availability in most urban areas. (66) Neither levels of evidence nor recommendations with strength of evidence grades are included in the 1997 NZ guideline.

The 1997 guideline was reviewed with six other HF guidelines that referred to diagnosis (AHA, AHCPR, Canadian, European, Irish and SIGN) (93), using an early version of the AGREE tool. (94) The guidelines were scored on the following dimensions (94):

- Rigour of development (20 items that assess validity and reproducibility, the composition of the development group, interpretation of evidence, links between evidence and recommendations, peer review and updating)
- Content and context (12 items that relate to reliability, applicability, flexibility and clarity)
- Application (5 items that look at implementation, dissemination and monitoring strategies)

The 1997 NZ HF guideline scored poorly on rigour of development (score 10%) and on application (0%) but highly on context and content (83%). (93) The final score was 32.4%. It was ranked 4th equal out of seven HF guidelines, with three of the top five

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a This is the version of the guideline was supplied to the study in late 2001 and the formatting differs from the one that was published. Any references in this thesis to the NZ HF guideline are to the format that is included in Appendix 4E.

b The AHCPR guideline was ranked first with a score of 78.4%, second was the SIGN guideline with 70.3% and third was the AHA which scored 37.8%. (93)
guidelines pre-dating the NZ guideline. (93)

The 2001 HF guideline (29) describes the AHCPR quality rating scale and uses the strength of evidence grades for recommendations. The statement regarding echocardiography changed; echocardiography was now described as not widely available and subject to considerable delays in patient access. The diagnostic algorithm stated that if assessment of left ventricular (LV) function is delayed due to local resource constraints, then treatment should continue on an empirical basis. (29) This concept of treatment is a cornerstone of this thesis given the real concerns over waiting times for echocardiography.

The following statement is a brief description in response to each of the questions in the current AGREE tool (95) for the 2001 NZ HF guideline. (29)

**SCOPE AND PURPOSE:** The objectives of the 2001 NZ HF guideline were to reduce morbidity and mortality which would be demonstrated through either functional scores or hospital admission. Another stated objective was to improve patients’ understanding and satisfaction with their health care. The clinical questions covered by the guideline are not specifically described at the beginning. Recommendations are given throughout the guideline, usually accompanied by key points and comments. The introduction gives details of the areas that required further review since the last version of the guideline. The guideline applies to patients with an established diagnosis of congestive HF due to systolic ventricular dysfunction and also states that there is a commentary on diagnosis to further define the patient population to which the guideline refers.

**STAKEHOLDER INVOLVEMENT:** The guideline development group included GPs some of whom held positions within their local GP Associations, cardiologists, and staff members of the National Heart Foundation. The medical director of Merck, Sharp and Dohme, Ltd was also on the guideline team. There was no evidence that patient perspectives had been sought. The title page referred to the guideline as the ‘Doctor’s Guide’ but it was not further defined as primary or secondary care or both. The wording was changed to ‘Health Professionals Guide’ at the final printing.

It is not clear whether the guideline was piloted among target users. There was reference to the development of the guideline using a systematic approach developed by the NZ Guidelines Group and the Guidelines for Guidelines Trust.

**RIGOUR OF DEVELOPMENT:** A systematic search identified explicitly developed evidence-based guidelines. The 1994 AHCPR guideline was selected as being
systematically developed from a review of external evidence and the strength of
evidence was given for each recommendation. Areas requiring further review and
evaluation were then selected. A Medline search was run by a medical librarian and
each paper was reviewed by a member of the guideline team and strength of evidence
grades (AHCPR) were assigned. Final decisions were by consensus. The health
benefits, side effects and risks were given for the medications and interventions
described. The recommendations included strength of evidence grades and generally a
link to the references on which they were based. The draft guideline was peer reviewed,
but only for format, presentation and utility. An annual review of the guidelines will be
undertaken by a Heart Foundation Committee, to decide whether updates are required.

**CLARITY AND PRESENTATION:** The recommendations were concise and specific.
There were few options for management. The key recommendations were in bold and
there were colour-coded algorithms for diagnosis and treatment. The additional tools
for application apart from the algorithms included a patient booklet on living with HF and a video for Maori patients.

**APPLICABILITY:** The only potential organisational barrier referred to was the delay in
assessment by echocardiography. There was no discussion of potential cost
implications apart from a suggestion that future research could assess the cost-
effectiveness of echocardiography in improving outcomes for patients with LV failure.
No review criteria were specified but referring patients for echocardiography and target
doses of medications could be assumed to form monitoring criteria.

**EDITORIAL INDEPENDENCE:** A one-off grant from Merck Sharp and Dohme
(MSD) sponsored the initial guideline development but expenditure was at the
discretion of the guideline team and remaining funds were returned to the sponsor. The
only team member to receive remuneration from the sponsor was the MSD
representative. MSD and Roche supported the printing of the revised guideline. No
other conflicts of interest were recorded.

**SUMMARY:** The 2001 NZ HF guideline was clearer in the recommendations for
management than the 1997 version. The 2001 guideline seemed to be directed at all
members of the health professions who assist in the care of HF patients. The
development of the 2001 guideline was based on another comprehensive guideline. It is
not clear whether the guideline had been piloted on the target users or if patients’ views
or preferences had been sought. The algorithms were a clear and concise reference tool.
2.2.3 Primary care attitudes towards guidelines and EBM

GPs hold personal views on the worth or value of guidelines and EBM which reflect factors relating to the guidelines themselves, as well as characteristics of the GPs and how they practise medicine. A systematic review (not restricted to GPs) found that clinicians’ attitudes to clinical practice guidelines were overwhelmingly positive. (96) “Guidelines are a helpful source of advice” was agreed with by 80% of respondents, followed closely by “guidelines are good educational tools” and “guidelines are intended to improve quality of care”. (96) Only 26% of respondents agreed with the statement “guidelines are impractical and too rigid to apply to individual patients”. (96)

In surveys of GPs about attitudes to guidelines and EBM, feelings tended to be positive with most respondents agreeing that:

- Positive attitudes are held towards EBM/guidelines (61, 97, 98)
- Practising EBM/following guidelines improves patient care and quality improvement (59, 61, 97, 98)
- EBM/guidelines are useful in day-to-day patient management and will help with diagnosing and managing particular conditions (61, 97, 98)
- Guidelines enable GPs to use the latest knowledge derived from research and are a useful method of accessing expert information (59)
- Evidence-based guidelines are more accessible as a guide to current best practice than research articles (60)
- Levels of evidence highlight what the GPs should know and what areas of practice they should change. (60)

However there were also mixed feelings about the worth and applicability of clinical trials. In one study 93% of GPs felt capable of adapting the guidelines to individual patient needs. (61) In another study clinical trials were seen to be far removed from primary care and the exclusion criteria applied to trials limited their generalisability. (60) (See Chapter 3, section 3.2 for a further discussion of this view.) Clinical experience was thought to influence decision-making, possibly more than research evidence, and what had to be added to this were the context and psychosocial issues that occur within primary care. (60, 99) Contrasts were made between the ‘art’ of medicine and the ‘science’ of EBM, and the fear that EBM was promoted to the exclusion of other skills. (60)
EXTERNAL INFLUENCES ON USE: A range of different factors to improve the use of guidelines and EBM were suggested by GPs. The most pressure to use guidelines came from the Department of Health. (59) Endorsement by Government or health regulatory bodies had a negative effect on possible uptake but endorsement by professional colleges or networks was seen to be positive. (100) Other highly rated methods that would persuade GPs to use guidelines included Continuing Medical Education (CME) events, discussions with local colleagues, one-to-one meetings and feedback on practice. (59, 100) Some GPs were highly influenced by hospital and specialist practices (described in section 4.8 of this chapter). (60) In one study 56% of GPs strongly agreed that personal contact with specialists was much more useful than guidelines. (61) The opinion of colleagues and local experts (see section 4.5 of this chapter for a fuller description) may improve the uptake of EBM more than the publication of level 1 evidence (see section 2 in this chapter). (60)

GPs were asked how to move from opinion-based practice to evidence-based practice and almost 60% of respondents suggested using evidence-based guidelines or protocols developed by colleagues. (97) The source of the guidelines/evidence that influenced the GP and those that were trustworthy included guideline developers, local specialists and meta-analyses, but evidence from pharmaceutical companies was not trustworthy. (60) GPs preferred short guidelines or flow charts (one to two sides) and guideline use could be increased if the guidelines were clear, simple and of high quality. (61) A greater understanding of recommendations may produce a more positive attitude towards recommendations and a greater likelihood that these will be implemented. (101)

However, these studies assume that GPs are aware that guidelines exist. When compared with cardiologists, GPs were overall significantly less aware of the HF guidelines (AHCPR / ACC / AHA) with 30% of them and 9% of cardiologists (P < 0.01) being aware but not having read the guidelines. Only 8% of GPs had reviewed the guidelines in full compared with 38% of cardiologists. Approximately equal proportions of these specialities had read the guidelines briefly and 8% of cardiologists were unaware of the guidelines compared with 17% of GPs. (102) These findings may be explained by the sheer volume of guidelines that are applicable to primary care.

Guidelines in general practice have been described as the new ‘Tower of Babel’ after a study asked a third of local practices to produce copies of the guidelines they had. (103) There were 855 guidelines of which about half were one to two pages long, and 20%
were 10 or more pages. 38% of the guidelines were undated, making it difficult for GPs to know if they were practising current or obsolete medicine. The amount of paper represents an unmanageable resource which is of little use in decision making at the point of clinical contact. (103)

**DEMOGRAPHIC INFLUENCES:** Outstanding factors that influenced attitudes towards guidelines and EBM tended to be GP age and whether the GP practised alone. Younger GPs were more likely to have a positive attitude to guidelines than were older GPs and to prefer guidelines based on EBM. (61, 104) Older GPs were less likely to agree that guidelines would assist with quality improvement and also to improve their diagnostic and management skills. (59) Solo GPs were also less likely to agree with those statements, and less likely to agree that guidelines would improve work satisfaction and that discussions with colleagues would facilitate uptake of guidelines. (59) The preference for EBM guidelines was higher in university affiliated GPs (73%) and lowest in solo GPs (30%). Overall use of guidelines was lowest in solo practice (23%). (104)

**SUMMARY:** There is a wide range of opinion over the worth of EBM and guidelines. GPs have definite opinions about what would influence them to use guidelines and the credibility of different sources of guidelines. Specialist contact was seen to be important and in some instances more useful than guidelines.

### 2.2.4 Summary

Evidence-based guidelines present what is currently known about a topic. This information has been critically appraised and graded. The graded recommendations indicate the quality of the evidence and the likelihood of benefit. The definitions of the terms ‘guideline’ and ‘recommendation’ must be borne in mind when applying this evidence. Clinical experience and judgement need to be used judiciously when applying the guideline. Attitudes to guidelines and EBM have been shown to be generally positive, with the belief that they will improve patient care and can be used in day-to-day management. However, guidelines have been perceived as prescriptive and simplistic. The polarisation of views may be due to variables including the age of the GP, solo practice, and academic affiliation. There is also the danger of guidelines becoming obsolete which may leave GPs practising poor quality medicine.
2.3 Implementing change in the primary care setting

The advent of a new guideline or an updated version of a guideline signifies a change in how patients should be managed. The issues that arise are how to ensure and evince adoption of the recommended changes in what may be firmly established management strategies.

It has been suggested that a doctor would have to read 17 journal articles per day to keep up to date. (105) Doctors seem to spend between <1 hour per week reading (straw polls taken at a variety of CME events) (106) and 111 minutes per week (283 Norwegian primary care physicians, medical journals and articles) (107). Hence, the traditional model of disseminating research findings in peer-reviewed journals is unlikely to be of value in relating advances in medicine. Collections of research reviews are available but their increasing number is creating an information overload (108) and the provision of information alone may not cause change in practice. (109) Even if doctors had sufficient time to read all the available evidence, there are the added problems associated with the variable quality of published research. Many doctors have no training in how to critically appraise published research and nor do they have the skills to understand terms used regularly in EBM, such as NNTc. (106, 110)

No uniform applicable theory can account for behaviour change among doctors and nor has any single theory been proven to be effective among practising doctors (111, 112). There are also no cogent arguments for why one theory should dominate. (42, 112) Theories may overlap and contradict each other and other factors such as prior beliefs and context will moderate the effect of the theory. (42, 112, 113) No model or theory can answer all questions and a single theory may explain only a small variance in targeted behaviour. (112)

The implementation of clinical evidence may also reflect previous personal and professional experiences and beliefs. (60, 114-116) It is easy to return to past practice patterns unless there is good motivation not to and there are recurrent reminders of the change to implement. (116-118) For example, even if a practice has agreed on a

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Some examples given by GPs as definitions for NNT were ‘sample size required in research to determine an effect’ and ‘number of people in a trial who did not respond to treatment, therefore still need to be treated’. The definitions required were ‘the reciprocal of absolute risk reduction’ and ‘the number of patients needed to be treated to achieve one good outcome (or to prevent one bad outcome)’. (110)
prescribing protocol it may be easiest during patient visits to revert to personal experience. (117)

2.3.1 Sources of information

Doctors choose resources that are familiar, clinically oriented, accessible and efficient. The proximity to the doctor’s clinical site and the ease with which knowledge can be understood influence perceptions of the accessibility of a learning resource. The time and energy needed to access a resource are important determinants of use, perhaps even greater than the quality of the information source. Doctors also rely heavily on colleagues for advice. (119) A recent NZ study found that critical characteristics of useful information that GPs identified were brevity, timeliness, recognition by authorities, ownership of the information, and presentation. (120) Another recent NZ study (in 2004) discovered that rapidly accessed resources were more likely to be used in clinical decision making than ones that are slower to access but of higher quality. (121) German GPs ranked the following aspects of CME as very important: relevance to practice (93.3%), reliable/scientific (90.9%), concise (71.8%), user friendly (68.1%) and fast (58.4%). (122)

**POPULARITY OF SOURCES:** GPs in North West England were interviewed approximately 10 years ago about their sources of information when prescribing new drugs. A total of 86% of them saw drug representatives whom 70% considered as a quick way to obtain and process drug information and keep up to date. (123)

While drug companies’ information was seen as an important source and useful for keeping GPs’ knowledge current, GPs did question the objectivity of the information presented. They reported that the data presented were selective rather than inaccurate and that they could separate what was credible from what was not. (123) A NZ study similarly found that information from the pharmaceutical industry was concise and clear, allowing rapid understanding, and that this information, though it ranked as low on trust, was read and considered more than more trusted sources. (120) These examples agree with research regarding the efficacy of face-to-face communication (see this chapter, section 3.3).

Despite the views illustrated above, one study found that quite often the information from the drug company was the only source used prior to prescribing, with dose and
interactions sometimes checked in the British National Formulary (BNF<sup>d</sup>) or Monthly Index of Medical Specialties (MIMS). (124) Another study likewise found that in a third of prescribing incidents, the initial informant (pharmaceutical company or drug representative) was the only information source and the major prescribing influence. (123) Another study suggested that CME meetings were seldom a source of information about new drugs. (124) Additional assistance in prescribing has been offered by the establishment of organisations such as NICE. (124) However, GPs also use a number of other information sources. Table 2.3.a presents data from:

- the North-West England study (123),
- a study of NZ GPs (99, randomly selected) who identified standard prescribing information (121),
- a study of the source of information for the last new drugs that GPs prescribed (125)
- a NZ study of the most frequently used sources of clinical information identified by GPs (120)
- and a Norwegian study of 283 primary care physicians about perceived importance of CME and information sources. (107)

Table 2.3.a illustrates the different sources of information that are most important when prescribing drugs and those which are important for clinical information. Pharmaceutical company information is popular for new drug information (121, 123, 125) however primary and secondary care colleagues and further education are highly used sources for clinical information (107, 120).

Specialists were valued for their knowledge (specific to topic or area, and/or patient- or drug-specific) and their ability to answer GPs’ questions. (121) In particular, greater access to specialists such as hospital pharmacists was considered beneficial. (121)

As discussed above, while pharmaceutical company information is frequently referred to, this is not always seen as useful whereas reference materials, less frequently referred to become more important in their usefulness for prescribing.

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<sup>d</sup> The BNF provides UK healthcare professionals with authoritative and practical information on the selection and clinical use of medicines in a clear, concise and accessible manner. www.bnf.org/bnf/
Table 2.3.a. Information sources used in primary care

<table>
<thead>
<tr>
<th>Information source</th>
<th>Initial information source – new drug (123)</th>
<th>Commonly used prescribing information sources (121)</th>
<th>Information source for last new drug prescribed (125)</th>
<th>Clinical information sources (120)</th>
<th>Important professional updating and maintenance activities (107)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Professional contact</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>1%</td>
<td>7%</td>
<td>90% (outside workplace)</td>
<td>60% (referral information, collegial feedback)</td>
<td></td>
</tr>
<tr>
<td>Hospital</td>
<td>10%</td>
<td>36%</td>
<td>80%</td>
<td>38% (informal contact with colleagues)</td>
<td>30% (formalised workplace meetings)</td>
</tr>
<tr>
<td>Pharmacist</td>
<td></td>
<td></td>
<td></td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td><strong>Reference Material</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MIMS/New Ethicals</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BNF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHARMAC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical journals</td>
<td>1% (16% non-peer reviewed)</td>
<td>65%</td>
<td>9%</td>
<td>57%</td>
<td></td>
</tr>
<tr>
<td>Guideline</td>
<td>67% (Local guidelines IPA/PHO CME)</td>
<td></td>
<td></td>
<td>49% (PHO material)</td>
<td>54% (NZGG)</td>
</tr>
<tr>
<td><strong>Pharmaceutical company</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug representative</td>
<td>33%</td>
<td>42%</td>
<td>50%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mailed material</td>
<td>15% (includes advertising)</td>
<td>85%</td>
<td>5% (includes advertising)</td>
<td>30% (includes advertising)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>3% (patient)</td>
<td></td>
<td></td>
<td>87% (Continuing professional education)</td>
<td>76% (Congresses, CME )</td>
</tr>
</tbody>
</table>
INFLUENCES ON DECISIONS TO PRESCRIBE NEW DRUGS: There are many influences on the decision to initiate a prescription for a new drug, some more important than others. There seemed to be little discussion of new drugs between GPs owing to lack of time, and changes to prescribing were often influenced by seeing another GP’s patient, and following the prescribing lead of the other GP. (123)

In a study during the late 1990s (54% response rate), GPs were asked to rate the importance of sources of prescribing information, and to state from which of these sources they first found information about the last new drug they had prescribed. (125) The pooled responses of ‘most important’ or ‘important’ regarding information sources for new drugs are presented in table 2.3.b below. (125)

Table 2.3.b. Influences and important information sources for new drug prescribing

<table>
<thead>
<tr>
<th>Influences on decision</th>
<th>Important or Most Important information source for last new drug prescribed</th>
</tr>
</thead>
<tbody>
<tr>
<td>to initiate new drug</td>
<td>(123)</td>
</tr>
<tr>
<td>%</td>
<td>% a</td>
</tr>
<tr>
<td>Professional contact</td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>4%</td>
</tr>
<tr>
<td>Hospital</td>
<td>32%</td>
</tr>
<tr>
<td>Pharmacist</td>
<td></td>
</tr>
<tr>
<td>Reference Material</td>
<td></td>
</tr>
<tr>
<td>MIMS</td>
<td>3%</td>
</tr>
<tr>
<td>BNF</td>
<td></td>
</tr>
<tr>
<td>DTB b</td>
<td></td>
</tr>
<tr>
<td>Medical journals</td>
<td>4%</td>
</tr>
<tr>
<td>Guideline</td>
<td>15%</td>
</tr>
<tr>
<td>Pharmaceutical company</td>
<td></td>
</tr>
<tr>
<td>Drug representative</td>
<td>39%</td>
</tr>
<tr>
<td>Mailed material</td>
<td>4% (including advertising)</td>
</tr>
<tr>
<td>Other</td>
<td>6% (GP press)</td>
</tr>
</tbody>
</table>

a 11 potential sources of information were listed, with a 12th option of ‘other’. GPs were asked to rate these sources from 1 = most important through to 5 = unimportant. (125) b DTB’s aim is to provide information and unbiased information on medical conditions, medicines and other treatments to enable people to make informed choices. True to this objective, DTB has always been wholly independent of the pharmaceutical industry, Government and regulatory authorities. DTB is also free of advertising and other forms of commercial sponsorship. http://dtb.bmj.com/info/independence.dtl
Sources of information that were rated as important e.g. DTB, medical journal articles, were seen to have little use in actual practice. The opposite situation was seen with pharmaceutical representatives (i.e. not ranked so highly in theory but ranked highly in practice). This result may reflect the GPs’ lack of awareness of the influence of drug company representatives, or their unwillingness to admit reliance on them as a source of information. (125) Moreover, since GPs work alone or with a few colleagues, contact with hospitals or consultants and with pharmaceutical representatives may represent their main opportunities to come across ‘change agents’. (125) Whereas GPs interact face-to-face with pharmaceutical representatives (see section 2.3.3 regarding media rich information) and can have personal contact with specialists, academic communication uses weak methods e.g. unaddressed documents. (125)

However, GPs felt that contact with consultants was limited, and mainly by letter (124). Some GPs found that hospital specialists were less easy to access than private consultants. (121) It was important to GPs to see consultants using a drug and follow their example. (124) This approach reduced uncertainty and risk and the authors suggested that “GPs used this device to simplify decision rules, especially when judgement is difficult and complex.” (123) Seeing hospital-prescribed drugs be successful in other patients also influenced GPs’ prescribing decisions, although GPs were reluctant to change from a standard prescription item to a new drug. The factors described above may contribute to time delays before new drugs are used as first-line agents. (123)

The continued use of a drug depended on early experience in a few patients. Prescribing data indicated that the GPs were not consistent in the rate of uptake and subsequent use of new drugs, but seemed to consider each drug individually. (124)

**EFFECT OF PRESCRIBING BEHAVIOUR:** High prescribers and low prescribers of new drugs (30 GPs from North West England, interviewed mid-1999 to early 2000) were asked about prescribing influences to explore the differences between these groups. (126) The figures report the frequency of cited influences rather than their relative importance; however there are striking differences between the two prescribing groups in the frequency of certain influences. Pharmaceutical representatives were mentioned as influences in 46% of new drug initiations, compared with only 10% for low prescribers. High prescribers were less likely to be influenced by
hospital/consultant colleagues (13%), guidelines (10%), GP colleagues (9%) or peer-reviewed literature (2%). (126) For low prescribers, the most frequent influence on new drug initiations was hospital/consultant colleagues (58%), then guidelines (26%) with peer-reviewed literature influencing 16% of new drug initiations by low prescribers. GP colleagues did not seem to influence new prescribing for low prescribing GPs. BNF and MIMS had minimal influence on new prescriptions for high (3%) and low (5%) prescribers which may be due to the time delay between the release of a new drug and the publication of these sources. (126)

**EFFECT OF GP AND PRACTICE SIZE:** The GPs who see drug representatives most often tend to practise solo. (127) Frequency of contact was positively associated with willingness to prescribe new drugs or drugs not clinically indicated in response to patients’ requests, mediated by receptiveness to information from drug companies. (127) This study also suggested that the visits may fulfil a pastoral need rather than educative one. (127)

No differences were seen between small and large practices in the sources (of prescribing information) rated important or those actually used. However, GPs who had been practising for <10 years were more likely to identify medical journal articles or primary care colleagues as their source of first information about the last new drug prescribed than were the GPs who had been in practice longer. (125) Practice size may affect the frequency of use of information sources as larger practices had more contact with colleagues within the workplace (p<0.001), but used CME (p=0.007) and New Zealand Guidelines Group (NZGG) (p=0.002) less than did GPs from smaller practices. (120)

**SUMMARY:** While GPs place most importance on information that is evidence-based and independent, this stated preference does not represent the information source that seems to be the most prevalent in the prescription of new drugs, namely the pharmaceutical industry. (125) However, this does not seem to be the case in some NZ studies. Different studies indicated variation in reliance on colleagues for advice. GPs cited information overload, inability to analyse the quality of research, intuition, and a focus on how the patient actually feels, rather than what the journals say, as reasons for not seeking evidence-based medicine. (123) Younger GPs may be more likely to refer to journal articles and solo GPs may be more likely to see drug representatives. GPs were also highly influenced by hospital prescribing. The ranking of these influential
sources changes depended on whether the GP was a high or low prescriber of new drugs. GPs may be unaware of the importance they place on the information they receive from drug companies.

2.3.2 Credibility of sources
As described above, GPs are exposed to many sources of information about medications. GPs may be more accepting of information if it is from a source that they believe to be credible.

24 Scottish GPs were interviewed (late 1990s) about their access to and use of statin trial data. (128) These doctors tended to judge the trustworthiness of the evidence source, rather than critically evaluate the method and contents. Familiarity with local guidelines writers meant that they were more likely to be used than national guidelines written (128) although national guidelines were highly trusted by 90% of GPs in one survey of sources of clinical information. (120) National agencies or policies such as NICE, SIGN and the National Services Frameworks also influence prescribing owing to the credibility of the source and the authority conveyed by their publications. (24, 129)

There is scepticism about the motives of guideline developers and the origin of guidelines, especially from the local health board due to their interest in cost containment. (128) This cynicism about guideline authorship becomes a common theme, with GPs regularly critical of guidelines produced for example, from secondary care, where the types of patients and management processes are very different to primary care. (24, 129) Yet even when a guideline is valued, GPs may not implement its recommendations when they question the relevance of the evidence base to their patients. (24, 129) (See Chapter 3, section 3.2 for a discussion regarding comparability of clinical trial patients and primary care patients, focussing on HF.)

Pharmaceutical company guidelines tended to be ignored as their intentions were not trusted and because of the company’s economic interest in the product. (128, 129) This finding was also revealed by a survey of GPs which identified that drug representatives and unsolicited advertisement were not highly trusted (7.8% and 2.6%), and were accompanied by high levels of distrust (33% and 5.9%). (120) However a GP who had a long-standing and trusted relationship with a drug company or representative was more likely to accept their information than a GP who did not have such a relationship. (123)
The small number of GPs who used peer-reviewed medical journals rated them as very credible sources. (123) The opinions and prescribing behaviour of colleagues, especially of consultants, were more frequent sources of information. Perceived specialist endorsement was also an influence – the status of the communicator being the influential factor. (123) A survey of clinical information sources found that the most trusted source was hospital colleagues (95%), while peers within and outside the workplace were also highly trusted (85% and 84%). (120) Change may happen as a result of peer group influence than on the basis of scientific evidence. (130) Specialists’ influence went as far as some GPs being reluctant to prescribe new drugs unless ‘respected’ consultants were using them (not all specialists were seen to be equally credible – see sections 4.5 and 4.8). GPs rarely received actual pharmacological information from the specialists, saying that hospital letters did not often explain much, and did not put a logical argument as to why that particular drug should be prescribed. (123)

**SUMMARY:** GPs’ response to evidence may be swayed by the credibility of the source and the relationship the GP may have with the source. Peer and certain specialists are highly trusted and specialist prescribing behaviour can exert a strong influence over GP prescribing. The source of the guideline is viewed by GPs as important in persuading them to apply the guideline to their practice.

### 2.3.3 Format of information

The format in which GPs prefer to receive information about changing practice may not necessarily be the format that causes the greatest change in practice.

Characteristics of how information is delivered can be described by ‘media richness’. (131) These characteristics are:

- **Immediacy of feedback / interactivity.** An example of this would be real-time conversation or feedback between participants.
- **Multiple cues.** These would include verbal and non-verbal cues, which would be evident in a face-to-face meeting.
- **Natural language.** Where participants can talk as if they were in conversation.
- **Personal focus.** Where what is being communicated can be directed at individuals. Media richness reduces with an increased group size.
The ‘richness’ is determined by the number of characteristics. This is described as a continuum with one-on-one being rich, followed by group meeting, telephone, e-mail, written memo, and data reports being ‘lean’. (131) The media richness concept can be applied to the implementation of guidelines or any other change in practice. If all the characteristics described above cannot be included in one form of implementation, they could be found in an additional method of implementation. (131)

Another concept that should be considered is social information processing theory, which looks at participants’ usual format of communication and their familiarity with specific media types. Providing different media types could cater for different preferences, as well as providing multiple education methods as suggested above. (131)

**PREFERENCES:** When American doctors and related healthcare professionals were asked about their learning experiences in the past 12 months (in 1998), 93% had attended an in-person conference, 66% had learnt from print-based materials (journal review), and only 13% had learnt by using the Internet. Asked to rank their preferences of continuing education delivery mode, the in-person conference received the highest score followed by print-based self-study. Internet received a low score but more than 75% of respondents were interested in being taught how to use the Internet. (132) In a study of almost 1000 American physicians the most frequently reported reason for not using self-directed learning was a preference for in-person education. (133) The second reason given was the time-consuming nature of self-directed learning. (133)

A 6 month survey of doctors participating in on-line CME courses found that the quality of content was the most important characteristic, followed by on-line accessibility and ease of use. (2) The least preferred features were having little opportunity to interact and the need to download additional software. (2)

The overwhelmingly preferred format for receiving CME was through personal interaction (a 2001 in-depth study of 24 NZ GPs). (134) ‘Interaction’ ranged from a specialist lecture with questions, to leaderless small groups, problem solving, a journal club or critical event analysis. A preference for lectures could indicate laziness as interaction was not essential. Face-to-face delivery with discussion was preferred over reading or the Internet. Any CME that was not interactive on a personal level was seen as an adjunct. (134)

The preferred format of CME was GP-focused, not combined sessions with practice nurses, pharmacists, or practice managers, as their needs were seen to be different to
those of the GPs. GPs also asked for succinct courses on common conditions, with only a few ‘take home’ points. (134) The same preference applies to guidelines – namely, simple recommendations and clear guidance (129), with almost two-thirds of GPs preferring a ‘small books’ format and 59% favouring ‘one official manual of all guidelines’. (135)

**PERCEIVED EFFECTIVENESS:** When given examples of different strategies and asked to select which would increase, decrease or have no effect on their use of a particular guideline, provision of feedback on performance measured against the guideline was thought to be influential by only 6% of GPs. Endorsement by a recognised expert was thought to increase use by 9% of GPs (although 12% said this would decrease their use). Local adaptation of guidelines was seen to be useful by 22% but would not change use in 47% of GPs who responded to the survey. (135)

The most likely way of increasing guideline use was reported to be a personal visit by a trained nurse who answers questions (55%), followed by a lecture about the content of the guideline (48%, but 38% said that this method would not produce change). A personal visit by a GP to answer questions was seen to be useful by 31% of GPs, although 37% were not sure about the outcome of this method. 18% of GPs thought that small group workshops to discuss and review guidelines would increase their use of a guideline. Prompts in computerised medical records were least popular at 5%. (135)

A further study in primary care that investigated the perceived effectiveness of interventions to implement guideline prescribing recommendations found that audit and feedback, peer review, participatory guideline development and local opinion leaders were considered effective, while the effectiveness of inter-professional shared care was thought to be context-specific. (129) Educational outreach and financial incentives were perceived as having limited effect, and mailed printed material was thought to be not effective. (129)

**SUMMARY:** GPs tend to prefer information delivered in formats that are media rich i.e. with personal contact. Providing different formats of information may cater for different preferences and familiarity with particular types of media. The information delivered should be concise. However, the approaches thought to be effective by GPs are not necessarily those which have been proven to be effective in practice.
2.3.4 Uptake of therapies without an implementation strategy

Personal differences can influence how health professionals integrate new therapies that are not accompanied by deliberate implementation into their practice (i.e. diffusion). The uptake of new knowledge has been described by the concept of five adopter categories, derived from the S-shaped curve of diffusion – the cumulative number of adopters plotted over time. The types differ according to personality, communication, and education strategy:

- **Innovators** Supposedly more sensitive to passive education strategies and evidence-based medicine, possibly opinion leaders. (52, 136)
- **Early adopters** Require a more intensive approach to alter attitudes more quickly and are more influenced by peers and opinion leaders. (52, 136)
- **Early majority**
- **Late majority**
- **Late adopters** May respond to incentives or official statements. (52)

The innovators represent 2.5% of a population, followed by early adopters 13.5%. Early majority and late majority each represent 34%, and the late adopters represent 16%. (130) Making the innovators and early adopters more visible assists momentum for change and helps the innovation gain critical mass, at which point the change becomes self-sustaining. (130) The question is how to identify the innovators and early adopters.

A study (136) looked at the diffusion of new drugs into general practice and the concept of the five adopter categories. The questions the study addressed were whether the GPs retained the same adopter category for different drugs, and whether the categories had any relation to general prescribing behaviour, practice activity and demographics. (136)

The five categories were compressed into three for analysis:

- Early prescribers = innovators and early adopters
- Intermediate prescribers = early and late majority
- Late prescribers = late adopters.
**CATEGORY:** The consistency of adopter categories, measured for the 95 solo-GP practices over the different drugs, was mixed. Late prescribers were more consistent in their responses than early prescribers, but GPs did move between early and late prescribers for different drugs. (136)

**PRACTICE SIZE:** Partnership practices \((n = 79)\) showed an initial steep rise in prescribing with no slow initial phase whereas solo GP practices did show an initial slow phase, followed by a less steep rise in prescribing. The time taken to reach 80\% of practices prescribing was 6 weeks for group-practices and 21 weeks for solo-practices. (136)

The diffusion time (day of drug release for GP prescription to the day of the first written prescription) for group practices was a median time of 10 days (mean 41 days) whereas for solo practices the median diffusion time was 52 days (mean 119 days), \(P < 0.0001\). (136) These figures were for one drug but the researchers say that the other drugs followed the same pattern. (136)

Group practices were seen to adopt new drugs more quickly (although the measure was set at just one GP in the practice prescribing the drug for the whole practice to be considered as adopting it). It has been suggested that continuous professional stimulation and other social factors (i.e. practising in a manner similar to the other GPs in the group) play a part in this. (136)

**GP AND PRACTICE DEMOGRAPHICS:** Compared with intermediate prescribers (reference group), female GPs and small list size were associated with late prescribing. (136) Female GPs were more likely to practise part-time and have less exposure to the practice of other GPs. The number of other prescriptions per patient was a strong negative predictor of late prescribing. A large number of diagnostic procedures per patient was positively associated with early prescribing and negatively associated with late prescribing (not significant difference). (136)

**OTHER:** Differences in adoption may also have been due to the marketing (mass media) of one particular expensive drug, public demand, cost and duration of therapy. (136)

**SUMMARY:** The data here suggest that adoption of new drugs depends at the very least on both doctor and drug characteristics. (136) The authors describe the associations
between late prescribing and female GPs, smaller list size, lower diagnostic activity per patient, and a strong general restrictive attitude towards pharmacotherapy. This fits well into the typology of a ‘conservative’ physician who is more likely to be in solo practice. If the need is to achieve rapid diffusion of drugs into primary care, then the focus should be on the late prescribers, who at the outset of this study of diffusion and adopter characteristics were described as needing more intensive educational strategies to help change their prescribing adoption habits. (136)

2.3.5 Type of GP most likely to implement change

The study quoted above of diffusion of new drugs into general practice also indicated differences in the speed of adoption of new drugs on the basis of solo/group practice, sex, list size, existing level of prescribing and attendance at medical meetings. (136) A qualitative study on new drug prescribing found that the willingness to prescribe a new drug depended on the perceived risks and special interests and that the continued use of new drugs depended on early experience in a few patients. (124)

**PRESCRIBER TYPE:** In-depth interviews regarding prescribing were carried out with the highest (≥9 new drugs, n = 17) and lowest (≤2 new drugs n = 13) prescribers of drugs that had been introduced within the preceding 48 months. (126)

There were no significant differences in demographics (sex, years since qualification, full or part-time) between the groups of low and high prescribers. Most GPs described themselves as ‘cautious’, although the way this term was defined differed between the two prescribing groups. This fitted with the conservative nature of the low prescribers who were reluctant to introduce new drugs, preferring instead established and familiar drugs. While high prescribers were less reluctant to prescribe new drugs, there were differences in attitudes with some keen to keep up to date, and try completely new products. Others were willing to try new drugs when they had some knowledge of them or to trial the new drug in a small group of patients. Some high prescribers would try new drugs where previous treatments had been ineffective or there was no existing treatment. (126) Both groups exhibited similar characteristics with clinical experiences and increasing familiarity which reduced uncertainty. Successful outcomes of initial trials of the drug were important in the continued use of the new drugs. (126) High

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*30 GPs in North West England participated in interviews between August 1999 and February 2000. The interviews focussed on drugs that had been introduced between January 1998 and May 1999. (126)*
prescribers were less likely to use independent information sources, and expressed some criticism of medical journals as being out of date or overly complex in reporting research. (126)

**Risk:** Low prescribers were often more sensitive to risk than high prescribers, and expressed views that the profile of the drug could not be evaluated at introduction, only once it had been in clinical use for a while. They would adopt a ‘wait and see’ attitude until there was peer-reviewed evidence or validation by consensus or expert opinion (e.g. hospital doctors). High prescribers were more accepting of uncertainty and risks or viewed risks as minimal (since the licensing authority had approved the drug). Drugs would first be tried in patients for whom existing therapy had been sub-optimal or in areas they felt knowledgeable about. (126)

**Pharmaceutical industry:** None of the low prescribing GPs saw more than one drug representative each week. Four never saw representatives, describing their information as distorted and selective, and saying that major therapeutic innovations would be reported in medical journals. (126) High prescribers were more likely to rely on the pharmaceutical industry, with some prescribing predominantly influenced by drug advertisements or mailing. All but one saw drug representatives, with others receiving at least weekly visits (seven high prescribing GPs saw at least three per week). (126)

**Specialists:** Low prescribers were more likely to accumulate information gradually and relied on specialists as guides to prescribing. However, some described local consultants as “too innovative” in prescribing new drugs without sufficient evidence of their benefit. High prescribers were influenced to a lesser degree by hospital decisions, although one prescriber thought that local hospital consultants were ‘out of touch with pharmacological advances’, while other GPs were keen to follow prescribing trends of innovative hospital colleagues. (126)

**PERSONALITY:** GPs from a study of implementing evidence-based changes in prescribing were given a personality test which assessed their preferred approach to problem solving. The scale runs from ‘highly adaptive’ (preferring change that is a development of existing methods) to ‘highly innovative’ (preferring change that is radical and novel). A ‘cognitive climate’ for each practice can be estimated from averaging individual scores. (137)

The problem solving data indicated that GPs tended to adopt an adaptive, cautious approach with a mean score below that of the general population. This result may
suggest a resistance to new ideas, with a focus on implementing small changes that improve current treatment. This is perhaps a reasonable response as 71% and 76% agreed with the following statements:

“...there is too much information available on prescribing changes to assimilate.”

“...under present working conditions, I do not have enough time to keep abreast of recent recommendations in drug usage.” (137)

Problem-solving data and questionnaires relating to practice structure and organisation, GPs’ demographics and professional behaviour were used to further analyse the implementation of evidence-based prescribing. A weak negative relationship was seen between the mean age of doctors and the ACEi implementation score ($r = -0.33$, $P = 0.04$). (137)

**FAMILIARITY:** A fairly simple factor that will impact on how GPs manage conditions such as HF, is familiarity with the condition and management. A comparison of the proportions of GPs and cardiologists who saw >5 HF patients/week found a significant difference between GPs (31%) and cardiologists (79%, $p < 0.01$). (138) Another study which looked at the number of HF patients seen per week found that on average GPs saw four HF patients per week, but cardiologists saw an average of 12 ($P < 0.01$). (102) HF guidelines may not be of foremost importance to GPs if HF patients represent only a small proportion of their total consultations. (139)

**SUMMARY:** The speed at which GPs implement change depends on their personal and practice characteristics. GPs can also be categorised by their initial approach to prescribing new drugs, with some being much more likely to accept uncertainty and identify different information sources compared with GPs who were more cautious with prescribing. Overall, GPs tended to prefer gradual change. The approach to changing prescribing may be to show how new therapies, or new uses for old therapies such as metoprolol succinate, are an improvement on existing therapies, rather than underline their novelty. However a factor that may be difficult to moderate is the frequency with which GPs are able to put new knowledge into practice, which may not be often. How to proceed with change should be detailed and new recommendations should be given priority to avoid being drowned by the volume of information available. (137)
2.3.6 GP and Practice characteristics

This section will focus on GP and practice characteristics that may have an effect on the way GPs implement change and manage patients and how the advent of computerised medical records may or may not be assisting in management. GPs within a practice can affect each other’s behaviour. This can be significant in large practices when there may be the influence of partner GPs. (24) Therefore it is important to include all GPs (or as many as possible) within a practice in any implementation. (24)

**GP DEMOGRAPHICS:** Cross-sectional survey data on NZ GPs suggested that male GPs are more likely than female GPs to be in solo practice (29% vs. 12%, p = 0.0001) and overall 25% of GPs are in solo practice. (140) Male GPs are also more likely to be full time rather than part time (88% vs. 40%, p = 0.0001). (140)

Data from a large, continuous study of doctor-patient encounters from a representative and random sample looked at the effect of practitioner age, as age may have an effect on the way that GPs practice. (141) See table 2.3.c below.

Table 2.3.c. Comparison of demographics of oldest and youngest GP groups

<table>
<thead>
<tr>
<th></th>
<th>&lt;35 years (%)</th>
<th>≥65 years (%)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>48%</td>
<td>92%</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Solo practice</td>
<td>3%</td>
<td>31%</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Large practice (≥5 GPs)</td>
<td>64%</td>
<td>33%*</td>
<td>See note</td>
</tr>
<tr>
<td>FRACGP</td>
<td>82%</td>
<td>17%</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

* Similar proportions of GPs in younger age groups were also working in larger practices. 40% for GPs 45 to 54 yrs, 31% for GPs 55 to 64yrs.

There were similar proportions of age groups in medium sized practices (2 to 4 GPs) in the age groups studied (range 33.3% to 42.5%) (see box 2 of this reference for more details). (141) The proportion of visits of male patients increased as the age of the GP increased and GPs in the oldest age group (≥65 years) saw patients in this age group at just over twice the rate of the youngest age group of GPs. Older GPs were more likely to manage circulatory problems (P = 0.002), manage a chronic condition (OR = 1.25; 1.16 to 1.35) prescribe more and have lower rates of pathology orders or procedural treatments (all P < 0.001) when compared with GPs <35 years. (141)

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*d* BEACH. Bettering the Evaluation And Care of Health, Australia.
A survey of general practice in the Netherlands found that group practices (>2 GPs) were more likely to employ female GPs, part-time GPs and to have a larger proportion of younger GPs. (142)

Solo GPs may have little contact with peers and may have less information and change less than those who work closely with others. (143).

**PATIENT LIST:** Practice size may restrict whether patients see their own doctor, and GPs may be reluctant to alter the management of patients they see who are not their own. This was seen in a smoking cessation study where under half of the doctors surveyed did not discuss smoking with patients other than their own. (144) If this simple, benign intervention is not implemented by doctors when seeing other doctors’ patients, it suggests they are unlikely to change medications, which is a great deal more complex.

One study found that for practice sizes of 3000 to 6300 patients, patients had an OR of seeing their own GP of approximately 0.50 compared with practice list sizes <3000, with OR less than half this as practice size rises over 6300. (145)

Patients who wished to discuss a longstanding problem were more likely to see their usual doctor compared with patients who had a new or urgent problem, and increasing age increased the likelihood that the patient would see their usual doctor, as were visits where the patients had been asked to attend. (145) But there was great variability as overall 61.6% of patients saw their usual doctor but the range within practices was 39% to 98%; some of the variation is due to patients rather than differences between GPs and practices. (145)

**PRACTICE SIZE:** An English study compared regional demographics of single-handed and group practices. (146) The regional proportion of single-handed practices was 25.4% higher (data taken at 1 April 1998) than the quoted national figure of 10%. The age-sex distribution of practice lists was the same between single and group practices. However there were differences between single-handed practices in age, and mean practice list size / FTE GP, and female GPs (see table 2.3.d below). (146)
Table 2.3.d. Demographic differences between single-handed and group practices

<table>
<thead>
<tr>
<th></th>
<th>Single handed</th>
<th>Group practices</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP mean age</td>
<td>52 ± 8 years</td>
<td>44 ± 6 years</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Female GPs</td>
<td>25 (12%)</td>
<td>462 (76%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mean practice list size / FTE GP</td>
<td>2299 ± 708</td>
<td>2058 ± 535</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Single-handed practices were less likely to be involved in vocational training, and less likely to have a practice nurse. The number of FTE GPs in group practice was smaller than the actual number, suggesting that part-time GPs work in group practice. (146)

A comparison of single handed and group practices found no significant differences in disease prevention and health promotion, avoidable admissions or inappropriate surgery. (146) Significantly higher admission rates for chronic conditions (asthma and epilepsy but not DM) were observed in single handed practices. (146)

Results of immunisation and preventive health care indicators and hospital admissions do not support concerns of professional isolation and quality standards for solo GPs. (146) However this study illustrates demographic differences between GPs in single and group practices.

The organisational management differences in single (n = 518) and group practices (n = 248) (Netherlands, 1998 to 2003) (142) were assessed with data collected at practice and GP level, with GP level data aggregated at practice level. After factor analysis, 56 dimensions were constructed from the 303 indicators and the outcomes were analysed for 4 main areas: (142)

1. Infrastructure (premises, equipment, service and organisation).
2. Team (task division, workload and job stress of the GPs).
3. Communication (with colleagues/care providers, meeting time, patient information, computerised patient records, IT).
4. Quality assurance activities (CME, audit, QA-activities).

Outcome data were adjusted for deprivation, % of Asian and black residents, proportion of men and women >75 years, rurality, presence of a female GP and training status. (146)

Disease prevention and health promotion included immunisations, preschool booster, and cytology. Chronic care management included admission rates for asthma, diabetes and epilepsy. Avoidable admissions were for ENT, UTI, and congestive HF. Inappropriate surgery included D & C in women <40 years and rates for grommets and health outcomes looked at teenage pregnancy. (146)
The table below compares how type of practice was scored in the areas measured by the indicators.

Table 2.3.e. Comparison of practice indicators for single-handed and group practices

<table>
<thead>
<tr>
<th>Practice Indicators</th>
<th>Practice size</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group</td>
</tr>
<tr>
<td><strong>Infrastructure</strong></td>
<td></td>
</tr>
<tr>
<td>Sophisticated equipment and diagnostic equipment</td>
<td>✓</td>
</tr>
<tr>
<td>Frequent use of equipment</td>
<td>✓</td>
</tr>
<tr>
<td>General organisation of practice (e.g. practice routines, protocols, and logistics of patient information)</td>
<td>✓</td>
</tr>
<tr>
<td>Preventive-care services</td>
<td>=</td>
</tr>
<tr>
<td><strong>Team</strong></td>
<td></td>
</tr>
<tr>
<td>Collaboration in the local GP group (structuring of the meetings, discussing medical and organisational topics)</td>
<td>=</td>
</tr>
<tr>
<td>Longer meetings with colleagues, partners in primary care and consultants / specialists</td>
<td>✓</td>
</tr>
<tr>
<td>Protocols on care and formal collaboration with the hospital (policy on referring to hospital and back to GP)</td>
<td>✓</td>
</tr>
<tr>
<td>Monitoring patients with chronic diseases and prevention</td>
<td>=</td>
</tr>
<tr>
<td><strong>Communication</strong></td>
<td></td>
</tr>
<tr>
<td>Computerised (problem list, mediation, episode registration, agenda for appointments, coding)</td>
<td>✓</td>
</tr>
<tr>
<td>Quality of computerised records (SOAP-method)</td>
<td>✓</td>
</tr>
<tr>
<td>Computer use (generating prescriptions, referral letters, letters to other care providers, disease management, registration of drug intolerance and contraindications)</td>
<td>✓</td>
</tr>
<tr>
<td>Electronic communication (hospital and pharmacy)</td>
<td>✓</td>
</tr>
<tr>
<td>Internet use</td>
<td>✓</td>
</tr>
<tr>
<td><strong>Quality assurance activities</strong></td>
<td></td>
</tr>
<tr>
<td>Audit, assessment and other quality improvement activities</td>
<td>✓</td>
</tr>
<tr>
<td>Indicators for quality assurance in the practice (e.g. practice protocols, staff education, and equipment maintenance)</td>
<td>✓</td>
</tr>
<tr>
<td>Participation with audit with feedback on outcomes (prescriptions, referrals, diagnostic tests) and quality assurance activities</td>
<td>✓</td>
</tr>
<tr>
<td>Training/year (hours)</td>
<td>=</td>
</tr>
</tbody>
</table>

✓ better e.g. more sophisticated equipment, more frequent use of equipment
= practice types were equal
But for the indicators described above it is difficult to answer where statistically significant differences are clinically relevant.

Patients considered most aspects of practice management (and premises) to be better in single-handed practices. There may be a ‘halo effect’ of having a continuous, personal relationship with their GP. (142)

The authors suggest that the high score on infrastructure indicators achieved by group practices points to provision of evidence-based care for the medical needs of patients, rather than emphasis on service. But some aspects of quality of care that were studied may not be as relevant in single-handed practices (e.g. use of protocols, communication between team members). (142)

A survey (44% response rate) in southern England looked at implementation of evidence-based prescribing recommendations (use of warfarin or aspirin in AF, use of ACEi in HF) and the relationship between practice and doctor characteristics. (137) The types of evidence-based prescribing were studied as they had been consistently recommended to GPs within the past 5 years, and based on research evidence there was a consensus that change was needed. (137)

The proportion of patients receiving the recommended treatment was averaged for each practice to give an ‘implementation score’. The proportion of HF patients receiving ACEi was 48% (± 16.3, range 20 to 94%) with 59 cases (median) (range 10 to 71) identified as eligible for treatment. For aspirin/warfarin the mean proportion receiving either drug was 66% (± 15.6, range 10 to 96%). The median number of cases identified was 48 (range 4 to 67). (137)

Factors such as number and type of staff in the practice team or the frequency of team meeting did not have any effect on the implementation scores. (137) Practices with disease management protocols did not achieve higher implementation scores, and nor

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(137) The survey was carried out over a 13 month period from 1996 to 1997 with 39 practices and 184 GPs participating. Practices with single GPs or without computerised prescribing systems were excluded. Practices were stratified for certain conditions which may have reduced the risk of a non-representative sample. (137)

(137) Computer audits for recorded diagnoses of AF or HF and for prescriptions of digoxin or loop diuretics were carried out and medical records reviewed to identify those currently treatment for AF / HF, and for eligibility / contraindications. Prescribing markers were assessed at practice level, and variables relating to individual doctors were aggregated. This is a realistic approach as a patient’s treatment may reflect decisions made by several different doctors as well as practice policy, but may mask the influence of characteristics such as a doctor’s age. (137)
did access to disease registers except in practices that produced an AF register [mean score 70% (65 to 74%) vs. no register 55% (44 to 60%); P = 0.04]. A significant correlation was seen between computer usage and AF audit (-0.39, P = 0.01). (137)

There was a weak correlation between the implementation score and the mean practice problem score (r = 0.33, P = 0.04). Innovative partners and fundholding\(^1\) practices were the most important independent variables but the final model explained only 16% of the variance. Other influencing factors were not consistent across the conditions. (137)

**SUMMARY:** The success of implementation of change may be due to influences within a practice or the type of GP working in a particular type of practice e.g. older male GPs are more likely than younger female GPs to work in solo practice. Male GPs are also more likely than female GPs to work full time. Some indicators show that group practices perform better than single handed practices but these results are not consistent. The effects of practice characteristics on the implementation of change may best be assessed on a study-by-study basis. The clinical relevance of practice indicators needs to be determined.

### 2.3.7 Responsibility to implement change

The objective of medical education is to focus on learning (not teaching) since learning will result in doctors changing their practice and improving patient outcomes. (98, 147) Education is no longer considered instruction (passive) but facilitation of learning (active). (98, 147) CME should intervene in aspects of medical practice that can be improved. (147) However, doctors must be responsible in keeping up their own continuing education by setting goals and choosing activities to realise them, (148, 149) as formal CME events may identify different priority areas from those the GP may identify. (98) To maintain competency, the GP first has to estimate the difference (or ‘gap’) between their current knowledge, skill and performance and what their levels should be. (147) However GPs need the skills to realise that the deficit exists, carry out a self-assessment, and then determine how to improve their performance. (98, 150) The impetus to reduce the anxiety created by the learning need (or gap) is the motivation to learn and change. (147) No improvements can be made if the GP is not aware of gaps in their knowledge. So the problem becomes ‘how do I know what I don’t know’, which may be more difficult for GPs who are practising alone. (150-152) To be

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\(^1\) Practices that receive budgets to cover prescribing, non-urgent hospital services and ancillary staff.
satisfied with their level of knowledge, GPs may need to ask themselves two questions: what should any GP know, and what information is needed to do their specific job well. (153)

What is known is that ratings of self-assessment correlate poorly with actual performance measures. (150, 151) A comparison of self-report (interview) and objective measures of adherence (medical record review) to guidelines found that, out of 37 comparisons, almost 90% of self-reported rates of adherence were overestimates when compared with objective rates. (154) 32 of the differences were statistically significant and the median over-estimation was 27% (absolute difference). These results underline the fact that self-reported knowledge (in this instance of guideline recommendations) can be biased and needs to be verified by objective measures. (154)

A similar NZ study compared GPs’ self-assessment with tests of knowledge on three areas of common practice. (155) The correlations for all three topics were low (r = 0.19 to r = 0.21), suggesting that GPs have difficulty in determining their specific learning needs which undermines the effectiveness of self-directed learning. (155) The effect of this has to be coupled with the likelihood that given the autonomy to select educational events, doctors will not go beyond their “comfort zone”. (156) What complicates GPs’ efforts to keep up to date and make changes in their practice is the wide scope of practice and the rapid information changes associated with primary care. (153, 155)

In a recent systematic review of health professionals, two-thirds of comparisons studied found weak, no or inverse relationships between self-assessment and objective assessment. (157) Trends also indicated that compared with objective measures of knowledge, doctors were not able to accurately self-assess their knowledge. (157) Some studies even reported that the worst accuracy in self-assessment was found in physicians who were the least skilled but most confident. (151, 158)

**SUMMARY:** GPs find it difficult to identify gaps in their practice and to accurately self-assess their knowledge and adherence to guideline recommendations. This will cause problems in circumstances were GPs learning is self-directed.

### 2.3.8 Summary

Implementing change in primary care is difficult due to the large amount of new research that is published. To read this research would be time consuming, sometimes
irrelevant and require critical appraisal skills. Apart from published journal articles, which form only a small part of a GP’s sources of information, most GPs also receive information from drug representatives, official drug compendia, hospitals and specialists, and depending on which study is quoted to a greater or lesser extent from their GP colleagues. However the usefulness and importance of these sources of information are two different things. While GPs tended to follow information provided by specialists, they felt that more detail could be supplied.

The credibility of the source of evidence may also influence whether the evidence is used. Information emanating from a national agency or professional body is not automatically assumed to be credible.

The format in which information is delivered by is also important. A media rich format (one-to-one i.e. interactive and immediate delivery) is preferred by GPs over reading, the Internet or didactic lecture.

Some GPs are more inclined to adopt new interventions, however the GP’s age, sex and practice characteristics may affect how GPs change practice. GPs may also be influenced by the results of early trials of new drugs in patients.

Change in practice will also depend on the evidence itself. Facilitators for adopting recommendations for change in practice have been suggested to include: support for the recommendation from scientific evidence, discussion of benefits, discussion of harms and risks, easy recommendations to follow, effects of change in practice that can be seen quickly, and recommendations that are compatible with existing norms and values in practice. (159) New evidence that does not fit these conditions will be more difficult to implement.

Implementing change as research is published could be unsystematic and may not improve overall management practices. There is a responsibility on GPs and practices to identify where knowledge and its implementation are suboptimal. Meeting this responsibility requires formal, structured assessment, not merely self-reported knowledge, so that targeted educational interventions can measure and address gaps.

Planned CME needs to be delivered through an implementation strategy and the factors that influence uptake need to be considered when selecting a method of implementation and delivering an educational intervention.
2.4 Methods of implementation

Dissemination brings information, e.g. a guideline, to the attention of intended users. (44, 116) Implementation aims to change actions or behaviours. (116) An educational intervention is defined as: “any attempt to persuade physicians to modify their practice performance by communicating clinical information”. (149, 160)

Implementation research is the scientific study of methods to promote the uptake of research findings and hence to reduce inappropriate care. (161) Systematic reviews of more rigorous evaluations have shown variable effectiveness within the same intervention type which may be due to the modifying effects of context and content or be explained by variation in the intensity or quality of the interventions tested. (161) Better designed trials, more usually based on cluster rather than individual randomisation, will produce more valid (trustworthy) results. Randomised trials of head to head comparisons are required to establish the relative effectiveness of interventions in the same setting. (162) However, this type of study is expensive to conduct, can be lengthy, and may not provide the possibility of comparing all options.

Strategies such as postal distribution or didactic educational sessions are commonly used in GP CME but have been suggested to be largely ineffective. (57, 156, 161-163) Passive learning in a didactic lecture has the rate and sequence of information controlled by the teacher (164) and gives no guarantee that the information will be internalised by the learner, assuming that in the example of a mailout it is read at all. (118)

These strategies compare with interactive and participatory educational sessions such as discussion groups, hands-on training, problem solving or case solving. (156, 165) Active learning utilises dialogue between the teacher and the learner which is to some extent controlled by the needs of the learner with the teacher interpreting the knowledge. (164)

IMPLEMENTATION THEORIES: Factors influencing the implementation of interventions can be viewed using different theories. The purpose of theories is to provide generalisable forms of understanding (41) where theories can be defined as:

“... a system of ideas or statements held as an explanation or account of a group of facts or phenomena.” (166)
Some theories that relate to implementation are described below. For example, Rogers’ innovation-diffusion theory – overcoming routine practice – considers three independent factors: (44)

1. **Product characteristics.** These include format and content which for a guideline would be decided on during development.

2. **Communication channels.** These are all the possible methods of dissemination.

3. **Characteristics and impact of the product on the target population (intended users).** This is the most complex of the factors comprising skills and expertise, attitudes towards change, impact of feedback on prescribing and individual and group approach. Identifying the intended user is important e.g. a GP will see a broad range of clinical conditions, and tends to work with symptoms, which differs significantly from a specialist’s caseload. (44)

While Rogers stipulated two product characteristics, a number of the attributes of recommendations can affect their implementation. Recommendations that were not controversial and were compatible with current values had the most influence on compliance with the recommendation, followed by clear and specific recommendations, concrete and precisely described, and those which did not demand change in existing routines. (167) However even these attributes only had a small effect on compliance. Recommendations were more likely to be followed if they were based on scientific evidence, had no consequences for practice management (e.g. organisation of care, additional resources), did not demand new skills and knowledge and would not provoke negative reactions in patients, but these attributes had less influence than those listed above. (167) Recommendations that were more likely to be followed stipulate behaviours e.g. what, who, when, where and how. (168) These recommendations influence comprehension, recall, planning and behaviour, and are clear. Moreover, it is easy to determine when they have been achieved. (168) Indicators of guideline implementability have been developed into the GuideLine Implementability Appraisal (GLIA). (169) This 10 dimension instrument can be used in guideline development and implementation stages. (169)

Another influential theory of implementation is described by the ‘Awareness-to-Adherence’ Model of Pathman et al. This approach suggests four steps to implementation:

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1 Questions from the following dimensions are applied to each guideline recommendation: Decidability and Executability, Global, Presentation and Formatting, Measurable Outcomes, Apparent Validity, Flexibility, Effect on Process of Care, Novelty/Innovation, and Computability. The recommendations either meets the criterion / does not meet / unable to determine / not applicable. (169)
1. Awareness
2. Agreement with recommendations
3. Adoption, and
4. Adherence, applying it to at least 90% of eligible patients (institutionalising or routine adherence). (44)

In his Social Cognitive Theory of individual and group learning and behaviour change, Bandura refined the adoption step. (44) This theory emphasises that change depends on self-efficacy (belief that one can perform the recommended practice) and outcome expectancy (belief that this practice will lead to the suggested outcome). (44) Such cognitions and other personal factors (affective, and biological events) interact with environmental factors and behaviour. (170) Other influences on the adoption phase are described by Fox et al. as being social and political forces (group norms, professional regulations), environmental (demographics, practice setting and location, patient issues) and intra-provider (age, sex, motivation, attitudes). (44)

Even for the construct of motivation, there are several theories – content theories, cognitive theories, psychoanalytic theories, environmental theories – each of which has sub-theories. (171) Yet the theories of motivation constitute only one of a set of psychological theories which also include organisational theories and action theories. (166) For example, the ‘stages of change’ theory (112, 172) overarches pre-motivation, motivation and action. Listing these few theories illustrates several problems: there are many theories, they may conflict or overlap, and as mentioned in section 3, there is no uniform theory and nothing to recommend unequivocally one over the others. (42, 111, 112, 173, 174)

**IMPLEMENTATION BARRIERS:** The flip-side of these theories and influences on implementation is the barriers to implementation, which should be assessed during the development phase of a guideline. (44, 158, 169) However, identifying barriers to implementing evidence requires input from the health professionals targeted by the guideline, which is not always straight-forward. Guidelines for use in primary care are often criticised for not including GP preferences. While GPs who had not been involved in guideline development think that guideline development lacks GP input, GPs who have participated in guideline groups are aware of the time-consuming meeting and work commitments of the groups. (100)

Developed from the theories listed above were the following barriers and solutions: (44, 158)
<table>
<thead>
<tr>
<th>Problem</th>
<th>Examples</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lack of awareness</td>
<td>• Not reaching the intended user&lt;br&gt;• High volume of information&lt;br&gt;• Complexity of information&lt;br&gt;• Lack of clarity of recommendations, readability and format&lt;br&gt;• No time to stay informed</td>
<td>• Multiple, simultaneous methods of dissemination</td>
</tr>
<tr>
<td>Lack of agreement</td>
<td>• Conviction that current practice is fine or that no relative advantage offered by change&lt;br&gt;• Interference with custom, habits&lt;br&gt;• Incompatibility in present practice setting&lt;br&gt;• [In]Credibility of guideline team</td>
<td>• Social influence approaches, &lt;br&gt;• Local opinion leaders&lt;br&gt;• Education</td>
</tr>
<tr>
<td>Lack of self-efficacy</td>
<td>• Strong adherence to existing practice&lt;br&gt;• Lack of skills, time, effort, expertise&lt;br&gt;• No acknowledgement of benefit</td>
<td>• Interactive tutorials to improve skills&lt;br&gt;• Audit and feedback on sub-optimal current practice&lt;br&gt;• Retrospective audit and feedback to demonstrate health gains. (44, 158)</td>
</tr>
<tr>
<td>Lack of outcome expectancy</td>
<td>• Patient population not representative&lt;br&gt;• Lack of co-operation of colleagues&lt;br&gt;• Lack of overview of impact on overall population&lt;br&gt;• Conviction that major input will lead to marginal outcomes</td>
<td></td>
</tr>
<tr>
<td>Lack of cueing mechanisms</td>
<td>• Competing issues at patient encounter&lt;br&gt;• Lack of constant reminders of the guidelines&lt;br&gt;• Lack of technical possibilities for implementation</td>
<td>• Electronic flag identifying eligible patients at the start of a consultation&lt;br&gt;• Attachment of guideline to clinical records or on desk-top. (44, 158)</td>
</tr>
</tbody>
</table>
The barriers listed above have been modified into ‘sequence of behaviour change’ and related ‘barriers to guideline adherence’. (175)

Table 2.4.b. Barriers that can impact on behaviour change and guideline adherence

<table>
<thead>
<tr>
<th>Sequence of behaviour change</th>
<th>Barrier to guideline adherence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowledge</td>
<td>• Lack of familiarity</td>
</tr>
<tr>
<td></td>
<td>• Lack of awareness</td>
</tr>
<tr>
<td>Attitudes</td>
<td>• Lack of agreement, guidelines in general</td>
</tr>
<tr>
<td></td>
<td>• Lack of agreement, specific guidelines</td>
</tr>
<tr>
<td></td>
<td>• Lack of outcome expectancy</td>
</tr>
<tr>
<td></td>
<td>• Lack of self-efficacy</td>
</tr>
<tr>
<td></td>
<td>• Lack of motivation</td>
</tr>
<tr>
<td>Behaviour</td>
<td>• External barriers:</td>
</tr>
<tr>
<td></td>
<td>• Patient factors</td>
</tr>
<tr>
<td></td>
<td>• Guideline factors</td>
</tr>
<tr>
<td></td>
<td>• Environmental factors</td>
</tr>
</tbody>
</table>

More details of each of the barriers are given in Cabana et al. (175)

This model illustrates that guidelines affect knowledge, attitudes and behaviours before they affect patient outcomes. (175) While behaviour can be changed without change in knowledge and attitudes, modifying knowledge and attitudes probably generates a more sustainable change. (175)

Barriers to implementation have been further defined as information management (overload), clinical uncertainty (also incorporating knowledge, attitudes and skills), lack of a sense of competence, perception of liability, patient expectations, standards of practice (new methods conflicting with old, and effect of the peer group), financial disincentives and organisational/administrative issues. (106, 172, 176) Barriers particularly important to the adoption of guidelines are lack of awareness and motivation (inertia) and perceived external factors. (113, 175) One study identified 41 types of barrier (177) and a systematic review identified 293 potential barriers to physician adherence to guidelines. (175) Personal characteristics of doctors and age, experience, competence and willingness to change can also influence change in practice and some of these characteristics are difficult or impossible to alter. (178-180)
Co-morbidity and potential drug-drug interactions are proven predictors for non-adherence to guidelines. Individual guidelines are written for individual conditions and as these publications do not “talk to each other”, there is an increased risk of drug interactions in patients who have multiple conditions. Guideline implementation that requires changing the existing medication of patients is viewed as less than desirable, especially if the guideline calls for initiation of a new treatment that requires stepped titration, and thus more patient appointments and greater GP workload. A guideline recommendation that challenges the ‘accepted wisdom’ may threaten GPs’ trust in evidence. Reversing a trend in prescribing can be extremely difficult.

While GPs may want to change practice, other barriers to practising EBM or using guidelines include patients (wanting treatment despite lack of evidence for effectiveness or refusing treatment despite the evidence) and insufficient time (to read and appraise articles, to search for evidence and time within the consultation to refer to guidelines).

**INTRODUCTION TO IMPLEMENTATION STRATEGIES:** There are many organised methods of implementing initiatives to change practice but the success rate of measured change is not constant for each of these strategies. The strategies are listed below (see table 2.4.c) in alphabetical order, and could be used to change the management of HF in primary care. The relative costs, resource use and feasibilities of each strategy will not be discussed [however the feasibility and possible resource requirements of guideline dissemination and implementation strategies have been covered in interviews (182)].

The strategies are needed to avoid situations such as the 13 year time delay seen between research evidence and its implementation in practice as characterised the use of thrombolytic therapy. Similarly there is a need to reduce the estimated 6 to 12 months that it can take before a guideline is adopted into primary care. An extreme example is the eradication and prevention of scurvy in sailors. An initial experiment in 1601 with citrus (lemon juice) as a dietary supplement succeeded but did not change practice. The experiment was repeated in 1747 with oranges and lemons but daily citrus (again, lemon juice) was not added to the Navy diet until 1795. Different methods of implementation can have different effects on variables that may be required for this transfer and embedding in practice, e.g. knowledge, skills, competency, goal setting, social or group norms, motivation, and behaviour change.
Other factors to consider are the interaction between the personal attributes of the learner, the type of implementation strategy and the instructor (depending on the strategy). It can be challenging to determine which constructs are crucial to be addressed and how to evaluate them.

**RELEVANCE TO CURRENT THESIS:** The strategies that were tested in this study are in bold text. The Internet is a relatively new method for educational interventions, so predominance was given to this implementation strategy. It was selected because at the conceptualisation of this research project it was a novel method of delivery. Moreover, on inspection of the literature there seemed very little robust analysis of the effects of Internet education, i.e. randomised trials with objective outcome measures, rather than pre- and post-intervention knowledge tests. The Internet CME was delivered on the Goodfellow Unit platform, which was familiar to GPs within NZ.

The small group format used in this research was also familiar to GPs. They could discuss aspects of their practice and identify norms of practice and learn through the experience of others. These aspects show similarity to social cognitive theory – the interaction of environmental factors, behaviour and personal factors (cognitive, affective, biological events). (170) The small group format of CME is a commonly used arrangement in NZ. Several GPs meet together on a regular basis to discuss cases, listen and learn about topics with an invited speaker. All participants can contribute, and discuss management patterns, new developments or concerns. They also receive peer support and there is a social aspect. GPs are comfortable using this format to help meet their CME needs. In addition, small group format can significantly improve prescribing decisions, professional practice and practice outcomes compared with no intervention. (39, 40) Knowledge may also be retained longer after small group CME than didactic lecture. (187)

The control group (guideline mail-out) received no active intervention or teaching instructions. At the time this research project was developed, passive dissemination was viewed as unlikely to change practice. (106, 109, 188, 189) Research has since shown that passive dissemination is more powerful than expected. (182) Guidelines were mailed out in the belief that this was critical for patient safety and provision of the most recent EBM was seen as ethical.

Small-group and Internet were selected as the two active interventions on the basis that almost identical information could be delivered in the educational intervention, thus
testing the implementation strategy. There was also a pragmatic reason for this selection – to assess the usefulness of these two strategies, which were employed by the primary health care community to access information on EBM. These forms of educational intervention were commonly used to deliver education to NZ general practice.

The strategies listed below could have been feasibly used to deliver an intervention around the new HF guidelines (those selected for this research project are in bold). They exemplify what is involved in delivery and how effective the strategies have proved to be. (Cochrane reviews from the EPOC group have been used wherever possible to provide robust evidence.) Nevertheless as discussed in section 3.3, the strategies that GPs perceive as effective in changing practice and implementing recommendations are not necessarily those which have evidence of being effective.

Table 2.4.c. Methods used to change practice

<table>
<thead>
<tr>
<th>Method</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Academic detailing</td>
<td>Face-to-face meeting (one-on-one or small group) about a specific topic, using evidence-based medicine to inform change in practice, possibly after a review of current practice, by a trained healthcare professional.</td>
</tr>
<tr>
<td>Audit and feedback</td>
<td>Medical records are reviewed for information on a particular topic and the GP receives information about the results. This may be compared with data from local GPs or with guideline recommendations.</td>
</tr>
<tr>
<td>Didactic lecture</td>
<td>Traditional format with lecturer delivering a talk on a topic to an audience.</td>
</tr>
<tr>
<td>Internet</td>
<td>This could take many forms, but here is used to mean material read online and potentially a quiz where answers and reasoning are provided as feedback.</td>
</tr>
<tr>
<td>Opinion leader</td>
<td>Well respected expert in their field.</td>
</tr>
<tr>
<td>Passive dissemination</td>
<td>Information sent, without any interactive component, e.g. mailed guidelines, journals, pharmaceutical reference manuals.</td>
</tr>
<tr>
<td>Small group</td>
<td>Group of GPs, quite often established to meet with or without an expert to discuss a topic.</td>
</tr>
<tr>
<td>Specialist – GP Interaction</td>
<td>Examples include discharge letters from hospitals or from specialists sent back to a GP about a particular patient.</td>
</tr>
</tbody>
</table>

Besides the mechanical aspects of these implementation strategies, the influences that can cause or hinder change in practice must also be considered. The following table has
been developed from twelve domains that have been used to explain behaviour change (166). It contrasts the effect of each domain in relation to the implementation strategy (see table beneath for the grouping of the domains).

Table 2.4.d. Strength of influences seen in each implementation strategy

<table>
<thead>
<tr>
<th>Implementation strategy</th>
<th>DOMAIN†</th>
<th>Understanding</th>
<th>Psycho-social and cognitive</th>
<th>Resources</th>
<th>Change in practice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Academic detailing</td>
<td>★ ★</td>
<td>★ ★ ★</td>
<td>★</td>
<td>★ ★ ★</td>
<td></td>
</tr>
<tr>
<td>Audit and feedback</td>
<td>?</td>
<td>★ ★</td>
<td>★</td>
<td>★ ★</td>
<td></td>
</tr>
<tr>
<td>Didactic lecture</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Internet</td>
<td>★</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Opinion leaders</td>
<td>★ ★</td>
<td>★ ★</td>
<td>★</td>
<td>★</td>
<td></td>
</tr>
<tr>
<td>Passive dissemination</td>
<td>★</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Small group</td>
<td>★ ★</td>
<td>★ ★ ★</td>
<td>★</td>
<td>★</td>
<td></td>
</tr>
<tr>
<td>Specialist-GP interaction</td>
<td>★</td>
<td>★</td>
<td>★</td>
<td>?</td>
<td></td>
</tr>
</tbody>
</table>

★ Influence and strength of influence on the Domain

? Unclear influence on the Domain

† The twelve domains were collapsed into similar concepts for ease of use. See next table (2.4.e) for the groupings.

Table 2.4.e. Expanded domains of Understanding, Psycho-social and cognitive, Resources, and Change in practice

<table>
<thead>
<tr>
<th>COLLAPSED DOMAIN</th>
<th>EXPANDED DOMAIN (166)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Understanding</td>
<td>Knowledge, Skills</td>
</tr>
<tr>
<td>Psycho-social and cognitive</td>
<td>Social/professional role and identity, Beliefs about capabilities, Beliefs about consequences, Motivation and goals, Memory, Attention and decision process, Emotion, Behavioural regulation, Social influences</td>
</tr>
<tr>
<td>Resources</td>
<td>Environmental context and resources</td>
</tr>
<tr>
<td>Change in practice</td>
<td>Memory, attention and decision process, Nature of the behaviours</td>
</tr>
</tbody>
</table>

Referring to tables 2.4.d and 2.4.e the impact of the implementation strategy reaches deeper than the ‘Domains’ suggested by Michie et al (166). For example focusing on
the Domain of ‘Emotion’ an audit and feedback approach may be accompanied by the ‘Constructs’ of stress, fear, anxiety and threat (see pp30, 31 ibid).

The effect on Domains and Constructs may also depend on who instructs the implementation strategy. An audit and feedback session (see section 4.2) conducted by a PHO or DHB could have a greater effect on Environmental contexts and resources, and the need for Behavioural regulation. In contrast if the audit and feedback were delivered by a peer, there might be greater emphasis on Social/professional role and identity, Beliefs about capabilities, Beliefs about consequences, and Social influences.

As described in section 2.4.5, different types of opinion leader have been identified. They influence different aspects of the pathway from evidence to practice. Knowledge is gained from the ‘expert’ opinion leader who influences knowledge. The ‘peer’ opinion leader produces Social influences on others and can shape Beliefs about capabilities and Beliefs about consequences (see Table 2.4.d). These are examples of potential influences that can improve or obstruct the implementation of evidence in practice and should be considered in relation to the next sections on implementation strategies.

2.4.1 Academic detailing

Educational outreach visits (aka academic detailing) involve a personal visit to a healthcare professional in their own setting, and is also a strategy used by pharmaceutical companies. (190) Avorn and Soumerai based the concept of ‘academic detailing’ on the detailing visits used by pharmaceutical representatives. (191) They initially trialled this method in 1983 successfully reducing prescribing of excessively prescribed drugs (192) and later described eight important techniques of academic detailing: (193)

1. Conducting interviews to investigate baseline knowledge and motivations for current prescribing patterns
2. Focusing programmes on specific categories of physicians
3. Defining clear objectives
4. Establishing credibility using a respected organisation, referencing quality information and impartial presenting of controversies
5. Using interactive educational interventions
6. Using concise visual educational materials
7. Highlighting and repeating the essential messages and
8. Providing positive reinforcement of improved practices in follow-up visits. (193)

**EFFECTIVENESS:** A Cochrane review looked at educational outreach visits and their effect on inappropriate prescribing.\(^k\) (190) There were comparisons of outreach visits (including educational materials and conferences) with no intervention (± educational materials or conferences) which demonstrated that outreach visits were effective in reducing inappropriate prescribing, and comparisons of outreach visits combined with a complementary intervention (reminders, audit and feedback, local opinion leaders, marketing strategies, patient-mediated interventions) against no intervention. Most of the trials that combined outreach visits with an additional intervention showed that the intervention had positive effects. (190) The positive effects of educational outreach visits, especially when combined with social marketing, were small to moderate but potentially of practical importance (e.g. reducing inappropriate prescribing). (190)

A further review of multiple educational outreach (vs. no intervention or vs. intervention control, most commonly dissemination of educational materials) found that most studies reported improvement but that this effect was only modest (effect size >5% but ≤10%). (182) Further analysis of multiple outreach vs. no intervention indicated that combinations of educational materials, meetings and outreach may have modest to moderate effects (effect size >10% but ≤20%), but that educational materials and outreach may be relatively ineffective on their own. (182)

The evidence for educational outreach may not be as clear cut as shown by a cRCT that compared:

- a postal bulletin with
- a postal bulletin followed by an outreach visit (individualised prescribing feedback and guidelines-based educational information) consisting of a 10 minute presentation on the bulletin data plus additional information and a 5 to 20

\(^k\) It included only RCTs (n = 18) of healthcare providers responsible for patient care (mainly primary care doctors practising in community settings in North America, Europe, and Asia-Pacific regions) that objectively measured provider performance in a health care setting or health care outcomes. Studies that measured knowledge or performance in a test situation were excluded. The majority of trials assessed prescribing practices, the rest preventative services or general management of a variety of common problems encountered in general practice. All studies showed some risk of bias which ranged from inadequately concealed allocation, differences in baseline measurements following randomisation, unblinded outcomes assessment, or differences between the unit of analysis and the unit of randomisation. (190)
minute Q&A opportunity. Most academic detailing was one-on-one, with some group meetings held with other GP partners and a practice nurse. (194)

The outcomes were prescribing preventative therapies in CVD and DM, and there was an increase in prescribing for both the postal bulletin group and the academic detailing group but no significant benefit for academic detailing over the posted bulletin alone. (194)

To further blur the effects of academic detailing, this paper then quotes four other studies that used academic detailing in CVD (one in asthma). Two of them found positive effects, but the other two studies were negative. (194)

While educational outreach can be effective in changing clinical behaviour, the change may not always be in the anticipated direction. A pragmatic cRCT compared an educational outreach programme\(^1\) with postal dissemination of the guidelines. (195) The proportion of appropriate referrals was higher in the intervention group (P < 0.05), but the proportion of major findings at referral did not change significantly and this latter finding was unexpected. Another unanticipated finding was the significant rise in drug prescription and expenditure in the intervention group and there was no clear reason why some of the results deviated from the anticipated outcomes. (195)

**PRACTICE DEMOGRAPHICS:** The practice composition may also influence effectiveness. Educational outreach visits promoted 2 of 4 guidelines and looked at the improvement from baseline in the number of patients treated according to guideline recommendations. (196) The overall effect was a 5.2% increase (95% CI 1.7% to 8.7%) in the number of patients treated within guideline recommendations with changes for the four guidelines ranging from -3% to +7%. When analysed by practice size, small practices (≤2 FTE GPs) improved their practice by 13.5% (6% to 20.9%) but larger practices showed an increase of only 1.4% (-2.4% to 5.3%, interaction P <0.001). (196)

Smaller practices may be more relaxed about changing practice with larger practices experiencing more organisational complexity, with possibly a more embedded approach to prescribing. The educational outreach may have been less intensive in larger practices as not all GPs attended the intervention visit or were available for follow-up visits. When counted by practice size, 96% of small practice GPs and 48% of large

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\(^1\) The educational outreach programme consisted of small group GP meetings chaired by a local hospital specialist with a 15 minute presentation of guidelines plus one hour discussion including testing services available and summaries of local prescribing data. These were followed up by a visit at three months. (195)
group GPs attended the outreach visits but analysis showed that the dilution effect of reduced attendance could not explain the differences in observed effect. (196)

Another study found that larger list size had an association with poorer prescribing. (197) A RCT compared practices receiving an educational outreach visit (evidence-based visual presentation, printed referenced summary, flowchart of management and a six month follow-up visit) with control practices receiving a ‘monitoring visit’. The educational intervention gave GPs the opportunity to learn about new drugs in more depth even though positive trial data had been published. The intervention practices had significantly fewer referrals and a significantly higher use of a new and effective drug than the control group. There was no overall difference between the control and intervention groups for prescription rates of a relatively ineffective drug. Despite evidence being presented regarding the negative effects of this drug, it was difficult for the GPs to stop prescribing it. Poorer prescribing seemed to be associated with larger practice list size and the proportion of male partners in the practice exceeding 75% (although the topic was menorrhagia and male doctors may see fewer patients with this presentation). (197)

**DURABILITY AND PERCEPTION:** Consideration also needs to be given to the cost and time-consuming nature of educational outreach interventions. (195) The importance of the number of visits or how long the effects of the outreach visit last is not certain, with one trial indicating that at the end of one month of follow up the effects of the outreach had lessened. (190) But information from a study (198) that had been excluded from the Cochrane review (see above) suggested that reductions in inappropriate prescribing were still seen two years after the outreach visit. The relationship between the number of interventions and effect size was not obvious for either multiple educational outreach vs. no intervention or vs. intervention control. (182)

The outreach visit regarding preventative therapy prescribing for CVD and DM was viewed as useful by 81% of GPs who received this visit and wanted to continue to receive the postal bulletin. While the limitations of a one-off intervention on sustained change in practice were acknowledged, 94% of GPs felt that the feedback had some impact on their practice. (194)

There is also uncertainty about the cost-effectiveness of time- and labour-intensive outreach visits as an intervention strategy, which is essential to take into account given the previous point regarding the durability of the information conveyed in the visit.
SUMMARY: Academic detailing allows the recipients to discuss in-depth the contents of the intervention. While some improvements have been seen as a result of academic detailing, these may depend on the size of the practice involved. The knowledge gained through academic detailing may not persist in practice.

2.4.2 Audit and feedback
Audit and feedback comprise monitoring of individual physician performance and comparing it with that of a peer group or providing a summary of clinical performance that may include recommendations for clinical action. (189, 199) These approaches seem most effective in changing prescribing and test ordering. (189) Audits need to be planned in advance with appropriate standards, and by someone who has knowledge of audit methodology and analysis. (200)

The benefits of audit have been described as improved communication between colleagues and other health professionals, improved patient care and service delivery, increased professional satisfaction, and better administration. (200) However, the negative aspects of audit have been described as increased workload, restrictions of clinical freedom, fears of litigation, and unwillingness to criticise other doctors. (200)

EFFECTIVENESS: A 2003 Cochrane review of RCTs of the effectiveness of audit and feedback included health care professionals and objectively measured provider performance or health care outcomes. (199) The types of targeted behaviours and outcomes measured varied greatly between trials as did the content of feedback and the timing (frequency and duration). Comparing audit and feedback alone to no intervention found the effects of the intervention were small to none. Short term effects were compared with long term effects after feedback stopped but effects were variable across the studies reviewed (different outcome measures used). A few studies compared audit and feedback to other interventions but audit and feedback were inferior, equal or superior. Ten high quality studies (that included a number of different comparisons) were assessed as individual studies and the comparisons again showed mixed results. It was not possible to determine which features of audit and feedback

---

\[a\] The review excluded studies that measured knowledge or performance in a test situation. 85 studies were included with over half of these set in North America with two-thirds set in general practice, family medicine or in community care. Non-compliance with the targeted behaviours was measured before and after the outcome and used to determine the effect of the audit and feedback. (199)

\[b\] The authors indicated that some studies used the incorrect unit of analysis and that many were only of moderate quality (i.e. protection against bias). (199)
related to its effectiveness. The overall conclusion was that audit and feedback can produce small to moderate improvements in professional practice but these improvements are more likely to be seen in situations where the baseline adherence to recommended practice is low. (199)

Reviews of audit and feedback, and audit and feedback plus educational materials, found modest improvements in care (effect size >5% but ≤10%). However some studies had indeterminate statistical significance or unit of analysis errors. (182)

Other studies also discovered mixed results seen in the Cochrane review. One review of audit and feedback found included studies had 10 positive and 14 negative outcomes, but the outcomes were more consistently positive when feedback was in the form of chart review. (160) A comparison of 15 studies of chronic disease management (audit and feedback vs. no intervention) found that in only half the studies was the intervention better than nothing. (201)

A single study of peer review medical audit (four meetings, two of which were feedback sessions, over two years in six general practices) looked at improving the diagnosis and management of hypertension. (202) It showed improvements in GPs’ clinical behaviours (e.g. recordings of BMI, lipids, urea and electrolyte values) but not complex behavioural changes (described as the diagnosis of hypertension and better BP control which require cognitive actions by the GP and patient cooperation). (202)

The Echocardiographic Heart of England Screening (EcHoES) study (203) used education and feedback. Patients with LVD were identified and letters were sent to their GPs, which said that the patient had LVD and recommended ACEi prescription. This feedback resulted in “significant uptake” of ACEi treatment for most practices. (203)

**VARIATION:** Factors that may explain the variation in results are the: type of intervention (or combination of interventions), intensity of feedback, complexity of the targeted behaviour, seriousness of the outcome, baseline compliance with best practice, and quality of the study. (201, 204) There seems to be no increased effect with increased intensity of feedback. (201)

**SUMMARY:** Audit and feedback can improve certain aspects of practice but overall the effects have been variable and the variation may be explained by the implementation strategy, the target behaviour and the context. The audit and feedback will need to be well planned and may be an iterative process.
2.4.3 Didactic lecture

Didactic education sessions have been defined as predominantly lectures or presentations with minimal participant interaction or discussion. (165) Lectures or presentations alone are unlikely to change professional practice. (40) This type of educational intervention is often used as a default control group in studies.

An assessment of three didactic lectures aimed at primary care physicians found that overall none of the interventions changed physician performance. (40, 165) A comparison of a didactic intervention with an ‘active’ learning session found no difference in knowledge gain, but the education of the didactic session was valued more highly. (164, 205)

**SUMMARY:** Didactic interventions do not promote change. They may promote knowledge, skills, or attitudes or be predisposing elements to change but by themselves they do not improve doctors’ performance or patient outcomes. (165)

2.4.4 Internet

The success rate of Internet education has usually been measured by pre- and post-tests of knowledge but in studies reviewed, the translation of knowledge to practice is not actually measured. This was a key reason for this study testing Internet educational intervention against small-group format and control. The doctors who are using the Internet have been described as innovators and early adopters (see section 2.3.4), and are the ones who will assist the uptake of the Internet as a mainstream educational tool. (206)

**USAGE:** At the end of 2001, a year before this study started, in-depth interviews with 24 GPs in New Zealand regarding their experiences of and preferences for CME ascertained that their Internet use was not widespread (factors contributing to use were interest, expertise and availability). (134) A number of GPs predicted using the Internet more in the future. (134) As can be seen from Table 2.4.f, access to the Internet has become more widespread, and use of the Internet by medical professionals has increased over time.
Table 2.4.f. Access to and use of Internet (general and specific) over time

<table>
<thead>
<tr>
<th>Country, Year</th>
<th>Internet Access</th>
<th>Internet Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia, 1995 (135)</td>
<td>9% (home or work)</td>
<td></td>
</tr>
<tr>
<td>America, 1998 (207)</td>
<td>74%</td>
<td></td>
</tr>
<tr>
<td>American Medical Association, various years (208)</td>
<td></td>
<td>10% (1997)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>37% (1999)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>70% (2000)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100% (2001)</td>
</tr>
<tr>
<td>Canadian Medical Association, 1999 (132)</td>
<td></td>
<td>41% (CME)</td>
</tr>
<tr>
<td>New Zealand, late 1999 – early 2000 (140)</td>
<td>40% (work)</td>
<td>56% (patient care)</td>
</tr>
<tr>
<td></td>
<td>76% (home)</td>
<td></td>
</tr>
<tr>
<td>Germany, 2001 (122)</td>
<td></td>
<td>54%</td>
</tr>
<tr>
<td>America, 2001 (4)</td>
<td></td>
<td>53% (medical information)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29% (specific patient information)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31% (CME)</td>
</tr>
<tr>
<td>America, 2002 – 2003 (133)</td>
<td></td>
<td>67% (patient specific information)</td>
</tr>
<tr>
<td>Germany 2007 (122)</td>
<td></td>
<td>82%</td>
</tr>
</tbody>
</table>

As might be expected, the early users of the Internet, who enjoyed reading journals or participating in online CME, were termed ‘enthusiasts’. (134)

**DEMOGRAPHICS:** Some GP demographics influence use of the Internet. A NZ study (140) (conducted between November 1999 and February 2000) identified that GPs aged <35 years had the highest use of the Internet for queries about patient care (71%) (see table 2.4.g). The age and sex of the GP were independent predictors of using the Internet for patient care. (140)

Table 2.4.g. Effect of age on use of Internet for queries about patient care

<table>
<thead>
<tr>
<th>Age</th>
<th>Odds Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 year age band*</td>
<td>0.78</td>
<td>0.60 – 0.90</td>
</tr>
<tr>
<td>GPs &lt;35yrs</td>
<td>2.69</td>
<td>1.10 – 6.60</td>
</tr>
<tr>
<td>GPs &gt;55yrs†</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

* Other variables not associated were sex, solo practice, rural, part-time status, years in practice or patients seen per week.
+ Variables of solo practice, urban location, and patients seen per week were controlled for.
Similarly, length of time in practice also had an effect on Internet use with doctors who had spent less time in practice (mean years 13.9) tending to make greater use of the Internet than those who had been longer in practice (mean time 17.0 years, \( p < 0.001 \)). (132) In addition, younger or more recently graduated doctors were more likely to use and have more confidence in using the Internet to find medical information, (1, 4) access on-line journals and place greater professional value on the Internet (4). They were also more likely to participate in Internet education or use personal computers. (1)

Studies on the effect of the sex of the GP on Internet use are not as clear. One NZ study found that the age (see above) and sex (see table 2.4.h below) of the GP were independent predictors of using the Internet for patient care. (140)

Table 2.4.h. Effect of sex on use of Internet for queries about patient care

<table>
<thead>
<tr>
<th>Sex</th>
<th>Odds Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>1.72</td>
<td>1.02 – 2.90</td>
</tr>
<tr>
<td>Female†</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

† Variables of solo practice, urban location, and patients seen per week were controlled for.

Another study of online CME users found higher use by women doctors than expected from the proportions seen in comparative populations. One example is that of an IPA that had 8% female doctors, but 43% of the online CME participants were female. Demographic data indicated a tendency for female primary care physicians to be younger than male doctors, ranging from approximately four to nine years depending on location. (206)

A large scale survey of 2200 American physicians in 2001 about Internet use found that male physicians reported more experience and confidence in using the Internet (4) and were more likely than female physicians to use the Internet for CME and to have accessed it for CME in the past year (sample demographics: 79% male and 32% primary care). (4) A Danish study (11/2006 to 03/2007) found that male GPs and members of the Danish College of GPs were more likely to use an e-learning programme female GPs or non-members of the College. (209)

**RESISTANCE AND EXPECTATION:** In a study conducted in 1998, some of the barriers to using the Internet for learning were reported by doctors as “don’t know how
to use it” (20%), “prefer in-person instruction” (17%) or “too time consuming” (9.5%).

(132) Other perceived major barriers to Internet use (2001 American study, 2200 physicians and 2003 American study 457 family physicians) have been excess information to scan (30% of survey respondents, 59%), unable to find information (28%, 44%) and lacking searching skills (23%, 61%). (4, 210) Even if well respected online CME providers exist (e.g. the Goodfellow Unit© CME club site, RNZCGP-endorsed) there may be limited knowledge or use of this. (134) A NZ study also reported that some GPs were not interested in learning via the Internet. (211) But some of these GPs would have needed to upgrade their hardware or software and improve their technical skills to benefit from participating in Internet CME. (211: #3095) This likelihood was echoed in a review which found that the main barriers to using Internet courses were the lack of competence among users and technical difficulties with programmes. (1) Some family physicians reported not using on-line CME as they had adequate access to high quality face-to-face CME. (133, 212)

A large American survey of physicians and Internet usage (2001), repeated in 2003 found that traditional activities, journals and local CME meetings were still popular and the majority of doctors using the Internet did so to supplement other learning activities and look up medical information. (4, 210) While in-person education was the most popular, trends indicated increasing use of Internet education. (1)

Doctors using the Internet for professional development expected it to be immediate, credible, easy to use and relevant. (4) When asked what factors were important in using on-line CME, (ranked from 1 = most important to 6 = least important) American physicians placed provider credentials as least important (mean score 6) while ease of programme use was ranked highest (mean score 2.5). Validity of content scored 2.6, while evidence-based content received a mean score of 4.2 and was ranked 4th out of 8 factors. (4) A later study reported credibility as the most important factor related to clinical information on the Internet, (210)

While it could be argued that use of the internet may be no different from reading, online courses can be interactive; for example online quizzes can provide immediate feedback where students can learn from their mistakes and not worry about appearing ignorant when asking or answering questions. (212, 213) There is also the opportunity

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© The Goodfellow Unit is located within the Department of General Practice & Primary Health Care, University of Auckland, and provides continuing education to GP, nurses and other primary care health professionals. The web-based CME club was established in 1998.

http://www.fmhs.auckland.ac.nz/soph/centres/goodfellow/default.aspx
for reflection with GPs able to work through parts of a course, see patients, put the
learning into context, and if required, review the course in light of the patient contact.
(212) However, the independent nature of Internet CME also meant that GPs felt that
they could skip parts of programmes that they felt they knew enough about. (212) For
those participating in online learning, they could participate at a time convenient to
them and work for the length of time that suits them, which could save time. (211-213)

**EFFECTIVENESS:** Studies of online-CME generally indicate increased knowledge
(see below) and while the sustainability of this knowledge and transfer to clinical skills
and patient outcomes are uncertain, Internet CME (despite the heterogeneity of
reviewed studies) seems to be on a par with other traditional CME methods.

**Meta-Analysis:** A comparison (201 studies) of Internet-based interventions for health
professionals vs. no intervention and vs. non-Internet formats, measured outcomes of
knowledge, skills, satisfaction, learner behaviours, and effect on patient care.⁷ (214)

For Internet education vs. no intervention the pooled effect sizes were high for
knowledge, skills, and behaviour and patient care⁴, indicating that Internet-based
educational interventions are better than no intervention. (214)

The comparison of Internet-based education with non-Internet education suggested no
participant preference for any modality. The knowledge outcome favoured Internet-
based education but the skills outcome did not reach statistical significance and there
was a wide range of individual effect sizes for these two outcomes. Learner behaviours
and effect on patient care were not statistically significant and individual effect sizes
ranged widely. (214)

Some of the difference may be explained by the learner groups, education methods and
other aspects of the studies. The inconsistency of individual results suggests that some
methods of implementing Internet courses are more effective than others. Overall
effectiveness of Internet-based education may be similar to traditional methods and

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⁷ The health professionals included physicians, nurses, pharmacists, dentists, occupational and physical
therapists, and veterinarians at student, trainee and graduate levels. Internet-based instruction (including
local intranet) was defined as Web-based tutorials, virtual patients, discussion boards, e-mail and Internet-
mediated video conferencing. Non-Internet interventions included face-to-face courses or paper modules,
satellite-mediated videoconferences, and slide-tape self-study modules. Knowledge outcomes tended to
be multi-choice tests, few objectively scored methods, and some self-report measures of knowledge,
confidence or attitudes. Skills outcomes were objectively assessed for about three-quarters of outcomes.
Almost two-thirds of behaviour and patient effect outcomes were self-reported. Objective assessment of
these was generally by medical record review. (214)

⁴ There was large variation in the outcomes from individual studies which indicated considerable
inconsistency.
there may not be a global effect of Internet-based education across learners, content and outcomes. (214)

**Knowledge:** A review of 17 eligible studies of online education for different health professionals\(^1\) focussed on areas such as practices and preferences for Internet education, and evaluation of outcomes of Internet education. (1) A number of studies found that the online programmes were effective but not superior to traditional formats in imparting new knowledge but less evident was how beneficial the online education was in changing practice. (1)

A comparison of Internet-based and print education looked at six studies (all RCTs, participants were practising or training healthcare professionals) which showed mixed results. In three studies, post-test scores were higher for the Web participants than the students who used print-based materials. Two studies found that the web-based courses were more ‘learning efficient’. One study found that more time was spent on the modules by the Web-based group but another study found no difference between the groups in time spent on learning. Three studies found that the participants had greater satisfaction with web-based learning. (3)

While interactive sites are the ideal, a 2001 survey found most American CME sites were text-based with less than 20% containing interactive content. Rather than transforming traditional lectures into on-line CME, case-based learning as a component of on-line courses will improve their effectiveness. (4) Case-based courses have shown an increased knowledge effect size larger than text-based courses immediately after the course. (2)

One review (six studies) comparing Web-based education and didactic / traditional lecture format found a statistically significant advantage for the Web-based programme in one study. (3) In three other studies the Web-based courses were better on a ‘unit’ basis, but this was not seen on final exam for any of the studies with both types of intervention proving effective in improving final exam performance. (3) Overall, Internet CME was as effective in transferring knowledge as the comparison interventions. (3)

\(^1\) The authors commented that there was difficulty in generalising the findings as there was a small number of studies, a lack of RCTs, heterogeneity of study designs and participants, and a wide range of sample sizes. The studies used surveys or questionnaires, or pre- and post-tests, with post-tests carried out at differing times after the online education.
**Knowledge, effect on practice and sustainability:** A study which compared knowledge before and after participation in any of 30 on-line CME courses over a 6 month period (August 2002 to March 2003) found that mean knowledge scores were significantly increased at post-test compared with pre-test, although when re-tested 4 weeks after the course, knowledge scores had significantly decreased compared with post-test but were still significantly higher than pre-test. (2) Nearly all the doctors reported making a change in their practice related to course content. However, the changes that were made differed significantly from those the participants intended making. (2)

One online course found that post-test knowledge and clinical skills were significantly increased, and attitudes and confidence in management were also increased. But these outcomes need to be weighed against the course not being widely advertised so only doctors capable of using the Internet would have accessed it, and they may not be representative of the wider population of doctors. (5)

Overall, the review (3) showed that Internet CME was as effective in transferring knowledge as were the types of intervention with which it was compared. However, one study showed that over a four to six month follow-up period the changes in behaviour were not sustained, and the behaviour and knowledge had decreased to the level recorded by participants in the didactic arm. (3)

**COMMENTARY ON CURRENT RESEARCH:** Improvements in knowledge and skills, as seen in studies of online education, are prerequisites to improvements in performance. However, it is not known if this learning will be translated into clinical practice (patient outcomes) or the sustainability of knowledge and practice will change. (1, 2, 5) Another review paper (3) talked about the dearth of RCTs in the research on Internet-based CME, and several papers have identified a need for future studies (time series or RCTs) to objectively assess whether online CME is effective in changing behaviour and the length of time these changes are maintained. (1-3)

**SUMMARY:** The Internet has rapidly become more accessible as a tool for delivering educational interventions. Interventions delivered by the Internet seem to be on par with other active interventions (for knowledge and patient care). The format is appreciated as it allows participants to utilise it at their own convenience, however it can lack any interactive or social nature. The popularity of Internet-based education is greater in younger physicians than in those who are older.
2.4.5 Opinion leaders

Opinion leaders are also referred to as “educationally influential physicians” and represent informal learning in continuing education, assisting in improving the use of evidence-based medicine in practice. (189, 215, 216) This type of intervention can often be effective. (189) Traits that have been used to describe them include having a broad general knowledge, being likeable, trustworthy, credible, a willingness to answer questions and a humanistic, effective communication style which informally facilitates learning and change in their colleagues’ practice behaviour. (217-219) It is these traits that are suggested by Social Learning Theory to be persuasive agents of behaviour change. (215) Opinion leaders enjoy sharing knowledge and clearly express information, giving practical information first, and then, if time permits, an explanation or rationale. They treat people as equals and are not condescending when asked for information. They are described as having a “high level of clinical expertise” and “always seem up-to-date”. (217)

**EFFECTIVENESS:** A Cochrane review shows that local opinion leaders promote evidence-based medicine and that these results are based on moderate quality evidence. However, a caveat is placed on the widespread use of opinion leaders with regard to feasibility. (215) This Cochrane review included only cRCTs, healthcare professionals in charge of patient care, in studies that objectively measured professional performance and / or patient outcomes. Studies that compared opinion leaders with no intervention varied in their success with some favouring the control group. Overall there was a small benefit favouring opinion leaders. Studies that compared opinion leaders against a single intervention (audit and feedback, standardised lecture), or opinion leaders plus an additional intervention (audit and feedback, standardised lecture, educational materials) compared with the additional intervention, all had positive results, although the range of results was as wide as seen in the comparison of the opinion leader vs. no intervention. An overall figure calculated for the review suggested a 10% absolute decrease in non-compliance with best practice in the intervention groups. (215)

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\(^a\) Any study that measured knowledge or performance in a test situation alone was excluded. Most studies were carried out in the United States and in hospitals. Studies were also included if they defined the method used to identify the opinion leader. Unfortunately some studies scored highly in a risk of bias assessment. Three trials were directed at primary care doctors and did not account for design effect in their analysis. The studies that were reviewed had selected a widely differing range of clinical problems to monitor. The measure that was used was non-compliance with desired practice, measured by Adjusted Risk Difference. A negative value favoured control and a positive value favoured intervention. (215)
A small randomised but not clustered study (patients N = 171, HF n = 54) found that patients randomised to GPs who received opinion-leader endorsed evidence summaries were more likely to receive therapy within six months, and HF patients were more likely to receive an ACEi or ARB. (220). The differences between control and intervention groups were probably clinically significant but did not reach statistical significance.

While the use of opinion leaders was seen to be successful in improving evidence-based practice, the methods used by the opinion leaders to deliver education and to promote evidence-based practice are still vague. What adds to the complexity of the use of opinion leaders is how they are identified, that their influence may be disease-specific and that they may enjoy only transitory status as opinion leaders. (215, 220)

**INFLUENCE:** Behaviour change seems to depend on who the opinion leader is. An opinion leader who is identified as such is more likely to cause a change in behaviour than an opinion leader who is selected from a pre-defined group. (215) The closeness of the social networks of the doctors they are trying to influence is also important, as a tight-knit community is easier to influence than a loosely connected group of doctors who rarely meet. (218) A study of the management of cardiovascular problems in general practice indicated that behaviour change more often took place when it was supported by key individuals chosen for their influence rather than formal position. (216) This theory has been reinforced in previous sections of this thesis where other research has shown that GPs identify consultants that they believed to be educationally influential.

Mobilising the support of an opinion leader greatly improves the chances of successful innovation, but there is a fine line between success and failure. If opinion leaders deviate too far from the norm, then respect for them may be lost. (216)

**APPROACH IN PRIMARY CARE:** However, the situation is not as clear in general practice, where the tendency is for GPs to approach other doctors they know, rather than doctors who are known to have expertise. When GPs were asked\(^1\) about their information seeking behaviour in relation to their peers, almost 80% used informal consultation with their peers as a first resource in seeking clinical information. (119) Over half of the participants said they would consult with the peers whom they thought

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\(^1\) 45 GPs participated out of 270 GPs who were invited. The sample did approximate regional norms apart from an under-representation of female doctors and an over-representation of participants with the College of Family Physicians certification. (119)
would be most readily available and approachable. (119) These qualities were seen to be of equal or greater importance than expertise. Most of these doctors were reluctant to ask doctors outside their communities or to ask for advice from innovators. (119) Most of them either did not know how they evaluated the information they received, or they followed intuition or they trusted the knowledge of the doctor they were asking. These GPs frequently sought advice, practised conservatively, were the least critical of the advice they received and were more concerned with maintaining norms. Doctors who identified with this information-seeking behaviour were older than the others. (119)

Almost a quarter of doctors approached peers whom they considered to have expertise in the area of interest. These GPs were unlikely to be concerned about how their questions would be received, and more likely to approach innovators and peers outside their community. They tended to use a more systematic approach for appraising information. Less than a quarter reported first searching literature before or in conjunction with asking expert peers or innovators. Specialists were consulted more frequently for advice than other GPs. The doctors in this subset were more likely to seek advice outside their community. While the literature was their primary source of clinical information, half were sceptical of the advice they received from their peers. Participants who identified with this group were more likely to be CFPC-certified⁹. Proportionately more female doctors described systematic approaches to evaluating advice. (119)

**STAGES OF IMPLEMENTATION:** The roles of opinion leader may differ at different stages of implementation. Broadly these roles could be described as expert and peer. (216) The expert, generally an academic or consultant, is seen as a credible source who can explain the evidence or whose support is seen as sufficient endorsement. It would be an opinion leader at this level who started the transfer of research evidence into clinical practice. (216) The responsibility for assisting the practical implementation of evidence would then transfer to the peer opinion leader. The peer opinion leader is considered to be an ordinary GP who is seen to understand the working lives of their colleagues, and can give their colleagues confidence that they could implement the change as well. (216) However the validity of guidelines in primary care may be undermined if the recommendations are not used in secondary care, giving the impression that the specialist’s knowledge is ‘higher’ than the evidence on which the guidelines are based. (216) What cannot be underestimated is the influence of personal

⁹ CFPC is the acronym of the College of Family Physicians of Canada.
interaction; for example an opinion leader may work well with some but not other GPs. (216) Other opinion leaders who may be negative or not committed towards a new implementation will counteract the influence of an opinion leader in favour of change. (216)

**SUMMARY:** The opinion leader approach to implementing evidence can have some positive effects. However the selection of a leader can be problematical and more than one type of leader may be needed to implement change. There is also a need to ensure that information discussed is evidence-based. The personal contact between the opinion leader and other doctors can improve community interaction. Studies of opinion leaders and the effects on change in practice can be difficult to conduct and evaluate.

### 2.4.6 Passive dissemination

Passive implementation includes the most basic form of dissemination: mailing out a guideline or educational material or the publication of research findings. (109, 189) Passive dissemination can be described as a one-way information flow, with no interactive component (188). It may raise awareness of new materials and desired behaviour change, but has been found to be generally ineffective and unlikely to cause any change if used in isolation. (106, 109, 189) However the distribution of printed educational materials is a common approach to achieving change in practice. (109, 188)

**EFFECTIVENESS:** An HTA systematic review of guideline dissemination and implementation strategies (which addressed some of the methodological problems of previous reviews) compared 18 studies of educational materials (mainly in primary care settings, against no intervention). It found that the improvements were ‘modest’ (effect size >5% and ≤10%) although individual outcomes were mixed. (182) The authors stated that the evidence base was sparse and of poor quality. (182) The concluding comment was that educational materials have a modest but transitory effect on guideline implementation which could make their use a worthwhile strategy. (182, 221)
A Cochrane review assessed the effects of printed educational materials\(^\text{v}\) on improvements in the clinical practice of healthcare professionals and patient outcomes\(^w\). (188) The number of studies reviewed was small and the approaches were very context-specific. The first comparison was printed materials vs. non-intervention control, which found that changes were small (some in favour of control), the practical importance of these changes was small if not uncertain, and no outcome estimates were statistically significant. (188)

The second comparison – printed materials plus additional strategy vs. printed materials – yielded mixed results (some positive for the combination of interventions some positive for educational materials alone). A few outcomes reached statistical significance (the inclusion of an opinion leader), but other additional interventions (educational outreach, audit and feedback, traditional teaching methods i.e. conferences and workshops) did not or seem to have practical importance. (188)

Printed educational materials will achieve only a small impact on practice (if at all), and changes are unlikely to be rapid or substantial. Their use where practice is near-optimal is likely to be ineffective. (188)

The effect of a regular and expected printed educational material (a short drug bulletin with question-and-answer format called the Therapeutics Letter from the Therapeutics Initiative of the University of British Columbia) was assessed in a paired cRCT. (222) New prescriptions for patients >65 years for drugs examined in the Letter were compared before and after the receipt of the Letter and compared over the same time period with a control group of GPs. Half of the bulletins were expected to increase prescribing while the others were expected to decrease prescribing. The prescription of all but one of the drugs moved in the expected direction, but the move was greater for the drugs that showed increased prescribing. No one letter resulted in prescribing changes that were statistically significant. Some drugs had few prescriptions, and other drugs showed little difference in change, resulting in wide CIs. When all the changes were combined (and adjusted) the probability of the recommended drug being

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\(^{v}\) Defined as publication in journals or targeted mailings, national programmes for guideline development and distribution

\(^{w}\) Studies that used measures of knowledge, attitudes or satisfaction were not included. The only study designs that were included were RCT, interrupted time series, non-equivalent group designs pre-post measurement. All RCTs had some methodological problems. Other studies used incorrect analysis methods, had insufficient power to detect small but worthwhile effects or did not meet all quality criteria. The studies included for comparison were carried out either in North America or Europe with the large majority in general practice. (188)
prescribed as opposed to another drug in the same class in the 3 months following the bulletin was 30% (RR = 1.30, 95% CI 1.13 to 1.52). (222)

**DURATION:** Distribution by mail of a CME programme on hypertension increased GPs’ knowledge in the short term, but by the end of the study there was no difference between the intervention and control groups and while blood pressured dropped in both groups, there was no difference in the amount of decrease. (223) Some studies in an HTA review indicated decay in effects over time. (182)

**ATTITUDES TO PASSIVE DISSEMINATION:** The response to a mailed guideline was reported in a NZ survey of 422 GPs (729 were contacted). (224) A copy of the recently published guideline was included with a weekly GP ‘newspaper’ (dated 09/10/2002). Two weeks later, GPs in three regions were contacted about the guideline and 70% recalled receiving them. In response to further questions about the guideline, approximately a third of answers were coded as missing: for those who had not received the guidelines or did not answer the question. When asked where the guideline was currently, 44% said it was in a desk drawer, a pile on the desk or on an office shelf. 11% either did not know or said it was in the bin. Of the 15% who selected ‘other’ for the location of the guideline, one of the more unexpected responses suggested that it was in a hutch (presumably a guinea pig or rabbit’s) helping to keep the floor dry. (224) One third of the GPs had skim-read the guideline, and just over 10% had read it in detail. 20% of the GPs said they would read the guideline at a later date. A small number of GPs (6%) had either not read it or had no intention of reading it. (224) GPs were asked if they thought the guideline would change their practice: 10% did not know, 20% said yes and 37% said no. Some of the GPs who said that it would not change their practice commented that they were already practising according to guideline recommendations. (224) There is no objective measure of this and studies have indicated that GP recall can overestimate how well they are managing conditions. The guideline was sent out within a month of publication which makes the assertion that the practice was already in accordance with recommendations slightly unconvincing. (More information relating to passive dissemination is discussed in the section (3.4) relating to the uptake of therapies without and implementation strategy.)

**SUMMARY:** Mailed or unsolicited printed educational materials will need to compete with all the other post that a doctor receives. It takes self-discipline to read and reflect on the information, compare it with current practice and then modify this practice. The
effect of passive dissemination on change in practice is not uniform. There is some potential for improvement but gains may not necessarily be sustained in the long term.

2.4.7 Small group

Continuing education meetings and workshops are common types of educational interventions in which health professionals participate. Their aim is to improve professional practice and patient care. (40) A format that a small-group session could take is that of a pre-selected topic with an invited specialist who provides an overview of the topic. (225) Another small-group format is peer-led, where the educational intervention is run by GPs and the knowledge gaps to be targeted are identified by the GPs in the group. (39) A discussion may follow lasting one to two hours during which cases may be considered, and salient journal articles may be provided for further reading. (225) The session may be moderated by one of the GPs. (225) The objective is to create a relaxed, enjoyable session where discussion is free-flowing and highly interactive and all participate. (212, 225) The sharing of experiences is regarded as valuable. (212, 226) The interactive nature of the interventions can effect change as information flows in two directions in contrast to a didactic lecture where there is one way flow only. (189)

**EFFECTIVENESS:** GPs who were already receiving prescribing audit and feedback, academic detailing and educational bulletins were randomised to peer-led small group education or a control group. (39) Eight key messages were studied. The prescribing moved in the expected direction for six messages. For five of them the change was statistically significant, and it was the greatest for short-term prescriptions. (39) Assessment of the effect of the decay of the effect of the message revealed a need to reinforce or repeat messages at one to two year intervals, although messages that related to dose had the shortest duration of effect (six months). (39)

A comparison of 32 educational meetings compared with no intervention found that 24 studies reported significant improvement in professional practice in the intervention groups. (40) And improvement in the intervention groups was also seen in three of the eight studies that reported patient outcomes. (40) The results of this Cochrane review were a significant influence on the selection of small group learning as a comparative implementation strategy for this thesis.
Eight studies of interactive workshops, the majority of which included community-based health professionals, were compared with a no intervention control group, a lecture format or large group sessions. In seven studies, the practice outcomes of the interactive workshop groups showed statistically significant improvement, and in six studies the effect scores were moderate or moderately large. (40)

A RCT that compared problem-based learning (PBL) for dissemination of evidence-based guidelines against a didactic seminar found that the GPs who participated in the PBL rated the educational value of their session more highly than the lecture participants (statistically significant difference). (187) Both groups demonstrated significant increases in topic knowledge post-intervention, however the attrition rate of this knowledge after three months was higher in the didactic group. (187)

The way in which GPs use small group sessions tends to reflect their behaviour in seeking information from peers. In the opinion leader section, a study was described in most GPs would approach accessible peers for information, fewer GPs would approach peers they knew to be experts, and even fewer GPs undertook their own literature search. Of the doctors who asked accessible colleagues, half used small study groups for both continuing education and peer support and a similar proportion of doctors who asked expert colleagues also belonged to study groups. None of the doctors who preferred literature searches relied on small study groups for clinical information. (119)

**MODIFIERS:** Group size may not have an effect on outcomes. One review compared outcomes from a study of individualised training sessions, three studies that utilised small group sizes (<10 participants), six studies that assessed medium group size (10 to 19 participants) and three interventions that looked at large group size (≥20 participants). (165) No relationship was found between group size and positive outcomes (change in doctor performance or patient outcomes). (165)

Sessions that included discussion or case studies were more likely than didactic lectures to change doctors’ practice or improve patient outcomes. (165) Series of sessions were found to have more impact as the intended change would have been learnt, then put into practice and reinforced in a subsequent session. (165)

**PERCEIVED BENEFITS:** Opinion was divided over the value of peer groups for improving practice, although they did give support and there was trust and confidentiality which made it easier to ask questions. The interaction allowed for clarification, personalisation of information, exploration, feedback and reflection. (134)
Although meetings rate highly in aspects such as being relevant to practice, a break from practice, beneficial both socially and for professional networking, when compared with personal development plans meetings have less of an effect in change in practice. (227)

The small-group format gives GPs a supportive and understanding environment in which to discuss the difficulties in translating new evidence into everyday clinical practice. (228) The format utilises peer discussion, specialist input, self-directed learning and practice-centred interventions. (228) GPs are also able to measure their current practice against their peers and establish practice norms. (225) The presence of a consultant will improve communication between primary and secondary care and discussions between GPs and specialist will clarify which management strategies are feasible to implement in primary care. (225)

**SUMMARY:** Small groups of GPs meeting to discuss cases, or a discussion with a specialist are popular with GPs as they can ask questions pertinent to their practice and receive feedback in a supportive atmosphere. GPs can compare their management with that of other GPs. The improvement in outcomes measured can be moderate.

### 2.4.8 Specialist – GP Interaction

Interactions between specialists and GPs can take two basic forms:

1. a referral letter in which a GP is asking for advice from an expert or
2. a discharge letter regarding a patient’s stay in hospital where an expert is giving advice.

The interaction between specialists and GPs offers a potential opportunity for educational interaction. (229) The reasons for referrals to specialists are to ask for advice on diagnosis and treatment or to ask that the specialist directs the patient’s care. (230) The information contained in a discharge summary should improve communication between hospital and primary care regarding changes in and reasons for prescriptions.

The specialist-GP interaction could be viewed as similar to that of an opinion leader; but opinion leaders are selected as such, whereas replies to a referral letter or a discharge summary may come from a specialist who is not identified as an opinion leader by the GP.
Effective communication is needed since letters (formal, structured) transmit more information than informal (e.g., phone call) means of communication. Well-structured reply letters could offer timely educational interventions that are directly related to GPs’ clinical work and improving patient outcomes. (230, 231)

Some medical societies and colleges go as far as to direct the specialist to place a high value on teaching the referring doctor so that care of future patients is improved. (230)

**GP LEARNING PREFERENCES:** A review of studies has suggested that between 25 and 50% of referral replies include educational content. A large proportion of GPs wanted information on follow-up plans, treatment rationale and options, side effects and their management, prognosis, and their responsibility in patient care. Aspects such as diagnosis, treatment options and plans, and follow-up care were rated as important or very important. (230, 232)

To determine the type of information required by GPs and how often it is supplied in discharge summaries 71 GPs (64% response rate) were surveyed. (233) These GPs received discharge letters from a local University hospital. While discharge summaries were routine, the reasons for medication charges were not often detailed. 30% of GPs questioned were happy with the information they received, 12% did not answer, but 58% were unsatisfied with the information provided. (233) When asked about changes in dose, drug choice, new drug or discontinuation, and side effects and allergies, almost 90% of GPs said that for each subject they would like to receive information, but only 14 to 30% of GPs believed they received information on each of these therapeutic areas. (233) The findings in this study were sufficiently convincing that a new discharge form was designed and implemented in some of the local hospital wards. (233)

Similar lack of detail was observed in hospital letters written, mostly from three major teaching hospitals, for patients who had been prescribed drugs that required regular monitoring. (234) Few details or reference to guidelines were observed (see table 2.4.i).

Table 2.4.i. Information recorded in hospital letters to GPs

<table>
<thead>
<tr>
<th>Information</th>
<th>% of letters</th>
</tr>
</thead>
<tbody>
<tr>
<td>No indication that regular monitoring was required</td>
<td>&gt;80%</td>
</tr>
<tr>
<td>No detail of the frequency of monitoring</td>
<td>&gt;90%</td>
</tr>
<tr>
<td>Hospital advice adhered to guidelines</td>
<td>36%</td>
</tr>
<tr>
<td>Mentioned guidelines</td>
<td>2%</td>
</tr>
</tbody>
</table>
This lack of medication detail in the letters (i.e. titration schedules, explanations for not prescribing guideline-specified drugs) may be an outcome of who prescribed the medication and the understanding of the importance of communication may be a function of seniority/experience. (234, 235)

Given more explicit information, GPs could avoid adverse drug reactions and errors in prescribing, could maintain the continuity of medication plans and would not need to repeat the time taken to rationalise them.

Studying educational interaction between specialists and GPs, GPs said that they wanted to learn information that was directly applicable to their clinical work, and that specialists had an important role in increasing GPs’ medical knowledge. (229, 236) GPs were critical of specialists for not using referrals to their full potential as “learning opportunities”, and that to a certain extent it was the specialists’ responsibility to keep them up to date. (229)

However the influences of hospital prescribing are not always positive. GPs may not always have been comfortable with hospital initiated prescribing but have followed the specialist’s example and have found it difficult to change or to refuse to continue with a medication as this may damage their relationship with the specialist and their patient. But at the same time, the influence over prescribing was affirming and a learning experience for their own practice. Some GPs challenged the hospital’s prescription. (237)

SPECIALISTS’ VIEWS: Specialists regarded lectures as the principal way of transferring information and few used their response to referrals as a chance to inform the referring GP as they thought this approach might be perceived as condescending or critical and because of the time and work required to formulate detailed feedback. (229) However, GPs are receptive to the use of referral replies as learning opportunities, and provided with appropriate follow-up plans, GPs may be more willing to assume care for patients being considered for discharge. (230) (236)

GPs and specialists disagreed on the preferred methods of educational interaction. Specialists preferred traditional didactic lectures in providing information to GPs, but this was the least popular method for GPs. Specialists did not consider guidelines an effective way to transfer information to GPs. GPs enjoyed interactive sessions based on clinical cases and the few specialists who had participated in these. Most GPs preferred informal and unplanned learning based on referrals. But this was the least popular
option for specialists. This research suggests a need for understanding how GPs and specialists can work together to improve educational interactions. (229)

**SUMMARY**: If secondary care has an influence on patient management in primary care, it is important that specialists provide information that is consistent with guideline recommendations and are seen to support the guidelines that GPs use. (24) There seems to be a divide between what specialists’ think GPs educational needs are and what GPs want from specialists. The information that specialists communicate to GPs needs to be evidence-based and promote optimal care.

### 2.4.9 Summary

Many approaches could be used in implementing evidence however some are more effective than others. Educational interventions that seem to have little or no effect include didactic lectures which have been used extensively as educational events. Audit and feedback, and local opinion leaders can have variable effects on changing practice. Educational outreach and interactive meetings show quite consistent positive effects on changing behaviour. (109) The observed effectiveness of passive dissemination has altered over time from ineffective (109) to at least as effective as other forms of implementation (182) The type of information that needs to be implemented and any barriers that need to be overcome may determine which implementation strategy should be used. (238)

The systematic reviews that have been discussed above include studies that utilised different health professionals, different educational interventions and different categories of outcomes. Some used different methodologies with varying degrees of bias, and some used incorrect units of analysis. The mixed results of these studies make them difficult to generalise. There is also the conundrum of identifying precisely which barriers to transforming evidence into practice exist and how to align them with a particular implementation strategy and particular theory to underpin the strategy. These shortcomings are summed up as such: (42, 239, 240) A drug RCT and an educational intervention RCT are contrasted: a RCT testing a drug will have comparable outcomes if the research is replicated in a new group of similar patients whereas outcomes for an educational intervention are likely to differ each time it is replicated. (42, 239, 240) In the latter situation there will be different interactions between the teacher and the students, the setting will be different, the curriculum will be different, and the leadership
will be different. (42, 239, 240) The context-specific factors of education will make extrapolation from RCTs on educational intervention challenging, and suggests that more closely controlled studies (in which intrinsic factors may be controlled for) could be more useful in determining crucial factors that effect change. (42, 118, 156, 239)

It may also be that some methods of implementing evidence work better among some health professionals than others, and at different stages of dissemination and implementation or even at the different stages of readiness to change. (239, 241) Educational meeting such as seminars and interactive CME events can be useful when there are barriers to knowledge. (238) If GPs are unaware that their practice is sub-optimal then audit and feedback could be used. (238) If existing culture, routines and practices are impeding change then social influence approaches such as educational outreach and opinion leaders could help. (238)

No single implementation strategy clearly improves all aspects of patient care. The answer may be to provide multiple types of intervention but GPs and their primary care teams need to acknowledge what may be preventing them from implementing evidence.

### 2.5 Conclusions

While GPs appreciate the value of guidelines and EBM, these may not be the first sources they turn to when seeking prescribing information. The pharmaceutical industry has been quoted as a quick and readily available source of information that many GPs utilise. Studies vary in the reported reliance that GPs have on their colleagues and on specialists for prescribing information. However one important factor in the prescription of a new drug is the early experience in a few patients. GPs’ personal characteristics also play a part in their prescribing habits as does the type of practice they work in, which may be a result of their personal characteristics. Added to this mix of factors influencing prescribing is the credibility of the source of information and the format in which it is delivered.

Drug companies may release new drug information quickly whereas the integration of this information into existing management protocols (either a new drug or a new indication for an old drug) in the form of a guideline may take several months or years. The time taken to develop the guideline ensures that evidence has been critically
appraised and graded for quality. GPs may not have these appraisal skills and have difficulty determining the accuracy of information presented by drug companies.

The lack of appraisal skills may also affect how GPs measure where the gaps are in their own practice. The scope of practice and the amount of new evidence that is published intensifies the difficulties in keeping up to date.

Many implementation strategies have been trialled to determine the best ways to translate research evidence into practice. The most recent of the implementation strategies is the Internet. It has progressively become more accessible and more acceptable as an educational tool. Its effectiveness in delivering sustained changes in practice needs to be compared against other implementation strategies. For example, the literature has suggested that small group learning is a well-known, well-liked and promising method of delivering education to GPs.

The results of studies of individual strategies have been disparate, making it difficult to draw these results together. There are differences in the delivery of the education, quality of the studies, the outcomes measured and the settings. The result is a mosaic of findings with no unifying theory guiding how to ensure the research is implemented. Instead of hard facts of what works and what does not, there are theories of what might work and these are highly context driven. While testing of these strategies must be robust, the studies are not biological models but population health models and the outcomes hinge on diverse factors such as psychology, economic drivers, and numerous other influences some of which the GP will be unconscious of.

While a combination of strategies – although which ones is not entirely clear – may result in greater improvements in outcomes, the knowledge that is gained during an educational event will decay over time. This means that there is an onus on those responsible for delivering the CME to keep the educational content current and to reiterate the information to keep clinical management at an optimal level.
3. Heart Failure

GPs are frequently the first to diagnose HF and subsequently treat it. However, HF is a complex syndrome to diagnose and thus is commonly overdiagnosed, misdiagnosed and underdiagnosed. The suggested overdiagnosis rate in primary care may be as high as 70% of suspected HF patients. (242-244) The underdiagnosis of patients who have major LVSD without symptoms is possibly equal in number to those with LVSD and signs of HF. (245) The leading concern is patients with suspected but unconfirmed HF. They have been estimated to number between 2 million and 3 million in the UK in 2000, which equates to 135000 to 200000 patients currently in NZ. (245, 246) These prevalences suggest that one barrier to managing HF is the sheer size of the problem. (245) The problems with diagnosis and treatment could be reduced considerably if objective testing were used more frequently.

The increasing number of HF patients can be explained on several fronts. As a condition most commonly observed in the elderly, HF is increasing in aging societies and where average life expectancy is rising. Younger persons who experience MI, and may have died in past decades, are now increasingly surviving, but with damaged myocardium, and an increased risk of developing HF. More patients have precursors for HF such as hypertension and CHD. The improved methods of diagnosis (echocardiography and BNP tests) can now identify more patients than previously. (245, 247)

HF cannot be defined by any one parameter, unlike conditions such as DM which is determined by blood glucose levels, or hypertension by blood pressure readings. (248) Signs and symptoms of HF tend to be non-specific, and subject to observer bias. (29, 248) The lack of accuracy related to diagnosis on clinical grounds alone will be demonstrated in section 3.2 X on echocardiography. This procedure is the current gold standard diagnostic test, but its utility is complicated by the lack of a standard value for EF.

This chapter examines the processes used to determine whether a patient has HF and describe the optimal management for HF and how GPs either adhere to or deviate from these processes.
Chapter 3 first defines HF before investigating the likelihood of HF in a patient presenting to a GP (section 2). Section 2 further discusses the usefulness of the diagnostic tests recommended by the NZ HF guideline (for patients with suspected HF), including echocardiography because this test was an outcome measure for this thesis. The role of the specialist and secondary care is acknowledged. Some GPs prefer specialists to manage certain types of patients (for example complex patients whose diagnosis is uncertain, or patients whose HF needs stabilising after an acute admission to hospital). Differences in approach between GPs and specialists are also referred to.

Section 3 details the trial evidence for the three outcomes tested: ACEi (and Angiotensin II Receptor Blockers), β-blockers for use in HF, and spironolactone. It then evaluates how patients are treated in primary and secondary care, emphasising difficulties that are real, imagined or due to misconceptions in managing HF in primary care.

**SEARCH STRATEGIES:** Medline and EMBASE were used for search strategies that included the terms or contractions of the terms: “General Practice”, “Primary Care”, “Heart Failure”, “Echocardiography”, “Diagnosis”, “Electrocardiography”, “X-rays” “Guideline” “ACE inhibitor”, “Spironolactone”, “β-blocker”.

The Heart Review Group Cochrane library was also searched for relevant implementation strategies.

English language articles only were used as it was not feasible to pay the additional costs associated with translation. Study populations were adults only. Searches were not limited by study type.

Additional papers were suggested by the study team as well as by other members of the Department of General Practice and Primary Care, University of Auckland. Other articles were identified during retrieval of articles identified in searches, or from reference lists. Some hand searching was carried out in the British Journal of General Practice, European Heart Journal, European Journal of Heart Failure, Journal of Continuing Education in the Health Professions, the New Zealand Medical Journal, and Family Physician (NZ journal, not indexed).
3.1 Defining heart failure

Problems in identifying HF may be partly explained by inconsistencies in defining HF. The NZ HF guideline does not define HF. (29) (Appendix 3A reports definitions given by other guidelines.) The European Society of Cardiology (ESC) short definition is:

“Criteria 1 and 2 should be fulfilled in all cases.
1. Symptoms of heart failure (at rest or during exercise), and
2. Objective evidence of cardiac dysfunction (at rest), and *(in cases where the diagnosis is in doubt)*
3. Response to treatment directed towards heart failure.” (76, 249)

This definition encapsulates the problems associated with defining HF and brings together the three critical aspects needed to make a diagnosis. However, studies use different criteria to determine HF (see Appendices 3B and 3C) in the absence of standardised criteria. Study populations may therefore be dissimilar, complicating comparisons between studies.

A review study (250) examined diagnostic heterogeneity in HF clinical trials published between 1977 and 1985. The review was to ascertain: whether diagnostic criteria were applied and reported; whether there was uniformity in these criteria among trials; and whether lack of uniformity produced conflicting results. Diagnostic criteria were defined as any group of specified symptoms, physical findings, and objective test results, but isolated references to NYHA, medications used or physician diagnosis were not accepted. (250) Of the 51 trials reviewed, 23 specified criteria for diagnosing HF with four using identical criteria (cardiomegaly, S3, pulmonary vascular congestion on CXR). The same research team conducted these trials. Of the remaining 28 studies, no two used identical criteria, despite five having the same first author. Several trials specified criteria within the same category, but precise criteria varied. 16 studies used CXR but again the criteria varied; with pulmonary vascular congestion (n=1), cardiomegaly (n=2), either (n=3), both (n=7) or CTR (>0.50 or >0.55) (n=3). 11 studies used LVEF as a criterion but values ranged from <30% to <49%. (250) None of the studies used standardised criteria (e.g. Framingham, Boston – see Appendix 4Q for further clarification of HF scoring systems). (250)

As described further in section 3.2.8, a survey of 2700 physicians in different fields revealed diverse opinions regarding diagnosis and management. (251) It may be inappropriate to use the one term to cover all aetiologies and pathophysiologic
presentations. If all studies specify diagnostic criteria then clinicians can easily
determine which clinical trials apply to their patients. (250)

3.2 Determining who has heart failure

Since HF is complex to diagnose, it is prudent to consider who is most likely to develop
it, what the risk factors are, what signs and symptoms are associated with HF and which
ones are also related to other conditions. The overlap of signs and symptoms between
conditions (see 3.2.3) and the deterioration of physical condition with ageing (examples
given in 3.2.9) make the clinical diagnosis of HF very inexact (see section 3.2.6.3). It is
therefore necessary to refer for objective testing any suspected cases of HF.

A distinction needs to be made between the methods used in primary and secondary
care to determine HF since the HF patient populations differ between each setting. For
example the patients seen in general practice have been shown to be older [79.2 years
vs. 64.2 years, p<0.001 (252), 77 years vs. 71 years p<0.01 (253)]; cardiology patients
are more likely to be male [78% vs. 42%, p<0.001, (254), 55% vs. 38% p<0.01, (253)]
and to have a history of IHDa [56% vs. 31%, p<0.001, (252)]. Moreover cardiologists
and GPs can value signs and symptoms and comorbidities differently in diagnosis, and
some signs may more easily be established by cardiologists. (252, 255) Given that the
HF populations that GPs and cardiologists diagnose are different, it is important for this
project to concentrate on studies that have been carried out using primary care patients.

3.2.1 Prevalence and Incidence

An important step in identifying HF is to know how often this condition occurs in a
population. As can be seen from the studies referenced below, these figures are not
always consistent, which can be due to the patient population sampled and the sampling
method (how HF was defined). The figures used below for prevalence and incidence of
HF were all generated by studies undertaken in primary care. Approximately 80% of
all the HF cases occur in people aged ≥65 years. (256) Overall prevalence has been
estimated at 3 to 20/1000 population, or >100/1000 population ≥65 years, and annual
incidence 1 to 5/1000, with relative incidence doubling for each decade after 45 years.
(257)

a Including prior MI, angina, CABG, PTCA.
3.2.1.1 Prevalence

A frequently quoted study is the Framingham, initiated in 1949, which had a patient follow up of 34 years. The prevalence is quoted in 10-year age-bands. See table 3.2.a derived from the Framingham reference. (258)

Table 3.2.a. Prevalences of HF by age in the Framingham study

<table>
<thead>
<tr>
<th>Age Bands (yrs)</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 – 59</td>
<td>0.8%</td>
</tr>
<tr>
<td>60 – 69</td>
<td>2.3%</td>
</tr>
<tr>
<td>70 – 79</td>
<td>4.9%</td>
</tr>
<tr>
<td>80 – 89</td>
<td>9.1%</td>
</tr>
</tbody>
</table>

The diagnosis of ‘overt’ HF was based on major and minor criteria (described in Appendix 4Q), and did not include patients with impaired sub-clinical function which would now be detected by non-invasive methods. (258) However the broad criteria may include patients with signs and symptoms who do not have HF.

The MONICA (Glasgow coronary risk factor survey)\(^b\) (259) calculated that the overall prevalence of definite LVSD (EF ≤ 30%) was 2.9%, with symptomatic prevalence at 1.5% and asymptomatic prevalence at 1.4%. The percentages increased with age, and were higher in men (4.0%) than in women (2.0%), p<0.001. (259)

Table 3.2.b. HF prevalence (determined by echocardiography LVEF ≤ 30%) by age-bands for symptomatic patients only

<table>
<thead>
<tr>
<th>Symptomatic HF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Age bands (yrs)</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>45 – 54</td>
</tr>
<tr>
<td>55 – 64</td>
</tr>
<tr>
<td>65 – 74</td>
</tr>
</tbody>
</table>

\(^b\) The MONICA study included participants aged 25 to 74 years who were invited to participate in an investigation of symptomatic and asymptomatic LVSD. Patients (n=1640) had LVSFx assessed by echo and LVSD was defined as EF ≤ 30%. Possible LVSD was defined as EF ≤ 35%. (259)
“Possible” LVSD, defined as EF ≤35%, was evident in 7.7% (n = 113) of participants although 77% (n = 87) of these patients were asymptomatic. Possible LVSD increased with age and was higher in men than in women. (259) However even an EF of 35% is low as a diagnostic value, which may therefore underestimate the true prevalence. It should be noted that the maximum age of the MONICA cohort was 74 years. This is a low age for HF, with a majority of HF patients usually being diagnosed around this age.

The CASE study (Cardiac Awareness Survey and Evaluation) (260) assessed 80 consecutive patients ≥60 years presenting to their GP (for 341 GPs). The prevalence of HF (total HF/total population assessed = 2905/22060) was 13.2%; previously diagnosed cases of HF (n=2485) were 11.3%; and newly diagnosed cases of HF (n=420) were 1.9%. This high prevalence may be due to the diagnostic method and greater use of objective testing, different populations or a widespread sample population. (260)

In 1988 a survey to establish the prevalence of HF used three general practices in north-west London, with a total of 30204 patients. (261) The mean age of patients who fulfilled the HF criteria was 73.7 years ± 12.2 years (range 5 to 99 years). The prevalence values are in table 3.2.c, derived from table 1, north-west London study. (261)

Table 3.2.c. Prevalence of HF (expressed as per 1000 patients) in north-west London

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>Practice A</th>
<th>Practice B</th>
<th>Practice C</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;65</td>
<td>0.7</td>
<td>0.8</td>
<td>0.4</td>
<td>0.6</td>
</tr>
<tr>
<td>≥65</td>
<td>33.4</td>
<td>22.7</td>
<td>26.2</td>
<td>27.7</td>
</tr>
<tr>
<td>All ages</td>
<td>5.0</td>
<td>3.7</td>
<td>2.9</td>
<td>3.9</td>
</tr>
</tbody>
</table>

However only about 30% of patients had objective evaluation of LVFx and the wide age range of the patients should be noted. (261) This level of objective evaluation of HF was similar (i.e. 30% of patients had echocardiograms) to that reported in another study that found contrasting figures: a prevalence of 15/1000 for all patients, and a prevalence

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*CASE study used Australian data from 1998. (260) No equivalent published NZ data were available.

* Potential patients in the north-west London study were identified by prescription analysis – previously shown to be a useful method in establishing the prevalence of angina. (262) It was presumed that all patients with HF were being prescribed a diuretic. The authors’ experience was that if HF is recognized, a diuretic is used as first-line therapy and that ACE inhibitors and digitalis are not used without concomitant diuretics. Practice records, including letters and discharge summaries, of all patients receiving diuretics were examined and as it is difficult to make clinical judgements by reading case notes and letters retrospectively, and in this study, the researchers erred in favour of a positive diagnosis if there was real doubt. (261)
of 80/1000 for patients aged ≥65 years. (263) The figures generated from these two studies should be viewed with caution given the lack of objective evidence for a large proportion of patients.

In the Rotterdam Study (264), the overall prevalence for HF in patients ≥55 years who had a ‘complete’ dataset (signs and symptoms, evidence of cardiac disease, and cardiovascular medication) was 3.9% (95% CI 3.0 to 4.7%), (using FS ≤ 25% ≡ LVEF = 42.5%). (264, 265) Table 3.2.d (264) shows that the prevalence of HF rose rapidly in the ≥75 year group but decreased for male patients at ages 85 to 94. (264)

Overall, variations in prevalence between studies may be due to the definition of HF, the sampling method or actual differences in practices in the studies. (259)

Table 3.2.d. Prevalence of HF by age band for all patients, males and females

<table>
<thead>
<tr>
<th>Age bands (yrs)</th>
<th>Total</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>55 – 64</td>
<td>0.7 (0.2 – 1.4)</td>
<td>0.7 (0.1 – 2.1)</td>
<td>0.6 (0.1 – 1.8)</td>
</tr>
<tr>
<td>65 – 74</td>
<td>2.7 (1.5 – 4.4)</td>
<td>3.7 (1.8 – 6.6)</td>
<td>1.6 (0.4 – 3.9)</td>
</tr>
<tr>
<td>75 – 84</td>
<td>13.0 (8.5 – 18.7)</td>
<td>14.4 (7.2 – 25.1)</td>
<td>12.1 (6.7 – 19.4)</td>
</tr>
<tr>
<td>85 – 94</td>
<td>11.7 (4.8 – 22.6)</td>
<td>5.9 (0.1 – 28.7)</td>
<td>14.0 (5.3 – 27.9)</td>
</tr>
<tr>
<td>Overall</td>
<td>3.9 (3.0 – 4.7)</td>
<td>3.7 (2.5 – 4.9)</td>
<td>4.0 (2.9 – 5.1)</td>
</tr>
</tbody>
</table>

### 3.2.1.2 Incidence

In a UK study looking at the incidence of newly diagnosed HF (dyspnoea and at least one other finding) in patients aged 40 to 84 years, the annual incidence of HF was 4.2/1000 person-years; 4.4 in men, and 3.9 in women. (266) The annual incidence observed in the Framingham study was 0.2% for patients aged 45 to 54 years, and 4.0% for patients aged 85 to 94 years. (258) Another study looked at the incidence of HF in the UK (267) assessing patients’ likelihood of HF on the basis of their medical records. For definite HF\(^{e}\) the rate was 9.3/1000 persons/year and for possible HF\(^{f}\) it was 20.2/1000 persons/year. The authors then estimated the incidences reported in table 3.2.e (267).

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\(^{e}\) Definite HF was described as a written diagnosis and clinic description in the patient’s medical record.

\(^{f}\) Possible HF was described as medical records where there was a description of signs and symptoms suggestive of HF or related cardiac abnormalities.
Table 3.2.e. Figures for definite HF and figures for possible HF

<table>
<thead>
<tr>
<th>Diagnostic likelihood</th>
<th>Age 55 – 64</th>
<th>75 – 84</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite HF</td>
<td>3.4 / 1000 persons / yr</td>
<td>25.2 / 1000 persons / yr</td>
</tr>
<tr>
<td>Possible HF</td>
<td>Approx 15 / 1000 persons / yr</td>
<td>Approx 38 1000 persons / yr</td>
</tr>
</tbody>
</table>

The Hillingdon study identified all new suspected HF cases referred to a rapid access clinic or, if acutely ill, to the local hospital Accident and Emergency Department using the ESC criteria for diagnosis (99% of patients had an ECG, 98% a CXR and 91% an echocardiogram). (268) The median age at presentation was 76 years (range 25 to 95 years), and the male:female ratio was approximately 1:1, although 64% of female presentations were aged ≥75 years compared with 47% of males. (268) A nationwide survey of computerised medical records in Scotland assessed the incidence of HF. It is not clear how HF was diagnosed other than through the coding in the patients’ medical records indicating HF. (269) Table 3.2.f compares the annual incidences reported by these two studies. It is unclear how well investigated some of these cases were, e.g. objective evidence of LVD with symptoms compared with just symptoms or a coded diagnosis. Nor was it clear which age groups were included in the calculations of overall incidence for a population; which could account for the differences between studies.

Table 3.2.f. Comparison of annual HF incidence rates for two studies

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>Hillingdon (268)</th>
<th>Scotland (269)</th>
</tr>
</thead>
<tbody>
<tr>
<td>45 – 54</td>
<td>0.2 / 1000</td>
<td>1.3 / 1000</td>
</tr>
<tr>
<td>55 – 64</td>
<td>1.2 / 1000</td>
<td></td>
</tr>
<tr>
<td>65 – 74</td>
<td>3.0 / 1000</td>
<td>6.1 / 1000</td>
</tr>
<tr>
<td>75 – 84</td>
<td>7.4 / 1000</td>
<td>16.0 / 1000</td>
</tr>
<tr>
<td>≥85 years</td>
<td>11.6 / 1000</td>
<td>22.4 / 1000</td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td><strong>1.3 / 1000</strong></td>
<td><strong>2.0 / 1000</strong></td>
</tr>
</tbody>
</table>

3.2.1.3 Knowledge of the extent of heart failure

GPs participating in the Euro-HF study (35) were asked the approximate prevalence of HF. The prevalence of 2/100 was selected by 54%. Almost 30% selected 2/1000, and 12% did not know. Most GPs selected the correct prevalence but a large proportion was incorrect. If GPs do not expect to encounter HF at the rate it occurs, this could explain
why they do not detect it. Also, only 42% of practices kept a disease register of HF patients. (35) The lack of a disease register and the lack of knowledge of the frequency of HF may contribute to the under-investigation and undertreatment of HF patients.

3.2.2 Patients at Risk of Heart Failure
Assessing all patients for potential HF would be time consuming and costly. Even though the likelihood of HF increases with increasing age, it makes sense to focus on patients with risk factors for developing HF. This task can be facilitated by a current problem list in the patient’s medical record, which can effortlessly be manipulated when the records are in electronic form.

The NZ HF Guidelines (29) mention history of HTN, previous MI, and cardiac murmur or other disease as key aspects of a patient’s history in the diagnosis of HF if these occur in conjunction with signs and symptoms. The following studies indicate the risk of developing HF associated with these conditions. All the studies based their calculations on primary care or community-dwelling patients. A comparison of risks calculated in the following studies is presented in table 3.2.g.

From the Framingham study, nearly 80% of patients had HTN or were treated with antihypertensives. Almost 50% of males and almost 30% of females had a background of CHD. 2.3% of patients had Rheumatic fever. Risk ratios for several risk factors were calculated (age-adjusted) for the incidence of HF. (258)

The MONICA study also calculated risk factors for LVSD patients (symptomatic and asymptomatic). Symptomatic LVSD patients had higher rates of IHD, MI, angina, ECG ischaemia, abnormal ECG, HTN and valvular abnormalities than asymptomatic LVSD patients. (259) Odds ratios (ORs) were calculated for the risk factors for both symptomatic and asymptomatic patients. (259)

A UK general practice case-control study of newly diagnosed HF patients aged 40 to 84 years was used to estimate the relative risk (RR) (as stated by the study) of various factors for HF. (266) Similarly, RRs were calculated for patients who had been referred to an open-access HF clinic. (207) The reason behind the calculations was to try to determine how to ‘streamline’ referrals to the service. Moreover, risk factors were calculated for the Connecticut cohort of the Established Population for Epidemiologic Studies of the Elderly programme. (270) This study of community-based elderly
evaluated 1,749 subjects aged ≥65 years who were free of HF, MI, and angina at baseline. (270) Follow up was over 10 years and amassed 13,811 person-years. (270)

The Rotterdam Study (previous section) assessed patients for impaired LVSFx (including asymptomatic) and found that these patients were more likely to be older, male, and have experienced an MI, angina or CABG or PTCA, or be taking cardiovascular or pulmonary medication (data not in table 3.2.g). (264)

Another approach to identifying possible patients would be to add probability values to risk factors which would simplify the decision to refer a patient for objective testing. A stepwise logistic regression analysis from the data generated by the study of open-access HF clinic identified four statistically significant factors associated with LVSD. (207)

- Abnormal ECG
- Cardiomegaly (CTR > 0.5)
- Male gender
- History of DM

The model predicted the following probabilities for the presence of LVSD, or P(LVSD):

- No ‘risk factors’ (of the 4 specified): P(LVSD) = 0.08
- 1 risk factor: P(LVSD) = 0.14 to 0.24 (dependent on the risk factor)
- 2 risk factors: P(LVSD) = 0.29 to 0.46
- 3 risk factors: P(LVSD) = 0.53 to 0.69
- 4 risk factors: P(LVSD) = 0.80

These studies repeatedly observed clear risk factors for the potential development of HF. Some factors such as being older and being male are relatively easy to determine but others need to be carefully and clearly noted in patients’ medical records. In the future it may be possible to use electronic medical record data and predictive modelling to diagnose HF as early as six months before the clinical diagnosis is made. (271)
Table 3.2.g. Comparison across studies of risk factors for developing HF

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Framingham (258)</th>
<th>MONICA (259)</th>
<th>UK general practice (266)</th>
<th>Open-access Echocardiography (207)</th>
<th>Community-based Connecticut cohort (270)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Study and RR or OR (95% CI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HTN</td>
<td>SBP: 2.3 DBP: 1.5</td>
<td>SBP: 3.0 DBP: 1.6</td>
<td>2.4 (1.1 – 5.1)</td>
<td>1.7 (1.4 – 2.0)</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.2 (p&lt;0.001)</td>
<td>1.7 (p&lt;0.001)</td>
<td>1.8 (1.4 – 2.3)</td>
<td>1.7</td>
<td>2.9 (2.0 – 4.3)</td>
</tr>
<tr>
<td>Relative Weight</td>
<td>NS</td>
<td>1.7 (p&lt;0.05)</td>
<td>2.1 (1.5 – 2.8)</td>
<td>1.3</td>
<td>BMI ≥28 kg/m²: 1.6 (1.0 to 2.4)</td>
</tr>
<tr>
<td>Angina/CHD/IHD</td>
<td>5.7 (2.8 – 11.4)</td>
<td>3.2 (2.7 – 3.7)</td>
<td>1.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal ECG^d</td>
<td>5.4 (2.6 – 11.1) (ischaemia or infarction)</td>
<td>Abnormal ECG: 2.5 AF: 1.9</td>
<td>MI: 21 (15 to 31)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>2.2 (1.1 – 4.5)</td>
<td>Male &lt;65 yrs: 2.1 (1.7 – 2.6)</td>
<td>Male &gt;65 yrs: 1.3 (1.2 – 2.4)</td>
<td>1.8</td>
<td>Male: 1.7 (1.3 – 2.4),</td>
</tr>
</tbody>
</table>

^a BMI ≥ 30.
^b Compared with <24 kg/m²
^c The low risk attributed to IHD may be due to good management post-MI.
^d The ECGs were performed and read by hospital staff. The RR may not be applicable to general practice as GPs may not be as experienced in interpretation of ECG printouts.
Table 3.2.g continued. Comparison across studies of risk factors for developing HF

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Framingham (258)</th>
<th>MONICA (259)</th>
<th>UK general practice (266)</th>
<th>Open-access Echocardiography (207)</th>
<th>Community-based Connecticut cohort (270)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Age  &gt;60yrs: 1.6</td>
<td>75 to 84 yrs: 1.9 (1.3 – 2.7)</td>
<td>≥85 yrs: 3.0 (1.7 – 5.5)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smokers Ex-smokers</td>
<td></td>
<td></td>
<td>2.0 (1.6 – 2.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.5 (1.2 – 2.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyspnoea &gt;6 months before diagnosis</td>
<td></td>
<td></td>
<td>1.8 (1.4 – 2.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valvular disease</td>
<td></td>
<td></td>
<td>2.3 (1.5 – 3.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td></td>
<td></td>
<td>2.2 (1.7 – 2.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CTR&gt;0.5 vs. &lt;0.5</td>
<td></td>
<td></td>
<td>2.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Compared with ≤74 yrs
SUMMARY: There is a need to identify potential causes of HF early and GPs should employ an “aggressive” approach to case-finding and management of identified risk-factors. There is a necessity to use a structured approach to identifying patients, e.g. stratified by risk. Action needs to be taken over primary and secondary prevention, over HTN, lipids and post-MI follow-up. Adopting these methods may delay the onset of HF and its subsequent severity. (183)

3.2.3 Signs and Symptoms
Chronic HF is a clinical syndrome consisting of symptoms and signs that may have potentially different causes and treatments. (248, 272) A wide range of signs and symptoms may accompany the development of HF, although not all of them are solely caused by the mechanisms of a failing heart. Different emphasis is given to the same symptoms by diverse researchers – as can be seen in appendices 3A, 3B, 4Q and in 3.1 – making it difficult to know which to rely on in the process of diagnosis. What is known is that signs and symptoms alone are too often used in primary care to presume that HF is present, and the use of signs and symptoms alone is not an accurate method of diagnosis.

The NZ HF guideline notes that not all of the symptoms commonly associated with the diagnosis of HF are due to HF, not all signs of HF are found in all patients and signs, and symptoms may not be highly sensitive or specific. (29) The recommendation is that patients who present with PND, orthopnoea or new onset of SOBOE should be evaluated for HF unless other causes can be found for these symptoms. (29)

Dyspnoea and fatigue were the two most frequent symptoms at the time of first diagnosis in 813 patients (87%), with pulmonary oedema present in 341 patients (36%) (266). When asked to list in order the three signs and symptoms that they believed were most suggestive of HF, these symptoms (see table 3.2.h) were most frequently quoted by GPs in the Euro-HF study. (35)
Table 3.2.h. Signs and symptoms thought by GPs to be most suggestive of HF

<table>
<thead>
<tr>
<th>Signs and symptoms of HF</th>
<th>GPs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oedema</td>
<td>75%</td>
</tr>
<tr>
<td>Breathlessness / dyspnoea</td>
<td>58%</td>
</tr>
<tr>
<td>Crepitations</td>
<td>28%</td>
</tr>
<tr>
<td>Tiredness / lethargy</td>
<td>20%</td>
</tr>
<tr>
<td>Shortness of breath on exertion</td>
<td>19%</td>
</tr>
</tbody>
</table>

But primary care studies suggest that breathlessness, thought to be HF, is frequently misdiagnosed. (273, 274) So, dyspnoea, used as a diagnostic tool for HF, can be misleading as a symptom as also seen from the following examples of initial GP diagnosis. Lung disease such as COPD frequently accompanies HF and its symptoms can easily mask HF. (275, 276) A NZ study of N-BNP and accuracy of diagnosis in primary care (277), found that after an initial visit to a GP by patients with symptoms of HF (dyspnoea ± oedema), 215 patients (70%) were thought to have HF. Once the patients had undergone testing, and a panel (of 3 cardiologists and 1 GP) evaluated ECG, CXR and echocardiography as well as clinical assessment, based on the ESC criteria only 77 patients (25%) were assessed as having HF.

The accuracy of a clinical diagnosis was further questioned in the Rotterdam Study which found that 60% of patients with LVSD had no signs or symptoms of HF (e.g. shortness of breath, ankle oedema, and pulmonary crepitations) and as such were underdiagnosed. (264) The study went on to investigate the relationship between left ventricular function (by echocardiogram) and signs and symptoms. While only a very small number (n=35) were diagnosed by signs and symptoms alone, less than a third had echocardiographic evidence of LVSD.

One survey of 18 practices found that most of them did not have systematic processes for diagnosing patients who presented with HF-like symptoms of breathlessness and ankle oedema, and that even within practices GPs differed in their views of which symptoms were important. (11)

Signs and symptoms are useful in the process of diagnosis but HF is difficult to diagnose clinically and many patients thought to have HF (by their GPs) may not have any “demonstrable abnormality of cardiac function on objective testing.” (278) Patients diagnosed solely on signs and symptoms could receive inappropriate management as
they may not have HF or the aetiology may require more than ‘standard’ HF management.

3.2.4 Electrocardiogram

As a recommended test for patients who have suspected HF (29) the ECG is quick and non-invasive. A patient with a normal ECG is highly unlikely to have HF/LVSD (248), with NPVs ranging from 82% to 98% (279-281) when performed and read by GPs with a special interest in HF, by cardiology registrars or by consultants rather than GPs. This variation is of note because the usefulness of the ECG in primary care may be ‘seriously undermined’ by a ‘lack of skills in interpretation’ (282) since referral for specialist opinion is usually still required; and delays the diagnosis. The ECG can only act as a “rule out test”, even in the best hands, and positive tests require further imaging. (282)

Many GPs use ECG rarely and not all practices own an ECG machine, ownership being less likely if the practice is small. (283) One study estimated that GPs review ECGs 1 to 2 times/week (203), and another study found that 40% of GPs used an ECG >once/month, 41% <once/month and 19% never. (283) Lack of training and experience may result in GPs missing minor changes that a specialist may see. (203) Interpretive ECGs (i.e. by computer) may assist GPs who are unfamiliar with reviewing ECGs but even they differ in accuracy from cardiologists’ (gold standard) interpretations. (284, 285) This makes it difficult to determine whether interpretive ECGs would improve ECG interpretation in primary care especially since doctors are becoming less experienced in interpreting ECGs and they cannot determine whether machine or human readings are more accurate. (285)

This underlines the comment that there is no optimal method of screening for LVSD in general practice. (286) However, some specialists still press for the use of ECG as “invaluable in any general practice screening programme for LVSD” as it is cheap and easy to perform as highlighted by NPV and sensitivity figures. (287) This may be true but the important aspect of using an ECG tracing as a diagnostic tool is the interpretation, and this skill does not exist in general practice to the required standard. ECGs are probably best left to highly experienced GPs or specialists. A recent systematic review which also used individual patient data concluded that in primary care BNP was more accurate than ECG, and that ECG assessment was not required to
determine the presence or absence of HF. (288) There is still a need for evaluation of heart function by echocardiography.

3.2.5 Chest X-Ray
The chest x-ray (CXR) is popular among GPs as a diagnostic tool. It is seen as easily accessible and relatively inexpensive, whereas the wait for an echocardiograph can be long (and if carried out privately and quickly can be costly to the patient). CXR seems more popular with GPs than cardiologists, being used by 73% of GPs and 47% of cardiologists (p<0.01) to establish a diagnosis. (138) Although as an overall diagnostic tool, CXR was used equally by GPs (80%) and cardiologists (82%). (138) Similar findings were seen approximately five years later by a study in which 84% of cardiologists’ HF patients received CXR compared with 51% (p<0.001) of GPs’ HF patients. (252) Similar importance was placed on CXR by cardiologists and GPs, who ranked it as the most important investigation, although cardiologists ranked it first equal with echocardiography. (255)

CXR can exclude pulmonary disease that may cause symptoms similar to HF. Patients without CHF but with COPD, neck-vein distention, or râles, can also meet the clinical criteria for HF set out by Framingham so these criteria may not be able to pin-point the cause of dyspnoea. (250) The attribution of symptoms of breathlessness to the wrong disease was demonstrated in Section 3.2.3. Various lung diseases such as pneumonia, pulmonary emboli, carcinoma or emphysema can be excluded through the use of CXR. (248)

In the NZ HF guideline CXR is one of the recommended tests and the findings of a CXR could consist of the following: cardiomegaly, pulmonary venous congestion, interstitial fluid, pulmonary disease. (29) Cardiomegaly and venous congestion are the most specific radiographic findings, but neither finding alone can adequately exclude or confirm LVD or reveal its cause. (274, 289, 290) There is some debate over whether one of the findings of CXR, the cardio-thoracic ratio (CTR), can predict LVEF. (207, 291-294) Most agree there is a weak negative correlation between CTR and EF, which is insufficient to replace tests such as echocardiography in determining systolic function; and the causes of an increased CTR cannot be determined by CXR alone. (289)
The CXR has its place in determining factors such as enlargement of the heart and various congestion and fluid patterns, but there is still a need for further testing using echocardiography to determine how the heart is functioning and potential aetiologies.

3.2.6 Echocardiography

Echocardiography is used to assess the severity of LV dysfunction by ventricular size and function and to give an indication of the potential cause of the HF. (29) Echocardiography provides a non-invasive, objective evaluation of cardiac structure and function with very little discomfort and without risk and as such is highly acceptable to patients. (278, 289, 290, 295) It should be used once patient history, physical examination and necessary ECG and CXR have been obtained and only in situations where there are adequately trained doctors and ultrasonographers and where there is sufficient patient volume. (295)

The evaluation of ventricular function is the most common recommendation for echocardiography. (295) The NZ HF guideline states that all patients with suspected HF should undergo imaging of the left ventricle (usually by echocardiogram). (29) Under the recommendations for the use of echocardiography in guidelines for the clinical application of echocardiography, the following recommendations are listed as Class I:\textsuperscript{a}: assessment of LV size and function in patients with suspected cardiomyopathy or clinical diagnosis of HF; and dyspnoea with clinical signs of heart disease. (295) These would be the most common referrals from primary care. Echocardiography may also uncover conditions contraindicated to ACEi use. (243)

EF measurement is an indication of the global systolic LV function. (296) EF can be reported quantitatively or qualitatively as increased, normal or mildly, moderately or severely reduced, or it may be quantitated (a quantitative value is preferred which is measured in percent, and if a range is measured then the mid-point is given). (295, 297) LVEF can be indirectly estimated from fractional shortening, or from a 9 segment model for assessing wall motion index score with LVSD indicated by a wall motion index score $<1.5$ or a fractional shortening $<0.26$ which roughly equates to an EF $<45\%$. (298)

\textsuperscript{a} Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective. (295)

\textsuperscript{b} EF calculated as fractional shortening multiplied by 1.7 (approximately). (265)
As early as 1994, echocardiography was recognised as the key investigation for diagnosing HF, such that a case of suspected HF should prompt a doctor to request an echocardiogram rather than a CXR as had been the case in the past. (299) The authors conclude their article on diagnosis and management of HF by saying that:

“The diagnosis and management of heart failure is rapidly changing. Interest is focussing on the early identification and prevention of progression of left ventricular dysfunction (the provision of easy direct access to echocardiography for general practitioners is crucial to this) . . . ” (299)

And this was reiterated in 1996 by the statement that: “Echocardiography is now considered to be the key investigation if HF is suspected, and should lead to improvements in clinical management.” (272) This statement suggests that more than merely confirming a diagnosis, echocardiography will improve patient outcomes. Section 3.3.3.1 discusses the effect of diagnostic certainty on management.

3.2.6.1 Proportion of suspected HF patients receiving echocardiograms

Since echocardiography is the gold standard diagnostic test for HF the question needs to be asked about its usage to diagnose HF in primary care. An echocardiogram will clarify a suspected diagnosis of HF and patients can then be managed appropriately.

Reviewing studies in chronological order (or as close as could be determined from the journal articles) suggests that echocardiography rates have been rising over time since the introduction of the echocardiogram as a more readily available diagnostic tool.

**PRIMARY CARE AND ECHOCARDIOGRAPHY:** Studies (early to mid-1990s) have indicated that 30% of HF patients have undergone echocardiography (261, 263, 268, 274, 300), with another study (late 1990s) suggesting that 34% of suspected HF patients had received echocardiography. (9) Echocardiography was performed in 36% of suspected HF patients in a review (late 1990s) of three group general practices. (8) A review of two general practices found that 42% of HF patients (2001 study) had undergone an echocardiogram. (7) Although in a 2004 to 2006 study only 31% of primary care HF patients were documented as receiving echocardiography. (6)

But these figures may not accurately represent the rates of echocardiography for male and female patients. One Swedish study of community HF patients (late 1990s) found that 31% of HF patients had their diagnosis based on objective echocardiographic
evidence, broken down to 38% of male patients and only 20% of women (p<0.001). (31) This was almost exactly replicated in a Dutch study (2004 to 2006) in which 31% of patients received echocardiography, 39% of male patients and 21% of female patients. (6) Hospital data (from Sweden 1995) found that 59% of HF patients had received echocardiography but again there was a significant difference (p<0.011) between men (68%) and women (55%). (301) A more recent study (EPISERVE) also found a significant difference in echocardiography rates between men and women (35% vs. 28%). (302)

The advent of the National Service Framework (NSF) has required practice-based disease registers for HF to be established. (7) But first a diagnosis needs to be confirmed, for which the NSF standard is confirmation by echocardiography. (7) Despite poor figures for the number of suspected HF patients receiving echocardiography as demonstrated above, the recently implemented NSF and GMS has resulted in considerable changes. Whereas in 2003/2004 HF diagnosis was confirmed infrequently by echocardiography, by 2005/2006, the national rate of confirmed diagnosis was 92.5% (local variations ranged from 69.6% to 100%). (303)

**DIFFERENCES BETWEEN PRIMARY AND SECONDARY CARE:** A survey was carried out in 1995, nine months after the release of the AHCPR guidelines, which recommended under “Diagnosis and Evaluation” that all patients with suspected CHF should have an assessment of left ventricular function. The survey found that in establishing a diagnosis of HF, 15% of GPs used echocardiography compared with 48% of cardiologists (p<0.01), and even when overall echocardiograph use was assessed, cardiologists still used echocardiography more frequently than GPs (83% vs. 43%, p<0.01). (70, 138)

**Importance and application:** The difference between use of echocardiography by GPs and cardiologists was even greater in a later European study which found that 97% of cardiologists’ HF patients had received an echocardiogram but only 12% of GPs’ patients. (252) There were also differing views on the importance of diagnostic tests for HF between the two specialities with cardiologists ranking echocardiography and CXR first equal (100% for each investigation), but GPs ranking CXR (98%, p=0.44) as the most important investigation, with echocardiography ranked 4th (62%, p<0.001). (255)

A case study of typical development of systolic dysfunction with abnormal results from a CXR was presented to GPs and cardiologists. (102) The majority of GPs said that
they would order an echocardiogram (89% vs. cardiologists 91%). However the perception of importance differed (p<0.01) between the two groups with 77% of cardiologists considering it very important while only 62% of GPs did. Their perception of importance of measuring LVEF correlated with the decision to refer for echocardiography and not with familiarity with the guideline recommendation. (102)

**SUMMARY:** While the early studies of echocardiography showed disappointing rates of use, there was a slow rate of increase over time which was accelerated at least in the UK by the introduction of a financial incentive for practices to confirm diagnosis by echocardiogram. However this strategy does not necessarily ensure that GPs know why echocardiography is so important in HF, as demonstrated in the following paragraphs.

### 3.2.6.2 Reasons given for not using echocardiography

While it may be easy to criticise the low levels of echocardiography it is necessary to try to determine the reasons for these low levels and if these reasons can be changed.

An early study (1994) found that while a majority of surveyed GPs graded usefulness of echocardiography as a ‘3’ on a scale of 1 = not useful to 5 = very useful, only 28% had ever referred patients for an echocardiogram. (304) Another study indicated that 80% of GPs agreed that it was important to discover the underlying causes of HF and two-thirds of GPs agreed or strongly agreed that to diagnose HF they should request an echocardiogram if possible. (20) But in a case study of typical HF presentation that 70% of GPs said they would manage, only 20% would request an echocardiogram. (20)

Problems with diagnosis were exposed in the pan-European IMPROVEMENT study (data from end of 1999 to mid-2000). The low value of echocardiography as a diagnostic tool is suggested with only 45% of GPs stating that they referred patients for ultrasound. (305) However, record reviews showed that in 82% of cases echocardiography was used for the diagnosis. Systolic dysfunction was found in 51% of HF subjects, with around half the doctors surveyed saying that they would differentiate systolic from diastolic HF. The remainder did not know how to differentiate or were not aware of the difference. (305)

UK GPs report problems with echocardiogram interpretation, quality of reporting and the meaning and interpretation of reports and EFs. (12, 304) Echocardiography was
introduced as a diagnostic tool only after some current GPs received their medical training and in the course of an interview, some GPs remarked about echocardiograms that they “just don’t know where they are with them” (12) and mentioned the need for further training around the use of such a service and in understanding the results. (304) There is also fear expressed of initiating the process of diagnosis and then being committed to a course of action. (12) Some studies have reported GPs not seeing the need for echocardiography in obvious cases or that their diagnostic skills did not need confirmation or further investigation. (9-11) This was confirmed in the SHAPE study (on primary care diagnosis of HF) where 75% of GPs stated that they often diagnosed HF on symptoms and signs alone. While 64% thought that echocardiography was necessary for the diagnosis of HF and 32% thought it supported a diagnosis of HF, only a third would often arrange an echocardiogram. (34) This may also be due to a lack of awareness of the importance of echocardiography or the doctor’s inexperience. (9, 11)

Other factors that may prevent GPs from referring patients are the long waiting lists, the difficulty in organising an echocardiography, the low utilisation at their local hospital, and unhelpful attitudes of their local cardiology service. (9-11) Patients who did not seem particularly unwell or older or sicker patients may not be referred when GPs do not believe that the benefit of echocardiography will be realised. (9, 12) A few GPs regarded echocardiography as a hospital or specialist tool. (10) One study found that predominantly solo GPs referred all their suspected HF patients for hospital assessment. (11)

If recommendations for HF management suggest obtaining an echocardiogram prior to proceeding with medication, there needs to be easy access, otherwise GPs may find it difficult to follow the prescribing recommendations. (24)

It may be possible to determine some reasons for using echocardiography or not. Table 3.2.i hangs these reasons on a theoretical framework that reduces the classification reported by Michie et al. 2005 into four main domains. (166) First, the GP requires understanding that comes from knowledge of the reasons for echocardiography, although not necessarily skill on the GP’s behalf. The second domain relates to psychosocial and cognitive influences. Social / professional role and identity may be relevant to whether the GP sees it as their responsibility to order an echocardiogram or as a responsibility of the hospital or specialist. Beliefs about capabilities are similar to skills as GPs would not perform the echocardiogram but may question their own ability
Table 3.2.i. Theoretical domains that can influence behaviour in ordering echocardiograms, initiating prescribing and increasing drug doses in HF

<table>
<thead>
<tr>
<th>Domain</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Echocardiography</td>
</tr>
<tr>
<td>Understanding</td>
<td></td>
</tr>
<tr>
<td>Knowledge, Skills</td>
<td>✓</td>
</tr>
<tr>
<td>Psycho-social and cognitive</td>
<td>✓</td>
</tr>
<tr>
<td>Social/professional role and identity, Beliefs about capabilities, Beliefs about consequences, Motivation and goals, Memory, attention and decision process, Emotion, Behavioural regulation, Social influences</td>
<td></td>
</tr>
<tr>
<td>Resources</td>
<td></td>
</tr>
<tr>
<td>Environmental context and resources</td>
<td>N/A</td>
</tr>
<tr>
<td>Change in practice</td>
<td></td>
</tr>
<tr>
<td>Memory, attention and decision process, Nature of the behaviours</td>
<td>✓</td>
</tr>
</tbody>
</table>

* refer to Chapter 3, section 3.3

✓ = this domain has an influence on the outcome

N/A = no influence on outcome
to interpret and understand the echocardiographic reports. *Belief about consequences* links into *knowledge*, and *motivation*, because the consequences of echocardiography are confirmed diagnosis and more appropriate and confident management. For GPs who rarely see patients with suspected HF, *memory, attention and decision processes* may play a part in not ordering an echocardiogram, which is also related to the *nature of the behaviour*. The third domain is resources. GPs may seek to avoid diagnostic delays produced by the *environmental context and public resources* in secondary care. The fourth domain relates to changes in practice. *Social influences* reflect how much GPs know of what others are doing or whether practice protocols are in place. *Behavioural regulation* is also associated with *motivation and goals*, and requesting an echocardiogram may not be a priority if it is believed that a patient may not benefit from an echo or if the advantages of an echocardiogram report to future management are not realised.

### 3.2.6.3 Accuracy of echocardiography compared with clinical diagnosis

The usefulness of echocardiography in confirming the accuracy of suspected HF has been shown to be very similar across a range of studies. Patients with clinical diagnoses of HF who had been referred for echocardiography have the diagnosis confirmed or evidence of LVSD identified in only 20 to 30% of cases. (203, 207, 243, 272, 274, 278, 281, 306-308) and up to 16% of patients referred have borderline impairment (203, 243, 278) These figures reinforce the difficulty of diagnosing HF on clinical grounds alone. The power of echocardiography as a diagnostic tool is described in a study of patients ≥65 years, who had a clinical diagnosis of HF. Only 56% of these patients had an ECG performed by the GP and 50% of patients had a CXR. The prevalence of HF, based on clinical assessment, was 29/1000 patients. Echocardiography was done to confirm LVSD and the prevalence then reduced to 9/1000 patients (≥65 years). The false positive diagnosis (71%) through clinical assessment indicates that signs and symptoms of other conditions are taken as HF. The authors quote figures that suggest up to 42% of these false positives could be accounted for by COPD and obesity together. (309)

Assessment by echocardiography may also uncover previously unknown HF as demonstrated in a cross-sectional study of primary care patients aged 70 to 84 years. (286) 1056 patients (mean age 76.1 years, 54% female) were examined clinically and
by echocardiography, and half of the patients with ventricular dysfunction (32/61) had never had HF documented in their medical records. (286)

Of 505 symptomatic patients identified as being treated with diuretics, 281 (55.6%) fulfilled the diagnostic criteria of specified signs and symptoms or dyspnoea and CXR evidence or dyspnoea and echocardiographic evidence. (300) The median age was 76 years, and the sex ratio was almost equal. Performing echocardiography on these patients demonstrated that 63% of patients had evidence of LV dysfunction. (300) These patients were already sufficiently symptomatic for their GP to prescribe a diuretic, possibly following the ESC criteria for HF diagnosis of response to therapy. (300) Some of these patients already had CXR or echocardiographic evidence suggestive of HF which could explain the high proportion with evidence of LVD on echocardiograph. (300) Another study of symptomatic patients who were prescribed diuretics found that 41% had echocardiographic evidence of LVSD. (243)

A concern that GPs may not identify clearly appropriate patients for referral and thus overload the open-access service was not a problem. In one case of an open-access echocardiography service there were 32 (12%) inappropriate referrals (that is, they did not have any of the indications on the request form). (306) An even lower inappropriate referral rate was seen in another study of open-access echocardiography where only 3 patients out of 250 referrals (1.2%) were seen as inappropriate. (272) Another rapid access HF clinic received 391 referrals for suspected HF over a 15 month period with 383 of these referrals appropriate to the clinic guidelines. (281) Despite fears that GPs would overwhelm such services, it would seem that the open-access type clinics are used wisely by GPs who have patients with suspected HF whom they wish to have further investigated.

In 2000 the cost in the UK was reported as £50 to £70 per patient per echocardiograph (278), although this needs to be compared with monitoring and treating incorrectly diagnosed patients which could cost £246 to £486 / year. (310)

3.2.6.4 Difficulties measuring echocardiograms

Some patients may not be suitable candidates for echocardiography, but this figure varies widely between studies. The MONICA study echoed all patients and EFs were measurable in almost 90% of participants. Patients who had measurable EF were significantly younger (p = 0.0002), were less likely to have DM (p = 0.004) or HTN (p
= 0.004), had a lower mean BMI (p < 0.001), and were less likely to have angina (p = 0.05). (259) Similarly, a study of an open-access echocardiography clinic found that 6 out of 250 patients (2.4%) were not able to have EF assessed. (272) However, another study assessing the value of open-access echocardiography found a much higher failure rate. Chamber dimension measurements (for precise measurement of LVFx) were not possible in 108 patients (42%) due to obesity or airways disease, although LVFx was then assessed as ‘normal’ or ‘impaired’. (306) Further figures were observed in the Rotterdam Study sub-study where 2823 HF patients underwent echocardiography. Almost 20% of recordings (n = 556) were not adequate to measure LV dimensions. These patients were more likely to be older, have a higher BMI, use medication for COPD and have CVD or DM. (264) A study of existing COPD and undetected HF found that 10% of echocardiographs were of poor image quality, with one unable to have LVEF estimated as a result. (275) In summary, while there will be patients for whom the quality of an echocardiography report is not good, these patients tend to be older and more unwell. They should still be sent for an echocardiograph to try to determine heart function.

3.2.6.5 **Provision of echocardiography reports to primary care**

If HF is suspected, then management needs to be guided by accurate diagnosis. In the CASE study, ACEi prescribing was higher in patients with echocardiographic evidence of LVSD than patients without echocardiogram. (260) However, a medical record review of 250 patients with a clinical diagnosis of HF (taking a diuretic without an ACE inhibitor) found that, two months after referral to an echocardiography clinic, ACEi had been started in only 50 patients; and a further 13 had been referred to hospital for supervised initiation. In patients who had EF<40%, 38 (78%) were eventually started on an ACEi. (272)

The rationale for open access diagnostic services is rapid diagnosis, and prevention of the delay which could lead to inappropriate treatment. (306) A positive diagnosis will allow appropriate treatment and negative findings will allow unnecessary treatment to be withdrawn. (306) However provision of echocardiography reports may not always guarantee this diagnosis (of HF) in primary care. The CASE researchers study noticed unusual diagnostic decisions by the GPs after echocardiography was performed.
● 466 patients (with a history of HF) had echocardiography. 108 reported systolic or diastolic dysfunction but only 77 patients were diagnosed as HF by their GPs.
● 420 patients were newly diagnosed and 162 had echocardiograms. 19 patients (of 162) were classified as HF by their GP but with no evidence of LVSD or LVDD. (260)

The reasons for these false positives and false negatives are not known, and suggests a need to supply highly specific echocardiography reports to GPs.

The unusual decisions made by GPs in the face of objective evidence of HF were also seen in a case study (102) of a typical development of systolic dysfunction which listed prior MI, signs, symptoms and raised CTR. Interpretation of the echocardiogram results (reported as ‘diffuse hypokinesis’ and ‘moderately to severely reduced EF’) was statistically significant between the cardiologists and GPs attributing the symptoms’ cause.

● HF from LVSD was considered the cause by 66% of cardiologists and 42% of GPs (p<0.01), with other possibilities given as:
● HF from combined LVSD and LVDD: 26% of cardiologists and 34% of GPs,
● HF from LVDD alone: 5% of GPs and 1% of cardiologists, and
● Silent ischaemia thought to be the cause by 15% of GPs and 7% of cardiologists. (102)

It would seem more difficult for GPs than cardiologists to interpret echocardiographic reports, which makes diagnosis more complex and identifying treatment options more difficult.

**STUDY DEFINITIONS OF LVSD:** However creating more confusion are conflicting reports of what level of EF defines abnormal LVSD. Studies quoted in this thesis use different values to define LVSD and these are presented in table 3.2.j. To add to the confusion, different studies use different descriptive terms for reduction in function; this can also be seen in the table in an effort to group similar reported dysfunction.
Table 3.2.j. Comparison of study definitions of impaired left ventricular systolic function

<table>
<thead>
<tr>
<th>Descriptive terms</th>
<th>Poole cross-section (286)</th>
<th>Canberra Heart Study (311)</th>
<th>MONICA Glasgow study (259)</th>
<th>Rotterdam study (264)</th>
<th>ECHoES (312)</th>
<th>SOLVD trials (14, 15), RALES (25)</th>
<th>MERIT-HF trial (313)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild LVSD</td>
<td>EF = 48%</td>
<td>EF 41 – 50%</td>
<td></td>
<td></td>
<td></td>
<td>Borderline LVFx: EF 40% – 50%</td>
<td></td>
</tr>
<tr>
<td>Moderate LVSD</td>
<td>EF = 38%</td>
<td>Moderate or severe: EF≤40%</td>
<td></td>
<td></td>
<td></td>
<td>Definitely impaired: EF &lt;40%</td>
<td></td>
</tr>
<tr>
<td>Severe LVSD</td>
<td>EF = 26%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal LVSD</td>
<td></td>
<td>EF &lt;35%</td>
<td>EF &lt;42.5%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial entry criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>EF ≤35%</td>
<td>EF ≤40%</td>
</tr>
</tbody>
</table>
**GUIDELINE DEFINITIONS OF LVSD:** The 2001 ACC/AHA HF guideline (77) quotes the 1997 ACC/AHA guidelines for the clinical application of echocardiography (314) as saying that patients with an EF<40% are generally considered to have systolic dysfunction. These values are further complicated by the 2005 ACC/AHA clinical data standards for chronic HF (297) that describe qualitative measurements of EF. Normal is considered to be LVEF>50%, mildly diminished as LVEF 41 to 49%, moderately diminished as LVEF 26 to 40%, and severely diminished as LVEF≤25%. The NZ HF guideline does not suggest EF values for systolic dysfunction. (29)

One paper endeavours to integrate a number of the studies quoted above and suggests the following cut-off values: mild dysfunction EF 41 to 49%, moderate dysfunction EF 35 to 40%, and EF<35% should represent severe dysfunction. (315)

The ultimate evidence of the usefulness of echocardiography is the NSF figures for echocardiography rate and ACEi prescription. While it may be difficult to differentiate the financial effects from the good prescribing decisions, the rate in increase of confirmed diagnosis of HF has been accompanied by an increase in the prescription of ACEi.

**SUMMARY:** There is a need to convince GPs that the gold standard diagnostic test for suspected HF is the echocardiogram. It is difficult to determine how to quicken access to this test given the resource constraints of funding, space and time, i.e. more ultrasound machines, more sonographers, more cardiologists to interpret and report back to GPs. Reports to GPs need to be standardised and give clear diagnoses and management recommendations. However, GPs also have the responsibility of transferring relevant information to the patient’s medical record and implementing the recommendations.

**3.2.7 Brain Natriuretic Peptide**

The current accepted use of BNP / NT-proBNP in primary care is that of a diagnostic test that is useful in ruling out HF. BNP is mentioned in the NZ HF guideline as a recommended test with HF likely if BNP is elevated. (29) It has been suggested that the test could be used as a triage for symptomatic adults to select which patients should go forward for echocardiography. A positive test would recommend echocardiography and
a negative test would be likely to exclude HF. It may be better used in selective groups (i.e. with higher probability of HF). (242, 307) While BNP concentration increases with HF severity / NHYA classification, it should not be used to predict the classification of HF. (316)

Values in between the BNP reference points are a grey area, where HF could still be possible but BNP levels may be altered by certain conditions. N-BNP levels may be raised by renal impairment, AF, LVH, COPD, after MI, in the elderly, and by treatment with β-blockers or digoxin. Conversely, N-BNP may be decreased by hypothyroidism, treatment with diuretics, ACEi or vasodilators. (277)

The popularity of BNP as a diagnostic tool has been demonstrated in Christchurch, NZ, as seen in the analysis of the numbers of tests requested in the 7 years since it was first offered in February 1995. (317) The number of BNP samples measured increased from approximately 500/year to almost 4000/year (2001). General practice accounted for 26% of these requests. (317) An English study looked at the comparative costs of BNP and echo and found that the cost of a BNP test was £6.60 whereas the cost of an echocardiogram was calculated as £54.00. (318) The cost of a BNP test in NZ is currently around $NZ50.

**SUMMARY:** Tests of BNP / N-BNP levels have been shown to be useful in determining which patients should be referred on to echocardiography, and which patients require tests for other conditions with similar symptoms, reducing concerns that GPs will overwhelm echocardiography services. However research is still needed on the levels of BNP / N-BNP that signify HF in different patient populations and the utility of BNP / N-BNP in monitoring the effectiveness of medication, which could encourage GPs to increase doses of HF medications.

### 3.2.8 Process of Diagnosis

The preceding sections set out the ideal process to follow for diagnosing a gradually deteriorating primary care patient who has suspected HF. However this is not necessarily the sequence of events that occurs in general practice. Early diagnosis of HF is usually based on symptoms that need to be confirmed. (266) No standard questionnaire or validated scoring system is recommended for diagnosing HF in clinical
practice. (249) The majority of diagnoses are made by GPs (267) and most HF patients in the UK are cared for by GPs, not cardiologists. (243) However GPs may only infrequently see new cases of HF and so the clinical presentation of cases may not be familiar to many GPs, further emphasising the need for standardised grading methods for signs and symptoms that GPs can easily access. Furthermore, the types of new HF patients that attend GPs and emergency departments or cardiologists may differ in presentation, and given that the diagnostic tests immediately available to these health professionals will be different, the diagnostic process needs tailoring for each specific area. As demonstrated in previous sections of this chapter, GPs and cardiologists diagnose different HF populations and use different tools to achieve the diagnosis. (252, 267, 319, 320)

There is a lack of consensus on diagnostic criteria as illustrated by a survey of 2700 practising physicians (251) (family physicians / general practitioners, internists, practising cardiologists and academic cardiologists) in order to determine their understanding of HF. There was a broad range of signs and symptoms considered to be diagnostic of HF, even by academic cardiologists. The “diagnosis” from different supposed signs and symptoms may lead to inappropriate treatment. (251) The authors re-emphasise that most RCTs have not defined the criteria used for diagnosis, and that there was no “universally accepted” definition of HF for either researchers or clinicians. They proceed to state that “further work to develop and disseminate validated clinical criteria for this diagnosis is long overdue”. (251) The problem identified by this survey over 20 years ago, and reiterated 10 years ago (319), still has not been resolved. The diagnostic procedure for HF in primary care has been of concern for many years as patients are frequently diagnosed without any objective evidence. GPs participating in the Euro-HF (35) were asked what proportions of their HF patients were diagnosed by the following methods:

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms alone</td>
<td>26%</td>
</tr>
<tr>
<td>Signs and symptoms</td>
<td>41%</td>
</tr>
<tr>
<td>Only after further investigations</td>
<td>21%</td>
</tr>
<tr>
<td>Only after referral to hospital specialist</td>
<td>12%</td>
</tr>
</tbody>
</table>
These figures indicate that two-thirds of patients were diagnosed as having HF without objective evidence from diagnostic tests. (35) However a study of newly diagnosed HF patients in the UK indicated that 5% were confirmed by GP assessment alone. (266) In the Australian CASE study (260) diagnosis was based on symptoms 73%, signs 66%, causative factors 61%, and investigations 49% (categories not mutually exclusive).

Concerns raised frequently by GPs were that multiple symptoms and other comorbidities masked the condition; that there was a difficulty in distinguishing the cause of symptoms (cardiac or respiratory); and that the early stages of HF had no or few specific symptoms. (10-12) There was also a lack of time during a consultation to fully investigate. (11, 12)

Tests routinely used by GPs (Euro-HF survey) for diagnosis were CXR (86%) and ECG (72%). Echocardiography was routinely used by 38% of GPs. (35) These figures are similar to those found in the UK newly diagnosed HF study (266) in which 75% of patients had some diagnostic test and 29% of patients had echocardiography. Figures of newly diagnosed CASE study patients (260) are not quite as high: 60.4% had at least one investigation in the 12 months before and after the audit.

Table 3.2.1. Diagnostic tests used in newly diagnosed HF by CASE study GPs

<table>
<thead>
<tr>
<th>Diagnostic test</th>
<th>Proportion of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG</td>
<td>50.9%</td>
</tr>
<tr>
<td>CXR</td>
<td>49.6%</td>
</tr>
<tr>
<td>Echocardiography</td>
<td>17.7%</td>
</tr>
</tbody>
</table>

When the CASE study investigators looked at patients who had a previous diagnosis of HF, they found a much higher use of diagnostic tools with 96% of patients having had an ECG, 64% an echocardiogram, and 66% referral to a specialist. (260)

GPs have claimed a lack of access to diagnostic tests and a fear of initiating the diagnostic process that would then commit them to an intensive course of action. (12) More likely to refer younger patients for investigation, (11) GPs also mentioned that some patients were reluctant to undergo further investigation and treatment. (12)

Given the rarity with which GPs see new cases of HF, their diagnostic skills may not improve over time, as suggested by a study of N-BNP and diagnostic accuracy, which assessed the appropriateness of more than one diagnosis of HF made by GPs. (277) GPs who referred two or more patients to the study were assessed for a learning effect,
i.e. a trend in diagnostic accuracy. Diagnostic accuracy did not change. Also, for GPs (n=7) who had patients randomised to the BNP group and to the control group, patterns of diagnostic accuracy were examined for contamination. Diagnostic accuracy did not change during the study. (277)

However GPs may tentatively treat suspected HF patients anyway. The patients (≥65 years) (in a study that evaluated the use of N-BNP and HF diagnosis in COPD) had COPD (according to their GP), but not known HF. (321) Cardiovascular medication (ACEi, ARBs, and diuretics) was more commonly prescribed to patients who were eventually classified by a panel as having HF (see table 3.2.m) although the differences in prescription between the groups (HF and not HF) were not statistically significant. (321)

Table 3.2.m. Patients with COPD, determined as having HF present or absent and medications and comorbidities

<table>
<thead>
<tr>
<th>Medication</th>
<th>HF present (N = 51)</th>
<th>HF absent (N = 149)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretics</td>
<td>16 (31.3%)</td>
<td>27 (18.1%)</td>
</tr>
<tr>
<td>ACE I or AT-II blockers</td>
<td>18 (35.3%)</td>
<td>34 (22.8%)</td>
</tr>
<tr>
<td>Patient History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IHD (including MI, angina, PCI, CABG)</td>
<td>28 (54.9%)</td>
<td>55 (36.9%)</td>
</tr>
<tr>
<td>DM</td>
<td>10 (19.6%)</td>
<td>11 (7.4%)</td>
</tr>
</tbody>
</table>

Although unclassified by the GP, these patients may be intuitively treated as HF patients.

The two studies discussed above indicate that complex processes are occurring during the diagnosis of HF. An aspect that needs to be considered is diagnostic reasoning. Areas in the process of diagnosis that cannot be quantified are the heuristics (also known as mental shortcuts or rules of thumb). (322, 323) GPs have described these as something appearing immediately to mind which help to simplify and to structure their work. (322, 324) They were quite often an expression of probability. They functioned unconsciously because their purpose was to save the GP from reasoning although GPs were able to differentiate between routines, guidelines and rules of thumb. (322, 324)

The rules of thumb tended to originate from word-of-mouth from colleagues, the workplace, medical education and experience although clinical experience was seen as a
prerequisite for applying the rules. While GPs are able to identify these rules, they have been referred to as ‘tacit knowledge’ which is defined by “we can know more than we can tell”. (322, 323, 325) The rules must also be simple and easy to convey. It may be due to these factors and the idiosyncratic nature of some rules that while new and complex information is gained practice remains unaltered. (322)

However some practices do not have a systematic method of diagnosing HF and GPs within practices do not concur about diagnostic pathways. (11) One method of determining how GPs use diagnostic data is through a think aloud technique. It was used to learn about GPs’ diagnostic reasoning and judgement in case studies of suspected HF patients. (326) GPs were asked to verbalise their thoughts on the probability of the case studies’ patients having HF. The case studies included information on comorbidities, lifestyle factors, signs and symptoms, ECG, CXR and echocardiography. The diagnostic reference standard was cardiologist assessment of the case studies. The ESC HF guidelines were used as a comparison. The case studies included commonly seen confounding factors such as obesity, COPD, clinical findings of HF but normal LVFx on echocardiography. (326)

During diagnosis, the most frequently used information was EF on echocardiography and pulmonary congestion. The most frequent argument against CHF was EF and the most frequent argument for CHF was pulmonary congestion on CXR (38 times with 32 for and 6 against). Out of the 90 diagnostic situations (15 GPs and 6 cases), EF / LVFx was used as an argument in 48 (with half arguing for and half against CHF), but in 33 situations no echocardiographic information was used to assist diagnosis which represents a third of diagnoses. (In 9 situations other echocardiographic information was used.) There was variation between GPs in the use of echocardiography with five GPs using EF to consider diagnosis in five cases, down to two GPs who never used information on EF. Interestingly, some GPs indicated that they were uncertain about the meaning of EF. (326)

Symptoms and signs were not often used in diagnostic reasoning. Not all GPs used signs correctly and one specific sign was used by only one GP. There was confusion about the implications of comorbidities – the same disease seen as explanations for symptoms and by different GPs as increasing the risk of CHF. (326)

There was large variation in diagnosis for the case studies. Clinical information presented was not used to the extent suggested by the ESC guidelines. Structuring
previous and current medical history of the patients into diagnoses may be indicative of a more experienced GP. (326) History of MI was presented in five case studies, but only used as an argument for HF 11 times. The authors suggest that the guidelines need to be more explicit on the use of past and current CVD (documented or investigated) in considering a diagnosis. (326)

The authors also suggested that diastolic HF be discussed in guidelines given the confusion over one case study in which the patient displayed signs, symptoms and radiological evidence of HF but who had normal EF. (326)

Difficulties in accessing echocardiography has been given as a reason for incorrect diagnosis or missed diagnoses. Opening up access to echocardiography may not improve the diagnosis of HF if GPs are unsure of the meaning of the reported variables. One third of the diagnostic judgements did not take into account any echocardiographic evidence. This suggests that GPs need more thorough education regarding the meaning of echocardiography findings and the integration of these in the diagnostic process of HF. (326)

**SUMMARY:** There is a complex interplay of factors which are ongoing in the process of diagnosis. There are issues of incorrect belief or lack of knowledge but these are coupled with a lack of standardised description of what constitutes HF. While these problems can most likely be overcome through education, it will be harder to determine what rules of thumb are operating if the GPs themselves are unaware of these.

### 3.2.9 Diagnosis in the Elderly

A difficulty in describing the ‘elderly’ is that there is no actual definition of the ‘elderly.’ This is compounded by the difference in chronological and biological age, affected by genes, lifestyle, wellness and an interaction of these three factors.

In diagnosing HF, typical signs and symptoms may be absent or more subtle or obscure than in younger patients. Dyspnoea on exertion may not be present if the patient has a sedentary lifestyle and, if symptoms do occur, patients may decrease their activities and the symptoms will reduce or disappear. (327, 328) Or the effects of ageing may be considered the reasons for some symptoms; e.g. weakness, tiring easily, or underlying lung disease may be thought to be the reason for respiratory symptoms such as cough,
slight shortness of breath, pulmonary crackles. (327, 328) Ankle oedema is a common symptom in elderly patients and should not be associated with HF unless there are other signs of HF. (328) AF is another common condition associated with ageing and may aggravate existing HF or tip subclinical HF into an overt state. (328) Other comorbidities and/or long-term medications may also affect signs and symptoms.

In a study of the incidence of HF, as patient age increased, the proportion of referrals decreased, which suggests that investigation is less thorough. The diagnosis of HF will be less certain in the elderly than in younger age groups leading to inappropriate management. (266) The effect of the rise in diastolic dysfunction with age also needs to be considered and the only objective test which will uncover this is echocardiography. The elderly should be investigated as thoroughly as all other patients by ensuring that every patient who is clinically suspected to have HF is offered a referral for an echocardiogram (300). Determining an accurate diagnosis will aid in management plans and has the potential to improve prescribing and patient well being.

3.2.10 Summary

HF is difficult to define and diagnose as there are differing criteria and different weight is placed on these criteria. What is certain is the increased likelihood of HF as patients age and in patients with multiple risk factors, but again the weight given to individual risk factors differs between studies. In these instances it is important to assess the patient population used for the study.

In general, signs and symptoms can also be suggestive of other conditions and for this reason a clinical diagnosis of HF is not dependable. Only a minority (20 to 30%) of suspected HF patients do actually have HF. The effect of aging further complicates the diagnostic process with signs and symptoms obscured or a result of increased biological age or other comorbidities.

Objective testing of suspected HF is required, which currently includes CXR (possibly), BNP and echocardiography. ECG is perhaps a more specialised tool used to identify more subtle abnormalities rather than evidence of LVSD/HF. BNP may become critical to determine which cases of suspected HF proceed to echocardiography. A recent HTA report proposed that a breathless patient, with suspected HF, should be referred directly to echocardiography if they have a history of MI or basal crepitations or are male with
ankle oedema. (288) For other cases of suspected HF, referral to echocardiography would depend on the BNP result. (288)

GPs do not seem to view echocardiography as a highly important diagnostic test. They may not understand its importance or may not see it as their responsibility to order echocardiographs. There may be deeper concerns regarding the consequences of echocardiography – that they would then have responsibility for managing this patient. GPs may not be aware of what their colleagues are doing and so do not feel pressured into referring suspected HF patients for echocardiography.

The echocardiography report may be confusing and inconclusive, with little indication or suggestions for future management. GPs can quite reasonably expect a lead from secondary care in the management of such a condition. But this does not relieve them of the duty of careful assessment and working in the patient’s best interests.

3.3 Treatment of Heart Failure

The 2001 NZ Guideline for the Management of Heart Failure states that its aim is to:

“... reduce morbidity and mortality from congestive heart failure. ... . Outcomes predicted are increased survival and reduced morbidity as represented by either functional scores or by hospital admission.”

These objectives can only be met if among other management strategies, medications for HF are optimised. While a ‘basic’ drug regimen could include a minimum of four drugs (e.g. diuretic, angiotensin inhibiting agent, specific β-blocker, spironolactone), these all act by different mechanisms to achieve different therapeutic aims.

The next section backgrounds the new trial evidence for the management of HF, which was used to develop the educational interventions used for this thesis. These studies relate to ACEi, as the evidence of beneficial effects of ACEi in HF has been available since the late 1980s and early 1990s and less than 10 years later studies on the effect of dose; β-blockers available in NZ for prescription in HF; and spironolactone. This is followed by a discussion illustrating the dissimilarity between trial patients and community HF patients.
The next section (3.3) examines the actual treatment (prescriptions and doses) received in primary care, some differences between primary and secondary care, and the effect of patient variables on treatment prescribed. GPs’ perceptions of barriers to prescribing are also discussed, and then applied to theoretical domains that may influence behaviour. Chapter 2 examined factors such as age, GP and practice variables that might have an effect on decision making and HF management.

The final section of this chapter (3.4) considers HF management in secondary care. It covers rates of diagnosis and treatment and the effects of age and sex thereon. The effect of care provider and physician attitudes to HF management are briefly discussed. Decisions regarding diagnosis and management made at this level of care will have a significant impact on how patients are subsequently managed when discharged back into primary care. This chapter ends with concluding points on the diagnosis and management of HF.

3.3.1 Trial evidence of beneficial treatment outcomes
Several clinical trials have demonstrated benefits from medications for HF, but what is of concern is the slow uptake of these medications into general use for HF. The following medications have proven high quality evidence regarding their benefits in HF.

3.3.1.1 ACE Inhibitor Dosage
The benefits of ACEi in HF have been demonstrated in several trials. (29) The two significant placebo-controlled trials of ACE inhibitors were CONSENSUS\textsuperscript{a} (published 1987) and SOLVD\textsuperscript{b} (published 1991). (13-15) A brief evidence summary of these is available in Appendix 4E which contains the NZ HF Guideline.

A 1994 review paper (299), that discussed the foundations of ‘modern’ management of HF gave this key message:

“[that] all patients with heart failure should be considered for such treatment [with ACE inhibitors] even if they have been rendered asymptomatic by a diuretic.”

\textsuperscript{a} CONSENSUS: Co-operative North Scandinavian Enalapril Survival study.
\textsuperscript{b} SOLVD: Studies of Left Ventricular Dysfunction.
Higher doses were also emphasised when the authors discussed enalapril 10mg twice a day and captopril 25 to 50mg three times per day; although one study that is quoted in their article aimed for captopril 100mg three times per day. The greater benefit conferred by higher doses is also referred to in two studies published in 1991 and 1993.

The ATLAS\(^c\) study (published in 1999) showed an advantage of high dose (30mg) lisinopril over low dose (5mg) in lowering the risk of death or hospitalisation (any reason) and reducing hospitalisations for HF (Classes II to IV). (16) While the high dose of lisinopril did not seem to confer mortality benefits (-8% all-cause mortality, p=0.128 and -10% cardiovascular mortality, p=0.073), the differences between high-dose and low-dose groups were clearly seen in the hospitalisation endpoints. Risk of death or hospitalisation for any reason was 12% lower (p=0.002) in the high-dose group, and hospitalisation for HF was 24% lower (p=0.002), 16% fewer hospitalisations (p=0.05) for any cardiovascular reason. Patients receiving high-dose lisinopril did experience slightly higher rates of dizziness, hypotension and worsening renal failure, but lower rates of worsening HF or cough. However, overall, 18% of patients in the low-dose group stopped taking lisinopril and 17% of the high-dose group. High-dose lisinopril reduced the risk of major clinical events in patients with chronic HF. (16)

A barrier to increasing ACEi dose in practice seems to be a belief held by doctors that a minimum BP measurement of 120/80 mmHg should be maintained rather than letting the patient determine the maximum dose suggested by side effects. Blood pressure control should not be relied on as a surrogate marker for adequate ACEi dose. (17) Successful titration and maintenance of high dose were possible in patients in the ATLAS trial who had low blood pressure, increases in baseline serum creatinine, and patients over 70 years. These results demonstrate that a high maintenance dose is possible in most patients. (18)

In the ATLAS trial, cost savings were associated with the high-dose group of patients. Mean days in hospital for high-dose patients were 18.5, and for the low-dose group 22.5. The mean cost of high-dose lisinopril was greater but off-set by fewer in-patient days, with a saving per patient of £397 (currently approximately NZD978), over the duration of the trial (4 years). Without discounting\(^d\), this figure rises to £465 (NZD1146). The treatment of HF by high-dose lisinopril has a high probability of being more cost effective than low-dose. (19)

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\(^{a}\) ATLAS: Assessment of Treatment with Lisinopril And Survival.

\(^{d}\) Discounting gives greater value to current costs and benefits than those occurring in the future. (329)
Too few patients are being initiated on or have ACEi up-titrated to optimal doses (330) which limits decreases in re-hospitalisation rates, the time to readmission and increases the likelihood of readmission. (331, 332)

3.3.1.2 Angiotensin II Receptor Blockers

At the time the NZ HF guideline was published (December 2001), little was known about the effects of Angiotensin II Antagonists (also known as Angiotensin Receptor Blockers or ARBs) in HF beyond from the ELITE\(^6\) and ELITE II trials. The recommendation from ELITE II was to use ARBs as an alternative RAS blocker if ACEi were not tolerated. (333) This recommendation was given in the 2001 NZ guideline. (29)

A positive aspect of the ELITE and ELITE II trial was the utilisation of an older age group (67% aged ≥70 years and 85% aged >65 years). (333, 334)

The two ARBs available in NZ are losartan (trade name: Cozaar) and candesartan (trade name: Atacand). Both were available only on Special Authority, with candesartan listed for use in hypertension with the application able to be made by a GP, and losartan listed for use in congestive HF, with the application to be made by a cardiologist.

3.3.1.3 β-blockers

The introduction of β-blockers to HF management represents an important development requiring a change in usual practice for most cardiologists and GPs. Due to their mechanism of reducing contractility (335), β-blockers have previously been contraindicated in HF. In 1973 it was observed that a low starting dose of β-blocker that was up-titrated improved patients’ condition. (336) However, a 1994 paper on the diagnosis and management of HF mentioned β-blockers under the heading of ‘Controversial Treatments’. (299)

International figures suggest that <30% of eligible patients are being treated with β-blockers. (337) Other authors also mention non-β-blocker HF trials (Valsartan, biventricular pacing, omapatrilat vs. enalapril) which had β-blocker prescription rates of

\(^6\) Evaluation of Losartan In The Elderly.
25 to 50%. (338) Usage rates in clinical trials (MERIT-HF\textsuperscript{f}, and U.S. carvedilol HF study) reached 88%, with no significant differences between drug and placebo discontinuation rates in the MERIT-HF study. Yet studies of clinical practice (SPICE, published in 1999; Achieving Carvedilol Excellence Project, published 2003) and insurance claims (published 2003) reported β-blocker use as low as 30% in eligible patients. (339) Information from participating GPs (surveyed mid-late 2002) indicated that not all may be aware of β-blocker use and some recommend only low doses of β-blockers.

Two β-blockers are registered for use in NZ for HF. These are extended release metoprolol (succinate) a β\textsubscript{1}-receptor blocker (trade name: Betaloc CR), and carvedilol, an α\textsubscript{1}-, β\textsubscript{1}-, and β\textsubscript{2}- receptor blocker (trade name: Dilatrend).

The MERIT-HF trial showed that use of metoprolol improved survival, with a reduction in hospitalisations due to deterioration in HF and a reduction in number of hospital days as well as improved well-being. (21) This trial was stopped early due to the substantial reduction in total mortality in the metoprolol group. The ANZ\textsuperscript{g} carvedilol trial demonstrated a reduced number of events resulting in death or hospitalisation admission and some improvement in ventricular function and size. (22) The COPERNICUS\textsuperscript{h} trial showed benefits of carvedilol in patients with severe HF. (23) Again, this trial was stopped early due to the marked benefit of carvedilol on mortality.

The β-blocker effect appears additive to the ACEi effect. (340) The addition of β-blockade to established HF therapy is well tolerated and should be possible for ambulatory patients. (313) Since treatment with β-blockers may produce an initial reduction in quality of life (337), the assistance of a cardiologist may be required when starting patients on low-dose metoprolol (341). However, a committed, trained non-specialist could initiate and titrate β-blocker therapy and experience would improve the success and ease of implementing these steps. (86)

The NZ HF guideline (29) lists survival benefits of β-blockers, derived from a meta-analysis which also included bucindolol and bisoprolol, neither of which is available in NZ. (See table 3.3.a, derived from p.15 of the NZ HF guideline.)

\textsuperscript{f} MERIT-HF: Metoprolol CR/XL Randomized Intervention Trial in congestive heart failure.
\textsuperscript{g} ANZ: Australia and New Zealand.
\textsuperscript{h} COPERNICUS: Carvedilol Prospective Randomized Cumulative Survival.
Table 3.3.a. Benefits of β-blocker treatment in HF

<table>
<thead>
<tr>
<th>Absolute risk reduction</th>
<th>4.5%. Annual mortality rate in placebo-treated patients 17.4% (approx) vs. 12.9% in β-blocker patients.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative risk reduction</td>
<td>28% (SD 4%)</td>
</tr>
<tr>
<td>Number needed to treat</td>
<td>22 to prevent 1 death during 1 year (approx.) of treatment.</td>
</tr>
</tbody>
</table>

### 3.3.1.4 Spironolactone

Spironolactone has been used in doses of up to 100mg per day as a potassium-sparing diuretic for many years. The RALES\(^1\) trial used spironolactone to suppress aldosterone. The CONSENSUS I trial noted that there were correlations between mortality and angiotensin II and aldosterone and that suppression of the renin-angiotensin-aldosterone system during ACE inhibitor treatment was not consistent. (342) Angiotensin II reactivation stimulates aldosterone release which increases sodium and fluid retention. (343) RALES showed that the spironolactone group\(^j\) had a 30% reduction in the relative risk of death from progressive HF and sudden death from cardiac causes, and a 35% reduction in the frequency of hospitalisation for worsening HF. (25) This trial was also stopped early due to the reduction in mortality in the spironolactone group\(^k\). Again, the beneficial effects of spironolactone are additive to those of ACE inhibitors. (344) The dose that is currently used is 25mg per day. This is not to underestimate the potential seriousness of the side effect of hyperkalaemia.

The NZ HF guideline (29) lists survival benefits of spironolactone (see table 3.3.b, derived from p. 17 of the NZ HF guideline).

Table 3.3.b. Benefits of spironolactone treatment in HF

<table>
<thead>
<tr>
<th>Absolute risk reduction</th>
<th>11%. Two-year mortality rate in placebo-treated patients 46% (approx) vs. 35% in spironolactone-treated patients.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative risk reduction</td>
<td>30%</td>
</tr>
<tr>
<td>Number needed to treat</td>
<td>9 to prevent 1 death during 2 years of treatment.</td>
</tr>
<tr>
<td></td>
<td>11 to reduce the number of patients needing hospital admission for cardiac causes over 2 years.</td>
</tr>
</tbody>
</table>

\(^1\) RALES: Randomised Aldactone Evaluation Study.
\(^j\) All participants were already being treated with ACE inhibitor and diuretic.
\(^k\) The current evidence of benefit of spironolactone is for NYHA Classes III and IV.
3.3.1.5 Summary
High grades of evidence exist for the treatment of HF. It has been shown that high-dose ACEi have benefits over low-dose, and that the use of specific β-blockers in HF provides benefits as does the prescription of spironolactone. However, these medications must be prescribed to appropriate patients. Of even greater significance are the cumulative benefits that are observed when β-blockers are added to ACEi and also when spironolactone is added to ACEi therapy. The benefits are improved survival, reductions in all-cause and HF hospitalisations. The time lapse in translating evidence into practice and the sub-optimal dosing that occurs leave HF patients vulnerable to poor clinical outcomes. The challenge is to improve the use of the above medications so that they are standard in the management of HF patients in primary care.

3.3.2 Comparability of HF patients in trials and in primary care
It is the homogeneity of clinical trial populations which makes their findings so consistent but also makes difficult the transferability of those findings into general practice. (345, 346) It has been suggested that those who produce guidelines are so different in their area of practice to those who are supposed to apply them that these two groups are in danger of operating in “parallel realities”. (345)

The participants in clinical trials tend to be dissimilar to patients seen in practice but the magnitude of the discrepancy in HF at a population level is not well quantified. (347) What is known is that there is an evidence gap between the clinical trials and the management of older HF patients in the community. (65) Thus applying research findings to these patients in practice may require a ‘leap of faith’ which sometimes may be greater than others. (346)

Clinical trials tend to recruit from secondary or tertiary centres, where there is support from specialist clinicians. The participants recruited tend to be younger, predominantly male with a single (or primary) diagnosis of LVSD. (65) HF patients who are more likely to be managed in primary care are older, more often female (i.e. more gender equality than seen in clinical trials), with significant co-morbidity and concomitant medication, with a mix of systolic and/or diastolic dysfunction. (65, 348)
The difference in age between patients enrolled in clinical trials and those in the community can be great, with population-based studies indicating the average age of a patient with HF is 76 years, whereas clinical trial patients tend to be in their 50s and 60s. (349) This is demonstrated in a comparison of older patients surviving hospitalisation with HF with study enrolment criteria of major RCTs (e.g. SOLVD, MERIT-HF and RALES). The community-based patients were ≥65 years, with a mean age of 78 years, and 57% were women. (347) The definition of HF also tends to be broader (impaired and normal LVSFx). (347) The researchers applied the study enrolment criteria to the community cohort (see table 3.3.c) and found that very few of the cohort would have been eligible to participate in these trials. Including patients with impaired systolic function increased the number ‘eligible’ for participation in such HF trials.

Table 3.3.c. Clinical trial enrolment criteria and application to and eligibility of community HF patients

<table>
<thead>
<tr>
<th>Study</th>
<th>Eligible by Study Criteria</th>
<th>Total cohort %</th>
<th>Males %</th>
<th>Females %</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOLVD</td>
<td></td>
<td>17</td>
<td>23</td>
<td>13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Impaired LVSFx</td>
<td></td>
<td>38</td>
<td>40</td>
<td>35</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>MERIT-HF</td>
<td></td>
<td>13</td>
<td>17</td>
<td>11</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Impaired LVSFx</td>
<td></td>
<td>25</td>
<td>26</td>
<td>23</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>RALES</td>
<td></td>
<td>25</td>
<td>32</td>
<td>21</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Impaired LVSFx</td>
<td></td>
<td>55</td>
<td>55</td>
<td>54</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Of the total cohort, 67% of patients did not fit the enrolment criteria of any 1 of the 3 RCTs. The proportion increased with age and was uniformly higher in women (75%) then in men (59%). (347)

The SOLVD and MERIT-HF had upper age limits (80 years). Removing this limit reveals that patients eligible for the SOLVD trial increased from 17 to 35%, and for the MERIT-HF from 13 to 22%. (For each of the trials approximately 20 to 25% of patients in the higher age brackets (80 to 84, ≥85 years) would have been included; total, male and female.) (347)

Drug-specific exclusions were 37% for ACEi, 49% for β-blockers and 40% for spironolactone. A trend of higher numbers of drug exclusions with increasing age was
seen with ACEi and spironolactone (each p<0.001) but was reversed for β-blockers (p<0.0001), which may be related to the decrease in COPD seen with age (p<0.0001). (347)

The authors considered the reasons for trial inclusion/exclusion criteria; (347) for example, testing hypotheses of pathophysiological mechanisms for HF and the effects of drugs to attenuate them, hence the need for homogeneity. It may not be possible to determine the benefit of treatments if there are high rates of unrelated outcomes. Patient safety also needs to be considered in developing exclusion criteria for clinical trials. (347)

While the importance of the findings of these clinical trials cannot be understated (i.e. efficacy), this study demonstrates a significant gap which evidence must somehow bridge, despite it possibly not being applicable. Primary care or community data will demonstrate the effectiveness of the trial medications. The difficulties voiced by GPs about translating trial findings to their patients (10, 60) need to be acknowledged.

This is not to detract from the impressive therapeutic gains as demonstrated in HF trials. (345) (350) Perhaps what is needed is an awareness of the difference between the internal validity required of RCTs (to answer the study question posed) and the external validity or generalisability of the findings obtained in general clinical practice. (350) Improved knowledge of RCTs would create an understanding of the mechanisms of clinical trials and what their outcomes mean for other HF patients.

**SUMMARY:** The demographic and clinical characteristics of HF patients enrolled in clinical trials and HF patients in primary care can be quite different. The trial outcomes may not be entirely applicable in other settings, which can create cognitive difficulties for GPs whose task is to apply this evidence. However, similar outcomes can be achieved in primary care if the administration of the drug is given careful attention.

### 3.3.3 Actual treatment in primary care

Most patients with HF are first seen and subsequently managed in primary care. Given that recall of prescriptions by GPs and adherence to guidelines can be variable, it is necessary to verify patient management through audit of medical records. The following sections will illustrate discrepancies between recall and actual practice.
Delivery of care may be split depending on the patient; the patients managed in primary care tend to be more complex than those managed by cardiologists. (351) Patients in general practice tend to: be older, female, have long term HTN, be less likely to have experienced an MI, and be more likely to have diastolic HF. Patients managed by cardiologists tend to be younger, male, have a history of MI or CAD, and have systolic HF. (252, 351-354) The type of HF patient seen in primary care may influence the overall management compared with specialists. Other factors such as the age of the patient, characteristics of the individual GPs as well as practice dynamics may also play a part in primary care management strategies.

There is a tendency to overstate primary care prescription rates for chronic conditions such as HF. One study suggested that when the results of recording actual prescribing and GP reported prescribing were compared, there was an over-estimate of ACEi prescribing by approximately 50% in the prescribing reported by the doctors surveyed. (35) This is one reason why research into chronic disease management needs to be audit-based rather than simply reported recall.

A review of journal articles suggests that for ACEi use and increased doses, there may be a trend upwards. However the latest data included here are restricted by the length of time required to analyse and publish. Limited data are available on β-blockers given their recent introduction to HF management.

3.3.3.1 Examples of prescription rates

The following examples are taken from primary care (except where comparisons with specialist prescribing are made) and presented in chronological order where possible to indicate how prescription rates have changed over time. Despite consistent beneficial outcomes described by ACEi trials, the following overview of ACEi prescription indicates that uptake was not immediate and has only slowly increased over time.

This overview indicates whether drugs that are new to the management of HF are being ‘normalised’ over time. Practitioners may or may not implement guidelines that have been updated to reinforce recommendations. The evidence of ACEi benefits in HF has been available since the 1990s. While changes in specific drugs may be available or subsidised, the general class of drugs (e.g. ACEi) will exist and substitutions can be made. Whatever variables are responsible for changes in prescribing patterns as can be seen in the data presented below, uptake of medication in primary care has been slow.
Comparisons between studies are complicated by the differing populations and methods used to determine diagnosis. Some studies have low response rates e.g. 18% (6), no date of data collection making it difficult to determine influences on prescribing (352), no definitions of diagnostic criteria or state that no formal validation of diagnosis was made (33, 36, 354). Other studies ensure that all patient participants have received echocardiography. (353)

**GENERAL PRESCRIBING:** In a survey between 1995 and 1998, 38% of primary care patients considered to have HF were taking an ACEi. (355) A Swedish study in primary care (late 1990s) found that 56% of HF patients received an ACEi. (31) The CASE study (260) reviewed the pharmacotherapy (data from 1998) received by newly diagnosed HF patients and found that 51% were prescribed an ACEi and 8% β-blockers (Carvedilol was specialist-only prescribing at that time). Prescriptions of HF medications to CASE study patients diagnosed with LVSD by echocardiography and of all HF patients were compared as seen in the table below derived from Box 5 of the CASE study.(260)

Table 3.3.d. Prescription of HF drugs by diagnosis group

<table>
<thead>
<tr>
<th>Drug</th>
<th>LVSD by echocardiography (n = 932)</th>
<th>All patients (N = 2905)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi</td>
<td>70.7 %</td>
<td>58.1 %</td>
</tr>
<tr>
<td>Angiotensin II antagonist</td>
<td>6.4 %</td>
<td>4.3 %</td>
</tr>
<tr>
<td>β-blocker &lt;50% carvedilol</td>
<td>13.9 %</td>
<td>11.8 %</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>6.9 %</td>
<td>8.1 %</td>
</tr>
</tbody>
</table>

As can be seen from the above table, the patients who had HF confirmed by echocardiogram had a higher prescription of angiotensin inhibiting agents (difference not tested). The beneficial effect of echocardiography on the management of HF using ACEI has been described in the Echocardiography section of this chapter (3.2.6). This effect was also seen in a study of HF in primary care where 54% of patients who had received a diagnosis confirmed by echocardiogram received ACEi. (7) A link between confirmation of diagnosis, use of ACEi and knowledge of HF was seen in another study.
in which the few GPs who referred suspected HF patients for echocardiography were more likely to report ACEi use and be aware of the poor prognosis of HF. (11)

Three pan-European studies have attempted to determine how HF is managed. The IMPROVEMENT survey (data from end of 1999 to mid 2000) found that 60% of patients were taking ACEi and that ARBs were prescribed to 5% of patients. A further 6% of patients had been initiated on an ACEi which was subsequently withdrawn. 34% of patients were currently treated with β-blockers, and an additional 5% had attempted β-blocker therapy. Only 20% of patients were prescribed an ACEi and β-blocker in combination. (32)

GPs participating in the Euro-HF study (pre 2002) were asked to estimate the percentage of their HF patients who were prescribed common HF drugs. ACEi use was estimated by GPs as 47 to 62%. When medical records were evaluated, actual prescribing of ACEi ranged from 25 to 43%, with some countries exhibiting a small gap between believed and actual practice (~ 10%) and others a large gap (36%). β-blockers were not commonly used – overall 6.2% but over a range from 2 to 14%. (35)

A later survey (SHAPE) surveyed doctors in 2002 to 2003. (34) There was a low response rate of 13% from primary care physicians, and the survey was not validated by an audit. (34) 43% of GPs said they always used an ACEi in HF, 51% said often. β-blockers were lower priority as only 5% said that they would always prescribe a β-blocker, 35% said ‘often’, 48% said ‘occasionally’ and 11% said ‘never’. There seemed to be a reluctance to prescribe β-blockers to patients with mild HF and taking an ACEi and diuretic, with only 35% of GPs saying they would initiate a β-blocker in these patients. (34)

Primary care database data (2000 to 2005) indicate that HF-specific β-blocker use (age-adjusted) has increased over time from 6.2% to 27.0% in men and 4.2% to 21.5% in women. (33)

More current data (2005 to 2006) indicate 61.3% ACEi/ARB prescription, 54.6% β-blocker prescription and 24.9% spironolactone prescription. (36) The Guideline Adherence Indicator (GAI: proportion of evidence-based recommendations followed by

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1 The IMPROVEMENT survey (32) reviewed 11 062 patient notes from 1363 GPs in 14 countries, all of which belonged to the European Society of Cardiology (ESC).
2 The Euro-HF study (35) included almost 300 GPs from six European countries that belonged to the ESC.
3 The SHAPE study (34) surveyed 2965 GPs in nine countries, who again all belonged to the ESC. SHAPE: Study group on HF Awareness and Perception in Europe.
the GP out of the total number of recommendations applicable to that patient) indicated that overall 53.3% of patients received optimal treatment, ACEi (all patients) 58.3%, β-blockers (HF-specific for patients with previous MI or NYHA class ≥II) 46.9%, and spironolactone (in patients with NYHA class ≥III). (36)

Analysis of Guideline Adherence (use of ACEi/ARB, β-blockers, spironolactone or equivalent) found that low guideline adherence (0 to 49% adherence) was determined by factors such as worsening renal function, age, and chronic respiratory disease. (353) High GAI (80 to 100%) was independently predictive of mortality risk compared with low GAI (HR 0.50, 0.32 to 0.74; p<0.001). The association was also seen in female patients (HR 0.42, 0.23 to 0.79, p=0.007) and older patients (HR 0.42, 0.27 to 0.66, p<0.001). (353) Treatment benefits of close adherence to guideline recommendations have been demonstrated in older and female patients.

Clear survival benefits have been demonstrated for prescribing according to HF guideline recommendations. Despite HF guidelines being written and distributed in the countries represented above, the change in management over time has been slow. Patients’ suitability for a particular drug does not guarantee it will be prescribed to them.

The aims of the UK’s GMS and QOF to increase HF prescribing in primary care have been effective.⁹ Reviews carried out in 2003/2004⁹ and in 2005/2006 around the National Service Framework showed differences in the management of HF. The 2003/2004 figures for ACEi/ARB prescription for confirmed HF were less than 50% but rose in 2005/2006 to a national rate of 82.5% (local variations for PCTs⁴ ranged from just over 90% to just under 80% and for practices from 100% to around 50%). The use of β-blockers was not included as one of the indicators. (303)

**INFLUENCES ON PRESCRIBING:** Age also had an effect on treatment. While any difference in prescribing for patients <70 years was only in ACEi, when prescriptions for patients aged ≥70 years were evaluated, the significant differences due to age were apparent for ACEi, β-blockers and spironolactone. (252) Similarly, a prescription

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⁹ The Quality and Outcomes Framework is part of the new primary care contract. Specified clinical targets have been developed and redefined to improve the quality of chronic disease management by way of financial incentives. (33)

⁷ The National Institute for Clinical Excellence (NICE) released their guidelines to the management of chronic heart failure in adults in primary and secondary care in July 2003.

⁴ PCTs stands for Primary Care Trusts.
review found that patients aged >77 years were significantly less likely to be prescribed ACEi/ARBs, β-blockers or spironolactone (or equivalent). (352) Extending the reference age to ≥85 years reveals that all other 5-year age bands had significantly higher odds of receiving β-blockers. (33)

There may also be prescription differences due to sex with female patients receiving significantly fewer prescriptions for ACEi and β-blockers than male patients (352) or having significantly lower odds of being prescribed β-blockers. (33)

One study found that patients with COPD were significantly less likely to be prescribed β-blockers (352) and a further study found that those with asthma and/or COPD received significantly fewer prescriptions for β-blockers (6) or were significantly less likely to be prescribed β-blockers. (33)

Compared with patients who had never received echocardiography, patients who had were almost twice as likely to be prescribed β-blockers (OR 1.70, 1.49 to 1.95). (33) Length of time since diagnosis of HF also reduced the odds of receiving a β-blocker. (33)

**COMPARISON WITH SECONDARY CARE:** Despite the uncertain extent to which GP prescribing for HF can be compared with cardiologists’ prescribing for HF, given the differing HF populations they see, there are still significant differences in prescribing rates. In a review of medical records, conducted in 2000, the differences between GPs’ and cardiologists’ prescribing were strongly significant with ACEi (40% vs. 76%), β-blockers (8.7% vs. 30%) and spironolactone (11% vs. 32%), all p<0.001. (252) Similar differences were seen between Hospital and GP prescribing of ACEi (40.4% vs. 23.5%, p<0.01), ACEi or ARBs (68.7% vs. 50.6%, p<0.01) and β-blockers (38.3% vs. 17.5% p<0.01) in a moderately large study (date of data collection not supplied). (253) Higher levels of guideline adherence were also seen in patients treated mainly by cardiologists compared with GPs (GAI 77% vs. 60%, p=0.01). (353)

GPs were seen to initiate medication less often: 29% for ACEi, 47% for ARBs and 37% for β-blockers. (6)

**MID-WAY CLINIC:** If GPs are not confident in management, a clinic mid-way between primary and secondary care, run by a GP specialist in HF found that it was
possible to use high levels of ACEi and β-blockers with 95% of patients with LVSD prescribed an ACEi or an ARB, of which 74% were taking target doses. β-blockers were prescribed to 70% of patients, with 63% of those patients able to take target doses. Patients who were not prescribed either of these drugs were either intolerant or had contraindications. Spironolactone prescriptions were limited to 8% with many patients discontinuing due to side effects. (279) Despite these good results, a mid-way clinic could be expensive to set up and maintain, notwithstanding the very real potential for overload. It also removes the management of HF from the domain of the GP and does not encourage any ownership of the condition in primary care.

**ACE INHIBITOR PERCEPTIONS:** The most likely reasons for the low use of ACEi and the use of low doses are the concerns over possible side effects, e.g. cough, postural hypotension, renal failure. (9, 10, 12, 203) In one study 51% of GPs thought ACEi had substantial risks and 62% thought ACEi had substantial side effects. (35) Another study found that a third of GPs thought that ACEi were a low risk treatment. (20) In a case study of typical HF presentation, fewer than 30% of GPs said they would start an ACEi. (20)

Studies reported that GPs believed an increased workload was involved in initiating, titrating and monitoring ACEi. (9, 11, 20) There may be lack of awareness of recommended practice or ignorance of benefits of ACEi to the extent that some GPs find it difficult to translate trial findings to their patients, and believed that some cases of HF did not warrant the use of ACEi. (10, 20) Some GPs believed that other medications were effective in controlling symptoms, that loop diuretics were sufficient for patients with mild HF, and that ACEi should be held in reserve until patients deteriorate. There was an unwillingness to add an ACEi if patients were well controlled on a diuretic. (9-12) These statements indicate a lack of awareness of the benefits of ACEi.

But some GPs believe that the evidence for ACEi prescribing is very good and that ACEi are generally well tolerated. (10, 20, 35) One study reported that 90% of GPs thought that treating HF could change prognosis and 60% thought ACEi were highly beneficial with another study reporting that 91% of GPs believed that ACEi could reduce mortality. (10, 20, 35) 76% of the GPs in one study thought that the benefits of ACEi outweighed the difficulties of initiation. (20)
GPs may also be reluctant to use ACEi if they question the diagnosis of HF e.g. because of lack of echocardiography. (9, 11, 12) Patient factors that were described as barriers to ACEi use included age, comorbidities and polypharmacy. (9, 11, 12) Polypharmacy carries the greater risk of drug interactions, with GPs mindful of minimising medications if a patient has been already prescribed multiple medications. (10, 12) Patients may also resist the addition of further medications or increased doses. (10, 12) GPs admitted to being more proactive with diagnosis and treatment in younger HF patients. (12) It was also mentioned that hospitals and specialists should ‘lead by example’ and promote the value of ACEi. (10)

As suggested in table 3.2.i, barriers to implementing evidence-based medicine can be categorised into theoretical domains. As seen above, knowledge of ACEi benefits is required, as is skill in prescribing. These two domains link closely with beliefs about consequences and capabilities (both immediate beliefs and consequences e.g. side effects and the ability to avoid these, and long term beliefs and consequences e.g. decrease mortality and morbidity). Given that ACEi are established as therapy for HF, prescribing ACEi for HF may fall under social/professional role and identity, motivation and goals, behavioural regulation, nature of the behaviour (routine) and social influences. Memory, attention and decision processes may not be pertinent if the behaviour of prescribing ACEi is normal practice. Emotion may have less influence on prescribing an ACEi, and environmental context and resources will not be relevant.

**β-BLOCKER PERCEPTIONS:** Few of the studies included were conducted late enough to incorporate information about the prescription of β-blockers. The studies indicated that GPs were not aware that β-blockers should be used; they were unfamiliar with the evidence and said that they did not have confidence in applying the findings or did not have experience in β-blocker use. (10). GPs also recalled that their medical training contradicted the new evidence and recommendations for β-blockers in HF. (10, 12) Factors that limited the use of β-blockers were described as the possibility of side effects, difficulties with comorbidities, polypharmacy, and contraindications (e.g. asthma, DM). (10) A majority of GPs have indicated concern about prescribing β-blockers to patients who had bradycardia or COPD/asthma, but age was not seen to be a contraindication. (34) β-blockers were viewed by some GPs as hospital-only initiated (12), although a letter to the editor, written in 2002, would have startled many GPs
when it recounted a study of initiating β-blockers in 264 mild-to-moderate HF patients, which concluded that β-blockers could be safely initiated at home. (356)

Introduction of β-blockers into treatment strategies for HF challenges accepted wisdom and threatens trust in the published evidence as the new recommendation is inconsistent with previous knowledge or ‘accepted wisdom’ of not using β-blockers in HF. (24) Reversing trends in prescribing can be difficult. (24) In this context the potential influences on prescribing β-blockers are complex. The knowledge of use of β-blockers in HF is new as they were previously contraindicated, and the nature of behaviour to prescribe them goes against previous behaviours. These two domains are connected to beliefs about consequences – β-blockers are known to worsen HF, which could still occur even with the newer HF-specific formulations. This in turn leads into skills and beliefs about capabilities – being able to identify suitable patients and the ability to manage adverse events. Social/professional role and identity and social norms may not apply to innovators (see Chapter 2, sections 3.4 and 3.5) who are comfortable in implementing new knowledge and are confident in their skills and beliefs about capabilities and consequences. This situation may be accompanied by positive emotions. Behavioural regulation and motivation and goals may not be seen as relevant by some GPs if they believe that β-blockers belong to secondary care management. However, reliance on this belief may conflict with social influence and social/professional role and identity (i.e. peer norms and messages given by opinion leaders e.g. that β-blockers can be initiated in primary care). This situation would probably be accompanied by negative emotions. For all GPs, addition of β-blockers is a new concept and action and will require memory, attention and decision processes.

SPIRONOLACTONE PERCEPTIONS: Knowledge about spironolactone has changed over the past few years from its use as a diuretic, at 100mg per day, to an adjunct to RAA inhibitor therapy at 25mg per day, and to patients who are in certain NYHA classification states. This change involves the nature of the behaviour and also memory, attention and decision process and may effect emotion. There is skill required to administer and monitor spironolactone (K⁺ and creatinine levels, effect of volume depletion). Combined with these two domains are beliefs about consequences and capabilities. Behavioural regulation and motivation and goals may not necessarily be immediately considered as time may be spent adjusting ACEi and diuretic doses and
GPs may not see patients at the stage when spironolactone could be prescribed – patients may have been admitted to hospital by this point, with spironolactone initiated in secondary care. GPs may not see prescribing spironolactone to these patients as part of their social/professional role and identity, and there may be few social influences.

**OTHER BARRIERS TO PRESCRIBING:** There is stress and confusion over rapidly changing management (12), as well as resource constraints in managing HF optimally resulting in a reluctance to refer, further complicated by the view that HF should be managed in primary care. (12) This leads back to the problem of a patient with suspected HF, without a definite diagnosis, whom GPs are hesitant to medicate for HF.

Issues around primary and secondary care communication were also described. The majority of GPs (83%) would have appreciated more information from the hospital about their patients e.g. why treatment had been initiated or altered. 83% of GPs also said that discharge letters often arrived after they had seen the patient. Just over half considered that discussions with specialists were more helpful than written guidelines. (20) This subject is critical for HF since it has been suggested that if secondary care has a major influence on treatment, then implementation of guidelines (or any evidence or recommendations) will need close collaboration with secondary care to succeed. (24)

**SUMMARY:** There is well documented evidence of the beneficial effects of ACEi use in HF, and for the careful initiation in almost all HF patients, but a significant amount of work still needs doing in primary care to provide sufficient coverage of the harmful effects of the RAAS in HF. Evidence is also widely available for the beneficial effects of β-blockers and spironolactone in selected cases of HF but this evidence is not necessarily translated into practice. As demonstrated above (and in 3.1.6.2), many theoretical domains may need targeting to improve understanding and prescribing. However these differ between the outcomes and may depend on how GPs approach new knowledge.
3.3.3.2 Examples of doses prescribed

Evidence from trials indicates that the best outcomes for patients result from high doses of ACEi but data from primary care suggest that these doses are not achieved. Examples can be seen in table 3.3.e. (6, 36, 355)

Table 3.3.e. Proportion of prescriptions at target doses

<table>
<thead>
<tr>
<th>Study and time period</th>
<th>ACEi</th>
<th>β-blocker</th>
</tr>
</thead>
<tbody>
<tr>
<td>1995 to 1998 (355)</td>
<td>18%</td>
<td>–</td>
</tr>
<tr>
<td>2004 to 2006 (6)</td>
<td>37.5%</td>
<td>10.5%</td>
</tr>
<tr>
<td>2005 to 2006 (36)</td>
<td>21.7%</td>
<td>12%</td>
</tr>
</tbody>
</table>

The differences may be due to the effect of time, with GPs becoming more confident in prescribing higher ACEi doses, but there may also be differences in patient demographics between the studies, the amount of management in secondary care, and reporting or registry requirements.

Similarly the researchers in the CASE study found that 60% of ACEi prescriptions were low doses, 31% were medium doses, and 9% were high doses. (260) They then compared the prescribed daily doses of the ACEi (see table 3.3.f). (260)

Table 3.3.f. Mean, median and recommended doses of ACE from the CASE study

<table>
<thead>
<tr>
<th>ACEi</th>
<th>Daily doses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Captopril</td>
<td>50 mg</td>
</tr>
<tr>
<td>Enalapril</td>
<td>10 mg</td>
</tr>
<tr>
<td>Quinapril</td>
<td>10 mg</td>
</tr>
</tbody>
</table>

* The mean doses prescribed represent half to one third and the median doses half to two thirds of the recommended daily doses.

There may even be differences in dose prescription due to the sex of a patient. A study of Swedish primary care patients (late 1990s) found that although overall 52% of patients received an optimal dose of ACEi, 44% of women but 56% of male patients were on maximal dose. (31)

Studies comparing GPs and cardiologists dose prescriptions also differ. There was no difference between the proportions of GPs’ and cardiologists’ patients receiving
maximal ACEi as used in the trials (32% vs. 44%, p=0.20). But another study found higher ACEi target doses for those diagnosed in secondary care (29.4% vs. 14.3%, p=0.03). Confirmation of diagnosis by echocardiogram and in secondary care also increase the likelihood that target doses will be reached. However ACEi/ARBs dose decreases were estimated to start at age 55 years (adjusted for sex, management in primary or secondary care, NYHA class, and setting of first diagnosis).

**BARRIERS TO TITRATION:** Reasons given by GPs for prescribing low doses of ACEi include concern over whether the diagnosis was correct, possible side effects associated with up-titration, lack of awareness of recommended practice or target doses, ignorance of benefits and lack of confidence in up-titrating. If low doses were seen to be controlling HF, it was thought unnecessary to increase the dose, and that any change in regimen may destabilise a stable condition. GPs were also mindful of minimising medications if patients had multiple drugs, and of patient reluctance to increased doses. It is unfortunate that these beliefs prevail when the evidence of benefit is for high dose ACEi and that either electrolyte imbalance or patient reported side effects should guide any decrease in dose.

**SUMMARY:** There are still serious shortfalls in the knowledge of ACEi that would be expected in GPs. An unwillingness to initiate ACEi in HF patients may be associated with the lack of positive diagnosis in some cases. The new evidence to prescribe β-blockers to HF patients is contrary to the education that most currently practising GPs previously received which presents a cognitive dissonance. The recent changes in concepts of HF management have left many GPs confused. Increased input from secondary care may assist primary care in improving management for HF patients.

### 3.3.3.3 Effect of age on treatment

Given the rise in prevalence and incidence of HF with age (incidence in men rises 9-fold annually between the sixth and ninth decades and 11-fold in women), age itself is a major risk factor for HF. There are age-related changes in the cardiovascular system and the pathophysiological changes (of disease) are overlaid on the age-related changes.
Additional factors to consider are the interaction of age, disease and lifestyle, and the genetic components of these. (357) Older patients are more likely to have renal dysfunction, renal artery stenosis and orthostatic hypotension. There may be increased risk of electrolyte disturbance, worsening renal function and hypotension, which need to be taken into account when initiating ACEi and during the entire period ACEi are prescribed. (358)

When prescribing β-blockers, greater consideration may need to be given to several influencing factors. These may include lower doses and a longer titration period in older patients, resting HR, low SBP, level of heart block, active bronchospasm, severe COPD, and HF that is decompensated or insufficiently diuresed. (358)

Diuretics pose another challenge as older patients are at greater risk of diuretic-induced renal insufficiency and electrolyte imbalance. Despite the symptomatic relief that they bring about, diuretics have not been shown to confer any mortality benefits and can activate the renin-angiotensin system. (358) Spironolactone is the exception as it does confer a mortality benefit but caution is needed in impaired renal function and in monitoring potassium levels. (358)

Over and above the medications prescribed for HF, older patients usually have multiple medications and the aim should be to simplify the regimen and dosing schedule, reviewing the need for all the medications and monitoring for adverse events of drug interactions. (358)

Although cardiovascular diseases are more common in the elderly and they have a high likelihood of death and disability from them, investigations are more likely to be performed in younger patients who also have higher rates of intervention. (359) This finding invites the question of whether policies in primary or secondary care that are age-based are due to rationing or prejudice. If resources are rationed, older patients may not qualify if they are considered to be more expensive to treat, or have a shorter life expectancy which would suggest that a younger patient would receive more benefit. (359)

**APPLICABLE TRIAL DATA:** Data on older patients may only be available from small trials and cohort studies. (360) Age limits have been used in clinical trials (see 3.3.2) as cut-off points to minimise analytical problems caused by comorbidities and related medications (adverse events and potential interactions with study drugs) and the greater
likelihood that patients would be lost to follow-up due to death. (359, 360) A smaller proportion of women tend to be included in clinical trials (see 3.3.2) and this research bias may influence GPs to be wary of treating older patients, especially older women. (359)

Differential treatment for male and female patients has been demonstrated in a number of studies. In a study of primary care patients in Sweden (late 1990s), 43% of female HF patients were prescribed ACEi compared with 64% of male HF patients (p<0.001). (31) A study of UK general practice (mid-1990s) found that compared with men, the OR for female HF patients being prescribed an ACEi was 0.7 (95% CI 0.5 to 0.9). (361) Studies have assessed the effect of age and prescriptions and tolerability of doses in older patients. One study (362) measured outcomes of survival, survival or HF hospitalisation and survival or all-cause re-hospitalisation over one year of follow up and compared these by dose of ACEi. These outcomes were assessed in 16539 patients \( \geq 66 \) years who survived 45 days post-discharge following their first HF hospitalisation. However in this cohort study in the United States, the effect was unclear for hospital, health insurance and/or socioeconomic status, attending doctor or medications for conditions other than HF. At 45 days 65% of patients were taking an ACEi, and the table below indicates the doses prescribed and titrations made (titration up to ‘high dose’). (362)

Table 3.3.g. Doses prescribed and proportion titrated to higher doses

<table>
<thead>
<tr>
<th>Doses</th>
<th>Prescription and titration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prescribed</td>
</tr>
<tr>
<td>Low (( \leq 25% ) of trial dose(^a))</td>
<td>36.5%</td>
</tr>
<tr>
<td>Medium (( &gt;25% ) to 99% of trial dose)</td>
<td>40%</td>
</tr>
<tr>
<td>High (trial dose or higher)</td>
<td>23.5%</td>
</tr>
</tbody>
</table>

\(^a\) 80\% of the high dose patients remained on this dose over 12 months. (362)

Using the low dose patients as the reference group, the study showed that non-prescription of ACEi was associated with a higher risk of death, and death or HF hospitalisation. The benefits of taking medium dose ACEi were similar to those for low

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\(^1\) The mean age of the sample was 79.1 years (± 7.4 years); over 50\% were women; 91\% had high comorbidity scores; 21\% had a history of hyperkalaemia, hypotension or renal impairment. Age, gender, comorbidities and use of other medications for HF were controlled for in analysis. (362)

\(^a\) The high dose was determined by trial data such as CONSENSUS, SOLVD, Captopril-Digoxin Multicenter Research Group, and doses suggested by the AHRQ for the management of HF.
High dose ACEi provided clear benefits and the comparison of no ACEi with high dose is shown in the table below adapted from table 3 of the reference. (362)

Table 3.3.h. Comparison of outcomes for no ACEi and high dose ACEi

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Hazard Ratio (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No ACEi</td>
</tr>
<tr>
<td>Death</td>
<td>1.12 (1.02 – 1.22)</td>
</tr>
<tr>
<td>Death / HF hospitalisation</td>
<td>1.08 (1.00 – 1.16)</td>
</tr>
<tr>
<td>Death / all hospitalisation</td>
<td>1.04 (0.98 – 1.10)</td>
</tr>
</tbody>
</table>

However with increased doses came an increased risk of dose reduction (≥50%) or discontinuation of ACEi (see table 3.3.i below). (362)

Table 3.3.i. Dose scale decreases or discontinuations for ACEi

<table>
<thead>
<tr>
<th>Dose decrease or discontinuation</th>
<th>Low dose</th>
<th>Medium dose</th>
<th>High dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of patients</td>
<td>13%</td>
<td>19%</td>
<td>25%</td>
</tr>
<tr>
<td>OR (95% CI)</td>
<td>1</td>
<td>1.57 (1.39 – 1.77)*</td>
<td>2.36 (2.07 – 2.69)*</td>
</tr>
</tbody>
</table>

* p<0.001

Low dose ACEi conferred some benefit over no ACEi, but clear benefits were seen for high dose ACEi, in patients who were older and disproportionately female, in a community-based population. (See also the study of GAI in 3.3.3.1 under General Prescribing.) The significant benefits conferred by a high dose of ACEi should encourage clinicians to attempt up-titration. (362)

It could be argued that older patients may have a greater absolute gain from treatments due to a greater baseline risk of morbidity and condition-related mortality. And while adverse events may occur more often in older patients this risk is balanced by the greater absolute benefit. Unless there are convincing biological reasons not to treat, it is reasonable to assume that treatment will benefit an older patient unless proven otherwise. However, this assumption does not discard the need for and importance of individual clinical judgement in managing these patients. It may not be unreasonable to suggest that a ‘default approach’ should be to assume that treatments do have an effect even when specific outcome data are lacking for older patients. (363)
SUMMARY: Elderly patients benefit from medical advances and there is a need for information for health professionals so they can prescribe appropriately for these patients. (360) It is highly likely that an older HF patient will be hospitalised and there is a responsibility on secondary care to establish diagnoses and initiate optimal management in all patients in the absence of contraindications. GPs need to be aware of the physiological changes that occur with age but not assume that all patients of a certain age will be affected. They also need to establish all potential morbidities (that become more likely as patients age) in order to determine clear treatment objectives.

3.3.4 Secondary Care

Many patients with HF will eventually be admitted to hospital with HF as a primary or secondary cause of admission. In older patients HF counts for more hospital admissions than any other one condition with approximately 50% being readmitted within six months. (357) Suboptimal use of evidence-based treatments and the consequent repeated readmissions can lead to increased dependency and institutionalisation in older people. (364)

Hospitals possess diagnostic equipment that GPs have difficulty accessing and patients will be reviewed by specialists in their field who have expertise in managing the particular conditions. Thus patients may even benefit from a hospital admission that confirms diagnoses, prescribes appropriate medication and sets ongoing management plans. The medication regimen set out in discharge summaries tends to be unaltered by GPs.

The following section contains a brief review of the hospital management of HF and the attitudes of specialists toward the management of HF. The survey data presented may be affected by respondent, recall and selection biases.

3.3.4.1 Diagnosis

Hospitalisation has been described as a key event that provides an opportunity to clarify diagnosis and optimise therapy. (365) This said, the first step would be to assess

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1 Secondary care is used here to describe hospital-based management, generally including admission to a ward.
ventricular function but as table 3.3.j indicates, this cannot be assumed. Examples include one study (300) where 74% (n=209) of the identified primary care HF patients had been admitted to hospital and just over half of them had been admitted acutely. Other diagnostic tests were performed frequently (ECG in 80% of patients, CXR in 75%). (300) Another study increased hospital use of echocardiography by implementing an in-hospital best practice in-patient management protocol (patients ≥70 years). (364) The large EuroHeart Failure survey \(^\text{\textsuperscript{a}}\) (365, 366) evaluated diagnosis and treatment in hospitalised HF patients (24 countries and 115 hospitals involved). (365) The EPISERVE study surveyed cardiologists, internal medicine specialists and primary care physicians. (302) More data and comparisons with primary care can be found in 3.2.6.1.

Table 3.3.j. Comparison of echocardiography rates in suspected HF patients admitted to hospital

<table>
<thead>
<tr>
<th>Study</th>
<th>Echocardiograph</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early 1990s (300)</td>
<td>31%</td>
</tr>
<tr>
<td>Late 1990s ‘before’ and ‘after’ (364)</td>
<td>42% vs. 62% (P=0.041)</td>
</tr>
<tr>
<td>Early 2000s (365)</td>
<td>67%</td>
</tr>
<tr>
<td></td>
<td>Range across countries 41% – 94%</td>
</tr>
<tr>
<td>2005 (302)</td>
<td>66% (internal medicine)</td>
</tr>
<tr>
<td></td>
<td>87% (cardiology)</td>
</tr>
</tbody>
</table>

An optimal figure for assessment of suspected HF patients would be 100% receiving echocardiography during admission to hospital. Many consultants expressed concerns over the use of echocardiography reporting and interpretation for two reasons: that interpretation could be subjective and that EF measurements tended to be a “one-off”. (367) Measuring parameters only once is not done anywhere else in medicine. (367)

**EFFECT OF AGE AND SEX ON DIAGNOSIS:** Differences in diagnosis due to sex were seen in patients admitted to hospital with HF – 55% of female and 68% of male patients received echocardiography (p<0.011). (301) This study population was 51%.

\(^\text{\textsuperscript{a}}\) The EuroHeart Failure survey was conducted in 115 hospitals in 24 ESC countries and evaluated diagnosis and treatment for over 11,000 patients over a six week period in 2000 / 2001. (365, 366) The mean age of patients was 71 years, half were women and there more women aged >75 years (51%) than there were men (30%). (365)
female and 49% male, median age 78 years and all (n=379) had been admitted and discharged from one of two city hospitals in the second half of 1995. (301) Another survey reported similar findings – 58% of female and 63% of male patients (p<0.01) received echocardiograms. (302) This study reported similar patient demographics (55% male, 45% female, mean age 72.3 years) despite its much larger sample (n=2249), however physician participation was only 16% One retrospective study of three Australian teaching hospitals (mid-1999 to mid-2001) included 438 HF patients, median age 79 years, 57% female. (368) The medical record review found that men and patients <80 years were more likely to have echocardiography recorded (both p<0.05). (368) Recording of NYHA in hospital records was sparse at <1%. (368)

Differences were also seen in discharge data. A higher proportion of males was discharged to the hospital outpatient clinic than to general practice (71% vs. 44%, p<0.001). (301) Patients discharged back to general practice were significantly older than in the outpatients clinic (p<0.001). (301) These findings on sex and age differences between primary and secondary care are not unique.

The age of the patient may influence referrals to a cardiologist, and evaluation of LVFx (see table 3.3.k). A significant inverse relationship between increasing age and investigation (referrals, and evaluation) was observed in a study of 1090 older HF patients\(^\text{\textsuperscript{y}}\). (369)

### Table 3.3.k. Comparison of investigation rates and patient age

<table>
<thead>
<tr>
<th>Investigation</th>
<th>65 to 74 yrs</th>
<th>75 to 84 yrs</th>
<th>≥85 yrs</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Referral to a cardiologist</td>
<td>36%</td>
<td>31%</td>
<td>24%</td>
<td>0.008</td>
</tr>
<tr>
<td>LVFx evaluation</td>
<td>66%</td>
<td>57%</td>
<td>51%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Patients aged ≥85 years with new onset HF (not taking an ACEi) had an OR of 0.54 (0.24 to 1.22) of having LVFx evaluated, compared with patients aged 65 to 74 years. (369) The younger patients were more likely to experience ‘common’ symptoms such

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\(^\text{\textsuperscript{y}}\) For this study, the patient population consisted of 1090 hospitalised patients (Medicare), 60% of whom were women. The mean age of the patient population was 79 ± 7.5 years. Overall LVFx evaluation was 58%. (369)

HF diagnosis was confirmed by one of the following: 1. History of HF; 2. Symptoms (dyspnoea at rest, dyspnoea on exertion, orthopnoea, PND); 3. Signs (jugular venous distention, 3\(^\text{rd}\) heart sound, displaced point of maximal cardiac impulse, or pulmonary râles); 4. Radiographic evidence (cardiomegaly, pulmonary venous congestion or pulmonary oedema); or 5. Treatment with digoxin and diuretics. (369)
as dyspnoea on exertion, orthopnoea and PND. In turn, these symptoms were associated with higher odds of receiving LVFx evaluation. (369)

**EFFECT OF DIAGNOSIS ON TREATMENT:** Echocardiography made a difference to ACEi prescription with higher ACEi seen on admission for patients with documented EF (p<0.05) and also on discharge (p<0.01). (368) The number of echocardiograms performed during the hospital stay was not documented. Male and female patients discharged with HF did not show any difference in rates of ACEi prescription (38%). (301) If their HF diagnosis was confirmed by echocardiography 72% of male patients but only 55% of female patients received an ACEi (no comparison). (301)

### 3.3.4.2 Treatment prescription and dose

Hospitalisation has been described as a critical point of care where diagnosis and management are most likely to be revised. (370) Specialists have the opportunity to optimise therapy, but as illustrated below, while ACEi prescription seems to be generally good, doses can often be low, and β-blocker prescription is variable. Patient variables of age and sex are also seen to influence prescriptions and doses.

A lack of confidence and reluctance to initiate therapy was even noted in consultants. Whether patients received evidence-based medicine was in part due to consultant experience and ‘interest’ in HF. (367) The barriers to implementation of evidence-based HF treatment included concerns about using it in older and frail patients, co-morbidities e.g. COPD and β-blocker use, drug interactions and polypharmacy. (367) The lack of understanding of trial evidence of medication benefits in older patients (16, 18, 371-374) and potential to prescribe β-blockers in patients with respiratory conditions (375, 376) is concerning. The admission of a patient to a ward, as stated above is an opportunity to revise medication regimens in a controlled environment.

**ANGIOTENSIN INHIBITION:** After referral to hospital with suspected HF, 31% of patients in one study were offered echocardiography but only 17% of confirmed HF patients were offered ACEi*. (300) The data below (table 3.3.1) present hospital

* These figures suggest the ‘rule of halves’ which describes the process in which half the task is completed at each management phase. (377) For example: “... approximately half of most common
prescription rates of angiotensin inhibition and are taken from a range of studies: acute hospital admissions for HF (278), evaluation of 17456 patients, mean age 78 years, hospitalised for HF with no contraindications to ACEi (381), 157 “closely monitored” patients i.e. seen by two consultants in a specialist clinic for HF (382), the EuroHeart Failure survey of 115 hospitals in 24 countries (366), an audit of a University teaching hospital (383) and a retrospective study of three teaching hospitals (368)

Table 3.3.1.  Hospital prescription rates on discharge of ACEi and/or ARBs

<table>
<thead>
<tr>
<th>Study</th>
<th>ACEi</th>
<th>ARB ± ACEi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute admissions (278)</td>
<td>51%</td>
<td></td>
</tr>
<tr>
<td>Closely monitored patients (382)</td>
<td>73%</td>
<td></td>
</tr>
<tr>
<td>Patients ≥65 yrs, EF&lt;40% or ‘moderate or severe LVSD’ (381)</td>
<td>68%</td>
<td>ACEi or ARB: 78%</td>
</tr>
<tr>
<td>Audit of medical records (383)</td>
<td></td>
<td>ACEi or ARB: 80%</td>
</tr>
<tr>
<td>3 teaching hospitals (368)</td>
<td>62%</td>
<td>ARB: 8%</td>
</tr>
<tr>
<td>EuroHeart Failure survey (366)</td>
<td>62% (range 40 – 85%)</td>
<td>ARB: 4.5% (range 2 – 14%)</td>
</tr>
</tbody>
</table>

Data from the three teaching hospitals indicate that the admission period was utilised with higher ACEi prescription on discharge (p<0.001), but not higher ACEi doses. (368) These admission/discharge comparative data are useful, and more hospital data should be presented in this manner.

Prescription rates of ACEi/ARBs did not change with number of hospitalisations unlike β-blockers and spironolactone (see below). (383) This runs contrary to the opportunity provided by hospitalisation to optimise medications, and also to the evidence of ACEi/ARBs reducing hospitalisations.

An in-hospital best practice in-patient management protocol (patients ≥70 years) improved prescription of ACEi to 44% up from 22% (p=0.016). (364) This demonstrates that change is possible even though the outcome could be higher.

chronic disorders are undetected, that half of those detected are not treated, and that half of those treated are not controlled” (377) It has been used to describe both diagnosis and treatment of HF. (215) This concept was developed in the 1960s, (377) and more recent data for HTN suggest that this has now become the ‘rule of two thirds’. (378-380)
A comparison of discharge destinations found that HF patients discharged to hospital outpatients’ clinics were more likely to be prescribed ACEi than those discharged back to primary care (76% vs. 53%, p<0.001). (301) This may be the foundation of the low rates see in primary care – GPs not adding medications if the hospital has not initiated them. Frequently, lack of prescription is not accompanied by explanation. This finding may also be associated with younger patients as well as more males patients discharged to outpatients’ clinics. (301)

**DOSE:** Even when ACEi are prescribed, the doses are likely to be lower than those recommended in clinical trials. U.S. hospital data (1998) showed 80% of enalapril prescriptions were <15mg, which is less than recommended dose (see table 3.3.m). (203)

The EuroHeart Failure study (countries belonged to the ESC) recorded daily dosages of ACEi (adapted from Table 4) (366) and compared these with doses recommended by the ESC 2001 HF guidelines (table 3.3.m). (76)

Table 3.3.m. EuroHeart Failure study ACEi daily doses and ESC 2001 HF guidelines recommended doses

<table>
<thead>
<tr>
<th>Drug</th>
<th>EuroHeart Failure</th>
<th>ESC guideline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>57.6 ± 37.1 mg/day</td>
<td>75 – 150 mg/day</td>
</tr>
<tr>
<td>Enalapril</td>
<td>14.3 ± 9.1 mg/day</td>
<td>20 mg/day</td>
</tr>
</tbody>
</table>

Another small study that looked at closely monitored patients (n = 157 seen by 2 consultants in a specialist clinic for chronic HF) found 54% of patients were taking lower than optimal doses (suggested by clinical trials). (382) Only 18% of patients were taking ACEi doses that were of benefit to improve mortality (the dose of ACEi was standardised to the lowest target dose shown to improve mortality). (382)

An in-hospital best practice in-patient management protocol (patients ≥70 years) had little effect in maximising ACEi by increasing prescriptions of maximal ACEi doses from 26% to 29%. (364)

**SEVERITY OF HF:** Patients with more severe HF (mean LVEF 26.0% ± 12.7%) were prescribed more ACEi than those with moderate HF (34.6% ± 18.6%, 95% CI for difference; 5.84 to 11.46; p<0.01). (382) Similarly, the EuroHeart Failure study found that 80% of patients with EF <40% received ACEi but only 63% of patients with EF ≥40%. (366) This finding of an apparent increased prescription in more severe HF may
be confounded by the ESC guidelines. The 2001 guideline states that EF≥40 to 45% (p 1532) is preserved LVSFx, and under Pharmacological therapy (p1540) that ACEi are recommended as first line agents for reduced LVSFx, i.e. subnormal EF <40 to 45%. (76) This pattern was repeated in patients with severe LVSD (EF<30%) who were more likely than those with moderate LVSD to receive ACEi or ARB (RR = 1.07, 1.05 to 1.09, p<0.0001)\textsuperscript{x}. (381) This was a highly selected American cohort (see footnote) who were mostly treated by general physicians in not-for-profit hospitals. (381). Patient, physician and hospital characteristics were included in models but the potential misclassifications of diagnosis and therapy are not known. (381)

Clinical variables did not seem to influence the dose or usage of ACEi. Patients in a specialist clinic for HF taking ACEi had a lower mean serum sodium concentration, and higher mean urea and creatinine concentrations. (382) A trend (i.e. not significant difference) to increased benefit from ACEi was seen with increasing creatinine levels. (384) And hospitalised patients with moderate creatinine levels (>120μmol/L) were just as likely to be prescribed ACEi as those with lower creatinine levels. (368)

**β-BLOCKER:** The EuroHeart Failure survey found an average β-blocker prescription of 37%, with a range from 10 to 66% in the participating countries. (366) Not all β-blockers were those specifically indicated in HF i.e. had no trial evidence for benefit in HF. Almost a quarter of β-blocker prescriptions were for Atenolol. (366) In a teaching hospital 32% of patients (out of 400 who had a primary diagnosis of HF, record review mid-1999 to early 2001) were prescribed β-blockers. (383) Only carvedilol was available as a HF-specific β-blocker but while the study refers to β-blockers it does not specify which. Another moderate size study (<400 patients) indicated that 31% received β-blockers. (301) This study was conducted in late 1995, so for a relatively early study the β-blocker prescription rate seems quite high. But the study does not define which β-blockers were prescribed. Differences in prescription were seen in discharge destination. HF patients discharged to the hospital outpatients’ clinic were more likely to be prescribed β-blockers than those discharged back to primary care (51% vs. 31%, p<0.003). (301) While the consultants presumably understood the

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\textsuperscript{x} The study consisted of 17 456 patients, ≥65 years who had survived hospitalisation for HF (fee-for-service Medicare), with no documented contraindication to ACEi and were evaluated for ACEi prescription at discharge. Patients had a discharge diagnosis of HF. All had either documented LVSD (EF <0.40) or qualitative description of moderate or severe LVSD. Patient population mean age 78 years, 20% ≥85 years. (381)
evidence base for β-blockers, the majority were hesitant in prescribing them given that only a short while earlier they would have failed their membership for using β-blockers in HF. (367)

During an audit it was identified that β-blockers were less frequently prescribed with increasing numbers of hospitalisations for HF. (383)

Table 3.3.n. Prescription rates of β-blockers by hospitalisations

<table>
<thead>
<tr>
<th>β-blocker prescription</th>
<th>≥2 hospitalisations</th>
<th>1 hospitalisation</th>
<th>No hospitalisations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12%</td>
<td>26%*</td>
<td>36%*</td>
</tr>
</tbody>
</table>

* Comparison of ≥2 to 1 hospitalisation and 1 hospitalisation to no hospitalisation, p=0.001

DOSE: Doses recorded in the EuroHeart Failure study (adapted from Table 4) (366) indicated that β-blocker doses were below those recommended by the ESC 2001 HF guidelines. (76)

Table 3.3.o. EuroHeart Failure β-blocker daily doses and ESC 2001 HF guidelines recommended doses

<table>
<thead>
<tr>
<th>Drug</th>
<th>Daily dose</th>
<th>ESC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carvedilol</td>
<td>17.6 ± 16.6 mg/day</td>
<td>50 mg/day</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>74.9 ± 43.4 mg/day</td>
<td>150 mg/day (tartrate)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>200 mg/day (succinate)</td>
</tr>
</tbody>
</table>

CONTRAINDICATIONS: In the teaching hospital data, only 20% of all patients had indications of pulmonary disease or severe peripheral vascular disease so contraindication to β-blocker use could not be a reason for the low β-blocker use. (385) The reference guideline, Diagnosis and treatment of Heart Failure due to Left Ventricular Systolic Dysfunction 1999 SIGN, does not make any mention of contraindications to prescribing apart from clinically unstable or severely symptomatic patients. (71) Instead it contains references to other texts, which seems a cumbersome way of promoting knowledge. The prescribing restrictions were that only hospital physicians could initiate and titrate HF β-blockers (in this case carvedilol). (71) During an audit (383), researchers reported that it was rare to find reasons in patient case records for not using ACE inhibitors, β-blockers or spironolactone. This lack of information is then perpetuated in discharge letters which could perplex GPs regarding appropriate management. (This is covered in 2.4.8 Specialist – GP interaction.)
**SEVERITY:** A comparison of severity of HF (EF<40% vs. EF≥40%) and β-blocker prescription EuroHeart Failure study found that the two groups had similar levels of prescription of β-blockers. (366)

**SPIRONOLACTONE:** The EuroHeart Failure study found spironolactone prescription of 20.5%, with a range from 6% to 58.5% in the participating countries. (366) Teaching hospital data indicated that spironolactone use was at 13%. (383) This figure seems low given that patients are likely to be in a decompensated state on admission to hospital, however the table below provides an interesting insight. Physicians may have been reluctant to add spironolactone to an ACE inhibitor due to the risk of hyperkalaemia. (383) The uptake of spironolactone after the RALES trial was associated with higher numbers of hospitalisations related to hyperkalaemia and more hospital deaths. (386) The authors note some inappropriate prescribing: 7% of patients taking spironolactone not qualifying by NHYA criteria. (383)

Increased spironolactone prescription accompanied increased numbers of admissions. (383) This may be due to increasing severity of HF/time spent in higher NYHA categories of HF.

Table 3.3.p. Prescription rates of spironolactone by hospitalisation

<table>
<thead>
<tr>
<th>Spironolactone prescription</th>
<th>≥2 hospitalisations</th>
<th>1 hospitalisation</th>
<th>No hospitalisations</th>
</tr>
</thead>
<tbody>
<tr>
<td>24%</td>
<td>16%</td>
<td>5%</td>
<td></td>
</tr>
</tbody>
</table>

*Comparison of ≥2 to 1 hospitalisation and 1 hospitalisation to no hospitalisation, p<0.001*

**EFFECT OF AGE AND SEX ON PRESCRIPTIONS AND DOSES:** As GPs find it difficult to alter medication regimens specified by the hospital, any under-treatment on discharge will continue into primary care. Common factors tend to be older patients and female patients as seen in the following studies.

Female HF patients discharged from hospital with a primary diagnosis of HF (mid-1990s) were prescribed ACEi less often than men (46% vs. 60%, p=0.011). (301) Similar numbers were seen in ACEi data from three teaching hospitals (females 55% vs. males 71%; p<0.001). (368) and in the EuroHeart Failure data (see table 3.3.q derived from table 8, EuroHeart Failure reference). (366) ORs were calculated for receiving various medications based on certain patient and hospital variables. (366)
Table 3.3.q. EuroHeart Failure ORs for receiving HF medications

<table>
<thead>
<tr>
<th>Influencing variable</th>
<th>Prescription OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ACEi</td>
</tr>
<tr>
<td>Patients &gt;70 years</td>
<td>0.77 (0.70 – 0.85)</td>
</tr>
<tr>
<td>Males</td>
<td>1.34 (1.22 – 1.48)</td>
</tr>
<tr>
<td>IHD</td>
<td>2.45 (2.21 – 2.71)</td>
</tr>
<tr>
<td>Respiratory and pulmonary disease</td>
<td>NR</td>
</tr>
</tbody>
</table>

† Reference groups for each variable are: patients ≤70 years; females; no IHD; no respiratory or pulmonary disease.
NR: not reported

Similar to the EuroHeart Failure survey, higher ACEi prescription was recorded in younger (<80 years) than older (≥80 years) discharged patients (68% vs. 56%; p<0.05). (368) There was no difference in doses (51% vs. 47%; NS). This latter study (record review in three teaching hospitals) did not document co-morbidities or other medications.

A large cohort study (17456 patients, 53% female, 47% male, mean age 78 years) found no difference in discharge prescription of ACEi/ARB between males (76.7%) and females (76.3%, p=0.62), but male patients benefited more in reduction in risk of death compared with female patients (see table 3.3.r). (381) The study indicated mortality benefit for especially the oldest patients (95% CIs not specified for younger ages but all <1 on the forest plot), although the p-value for interaction was not significant. (381) See table 3.3.r derived from the cohort study forest plot. (381)

Table 3.3.r. Mortality associated with ACEi prescription

<table>
<thead>
<tr>
<th>Variable</th>
<th>1-year mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RR</td>
</tr>
<tr>
<td>Overall</td>
<td>0.86</td>
</tr>
<tr>
<td>65 – 74 yrs</td>
<td>0.88</td>
</tr>
<tr>
<td>75 – 84 yrs</td>
<td>0.87</td>
</tr>
<tr>
<td>85 yrs +</td>
<td>0.83</td>
</tr>
<tr>
<td>Male</td>
<td>0.80</td>
</tr>
<tr>
<td>Female</td>
<td>0.93</td>
</tr>
</tbody>
</table>

NR: not reported
β-blocker prescriptions differed by age group, from 15.2% for <65 years, 9.8% for 65 to 80 years, and 5.3% >80 years (p<0.01 among the three age groups). (387) This was also seen in the EuroHeart Failure study (366) (see table 3.3.q) but not in discharge data from three teaching hospitals (<80 years 25% vs. ≥80 years 18%, p=0.08). (368) Two moderate sized studies (379 patients and 438 patients) found no significant difference in β-blocker prescription between males and females discharged with HF (33% vs. 29%) (301) and 20% vs. 22% (368). No differences were seen for age groups or by documented EF. (368) Neither study specified which β-blockers were prescribed.

Similar to the spironolactone findings of the EuroHeart Failure survey (see table 3.3.q) in the three teaching hospitals review, younger patients were more likely to be prescribed spironolactone (36% vs. 24%; p<0.01) as were men (36% vs. 25%; p<0.05), but also those who had documented EF (42% vs 24%, p<0.01). (368)

### 3.3.4.3 Effect of care provider

The type of ward to which patients are admitted can also influence prescription. Studies tend to indicate that cardiology care is better, but a few studies show varied results.

*Cardiology:* In the EuroHeart Failure survey ACEi and β-blocker prescriptions were higher in cardiology wards than in general internal medicine wards whose patients were older and had higher levels of comorbidity. (366)

Table 3.3.s. Differences in prescription of ACEi and β-blocker by hospital ward

<table>
<thead>
<tr>
<th>Drug</th>
<th>Cardiology</th>
<th>General internal medicine</th>
<th>OR (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi</td>
<td>71.5%</td>
<td>56.4%</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>β-blocker</td>
<td>50.7%</td>
<td>26.3%</td>
<td>2.69 (2.37 – 3.31)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>NR</td>
<td>NR</td>
<td>1.61 (1.31 – 1.99)</td>
<td>NR</td>
</tr>
</tbody>
</table>

NR Not reported.

The age of the patient may influence in-hospital care by a cardiologist (see table 3.3.t). (369) An inverse relationship between increasing age and actual care was observed in a study of 1090 older HF patients⁷. (369)

---

⁷ For this study, the patient population consisted of 1090 hospitalised patients (Medicare), 60% of whom were women. The mean age of the patient population was 79 ± 7.5 years. Overall LVFx evaluation was 58%. HF diagnosis was confirmed by one of the following: 1. History of HF; 2. Symptoms (dyspnoea at
Cardiologist care was independently associated with higher rates of LVFx evaluation. (OR 4.79, 95% CI = 3.5 to 6.47). (369)

The EPISERVE study found significantly higher prescription of ACEi and β-blockers by cardiologists than internal medicine specialists or GPs (ACEi: 62%, 50%, 45%, P<0.05; β-blockers: 70%, 40%, 40%; p<0.05). (302) Patients treated by a cardiologist were more likely to receive adequate treatment according to the ESC 2005 HF guidelines that those attending internal medicine (25% vs. 17%, OR 0.55; 95% CI 0.41 to 0.73). (302)

A study of patients discharged from hospital (Sweden, mid-1990s) discovered that those who were discharged to the hospital’s outpatient clinic were more likely to be male, younger, and have a prescription for ACEi (all comparisons p<0.001) than those discharged back to their GP. (301)

Other specialists and specialist wards: Another study found that compared with cardiologists, other physician types were more likely to prescribe ACEi, but internists demonstrated little difference. (381) An audit (383) of a University teaching hospital (August 1999 until January 2001) coincided with the recent release of the SIGN guidelines (diagnosis and treatment of HF due to LVSD). The audit revealed no difference between cardiologists and non-cardiologists in ACEi/ARB prescription but cardiologist prescribed almost twice the number of β-blockers than did non-cardiologists (37% vs. 21%, p=0.003). (383)

Primary and secondary care dialogue: Surveys of consultants found a split opinion regarding which sector should manage HF or be the lead provider. (367) Some thought

<table>
<thead>
<tr>
<th>Care by a cardiologist</th>
<th>65 to 74 yrs</th>
<th>75 to 84 yrs</th>
<th>≥ 85 yrs</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Care by a cardiologist</td>
<td>53%</td>
<td>45%</td>
<td>33%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 3.3.t. Comparison of specialist delivery of care and patient age
HF ought to be a general practice managed disease and others held the opinion that as it is such a difficult diagnosis to make that specialist HF clinics were the best option. (367)

Poor communication between primary and secondary care (see also Chapter 2, section 4.8, Specialist – GP Interaction) may impede evidence translation into practice. (388)
The NICE guideline (72) stipulates that each HF patient should have a clear management plan, known to all healthcare professionals involved in the patient’s care and to the patient. Adoption of the plan will clarify management decisions in each sector.

**SUMMARY:** The opportunity presented by hospitalisation to confirm diagnosis and establish or optimise a management plan is not always taken. Patients admitted to hospital with suspected HF are not all receiving echocardiography, a diagnostic test which may take weeks or months to access from primary care. A few countries in the EuroHeart Failure survey achieved near 100% for echocardiography. Studies seem consistent in their findings that female patients and older patients are less likely to receive echocardiography. Age may affect whether a patient sees a cardiologist and which medications they are prescribed.

Not examined was whether there was a ‘silo’ mentality, whether patients in cardiology were reviewed by a multi-disciplinary team and whether older patients and/or those with multiple comorbidities were automatically admitted to more general wards even if they presented with a cardiovascular diagnosis.

As seen in primary care, there can also be a hesitation to prescribe in secondary care. Patients are ‘lost’ on the clinical pathway between admission and diagnosis, and between diagnosis and treatment. ACEi use seems to be improving and can increase between admission and discharge, and patients with more severe HF receive more ACEi prescriptions.

It may be difficult to quantify the appropriateness of prescription/non-prescription since discharge letters do not necessarily give reasons for not prescribing, which may reflect poor medical record keeping during the patient’s admission. In one retrospective review ≤4% of patients had a documented history of ACEi intolerance and 5% had a history of symptomatic hypotension. (368) It is not known whether this is representative of all patients.
Confusing the prescribing data is a lack of reporting of β-blockers prescribed. Most studies are unclear whether these are HF-specific β-blockers or β-blockers for other indications. It is difficult to tell if the reported figures are representative of HF prescribing but true prescription rates are most likely to be lower than reported. There is scope for improvement and for secondary care to provide prescribing examples that GPs are seeking.

Spironolactone prescription rates vary across studies. It is not possible to determine whether patients may have been prescribed these when HF was exacerbated but then had them withdrawn.

Age and sex seem to be powerful predictors of medications prescribed, except possibly β-blockers, but this may be due to the type of β-blocker and the indication. Age and sex may also determine discharge destination. Prescription and post-discharge care illustrate primary care patterns of older HF patients and more females, who are less likely to be prescribed HF medication on discharge from secondary care.

The recommendations of the SIGN guideline and the ESC guideline have not been well implemented in secondary care, with underuse of evidence-based therapies.

The effects of management in secondary care will flow through into primary care and GPs may be reluctant to start medication if a diagnosis has not been confirmed during the in-patient stay or if the medication itself has not been started by the hospital. The results of these studies suggest that the opportunities provided by an in-patient stay, to clarify diagnosis and optimise medication have not been utilised and the hospital’s role in furthering education in primary care needs to be carefully considered by the teams that provide care to patients in the hospital environment. There is a need for local champions in both primary and secondary care to improve standards of diagnosis and treatment. (388)

3.4 Conclusion

HF is difficult to diagnose and it is clear that there needs to be a greater understanding by GPs (and indeed by some specialists) of the utility of echocardiography above and beyond clinical findings and CXR. Once echocardiography has been performed, the report received by the GP should give clear and concise findings so that it is evident which steps the GP needs to take in further management. There is potential use of BNP
to determine further management and diagnostic pathways but BNP should not be seen as an alternative to echocardiography.

A standardised diagnostic process in general practice would help to identify potential patients more quickly and more accurately. This is not to underestimate the time needed to evaluate patients and assess their suitability for all medications and the commitment required to initiate, titrate and monitor these medications. This is especially the case for the elderly who are less frequently referred for further investigation and less likely to receive HF medications, and this seems to be a similar scenario experienced by female HF patients.

There seems to be disagreement at specialist level whether HF is to be managed in primary or secondary care. GPs still anticipate that secondary care will take a lead in certain aspects of management. The responsibility for management will be different for different patients and at various stages of HF, and this needs to be clarified. There should be more leadership shown by secondary care, provided that the information given is consistent and evidence-based i.e. not experimental.

In primary care the missed opportunities for diagnostic testing and pharmacological therapy may be due to a range of reasons or motivations or influences. Knowledge and skills may be needed to perform a certain action. GPs may not see a particular aspect of management as part of their professional role and if they work in isolation may not have social influences from peers to persuade them otherwise. GPs also need to have belief in capabilities and belief in consequences, but also attention and motivation to implement a change. A change in routine may also be required, which the GP may need to monitor and set goals to identify whether change has been achieved.

HF should not just be viewed as a condition of the elderly for which there is no hope. There is sufficient evidence-based medicine to provide a convincing knowledge base to proceed with diagnosis and treatment. Accurate diagnosis and appropriate and optimised management can lead to improved quality of life and reduced morbidity and mortality which are important regardless of age.
4. Methods

This chapter describes the aims and methods of this study which implemented recommendations from the New Zealand Heart Failure guideline (NZ HF guideline), published by the National Heart Foundation of New Zealand, into primary care.

The first section of this chapter describes the aims and background to and development of the study.

The second section focuses on the setting, recruitment and randomisation of the GPs, and the educational interventions given to the GPs.

Sections three and four describe problems to overcome when defining HF in primary care, the search strategies to find potential patients, the algorithm applied to define HF, and the data collection in the review of patients’ medical records.

The fifth section describes the outcomes and methods of analysis.

4.1 Background

This study was designed to test the effectiveness of a new method (Internet) of delivering education to GPs, comparing this with a popular implementation strategy (small group session) against the background of a control group. A stratified cluster randomised controlled trial design was used to establish the effects of these three different implementation strategies on each of four recommendations in the management of HF in primary care. The four specified recommendations were from the National Heart Foundation’s Heart Failure Guidelines, 2001 (public release June 2002). (29) The aims of the study are described below.

4.1.1 Primary Aim – echocardiography and medications

The primary aim was to compare management of HF by echocardiography use and rates of RAAS inhibition and/or blockade and β-blockade (through recorded prescribing of ACEi, ARBs, HF-specific β-blockers and spironolactone) between intervention (Internet and small group) and control general practices.

The primary measure was whether the ‘task’ had been performed. It was generally not possible to determine if absolute contraindications existed, and the study was a measure
of what was done rather than why. Further details about the outcomes described by these aims are found in this Chapter in sections 4.4.3.2 to 4.4.3.4, 4.5, and 4.5.1.

4.1.2 Secondary Aim – doses of interest
The secondary aim was to compare the management of HF by the recorded maximal doses prescribed of ACEi, ARBs, β-blockers and spironolactone between intervention (Internet and small group) and control general practices.

Further details about the outcomes described by these aims are found in this Chapter in sections 4.4.3.2 to 4.4.3.4, 4.5 and 4.5.2.

4.1.3 Tertiary Aim – diagnosis, initiation of new medications and electrolyte monitoring, specialist input
The tertiary aims were to determine 1) whether GPs or specialists made the diagnosis of HF, 2) how many medications (ACEi/ARBs, β-blockers, spironolactone) were started during the study and the rates of testing electrolytes before and after spironolactone initiation, 3) which physician was responsible for these new prescriptions and 4) the specialist input to HF management which evaluated information that was given to GPs in hospital discharge letters.

Further details about the outcomes described by these aims are found in sections 4.4.2 and 4.5.3.

4.1.4 Post-hoc analyses – effect of listed HF diagnosis
The post-hoc analyses looked at the effect of HF diagnosis date (recorded in the medical record problem list) on the four primary outcomes (echocardiography use, prescription of angiotensin inhibition, β-blockers, and spironolactone).

Further details about the outcomes related to these aims are found in sections 4.4.2 and 4.5.4.

4.1.5 Other – GP feedback
Other aims were to: 1) test GP knowledge of HF management, before and after intervention, and 2) assess GP reaction to the implementation method.

1. GPs were sent a survey with questions about knowledge, attitudes and practice of HF management with the initial letter that invited them to participate. The survey was repeated three years after the educational intervention was given.
2. GPs were sent a questionnaire two and a half years after they participated in the intervention. The questionnaire asked about their experiences of the method of implementation they were randomised to.

The outcomes from this aim (GP feedback) are used in the Discussion chapter to illustrate possible reasons for the other aims outcomes and as a comparison with other international surveys on HF management.

4.1.6 Development of the study
The study focused on new advances in HF management and whether these advances had been taken up by GPs in their practices.

The use of β-blockers by GPs in the management of HF was the initial area of study, but after an unsuccessful grant application, the funding body suggested expansion of the topic. Areas generally known to be poorly managed in HF were then included, e.g. use of diagnostic tools (specifically echocardiography) and actual confirmation of diagnosis; use of ACE inhibitors and dose of ACE inhibitors; and new data on spironolactone produced by the RALES trial. These issues have been described extensively in Chapter 3.

The Internet had been used as a tool to deliver education but this study tested knowledge and implementation of this knowledge in actual practice. This was a marked divergence from other studies of Internet-based education at that time and these other studies had only tested “before and after” knowledge, as described in Chapter 2.

The use of the Internet as an implementation strategy was tested against small-group sessions which GPs participate in regularly and it is known that GPs appreciate the interactions of small-group format. The control group that was used was a passive dissemination of the guidelines. The assumption was that the GPs who participated in the small-group study arm would out-perform both the Internet and the control group when the four study outcomes were measured. Improvement was also expected in the outcomes seen in the Internet-group GPs, but not to the same extent that would be seen in the small-group arm. (Refer to Appendix 4C for the assumptions made regarding change seen in the study arms.) Dissemination may not be as powerless a change agent as originally identified at the start of the study (see Chapter 2, section 4.6). Another divergence from other studies of HF management and of implementation strategies is
the length of follow up. In this thesis, patient data was collected over a 4 ½ to 5 year period.

CME events such as small-group meetings are generally “one-off” events on a topic, and this study also attempted to show the effect of a brief intervention on practice management.

A study of these two issues – the use by GPs of new management techniques and the methods used to implement this new knowledge – was linked to the up-coming publication of the 2001 NZ HF Management Guidelines.

The study applied for ethics approval and this was granted, in December 2001, by the Auckland Ethics Committee Y, Ref 2001/280. This committee was disbanded in December 2004. The new committee name was Northern X Regional Ethics Committee and the ethics approval reference became AKL/2001/280. Figure 4.1.a on the next page illustrates the timeline of the study.

The study was piloted in a group of GPs who were not part of the study population. The pilot session was taped and the CME modified as well as the GP survey. (See this chapter, section 2.4.1.)

This study was also submitted to the Royal New Zealand College of General Practitioners (RNZCGP) for endorsement as a Practice Review Activity (PRA). A PRA is a form of practice audit, split into two cycles, that GPs are required to complete as part of their triennial recertification (known as Maintenance of Professional Standards [MoPS]) as GPs. College endorsement, as required for a PRA, was received by the study. The minimum number of points required over the three-year MoPS cycle is 150, with a minimum of 30 of from a Practice Review. The points associated with the PRA are considered the hardest to obtain, so offering participation in this study with the bonus of 30 PRA points seemed beneficial for the GPs. It was the opinion of the candidate’s main supervisor that the CQI points would be seen as attractive (at that time) to the GPs. GPs also commented that it was difficult to get audits accredited by the College.

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*The 2002 – 2004 MoPS cycle referred to PRA. In the 2005 – 2007 cycle, this had been changed to Continuous Quality Improvement (CQI) Activity.*
Figure 4.1.a. Study Timeline

**Mid 2001**
- Study outline

**December 2001**
- Ethics approval

**February 2002**
- Piloting small group CME

**Mid 2002**
- Initial letters sent to GPs
- Participation ascertained

**Until mid 2002**
- Modifying project, designing survey / questionnaire

**October 2002 to March 2003**
- Small group CME starts

**November 2002 to April 2003**
- Internet CME available

**December 2002**
- Guidelines mailed out

**Early 2004**
- Suspend registration for 3 months

**Mid 2004**
- Re-contact GPs
- Start to arrange times to visit
- Send 2nd round survey and questionnaire

**2003**
- Definition of HF developed
- Data collection sheets developed
- Follow up survey return
- Background reading

**September 2004 to end 2004**
- Data collection commences
- Testing of search strategies
- Modification of data collection sheets
- Patient mail out / consent form return system
- Hire research assistant – 3rd phase data collection
- Advertise extensively for further Research assistants
- Interview potential assistants
- Carry out data collection
- Follow up survey / questionnaire return
- Continue contacting GPs re data collection

**2005**
- Continue data collection
- Manage study and 3 part-time research assistants

**2006**
- Continue data collection
- Manage study and 3 part-time research assistants
- Suspend registration May – August
- Develop “Current Management” data collection sheets (3 years from intervention)
- Revisit earlier practices to collect 3-year data

**2007**
- Contact GPs, attempt to obtain equal numbers
- Create templates for data entry
- **Beginning December**
- Last of funding runs out
- Data entry

**2008**
- Return to data collection given loss of research assistants
- Manage data entry
- Clean data spreadsheets
- Writing
- Ongoing discussions with Biostatistician
- Final analysis received 22/12/2008

**2009**
- Writing until submission mid April

**Ongoing during study:**
- Annual reports to Ethics Committee
- Since mid 2001 until 2008, numerous grant applications written and submitted
- Annual reports to funding bodies for length of the grant
4.2 General Practitioners

4.2.1 Setting

General practitioners in North and West Auckland\(^a\) were invited to participate. These areas were selected after contacting the large Primary Health Organisations\(^b\) (PHOs) in Auckland in mid-2002 to determine which of them had recently offered CME courses regarding HF. Two PHOs – Comprehensive\(^c\) and HealthWest\(^d\) – had not yet offered any CME on HF and so they were asked if we could educate their members around implementation of the guidelines.

GPs decide whether they wish to belong to an IPA / PHO which provides benefits such as quality improvement, CME and peer group meetings, IT resources such as software, negotiation strength with health authorities and funding benefits. (389-391) GPs not affiliated with their local IPA / PHO were not contacted. It may not have been possible to identify these doctors as it is not a requirement to belong to the RNZCGP, and mailing lists (e.g. held by medical publishing companies) are not always updated regularly.

GPs who practised outside the Auckland dialling area were excluded owing to their distance from services such as hospitals, echocardiography, and specialists. The GPs were a convenience sample from the Auckland metropolitan region. The proportion of GPs who were Fellows of the RNZCGPs [a GP who holds a College Fellowship is vocationally registered (392)] in the randomised GP population and the participating population are compared with national figures in Chapter 5, table 5.1.f. Comparisons of study GPs’ length of practice and sex with national figures are in Chapter 5, table 5.1.e. The study and national comparisons were analysed using a one sample Chi squared test for a fixed population.

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\(^a\) North Auckland patients are more likely to be older, more affluent and European than patients in West Auckland.

\(^b\) Primary Health Organisation (PHO), formerly known as an IPA or Independent Practitioner Association. PHOs are umbrella organisations that bring together all primary care health professionals, and are subsidised to provide a range of health services. Their functions are to provide care for the unwell, preventive services to maintain health, determine and reduce health inequalities, co-ordinate care, and embark on continuous quality improvement based on good evidence.

\(^c\) Comprehensive PHO previously known as the IPA Comprehensive Health Services or CHS.

\(^d\) HealthWest PHO previously known as the IPA Integrated Primary Care Services or IPCS.
4.2.2 Recruitment

Comprehensive and HealthWest IPAs / PHOs provided lists of their GP members. The lists comprised 196 GPs in 80 practices. Every GP on the IPA / PHO membership lists was given a study identification number. Each doctor on the list in the Auckland metropolitan area was initially contacted by letter from the study team and their IPA / PHO (see appendix 4A) asking if they would like to participate in a study comparing three different types of CME dealing with HF. No details were given of the educational interventions to be tested. The letter was accompanied by a self-complete survey on knowledge of HF management⁶ (see appendix 4B). Some GPs, e.g. sports medicine specialists, Navy doctor, who did not have overall management for primary care patients were excluded. GPs had to have a ‘generalist practice’ and could be full or part-time (even 1/10th) regular practice and have the opportunity to attend HF patients.

The sending of the letter was followed a few days later by a follow up phone call to confirm whether the GP was interested in giving verbal agreement to participate. This phone call was made by senior members of the study team who had standing in the academic and medical fields and were likely to be “let through” to speak to the GP (as a key challenge is getting past the receptionist). The fact the principal supervisor (BA) was known personally to many GPs also helped in recruitment. GPs who were unsuitable for the study generally indicated this to us. These GPs were those who were not identifiable as ‘not GPs’ from the lists received from the IPAs / PHOs e.g. short-term locums, acupuncture specialists. The exclusion was made on the basis of the description of what they did. This was prior to randomisation, and exclusion was possible.

After excluding all ineligible GPs there were 69 practices with 177 GPs eligible for randomisation. Of these, there were 25 single-GP practices, 26 practices with 2 to 3 GPs and 18 practices with 4 or more doctors. The following figure 4.2.a indicates the recruitment process and an outline of the study.

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⁶ The information in the returned surveys prompted a new section to be written for the intervention arms about the basics of pathophysiology of HF and also the mechanisms of action of the main drugs used in the treatment of HF.
PARTICIPANTS
GPs that belong to IPCS/HealthWest or CHS/Comprehensive IPAs/PHOs

All GPs were sent a letter of invitation with details of the study and surveyed about their knowledge of current management of HF.

GPs were phoned to determine whether they would like to participate.

Yes
Practices randomised into one of three intervention arms:

Control or ‘usual care’
Guideline only, sent by post.

Small group session
Education session led by GP and cardiologist. Discussed outcomes, Guideline, relevant paper abstracts, diastolic dysfunction summary, GP testimonial on β-blocker use, quiz, Q&A time.

Internet CME
Small group information replicated on Internet site (named users and password protected administered by GFU), with interactive quiz, links to other sites, videos of echocardiograms, FAQs.

No
No further participation in the study.

36 months later
Researcher collects retrospective data from medical records (using algorithm to define HF) regarding medications, investigations. Outcomes (Echocardiography, ACE-I, β-blocker and spironolactone use) counted and compared.
4.2.3 Study design

4.2.3.1 Methodology and sample size

A cluster randomised controlled trial design was selected for this study. Cluster randomisation describes the situation where groups of subjects (who share a feature), rather than individuals, are identified as units of randomisation to intervention and control groups. (393-395)

This type of design is more appropriate for the evaluation of interventions such as educational interventions that target health care professionals to change their behaviour than is randomisation by individual patients. (395-397) Outcomes however are measured at patient level. (227, 398)

It would be complicated to randomise patients of one GP to intervention or control as it would be difficult to treat control patients according to previous practice and control patients could unintentionally receive the experimental intervention. (393, 398)

Randomisation by the next level up (i.e. the GP) may not be feasible as GPs within a practice could patient-share, exposing patients to both experimental and control group GPs. GPs within a practice may also share information about an intervention, resulting in some control GPs behaving like intervention GPs. (398) In this scenario, patients who were initially randomised as control patients may also be managed as if they were intervention patients. If there is contamination of control participants this will reduce the point estimate of the intervention effectiveness which may then lead to the (incorrect) conclusion that the intervention is ineffective as the observed effect size is not significant (either clinically or statistically). (399, 400) Contamination introduces bias and reduces power. (394) This can easily occur in educational interventions as the intervention arm is simple to transmit to others. (394) As GPs working at the same practice are likely to meet regularly, randomising entire practices to different study arms (cluster randomisation) will reduce the likelihood that GPs in the different study arms will meet and discuss or share intervention materials, and reduce the likelihood of contamination. (394)

Therefore the only option is to randomise at the next level which is the practice. All GPs within a practice participated in the same study arm. Any patient who attends several GPs within a practice would be managed according to the study intervention (assuming intention-to-treat principles), and practice GPs sharing information on the intervention would not contaminate the study. (393, 396, 398)
The statistical assumption (in patient-randomised trials) is that the unit which is randomised is the unit of analysis. (395) If standard sample size calculations and analysis methods are applied to cluster randomised trials, then studies will be underpowered and incorrect statistical significance values. (395)

In group or cluster randomisation the independent units of randomisation are the groups, not the individuals. (398) However randomisation by cluster may not take into account variables within the cluster, which could potentially lead to an imbalance in variables between study arms. Patient randomised trials are assumed to have independent outcomes where the outcomes for each patient are considered to be unrelated to those of any other patient. (401) This assumption of independence does not hold in a cluster randomisation as subjects within a cluster i.e. a practice may have similarities that could influence the outcome (e.g. characteristics of the doctor or location of the practice) and two patients from within a cluster are more likely to have similar outcomes than two patients from different clusters. (393-398, 401) The similarities in confounders or in outcomes of interest mean a loss of power to detect a difference between the intervention and control groups. (396, 397) Cluster trials are less efficient in design than individual randomised trials. (395) The lack of independence of the individuals within groups (clustering) increases the sample size required. (397, 398) To achieve equivalent power of a patient randomised trial, standard sample size calculations are inflated by a “design effect”. (394-397, 402)

The design effect (or inflation factor) is the ratio of total number of subjects required using a cluster randomisation to the number needed if using simple randomisation. (402) The equation uses the Intracluster Correlation Coefficient (ICC) which is estimated prior to the study, and the average cluster size (m). (394-397, 403)

\[
\text{Design Effect} = 1 + (m - 1) \times \text{ICC}
\]

A large design effect will require a large number of participants. (402) Greater gains in power can be achieved by recruiting a larger number of clusters in a study or increasing the average cluster size (the number of participants in each cluster). (395, 401) The latter strategy will only increase the power to a certain threshold. (395, 401)

The extent of clustering or the degree of similarity between the responses within the same cluster is referred to as the intracluster correlation coefficient (ICC). (395, 396,

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1 There were also repeated observations on the same patients recruited to the study which cannot be considered independent measures.
The ICC tends to be larger in smaller clusters. (395) As the size of the ICC increases the similarities between the individuals in a cluster increase (and the less they resemble individuals from other clusters) (397, 403). The larger the ICC the larger the design effect and the greater the number of subjects needed to detect a significant difference. (397, 403) If the cluster size is large then even a small ICC will have an effect. (401, 403) If the ICC is 0, then the individuals within and among the clusters are completely independent (with respect to that variable). An ICC of 1 means that the individuals within a cluster are identical but each cluster differs from the others. (397)

The calculations and Intracluster Correlation Coefficient (ICC) used to determine sample sizes for different effect sizes can be found in Appendix 4C. The medium and high levels of effect seemed feasible at the time however since then a significant systematic review has indicated that a maximum effect size of 10% can be expected from implementation studies. (182)

The initial estimate of the number of potential HF patients that could be recruited for this thesis was made with looser criteria than were eventually used in the study, leading to an over-estimate of potential participants. It was estimated there would be over 1300 HF patients in the study areas and the intention was to approach all these patients. As described below, this figure was not attained.

The anticipated number of participating practices, GPs and patients (low effect n = 1230 patients) made it feasible to detect the small change of 5% in change in maximal ACEi dose over the time period of the study. The amount of change observed needs to be weighed against the clinical significance of the change and the time, effort and funding required to effect a small change. During the study there was a significant drop-out rate of GPs overall. Some GPs actively withdrew from the study, but many others did not respond to several requests to collect data from their practices. Despite repeated grant applications, funding to employ a sufficiently large number of research assistants to collect data on over 1000 patients was never obtained. The time line of the study (see figure 4.1.a) lengthened considerably, and the decision was made to end the study once data had been collected from the GPs who had already agreed to this (see figure 4.2.b).
4.2.3.2 Stratification and randomisation

The practices were randomised to one of three educational interventions:

i. Small-group CME
ii. Internet-based CME
iii. Guidelines only (control)

**STRATIFICATION:** Before randomisation took place, practices were stratified by size\(^6\). The design of cluster randomised trials can include stratification where clusters are assigned to a combination of intervention and stratum. (395) Stratification by cluster size can balance the number of individuals (practices) assigned to each arm and is also useful as cluster size can be a surrogate for within-cluster dynamics that can be predictive of outcome. (395, 404) In this study the cluster size was taken to refer to the practice size, which could also operate as a proxy for the assumed number of patients per practice. Separate randomisation lists were used for each subgroup or stratum. (404)

Single GP practices were designated ‘small’ practices, 2 to 3 GPs were ‘medium’ practices and 4 or more GPs constituted large practices. All the small practices within the first IPA / PHO were sorted alphabetically by practice name. This was then done to the small practices in the second IPA / PHO. This pattern was then followed for the medium and large practices respectively. Stratification created a list of 69 practices sorted by size, prior to the intervention allocation. There were 25 solo-GP practices, 26 medium sized practices, and 18 large practices.

Stratification of the sample was necessary to attempt to distribute the ‘layers’ within the sample that could influence the outcomes evenly between the study arms. Different sized practice groups would have different characteristics, e.g. single-handed GPs tend to be older and male. Female GPs tend to be younger and work part-time in larger practices. Issues of clustering and confounding also need to be considered in this context – older GPs are more likely to have older patients and hence, greater exposure to patients with HF. Younger female GPs are more likely to have a younger, female patient population. Patients of large group practices are comparatively less likely to receive continuity of care from the same GP.

\(^6\) The stratification and randomisation was carried out by Victoria Andersen. Blinding was not possible since Victoria was managing the study and needed to give GPs details about their intervention and keep track of numbers.
**Allocation and Randomisation:** The randomisation of trial participants is the best method to distribute in a probabilistic sense known and unknown (confounding) factors evenly between the study groups, thus avoiding bias. (401, 404) These observed and unobserved confounders may independently affect outcomes of an intervention. (394, 401) This is especially important in the complex area of organisation or professional performance, where understanding of potential confounders is not complete. (401) Randomisation tries to achieve equivalence between the groups at baseline, apart from chance differences, and any difference in outcome can be attributed to the intervention. (394, 404) It also avoids selection bias or participant preference. It does not however control for any biases that may occur after randomisation. Using the Intention-To-Treat (ITT) theory in the analysis maintains the baseline comparability realised through randomisation. (394)

To allocate the study arm, the practices were numbered 1 to 3, starting from the top of the list. This numbering was sequential and did not restart for each IPA within a practice size or at each practice size. This was to try to achieve an even allocation of each intervention. The next step was to randomly assign an intervention to numbers 1 to 3. Each intervention was allocated a letter:

- A. Guideline only
- B. Small group
- C. Internet.

A random number was generated for each letter. The lowest was equivalent to 1 and the highest to 3. The number generated for A was the smallest and the number generated for C was the highest. These were linked back to the numbers given to the practices.

Practices, numbered 1 through 3 and then assigned an intervention, were randomised to one of the three implementation strategies. The numbers were evenly split (see table 4.2.a) between the three study arms with each arm comprising 59 GPs across 23 practices. Stratification and randomisation had been successful in distributing the different practice sizes evenly across the study arms.

Table 4.2.a. Results by practice size of randomisation after stratification

<table>
<thead>
<tr>
<th>Practice size</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single GP</td>
<td>9 practices</td>
<td>8 practices</td>
<td>8 practices</td>
</tr>
<tr>
<td>Medium</td>
<td>8 practices</td>
<td>9 practices</td>
<td>9 practices</td>
</tr>
<tr>
<td>Large</td>
<td>6 practices</td>
<td>6 practices</td>
<td>6 practices</td>
</tr>
</tbody>
</table>
This information would be useful at the end of the study to adjust for proportions of practice sizes in each arm if there had been uneven drop-outs.

No GPs who practised at more than one location were randomised to different interventions.

GPs were not left to select their own educational intervention. This would have biased outcomes as particular characteristics would select a particular type of educational intervention. Allowing doctors to select their own study arm also risked the possibility of different interventions within one practice, thereby contaminating any outcomes if they shared patients. The concept and effect of contamination is discussed in 4.2.3.1.

GPs were only told of the intervention that they were participating in and were not told of the intervention being tested in the other study arms. The academic GP (Bruce Arroll) who lead the small group sessions was aware of the GPs randomised to small group. Research assistants who collected data were not told of the study arm that the practices they visited were randomised to; however it may have been possible to identify which GPs were in the guideline study arm as their intervention dates (i.e. the date the guideline was sent out) were the same. Some research assistants did survey and questionnaire data entry and may have been able to determine practice randomisation.

**PARTICIPATION:** As can be seen in the following figure (4.2.b), some GPs did not participate in the educational interventions. This is the second problem that may occur in trials: that members of the intervention group do not receive the intervention (non-adherence or non-concordance). (394) These GPs continued to be participants in the study as the study approach was Intention-To-Treat. Other GPs actively withdrew after randomisation or participation in the interventions citing lack of time, lack of interest, or intention to leave general practice (at least one stated he was going to manage a vineyard). The drop outs occurred more quickly in the Internet arm. This may be due to unfamiliarity with the modality or resistance to this method of learning.

Some GPs participated in the data collection phase, but did not have any eligible patients to contribute towards the study.

At the end of the study, 53 GPs from 24 practices had participated in the data collection phase and supplied data. These numbers are represented in the following figure.
Figure 4.2.b. Flowchart\textsuperscript{a} of recruitment of practices and GPs. Practice\textsuperscript{b} and GP numbers at the start, randomisation, participation and conclusion of the study.

Assessed for eligibility: 196 GPs, 80 Practices

Randomised: 177 GPs, 69 Practices

Excluded: 19 GPs, 11 Practices
Locums, other specialities, GPs due to retire

Control arm
Allocated to intervention: 23 Practices, 59 GPs
Received allocated intervention:
23 practices (100%), median practice size = 2, range 1 – 9
Participants: 59 GPs (100%)
Did not receive allocated intervention:
Unable to determine: assumed all GPs and practices received guideline.

Small group arm
Allocated to intervention: 23 Practices, 59 GPs
Received allocated intervention:
21 Practices (91%), median practice size = 2, range 1 – 6
Participants: 46 GPs (78%)
Did not receive allocated intervention:
2 practices, 13 GPs (either did not respond or attend nominated session)

Internet arm
Allocated to intervention: 23 Practices, 59 GPs
Received allocated intervention:
20 Practices (87%), median practice size = 2, range 1 – 6
Participants: 28 GPs (47%)
Did not receive allocated intervention:
3 practices, 31 GPs (did not log in to the Internet education)

Lost to follow up: 16 practices, 42 GPs
Includes GPs/practices with no eligible HF patients, declined or did not respond to requests for follow up.

Control arm
Lost to follow up: 15 practices, 41 GPs
Includes GPs/practices with no eligible HF patients, declined or did not respond to requests for follow up.

Small group arm
Lost to follow up: 16 practices, 42 GPs
Includes GPs/practices with no eligible HF patients, declined or did not respond to requests for follow up.

Internet arm
Lost to follow up: 14 practices, 41 GPs
Includes GPs/practices with no eligible HF patients, declined or did not respond to requests for follow up.

Clusters: 8 Practices (35%), median practice size = 2, range 1 – 6
Participants: 18 GPs (31%), 137 patients

Clusters: 7 Practices (30%), median practice size = 3, range 1 – 4
Participants: 17 GPs (29%), 98 patients

Clusters: 9 Practices (39%), median practice size = 1, range 1 – 4
Participants: 18 GPs (31%), 124 patients

\textsuperscript{a} Based on CONSORT for cluster randomised trials (405)

\textsuperscript{b} Practice or cluster figures (e.g. number of practices, median practice size, range) are reported for practices that had at least one GP participate in the educational intervention.
4.2.4 Intervention

The educational intervention was delivered in two active arms, with a control arm that did not receive active intervention.

The small group format of CME is a commonly used arrangement in NZ. Several GPs meet together on a regular basis to discuss cases, listen and discuss topics with an invited speaker. The benefits of small group formats are that all participants can have the opportunity to contribute, management patterns can be discussed or any new developments or concerns, GPs also receive peer support and there is also a social aspect. This format is one that GPs are familiar with and are comfortable attending for their CME needs. Small group education has been shown to significantly improve prescribing decisions, professional practice and practice outcomes (compared with no intervention control). (39, 40)

The Internet was relatively new modality as a method of delivery of education and little was known about its effectiveness (beyond before and after knowledge tests) compared with established methods of CME. The Internet CME was delivered on the Goodfellow Unit platform which would have been known to a number of the participating GPs.

Guideline dissemination was used as the control arm – i.e. no active intervention or teaching involvement by the study team.

4.2.4.1 Development and piloting of the educational intervention

The educational intervention developed by the study team was based on the NZ HF guideline, December 2001, and was current at the time of implementationª. The information contained in the educational interventions was discussed with members of the Department of General Practice, School of Population Health, University of Auckland. The small-group format and educational intervention was tested during a CME meeting of GPs from another PHO. The meeting was taped and notes were made. These notes were then used to revise any areas of the intervention that required modification. The information was also discussed with the two cardiologists assisting with the small-group sessions.

ª A 2009 Update of the HF guideline has since been published.
4.2.4.2  Contents of the educational intervention

The information that was used in the educational interventions can be found in appendix 4E. The four objectives of the study (also the study outcomes) were selected as areas in primary care that were either new to HF management or were underperforming. These have been described in Chapter 3. These topics were presented to the GPs. The rest of the intervention included:

**READING MATERIAL**

- The Guidelines and the Algorithms, to be used in practice as a quick check list.
- The abstracts of the key studies used as evidence, ATLAS (16), MERIT-HF (313), and RALES (25).
- Single paragraph summaries of why each objective (echocardiography, maximizing ACEi, using β-blockers and spironolactone) was important.
- A brief pathophysiology review of the mechanisms of HF, and the action of the drugs used in HF.
- A description of diastolic dysfunction and the evidence (or lack of) for treatment.

**TESTIMONIAL – B-BLOCKER USE**

- Clinical experience with metoprolol succinate and spironolactone for patients with HF was described by one of this candidate’s supervisors who was able to speak to this testimonial during the small-group sessions.

**QUIZ**

This comprised a set of 18 questions with multi-choice answers. Fictional case studies were used to depict different stages of HF and therefore different management requirements.

In the two intervention groups, the sessions covered issues including:

- need for echocardiography to confirm or invalidate diagnosis,
- up-titration of ACE inhibitor doses to the maximum tolerated,

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<sup>b</sup> The guidelines and algorithms were received by the study team in electronic form prior to their publication. The printed copies of the guidelines supplied to the study GPs and in the appendices of this thesis therefore appear different to the copies distributed by the Heart Foundation. Any page number references will be to the page number in the copy that was printed for the intervention. The section on β-blockers is on p 15 and spironolactone on p 17 of the guideline.
• addition of β-blocker (Classes II & III for metoprolol succinate or Classes III & IV for carvedilol),
• addition of spironolactone (Classes III and IV), monitoring of electrolyte levels,
• patient education, e.g. daily weighing, symptom management, diet, exercise.

During the small-group sessions, GPs were given time to consider the answers and then discuss these with the academic GP and the cardiologist. There was also the opportunity for GPs to ask for advice regarding their patients. The answers and reasoning for these answers was supplied at the end of the session for small-group participants or on selection of one of the multi-choice answers for Internet-based participants.

The information folder itself, which was given to each small-group participant, was coloured neon green with bright red coloured dividers. The vivid colours were chosen deliberately to be easily recognised amongst the large amount of information that GPs receive.

4.2.4.3 Implementation strategies for the educational intervention

Once the practices had been randomised, each GP was sent a letter with details of which study arm they were in. In appendix 4F are copies of these letters that GPs in each study arm received. The GPs then proceeded to participate in their allocated intervention.

SMALL-GROUP STUDY ARM: GPs randomised to attend a small-group session were given a range of dates to select from and asked to fax return a form indicating which session they were able to attend. The sessions were held at the offices of the two PHOs involved. GPs were given an information folder on their arrival and food and drink were available. All sessions were held during the evening.

The small group sessions involved 5 to 12 GPs per session who worked through the quiz and relevant material led by an academic GP and a cardiologist (Guy Armstrong or Tony Scott, based at North Shore Hospital (at that time) the referral hospital for most of the GPs in the study).
INTERNET STUDY ARM: GPs who had been randomised to the Internet-based intervention were given their unique user name and a password to access the web page. If GPs preferred to have a hard copy of the Guidelines (rather than reading them online), a copy was provided.

GPs logged onto the Internet site through the Department of General Practice’s Goodfellow Unit. GPs worked through an interactive self-marking quiz based on questions about case histories, the answers to which were derived from the background resources. This was the same quiz as the GPs in the small-group session completed. The self-marking nature of the quiz and the rationales provided for why responses are correct or incorrect gave direct feedback regarding current knowledge and allowed for reflection on the learning that has occurred. A FAQ (Frequently Asked Questions) page was posted based on the questions that were asked in the pilot small-group education session and that were raised in the survey of HF knowledge and practices completed prior to the educational intervention.

GUIDELINES ONLY STUDY ARM GPs who had been randomised to the control group were sent a copy of the guidelines.

The guidelines were sent in the mail, and there was no educative session. This arm simulated usual care. Mail-out is a common means of guideline dissemination and was used here to promote patient safety i.e. to ensure that all GPs had access to the most recent evidence-based medicine.

ALL STUDY ARMS: The three interventions shared the following features:

- all GPs received a copy of the Heart Failure Guidelines and Algorithms,
- all were posted a copy of the survey about HF management, and
- all were requested to complete a Practice Review Activity.

All GPs were offered NZ$112.50 which was then the standard payment for participation in CME sessions. GPs were also sent information on how to complete the PRA Summary Sheet for the first cycle of the review (see appendix 4G for the PRA and
information sheet), and a Special Authority\textsuperscript{c} form for Dilatrend (carvedilol). A copy of this form can be found in appendix 4H.

**DATA COLLECTION:** GPs were re-contacted regarding data collection and sent a second copy of the survey, a questionnaire regarding their experiences with the mode of implementation they were randomised to (see appendix 4I), and the second cycle of the PRA (appendix 4G).

4.2.4.4 **Monitoring participation**

For the two active intervention groups (small-group and Internet-based) it was straightforward to monitor who participated. The small-group participants initially responded to the invitation to attend a CME session, and were faxed a reminder. They registered when they attended the session, and were followed up if they did not attend. Any GPs who did not respond to the initial invitation were contacted on at least two further occasions with the dates and times of remaining CME sessions. For the Internet-based participants, it was possible to see each individual log-in and which questions were answered, and also which GP returned PRA forms in the mail. GPs who did not log-on or who failed to complete the entire quiz were sent reminder letters with their user name and password and the numbers of the questions that had not been answered. It was assumed that GPs logged in under their own names and did not use a colleague’s details or complete the Internet intervention as a practice rather than as individuals.

The following table indicates participation rates at the time of study, and then as a measure of GPs who were involved at the conclusion of the study for the small group and Internet arms where attendance / participation could be relatively easily measured.

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Total (start)</th>
<th>Participated</th>
<th>%</th>
<th>Total (end)</th>
<th>Participated</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small group</td>
<td>59</td>
<td>46</td>
<td>78</td>
<td>17</td>
<td>16</td>
<td>94</td>
</tr>
<tr>
<td>Internet</td>
<td>59</td>
<td>28</td>
<td>47</td>
<td>18</td>
<td>11</td>
<td>61</td>
</tr>
</tbody>
</table>

\textsuperscript{c} A Special Authority is the application process for a prescriber to request a Government subsidy for a particular drug for a patient.
It was more difficult to confirm whether the guidelines-only group had made any attempts at self-directed learning. 14 completed PRA forms were returned but there is no reliable method of gauging whether this is a representative number of GPs who undertook self-directed learning of the guidelines. The only comparative measure is the PRA return rate for GPs who were randomised to the Internet arm. This group of GPs may be more similar to the control group in the rate of return of PRA, as in the small group sessions we assisted the GPs in filling out their PRAs so that they could be collected at the end of the session.

Table 4.2.c. Numbers and proportions of GPs who returned their PRAs comparing the start and conclusion of the study

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Total (start)</th>
<th>PRA rec’d</th>
<th>%</th>
<th>Total (end)</th>
<th>PRA rec’d</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guideline</td>
<td>59</td>
<td>14</td>
<td>24</td>
<td>18</td>
<td>7</td>
<td>39</td>
</tr>
<tr>
<td>Small group</td>
<td>59</td>
<td>42</td>
<td>71</td>
<td>17</td>
<td>15</td>
<td>88</td>
</tr>
<tr>
<td>Internet</td>
<td>59</td>
<td>21</td>
<td>36</td>
<td>18</td>
<td>11</td>
<td>61</td>
</tr>
</tbody>
</table>

The analysis of the data from this study would clearly be by intention-to-treat.

### 4.3 Defining HF in primary care

The study required a working definition of HF that could be used successfully in primary care. The initial problem with identifying HF patients from general practice records was the well established fact that a majority of patients are incorrectly labelled. This is predominantly due to over-reliance on signs and symptoms and under-utilisation of diagnostic tools.

Another problem that stemmed from this was whether GPs were coding their patients’ conditions, especially if the condition arose prior to a practice adopting fully computerised record-keeping.

Although determining HF patients through reported symptoms seemed feasible this had its own inherent challenges:

i. The constellation of symptoms that could be indicative of HF

ii. The decision to record these symptoms

iii. The diagnostic importance of each symptom or sign.
The confusion over the diagnostic importance of the various signs and symptoms possibly attributed to HF is demonstrated in Chapter 3. Many researchers try to determine the sensitivity and specificity of signs and symptoms in very specific patient subsets.

This study required criteria that could be utilised for different presentations of HF, e.g.

i. Over a wide age range
ii. Range of co-morbidities
iii. Slow onset ± severity
iv. Acute onset ± severity
v. Patients with minimal investigations
vi. Multiple and minimal symptoms (presentation versus recording)
vii. Patients in primary care – not pre-selected.

To standardise data collection for each research assistant and between research assistants a decision was made to use a scoring system with a defined list of criteria. Different HF scoring systems have been compared in Appendix Q. The Boston Score was chosen due to the high likelihood of the terms used in the scoring system being found in medical records (compared with other scoring systems). The wide range of categories would also take into account different recording preferences or importance placed on different symptoms by GPs. The Boston score also had a high PPV for definite HF and for possible HF.

The Boston Score was used as an initial measure of likelihood of HF. More up-to-date measures of HF were added to determine HF if the criteria of the Boston Score either were not available or gave an inconclusive diagnostic picture.

Each line of the Boston score was numbered and this referred to the instruction sheet on how to fill out the Boston Score. The instruction sheet also gave other acronyms for these symptoms / findings. (See appendix 4K for the full Boston Score sheet and additional measures used in the study and the completion instructions for research assistants.)

As described in Appendix Q, section 7, each group of symptoms could score a maximum of 4, regardless of how many symptoms were identified in each category. The three sub-scores were added, and the total determined the likelihood of HF.
- Total 0 to 4 was graded as ‘No’
- Total 5 to 7 was graded as ‘Possible’
- Total 8 to 12 was graded as ‘Definite’

Three supplementary measures were added to the Boston Score to update it and to give greater diagnostic ability, and also to collect necessary data for the study. This was labelled as Line 23 Additional Measurements. These measures were:

Line 24. **Response to diuretic.** As per the European Society of Cardiology, to be used as an objective criteria if diagnosis is in doubt. E.g. weight loss of ≥2kg within a few days to 1 week (to allow patients time to report back to their GP) with clear indication that diuretics were prescribed.

Line 25. **Echocardiography.** For echocardiography, a number of different variables were accepted as definitive of HF:

- Ejection Fraction (EF),
- Fractional Shortening (FS), or
- LVEDD / LVESD (Left Ventricular End Diastolic Diameter / Left Ventricular End Systolic Diameter).

These two last variables are then used to calculate Fractional Shortening (FS). An FS value of ≤28% is generally indicative of HF.

Line 26. **N-BNP.** This was not available at the time of the educational intervention but it became generally available in early 2003. (406) N-BNP was included in the diagnostic scoring system as it was deemed to be a developing tool and more easily accessible than echocardiography and as such would presumably have a quick uptake rate by GPs.

NT-ProBNP results initially sent back to GPs with the reference range of Normal <40pmol/l and Indicative of HF >220pmol/l. These were then reduced to <40pmol/l as the sole reference point. The reference point was then changed to <35pmol/l. However, there is no definition of what a value between the upper and lower values means or what the GP should consider next in diagnostic testing.

In 2005/2006 the cost per assay was $50, which rose to $53.10 in late 2007.


4.4 Identifying HF patients and data collection

Identifying potential HF patients and collecting data for the study was completed in three phases.

1. Patient identification. The practice was visited to compile a list of patients who potentially had HF, determined either by their problem list or by the combination of drugs in their prescription list. It was usually possible to compile this list within a few hours however the manipulation of the list may have taken several more hours.

2. The practice was visited a second time to evaluate all the patients identified in the first visit. Using a HF scoring system (the Boston Score) the likelihood of a patient having HF was determined and patients with possible or definite HF were then posted consent forms and participant information sheets with a letter from their GP on behalf of the study team. Identification of patients may have required several visits to the practice depending on the time allocated to each visit and the number of patients that had to be evaluated.

3. The third phase was the collection of data from the patient’s medical records (e.g. demographic data, comorbidities, physiological data, electrolytes and medications) at three time points: before the intervention (these data were called pre-intervention), in the 18 months after the intervention (these data were called post-intervention) and medication data at three years after the educational intervention (these data were called current management). This phase of data collection usually required several visits.

The different data collection sheets were printed on different coloured paper for ease of reference. The Boston score data collection sheet was coloured lime green. The patient demographics data collection sheet was light yellow, pre-intervention data collection sheets were light blue, post-intervention data collection sheets were light pink, and current management data collection sheet was burnt orange.

The data were collected by Victoria Andersen (VA) and three part-time research assistants. One (MH) was an overseas trained doctor, another (KH) was a research nurse, and the third (SB) had experience in collecting data in primary care settings. VA took responsibility for creating the patient identification lists. Evaluation of patients for the likelihood of HF was then completed by either MH or KH. Data from medical records were then collected by SB with some practices completed by KH avoiding practices she had already visited to evaluate patients. Two ‘summer students’ (students
who had completed the third year of medical training) assisted during the summer holidays of 2005/2006.

At the start of the study VA completed each phase of data collection in two practices in order to test and redevelop the data collection sheets and the methods of collecting data. When the funding for the study ceased, SB was no longer able to work, so her tasks were taken over by MH, KH and VA, which meant that the same researcher was collecting data at different phases of the study. This was also the case when funding for KH ceased – VA and MH were then the sole data collectors. VA was responsible for data collection at the end of the study (for incomplete or incorrect data).

Prior to data being collected, a working definition of HF needed to be developed for the study, which is where the work on scoring systems for HF, Appendix 4Q, becomes relevant. The scoring system used would need to include more current diagnostic tests for HF, which would need any of the scores reviewed in Appendix 4Q to be re-worked. Other difficulties in identifying potential HF patients are described in the sections below.

4.4.1 Search criteria to identify patients

Several different types of Practice Management Software are in use in Auckland, and there are different versions between practices. Three are Windows-based and one is run on Apple-Macintosh. The Windows based programmes are MedTech (most common is version 32), GDPat, and Next Generation. The Macintosh programme is Profile. All have different search platforms.

Med Tech 32 has a ‘generic’ drug search function which was used to eliminate the need to search under the different drug names and different doses.

Profile has an in-built HF search which:

“Provides a list of patients using prescribed HF medications as the search criteria”

This was run in practices that used Profile.

A typical search to identify prospective patients would include running separate searches on the following terms and medications:

- Heart failure
- Congestive heart failure
- Cardiac failure
- Congestive cardiac failure
- Left ventricular failure
- G58 + minor codes

(These would search the Diagnosis tables and consultation text depending on the programme.)

- All ACE-inhibitor names (brand and chemical)
- Spironolactone + brand name
- Frusemide + brand name
- Metoprolol succinate and Carvedilol + brand names
- Candesartan and Lorsartan + brand names
- Digoxin + brand name

Windows-based PMS generated lists were merged into one list containing the names of patients generated in all the searches. Some software allowed certain exclusion criteria to be built into the query, e.g. age, registration status at the practice. All other exclusion criteria needed to be searched for manually in the patient’s medical record. VA was responsible for running all searches to ensure consistency and that the searching would be completed.

The method of using specific medications to identify certain conditions has been used previously. A survey to determine the prevalence of HF in general practice in three practices in London identified potential patients by prescription analysis – previously shown to be a useful method in establishing the prevalence of angina. (261, 262) This PhD took this process one step further and applied diagnostic criteria to the patients who had been selected by medication.

### 4.4.1.1 Exclusion criteria

Any patient under the age of 45 years (before the educational intervention) was excluded as there was only a very small likelihood of patients under this age having HF. There was no maximum age. Patients who were visitors/casual were also excluded, as were patients who transferred out of the practice prior to or soon after the intervention.
and patients who transferred to the practice later than 18 months prior to the intervention. Patients solely on metoprolol succinate or ACEi + metoprolol succinate were excluded. Further exclusions occurred while patient records were being reviewed for evidence of HF. These were patients with severe psychiatric illness, alcohol/drug addiction, dementia, terminal illness, or insufficient data available e.g. <9 months on each side of the educational intervention date. In the cases of the first three illness-related exclusions, informed consent is required from each research participant or someone authorised to sign on their behalf. There was the concern that this may not occur with patients who were severely psychiatrically unwell. This was also a blanket exclusion as it was faster to exclude than to try and determine the diagnosis. Patients with severe psychiatric illness would have made up only a small proportion of HF patients and there is no reason to believe that there would have been unequal proportions between the groups. Two community-based HF studies report 4.9% and 8% of their sample with severe psychiatric disease. (6, 353)

Each patient on the generated list who did not meet any of the exclusion criteria then had their medical records evaluated for criteria stipulated in the Boston Score (see 4.3) and the additional measurements.

4.4.2 Evaluation of potential HF
Once potential patients had been identified by searching the practice database, the practice was visited again in the second phase of the study to collect data about whether the patient had HF (suspected or confirmed) using the Boson score.

A study referenced earlier (261) stated that it is difficult to make clinical judgements by reading case notes and letters retrospectively, and if there was real doubt the researchers erred in favour of a positive diagnosis. (261) Taken in context, we erred in favour of positive signs and symptoms, and CXR and echocardiogram report findings.

4.4.2.1 Diagnosis
While medical records were searched for evidence of HF, attempts were made to determine the date of diagnosis (Dx date), and the source of the diagnosis. (Refer to appendix 4K, Boston score, lines 28 and 29.) The earliest recorded indication of HF was taken as the date. The diagnosis date needed to be before the educational
intervention so that all patients were treated equally and in order to have continuous patient data before and after the intervention. The ‘Problem list’ indicates that the diagnosis was listed there; ‘Text note’ that HF was noted in the text of the medical records; ‘Hospital’ that the hospital described the patient as having HF e.g. in an admission or discharge report; and ‘Specialist’ that the patient was diagnosed with HF by a specialist outside of the hospital.

However, the need arose for a further Source description. There were occasions when the Dx date was after the educational intervention date, but there was evidence of HF prior to the intervention date or occasions when the diagnosis was listed under ‘Problems’ but no date was listed. The History and CXR dates were required to be close (i.e. no more than a few months apart), provided there was evidence of HF (graded as Possible or Definite by the Boston Score) but the diagnosis was not documented, then the History and Exam dates were taken as the diagnosis date. (Refer to appendix 4K, Boston score, line 31.)

In trying to date the HF diagnosis the following problems had to be considered.

1. If a patient had HF in the problem list, and the date was prior to the intervention i.e. had sufficient time (several months) to be started on HF medication and changes made, this was taken as the date of diagnosis. This was also the case if a letter was found in the medical records stating that the patient had HF.

   For patients whose date in the problem list was after the date of the educational intervention, see point 3.

2. If HF was listed under problems, but no date was given, efforts were made to find correspondence with a date. If this was found, then the date was taken as date of diagnosis. If no dated correspondence was found, the records would be searched for signs and symptoms.

3. For patients who did not have HF in the problems list, medical records were searched for evidence of signs and symptoms, as listed in the Boston Score, that occurred within a few months of each other. The date of these was then recorded. If HF was recorded as a diagnosis after the date of educational intervention this was also recorded.

   Provision would also need to be made for patients with “historical” HF, e.g. those who had transferred from another GP with HF as a condition but without any supporting evidence, or who had been labelled HF during a hospital admission. These were
patients whom the doctor believed had HF, and theoretically would be the patients who would be prime candidates for further investigation.

(See Appendix 4K for the guide for research assistants on completing the Boston Score.)

4.4.2.2  **Contacting participants**
Each patient who had a completed Boston Score that indicated that HF was suspected was sent information about the study from their GP. The information pack contained:

- A letter about the study signed by their GP/practice nurse/practice manager,
- An information sheet,
- A consent form,
- A Freepost envelope for return of the consent form.

The above documents can be found in Appendix 4J.

Each consent form included in the footnote the GP identifier number or GP identifier numbers (for practices with more than one GP) so it was clear which practice the patient belonged to. Ethics Committee regulations require signed consent from each research participant (patient), or someone with authority to sign on the patient’s behalf.

On receipt of consent forms from these patients, the practice was visited for the third phase of the study to collect data on patient demographics, electrolytes and physiological measurements, and medications.

**SAMPLE SIZE:** To measure a 5% change (from 20 to 25%) in maximal ACEi dose within a patient over time, 1230 patients (408 per study arm) was required. This was the largest sample size calculated for the different outcomes and effect sizes and different correlations between paired measures.

Power was set at 80% and all tests were two-tailed with 5% significance. The Design Effect was calculated as 1.2, the average cluster size (patients within a practice) as 17 and the ICC was 0.01.

Further details of sample size calculations are provided in Appendix 4C.
4.4.3 **Patient characteristics and management**

The data collected in the third phase of data collection centred on:

- Patient demographics
- Pre-intervention data including medication, electrolytes, blood pressure, heart rate, BMI, management around spironolactone initiation, in the 18 months prior to the educational intervention.
- Post-intervention data, as above, but collected from the period 18 months after the intervention.
- Current medication; medication at three years after the intervention.

4.4.3.1 **Patient demographics**

Data were collected about date of birth, gender, recorded ethnicity, and on other diagnoses, whether they were present and the date of diagnosis. (See appendix 4L for the data collection sheet and completion notes.)

**ETHNICITY:** Ethnicity data were collected from patients’ medical records. When ethnicity was not recorded or left as ‘Other’, it was obtained from the ethnicity data supplied by patients on their consent forms. This was recorded by patients as free text and was less straightforward to code. The ethnicity data in medical records was already standardised and thus concise to report. Ethnicities were recorded in medical records as free text. Ethnicity was grouped by those categories most likely to be found in New Zealand, based on Census questions. Patients could identify with more than one ethnicity. If a patient identified as Maori and any other ethnicity, the patient was deemed to be Maori (which is customary in NZ). For other patients who identified with more than one, the first ethnicity was considered to be the major ethnic group the patient identified with. Ethnicity was grouped as follows:

1. NZ European/Pakeha/European (or stated European ethnicity)/Other European
2. NZ Maori/Maori
3. Pacific (e.g. Tongan, Samoan, Nuiean)
4. Asian (Chinese)
5. Indian/Sri Lankan
9. Not recorded (where ethnicity was not able to be determined).
CO-MORBIDITIES: Information on additional diagnoses would be found in the ‘Problem’ list of the electronic records or inside the front cover of paper records. Specifically sought was information relating to hypertension, diabetes (Type 1 and Type 2), atrial fibrillation, rheumatic fever\(^d\), valvular disease, ischaemic heart disease and myocardial infarction. HF was also in this list to check the difference between the date first identified and the date it was added to the problem list.

The information gathered on valvular disease and rheumatic fever was used to delete these patients from the sample as they did not have ‘true’ HF, as valvular failure can be reversed by a replacement valve. Rheumatic heart disease can only be diagnosed by echocardiography. Since not all patients had undergone echocardiography, rheumatic fever was used as a proxy measure. This would have been important if one group had higher rates of echocardiography than the others. There may be potential bias of internal validity.

Other conditions that may have an adverse effect on HF were also noted. These were:

- Anaemia
- COPD / asthma
- Obesity
- Thyroid dysfunction
- Cardiomyopathy
- Smoking status
- Alcohol dependency.

For the ‘Diagnoses’ and ‘Other’ in the Patient Demographics, the year that these occurred has been recorded where available. The date may not indicate the actual diagnosis of the condition but may indicate the date that the condition was entered into the Problem List. For all conditions, the earliest recorded occurrence in the problem list has been noted. The exception is MI where all occurrences have been listed. Some of the listed diagnoses and the free-text diagnoses were categorised into similar groups for ease of reporting. The diagnosis groups and the listed and free text diagnoses that these were based on can be found in appendix 4O.

\(^d\) The highest documented rates of Acute Rheumatic Fever and Rheumatic Heart Disease are found in Maori and Pacific people in New Zealand, Australian Aborigines and Pacific Island nations. Figures from 1949 to 1953 show that the incidence of ARF in Maori children was 11 times that of non-Maori children. A study from 1956 to 1973 showed that the decline in incidence of ARF seen in developed countries was not seen in New Zealand. The highest rate of ARF recurrence is also in Maori and Pacific people. (407) Given this data the inclusion of Rheumatic Fever was seen to be important.
4.4.3.2  **Pre-intervention data**

The time period in which these data were collected was in the 18 months prior to the educational intervention taking place. The full data collection sheet is in appendix 4M with the completion instructions for research assistants.

4.4.3.2.1  **Physiological variables**

The following variables (where available) were recorded at the start and end of the 18 month period and then averaged:

- Blood pressure
- Heart rate
- Weight or BMI

Height was taken as one measurement.

Data gathered on Height, Weight, and BMI were limited so the decision was made to drop these variables from the data set as they would not add any useful information.

Electrolyte (Cr, K⁺, Na⁺) values were recorded. These values gave an indication of the general well-being of the patient and could also act as a comparison between practices and also between intervention arms.

The reference ranges for Sodium (Na⁺), Potassium (K⁺), and Creatinine (Cr) changed during the study period. The following table (4.3.a) presents the different reference points for diagnostic tests over time.

Table 4.3.a. Differences in electrolyte reference values during the study

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Lower reference point</th>
<th>Upper reference point</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cr</td>
<td>0.050 – 0.060 mmol/L</td>
<td>0.100 – 0.120 mmol/L</td>
</tr>
<tr>
<td>Na⁺</td>
<td>134 – 135 mmol/L</td>
<td>145 – 148 mmol/L</td>
</tr>
<tr>
<td>K⁺</td>
<td>3.5 mmol/L</td>
<td>5.0 – 5.3 mmol/L</td>
</tr>
</tbody>
</table>

Cr 0.050mmol/L was recorded in 2002, 2004 and 2005. Cr 0.060mmol/L was recorded in 2003. Cr 0.100mmol/L was recorded in 2002 and 2004. Cr 0.120mmol/L was recorded in 2003 and 2005.
Na⁺ 134mmol/L was recorded 2002 to 2005 and Na⁺ 135mmol/L was also recorded in 2005. Na⁺ 145mmol/L and Na⁺ 146mmol/L were both recorded in 2005 and Na⁺ 148mmol/L in 2002 to 2004.

K⁺ 5.3mmol/L was recorded 2002 to 2005 and K⁺ 5.0mmol/L also recorded in 2005.

The lowest of all measurements for any electrolyte and the highest of all measurements for any electrolyte were taken as the reference interval.

It is also worth bearing in mind the contraindications and recommendations as specified by the NZ HF Guidelines:

ACE inhibitors: K⁺ >5.5 mmol/L  
Cr >0.25 mmol/L  
Monitor initiation closely: Na⁺ <135 mmol/L

“It is recommended that for those who are not at risk of hypotension, low doses should be started (eg enalapril 2.5mg BID), and patients reviewed after a week to monitor blood pressure, renal function and serum potassium.” p.14. (29)

One of the study outcomes was spironolactone initiation. If Spironolactone had been prescribed, the date of initiation was noted as were the results and dates of laboratory tests for K⁺ and Cr measurements closest to the initiation date (before and after) in the patient's medical record or in hospital discharge summaries. This was done to see whether the NZ HF guideline was adhered to. Contraindications and recommendations for the prescription of spironolactone as specified by the NZ HF Guidelines are as follows:

Spironolactone: K⁺ >5.0 mmol/L  
Cr >0.25 mmol/L  
“Creatinine and electrolytes should be checked 3-4 days, one week and one month after initiation and then as indicated by renal function (6 monthly in stable patients).” p.17. (29)

4.4.3.2.2 Pre-intervention medications

The main medications of interest were;

- ACE inhibitors
- Diuretics (other than spironolactone)
- β-blockers (only metoprolol succinate and carvedilol)
- Spironolactone
- Angiotensin II antagonists.
The data collection sheet can be found in appendix 4M.

The chemical name of each drug prescribed was recorded for ease of identification. The maximum dose achieved was recorded. Earlier attempts to record all dose alterations within the 18 month period resulted in a complicated data set with unbalanced data points for patients and over different time periods.

The drug was ‘Continued’ if the prescription had started prior to the 18 month period.

The drug was ‘Initiated’ if the drug was started during the 18 month period. Attempts were made to determine who initiated the prescribing: the GP, the hospital, or any specialist seen outside the hospital. This would give an indication of which sector (primary, secondary) was responsible for commencing a new medication. This was of particular interest around β-blockers given their former status of being contraindicated in HF.

The drug was ‘Stopped’. This could either be determined from a prescription not being renewed or a text note in the patient’s medical records or a letter from a hospital / specialist. No attempt was made to establish why medications were stopped as in the majority of cases the reason was not documented.

Other medications of interest were recorded by class only. Short term prescriptions of the drugs of interest e.g. 1 month, were not included.

4.4.3.3 Post-intervention data

The above processes were repeated but for the 18 months following the educational intervention. (See appendix M for the data collection sheets.)

4.4.3.4 Current management data

Current Management data focused on medications prescribed at three years after the educational intervention (± a few months to capture relevant data). As above, the names of the main medications of interest (ACE inhibitors, diuretics (other than spironolactone), β-blockers (only metoprolol succinate and carvedilol), spironolactone, and Angiotensin II antagonists) were recorded, plus the doses prescribed.

The ‘Other Medications’ (anti-arrhythmic ± Digoxin, anti-platelet, anti-coagulant, β-blocker (not metoprolol succinate or carvedilol), Ca$^{2+}$ channel blocker (DHP and/or other), nitrate, lipid lowering, NSAID) were recorded in the same manner as for the pre- and post-intervention periods.
The data collection sheet for Current Management can be found at appendix 4N. This data collection time point was not originally part of the study but was added when it was realised that due to the length of time taken to collect patient data (researchers were in practices around three years after the educational intervention was delivered) there was an opportunity to collect data at another time point.

4.5 Outcomes and analysis

The primary outcomes were the four guideline recommendations that were emphasised in the two active study arms: small-group and Internet. The primary and secondary management outcomes are:

i. Echocardiography
ii. ACEi/ARB prescribed and highest dose achieved
iii. Metoprolol succinate or carvedilol prescribed and highest dose achieved
iv. Spironolactone prescribed, dose prescribed, electrolytes monitored.

These are described in more detail under the primary and secondary outcome headings.

Analyses of the outcomes were completed in two stages. The first analyses were conducted on the management outcomes being present or not. Then the doses of the medications were analysed.

It is beyond the scope of this study to determine whether patients were on medication appropriately or not. It was very rare to find NYHA classification in medical records – used as a basis for prescribing metoprolol succinate / carvedilol and spironolactone. There is also uncertainty around the accuracy and currency of the Problem list (potential contraindication or cautions) making it difficult to determine reasons for not prescribing. Medications are frequently stopped without any justification given.

Data were collected on participating HF patients for the time periods of 18 months prior to the educational intervention, 18 months after the educational intervention, and then at 3 years. Given that there are waiting lists of several months for echocardiograms and the length of time needed to up-titrate ACE inhibitors and the need to ensure patients are stable and euvolemic prior to starting β-blockers, assessment of management within an 18 month period after implementation seems reasonable.
The aim of the study is to assess the amount of change that is clinically possible to achieve. The data and patient lists are available should anyone in the future wish to conduct a longer-term follow-up to determine how clinically meaningful any changes in management have been.

**ANALYSIS METHODS FOR MANAGEMENT OUTCOMES:** Intention-to-treat analysis was used in this study for all GPs (whether they participated in the educational intervention or not) and for all patients at the end of the study (suspected or confirmed HF).

All of the analyses were carried out by Mrs Elizabeth Robinson, Research Fellow, Biostatistics and Epidemiology, School of Population Health, University of Auckland.

Descriptive statistics have been used to describe the demographics of the GPs participating in the study. All analyses have taken into account the size and clustering by practice. The demographics of the GPs and of the practices have been included in the analyses where possible.

The analysis (multi-level modelling) of cluster randomised trials can take into account the hierarchy of the data seen in primary care. In the primary care setting there are patients (level 1) with covariates such as age or sex, who are treated by GPs (level 2) whose covariates may include length of time in practice and sex, who in turn are nested within practices (level 3) which would have a covariate of size (number of GPs).

In this study of HF management and guideline implementation, the effect of the intervention, time and of intervention * time also need to be included in the model of analysis.

All statistical analysis of management outcomes was done using SAS v 9.1 (SAS Institute Inc., Cary, NC, USA), and the GLIMMIX procedure (Generalised Linear Mixed Model) was used. All tests were two tailed with 5% significance and power was set at 80%. The model investigated whether the change over time in outcome differed for patients assigned to the three study arms. These models allow for correlations between measurements within a subject and between subjects with the same GP to be modelled. The test of interest is whether the interaction between time period and study arm is statistically significant as this indicates whether the pattern of change over time is

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The MIXED Procedure was used to compare patient demographics, physiological variables and electrolyte values. 

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different for the three groups. For the outcomes of echocardiography, ACEi, β-blocker and spironolactone, a logit link was used, the GP was included as a random effect and the within patient measurements were included as repeated measurements over time.

The variables included in the model differed depending on the analysis and whether the model could run without becoming destabilised by the number of variables.

A priori analyses specified were the effect of the education programmes on the change in proportions of patients in each study arm having echocardiography, prescription of and maximal ACEi dose, prescription of and maximal dose of metoprolol succinate/carvedilol and prescription and dose of spironolactone. The proportions were compared within study arms over time and between study arms over time. Comparisons were also made on demographic data between study arms. The original comparisons of changes in doses over time for the different medications were revised from comparisons of single dose codes to the groups discussed below and in Appendix 4P.

The outcomes at each time period have been coded ‘1’ (present) or ‘0’ (absent). The proportions are the basis for comparison of effectiveness of the educational intervention. Some medication was initiated by a cardiologist but was counted as a positive response for a GP. This is a bias that will be equally distributed between the study arms.

4.5.1 Primary outcomes – echocardiography and medications

RATES OF ECHOCARDIOGRAPHY: The main objective of referring patients who have suspected HF for investigation by echocardiography is to confirm or disprove the clinical diagnosis of HF that has been made. Once diagnosis has been confirmed, this then clarifies the management pathway to follow, and gives the GP greater certainty in their prescribing decisions.

Information about echocardiograms, where available, was recorded on each patient’s Boston score. The echocardiograms dates were recorded and related to the appropriate time period (pre-intervention, post-intervention or at three years). Patients who had repeated echocardiograms had only the first echocardiogram counted.

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1 Pre-intervention refers to the 18 months before and post-intervention refers to the 18 months after the educational intervention was delivered. These were determined at the start of the study. During the study the ‘Current Management’ time period was added and was ‘current’ at the time of data collection i.e. at three years after the educational intervention.
The model used to analyse the binary outcomes (had echocardiogram or not) was the same as that used for medications of interest (see below). The study variables that were included in the model were intervention group, time period, group by time period. The demographics and clinical variables of the patients (see Chapter 5, section 2) that were included in the models were sex, age at first intervention, renal impairment, record of MI, record of CORD and record of asthma.

The GP variables included were size of practice, sex of GP and years since qualification. The GLIMMIX procedure was used to investigate whether the change over time in outcome differed for patients assigned to the three study arms. If the test of interaction between time period and study arm is significant this indicates that the pattern of change over time is different for the three groups.

**MEDICATIONS OF INTEREST:** The recorded prescription of ACEi, ARBs, β-blockers and spironolactone was recorded in the 18 months before and in the 18 months after the educational intervention and again at three years after the educational intervention. The analysis model processed the following variables:

- Intervention
- Time (period)
- Intervention by time
- Gender
- Age at first intervention
- MI
- Renal impairment
- CORD
- Asthma
- Practice size
- GP sex
- GP qualification (year).

The effects were multiplicative so any effect further down the list that was significant was an effect over and above any significant effects that occurred higher in the list.

For modelling of the spironolactone date the last three variables were dropped from the analysis as the numbers of patients were small and the model became unstable.
4.5.2 Secondary outcomes – doses of interest

For ACEi and Angiotensin II Receptor blockers, β-blockers approved for use in HF, and spironolactone, the maximum dose prescribed in the pre-intervention and post-intervention periods was recorded, and the doses prescribed at three years after the intervention were recorded for Current Management.

The number of dose codes originally derived for the study was too complex to be modelled so these were reduced to a maximum of two dose codes. These were dependent on the prescribed drug and the dose that was feasible to prescribe in general practice. The coding schedule is in appendix 4P.

For ACEi and ARBs, as it has been shown that it is possible to prescribe maximum doses in primary care, codes 1 and 9 (1 = dose recommended by the NZ HF guideline (29) and 9 = any dose above that recommended by the NZ HF guideline) combined vs. any other dose given were the two dose groups that were analysed.

The dose coding was used in order to standardise the doses prescribed to patients as eight different ACEi are available for prescription in NZ and the maximum doses of each of these are significantly different. For example the maximum recommended dose for captopril is 150mg per day, while the recommended dose for trandolapril is 2mg per day.

A large difference is also seen in the recommended dose for carvedilol (50mg) and metoprolol succinate (190mg). The tables (4.5.a and 4.5.b) on the next page indicate that the doses that patients seem to receive in primary care or in an out-patients’ setting are below those attained in clinical trials. Given the disparity between clinical trial and real-world doses i.e. community-based, the decision was made to analyse patients who received ≥50% of target dose for β-blocker.
Table 4.5.a. Comparison of carvedilol doses achieved between clinical trials and outpatient / primary care

<table>
<thead>
<tr>
<th>Doses</th>
<th>Clinical trials for carvedilol</th>
<th>Primary care / outpatient clinic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>COMET (409)</td>
<td>COPERNICUS (410, 411)</td>
</tr>
<tr>
<td>Target dose</td>
<td>25 mg bd</td>
<td>25 mg bd</td>
</tr>
<tr>
<td>Mean daily dose</td>
<td>41.8 mg</td>
<td>37 mg</td>
</tr>
<tr>
<td>% on target dose</td>
<td>75%</td>
<td>60%</td>
</tr>
</tbody>
</table>

Table 4.5.b. Comparison of metoprolol succinate doses achieved between clinical trials and outpatient / primary care

<table>
<thead>
<tr>
<th>Doses</th>
<th>Clinical trials for metoprolol succinate</th>
<th>Primary care / outpatient clinic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>COMET (409)</td>
<td>MERIT-HF (21)</td>
</tr>
<tr>
<td>Target dose</td>
<td>50 mg bd</td>
<td>200 mg</td>
</tr>
<tr>
<td>Mean daily dose</td>
<td>85 mg</td>
<td>159 mg</td>
</tr>
<tr>
<td>% on target dose</td>
<td>78%</td>
<td>64% 200 mg 87% 100mg</td>
</tr>
</tbody>
</table>
For spironolactone, the target dose was 25mg per day, so this dose was analysed against all other doses of spironolactone.

The variables were multiplicative so any variable lower down the order that was significant had an effect over and above any significant variables that were analysed earlier. The analysis variables were:

- Intervention
- Time (period)
- Intervention by time
- Gender
- Age (at first intervention)
- MI
- Renal impairment
- CORD
- Asthma
- Practice size
- GP sex.

The model investigated if the change over time in outcome (the two dose groups) differed for patients across the three study arms. If the interaction between time period and study arm is significant this indicates that the pattern of change over time differs for the three study arms.

4.5.3 Tertiary outcomes – diagnosis, initiation of new medications and electrolyte monitoring, practitioner responsible for prescription, and specialist input

RESPONSIBILITY FOR DIAGNOSIS: Information was collected on the Boston score about the signs, symptoms, CXR and other diagnostic tests for HF, date of diagnosis (the earliest definite mention of HF), and where this occurred (in the problem list of patient’s medical record, a text note in the medical record / endorsement on a
prescription / listed in a letter from the GP, in correspondence from a hospital e.g. admission sheet or discharge letter, or from a specialist e.g. outside of the hospital). (See section 3.2 of this chapter.)

INITIATION OF NEW MEDICATIONS AND PRACTITIONERS RESPONSIBLE:
The high likelihood that patients with HF will be admitted to hospital suggests that there may be significant changes to HF management outside of primary care. It was of interest to try to determine how many of the changes to patient management during this study were made by GPs, and how many could be attributed to specialists and whether there was any difference between the pre-intervention and the post-intervention time periods.

If the patient’s medical record indicated that their GP initiated the new prescription, then this was recorded as ‘GP’. If a hospital discharge letter or hospital clinic letter stated that a drug had been initiated, this was recorded as ‘Hospital’. If the patient had seen a specialist outside of the hospital setting who had initiated a prescription, this was recorded as ‘Specialist’. Any drug that was new in a patient’s list of prescriptions without any note in the medical records or letter from a hospital or specialist was noted as ‘Not recorded’.

There were few new prescriptions for these medications, so information on the initiating doctor is not shown by study arm but for the whole patient sample for pre-intervention and post-intervention time periods.

From records that have been reviewed, it is obvious that the NYHA classification system is not used in general practice despite widespread discussion of this system. The lack of NYHA classification does place a limitation in that it will not be apparent whether patients are in the class indicated for certain medication (although a number of prescriptions of β-blockers and / or spironolactone have been started while the patient has been in hospital. We are therefore relying on the hospital clinicians to prescribe appropriately). It is also used only rarely in hospital and specialist letters.

ELECTROLYTE TESTING AND INITIATION OF SPIRONOLACTONE:
Whenever a new prescription for spironolactone was identified in patients’ medical records, the date and values of K⁺ and Cr were recorded for the diagnostic tests done
immediately prior to and immediately after the date of the prescription. There were few patients who were initiated on spironolactone which makes comparisons difficult.

**SPECIALIST INPUT:** There were no initial plans to describe the information contained in discharge letters and letters from outpatient clinics. However in the late stages of the project when some of the initial data entry indicated that there was significant hospital initiation of some medications and VA recommenced data collection it seemed to be a useful adjunct to the study to collect examples of information that specialists gave. It is not known how representative the letters are that were collected. The extracts that are included were selected as they illustrate a wide range of the amount of information that was conveyed to GPs. The information given by specialists and HF nurses is compared with the information that was delivered in the small-group and Internet educational intervention. It was also of interest to see who assumed or was given responsibility for the management of these patients.

**A GP’S EXPERIENCE OF B-BLOCKERS:** An extract taken from a patient’s medical records was included as it demonstrated a clear example of knowledge being implemented in practice around the initiation of a β-blocker.

### 4.5.4 Post-hoc analyses – effect of listed diagnosis

Research that was done as the study progressed suggested that patients who had a definite diagnosis listed in their problem list were managed to a higher standard than patients who did not have a listed diagnosis. Given that information of the date of HF diagnosis had been collected, the patients were then categorised into groups according to their diagnosis date in relation to the date of their GP’s educational intervention.

HF diagnoses were grouped by date (using intervention date as the index date and comparing this against the date in the problem list) and these groups were compared with the study outcomes. Literature suggests that patients who have a diagnosis noted in their problem list will receive better care. The question was whether diagnosis date had any effect on the four outcomes identified for the study: Echocardiography, Angiotensin inhibition (ACEi or ARBs), β-blockers, and spironolactone.
The diagnosis date used was the date listed in the Problem List of the patient’s medical record as this would have been obvious to the GP each time they opened the record. The HF patients were split into four groups depending on the date when their HF diagnosis was listed in relation to the educational intervention date ¹.

The analysis used the GLIMMIX procedure and compared the four study outcomes across the three HF diagnosis groups (specified above), ‘early’, ‘mid’ and ‘late or no diagnosis’.

In the analysis of the effect of a listed diagnosis, the Odds Ratio (OR) was used to determine the likelihood of receiving each of the measured outcomes if a patient had a diagnosis of HF against the timing of this diagnosis. The ORs are generally reported with the 95% CIs in brackets after the OR.

The reference group for all HF diagnosis group analyses was the late or no diagnosis group (i.e. OR = 1). This patient group had documented evidence (clinical or objective) to suggest HF in the same way as the other two analysis groups. The pattern of OR for the analysis of the four study outcomes could not be predicted in advance; instead of changing the reference group for each analysis, the decision was made to hold it constant. There were also reference groups (see below) for the other variables tested (except age):

- Intervention: Internet
- Study time period: Current management
- Sex: Male
- MI
- CORD
- Asthma

¹ Some inaccuracies accompany this estimation as some dates for HF were only recorded by year and not by days and months. There are three possible reasons for this: a) the year was the only date recorded in the problem list, b) the instructions to research assistants who were collecting the data did not stipulate a dd/mm/yyyy format to collect this data (as the diagnosis date was less of a focus at the start of the study), and c) less than accurate data collection.

This meant that the decision had to be made to “force” the data (also because of later analysis requirements) into a format of dd/mm/yyyy. Thus any year of diagnosis that was in the years before the educational intervention received a proxy dd/mm date of 01/01/yyyy. If the educational intervention occurred at the end of the year (late December) and the diagnosis was listed only with a year, it was more likely that the diagnosis had been made in the days (approximately 350) preceding the date of the educational intervention. If the educational intervention date was early in the year, it was more likely that the diagnosis was made in the days after (approximately 300 to 270) the educational intervention.
The adjusted means were calculated from the data once the data were controlled for effects (clustering and other design variables). The adjusted means have CI calculated which gives an indication of the robustness and precision of the mean value. Raw means for each time period for the medication outcomes can be found in Appendices 5B, C and D.

The following variables were run in the model:

- Intervention
- Time
- Gender
- Age at first intervention
- MI
- CORD
- Asthma
- HF group.

The addition of further variables was constrained by the model’s ability to run. Too many variables resulted in the analysis failing to run.

The intervention effect was included to check if the HF diagnosis was independent of the intervention group. The intervention could act as a confounder on the diagnosis, echocardiography and medications and would lead to weakness in the model if not included. Time period was removed in the analysis of echocardiography as the model checked each time period to see if an echocardiogram had been given and then averaged the 1 (yes) or 0 (no) code which gave incorrect results (as echocardiography could only appear once in the data).

The effects were multiplicative so any effect further down the list that was significant was an effect over and above any significant effects that occurred higher in the list.

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b Time was not used as a variable in the analysis of echocardiography as the analysis was to look at whether an echocardiogram was given at all, not an average across the three time periods as was the case for the medication data.

c MI was used as a variable in the analysis of echocardiography. Patients who experience an MI are more likely to receive an echocardiogram as a diagnostic test to assess heart function.

d CORD was added to the model for analysis of echocardiography. The symptoms of CORD may obscure symptoms of HF and one of the few methods to determine cause is for patients to receive echocardiograms.

e Asthma was added to the model for analysis of echocardiography. Symptoms of breathlessness and wheeze may either be worsening asthma or HF.
4.5.5 Other – GP feedback

The minor outcomes of the study consist of the HF management survey first completed when the GPs were invited to participate in the study, repeated approximately three years later, and also the questionnaire of GPs’ experience and opinions of the educational intervention they participated in. Neither the survey nor the questionnaire was validated. The analysis of the two surveys and the questionnaire is mainly descriptive.

**LEVEL OF KNOWLEDGE OF HF MANAGEMENT:** GPs were surveyed (before and after the intervention) on their knowledge of current HF management and what changes they had implemented. The key responses that have been highlighted are knowledge of the study outcomes, implementation of the study outcomes, and problems with the implementation of these changes. Other areas of interest were what additional sources of information the GPs accessed, what proportion of patients they took responsibility for and which areas of management they preferred a specialist to initiate.

**GP EXPERIENCES/PROCESS EVALUATION:** GPs were asked to complete a questionnaire examining:

- their likes and dislikes of the intervention modality
- if they thought the intervention had changed their prescribing behaviour and what systems changes they had implemented e.g. computer prompts, utilisation of nurses
- whether they had done any further (independent) reading (and to give examples), and whether they had participated in other CME on HF,
- and if they had discussions with colleagues about HF management.

The questionnaire consisted of closed- and open-ended questions which the GPs completed in their own time. A structured questionnaire type format is preferable to an unstructured or semi-structured interview because standardisation of responses facilitates analysis.
This chapter will first describe the variables associated with the practices and GPs and the number of patients who participated in the study. The next section will give details of the patient characteristics including electrolyte values and comorbidities for the whole study as well as for each study arm. These sections establish the practice, GP and patient population demographics. Information on these populations is provided first because it informed the modelling for the analyses.

This chapter then proceeds to discuss the results of each outcome.

Section 3 discusses the primary outcomes: rates of echocardiography and the prescription of the three classes of medications of interest before and after the educational intervention.

Section 4 presents the secondary outcomes: results of the change over time seen in the doses of the medications of interest.

The tertiary outcomes are presented in section 5. These outcomes describe who had responsibility for the diagnosis of HF, how many initiations occurred in the study period and for spironolactone, how closely the electrolyte testing guidelines were followed. This section also presents which physician started the new medication prescribing, and what sort of information specialists gave to GPs in discharge summaries.

This chapter concludes with section 6 which discusses the effect of a listed diagnosis of HF on the four primary outcomes: echocardiography, ACEi prescription, β-blocker prescription and spironolactone prescription.

### 5.1. **GP and practice variables**

This section deals with information gathered from and about GPs which is relevant to the study and to the analysis of the outcomes.

#### 5.1.1 Practice and GP participation rate

The actual numbers of GPs who were recruited and randomised are shown in figure 4.2.b. 69 practices (177 GPs) agreed to participate and were randomised into the three
study arms. After randomisation each study arm contained 23 practices (59 GPs) (see table 4.2.a for the distribution of the practice sizes within the study arms after randomisation). Under assumption of normality every practice has an equal chance of going into any one of the three study arms. The randomisation was successful and acts to minimise differences between study arms. However the numbers in each study arm are not large.

At the conclusion of the study the control (guideline) group consisted of 8 practices (18 GPs), the small group consisted of 7 practices (17 GPs) and the Internet group consisted of 9 practices (18 GPs). Table 5.1.a reports for each study arm the response rate for both GPs and practices. The overall participation rate was 30% of GPs and 35% of practices.

<table>
<thead>
<tr>
<th>Table 5.1.a. Participation rate of GPs and practices by study arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (guideline)</td>
</tr>
<tr>
<td>GPs</td>
</tr>
<tr>
<td>Practices</td>
</tr>
</tbody>
</table>

Since the response rate was not 100% there may be some bias between practices. Dropout may be influenced by study arm. Randomisation was by practice so there may be differences in GP characteristics between the study arms. These differences have been investigated over the study period and are presented in section 5.1.2. Important factors that influence how the GPs prescribed have been investigated and were built into the model that analysed the effects of the intervention as described in chapter 4.

### 5.1.2 GP characteristics

Data gathered from participating GPs who had HF patients were compared with data about GPs who did not participate in this study. Where GPs did not return the demographic data sheet included with the questionnaire, information on qualifying year was obtained from the website of the Medical Council of New Zealand ([www.mcnz.org.nz/](http://www.mcnz.org.nz/)). The information listed on the MCNZ website was complex. The profile for each doctor included information on qualifications, scope of practice, vocational scope, general scope and provisional scope. Comparisons with the data

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*The guideline study arm acted as the control group. To simplify discussion and to clarify the different study arms, this group will be referred to as the guideline study arm.*
collected from GPs were not straightforward so the decision was made to compare only website data on year of graduation with an MBChB (or equivalent) for participating and non-participating GPs. (See table 5.1.b for qualification date as well as graduation date of participating GPs and table 5.1.c for graduation date of non-participating GPs.)

Table 5.1.b. Table of GPs’ sex and date of qualification as GP for those who had eligible HF patients

<table>
<thead>
<tr>
<th></th>
<th>Guideline n (%)</th>
<th>Small group n (%)</th>
<th>Internet n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td>12 (67%)</td>
<td>11 (65%)</td>
<td>12 (67%)</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>6 (33%)</td>
<td>6 (35%)</td>
<td>6 (33%)</td>
</tr>
<tr>
<td><strong>Qualified as GP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-1980</td>
<td>9 (50%)</td>
<td>6 (35%)</td>
<td>3 (17%)</td>
</tr>
<tr>
<td>1980 – 1989</td>
<td>7 (39%)</td>
<td>10 (59%)</td>
<td>8 (44%)</td>
</tr>
<tr>
<td>1990 – 1999</td>
<td>1 (6%)</td>
<td>1 (6%)</td>
<td>5 (28%)</td>
</tr>
<tr>
<td>2000 –</td>
<td>1 (6%)</td>
<td></td>
<td>2 (11%)</td>
</tr>
<tr>
<td><strong>Graduated</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-1980</td>
<td>11 (61%)</td>
<td>6 (35%)</td>
<td>3 (17%)</td>
</tr>
<tr>
<td>1980 – 1989</td>
<td>7 (39%)</td>
<td>11 (65%)</td>
<td>8 (44%)</td>
</tr>
<tr>
<td>1990 – 1999</td>
<td></td>
<td>5 (28%)</td>
<td>9 (22%)</td>
</tr>
</tbody>
</table>

Table 5.1.c. Table of GPs’ sex and time since graduation for non-participating GPs

<table>
<thead>
<tr>
<th></th>
<th>Guideline n (%)</th>
<th>Small group n (%)</th>
<th>Internet n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td>26 (63%)</td>
<td>25 (60%)</td>
<td>15 (37%)</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>15 (37%)</td>
<td>17 (40%)</td>
<td>26 (63%)</td>
</tr>
<tr>
<td><strong>Graduated</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-1980</td>
<td>14 (34%)</td>
<td>17 (40%)</td>
<td>12 (29%)</td>
</tr>
<tr>
<td>1980 – 1989</td>
<td>17 (41%)</td>
<td>16 (38%)</td>
<td>18 (44%)</td>
</tr>
<tr>
<td>1990 – 1999</td>
<td>10 (24%)</td>
<td>8 (19%)</td>
<td>9 (22%)</td>
</tr>
<tr>
<td>NR</td>
<td>1 (2%)</td>
<td></td>
<td>2 (5%)</td>
</tr>
</tbody>
</table>
The non-participating GPs in the guideline and small group arms displayed similar gender ratios to those seen in the participating GPs, however the ratio was reversed in the Internet arm favouring female GPs 2:1. The proportion of GPs in graduation years was more evenly distributed amongst the non-participating GPs than in the GPs who participated. The participating and the non-participating GPs were compared for the relationship between group and graduation. The association between non-participating GPs and graduation date was not different (P = 0.5). There was no association between non-participation and study arm (P = 0.8). There was no association between dropping out and graduation date (P = 0.4).

For comparison the data for all GPs who were randomised is also included. Once the GPs had been randomised, each of the study arms had a total of 59 GPs with data on their sex and graduation dates displayed in table 5.1.d.

Table 5.1.d. Table of GPs’ sex and time since graduation presented all randomised GPs

<table>
<thead>
<tr>
<th>Graduated</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>38 (64%)</td>
<td>36 (61%)</td>
<td>27 (46%)</td>
</tr>
<tr>
<td>Female</td>
<td>21 (36%)</td>
<td>23 (39%)</td>
<td>32 (54%)</td>
</tr>
<tr>
<td>Pre-1980</td>
<td>25 (42%)</td>
<td>23 (39%)</td>
<td>15 (25%)</td>
</tr>
<tr>
<td>1980 – 1989</td>
<td>24 (41%)</td>
<td>27 (46%)</td>
<td>26 (44%)</td>
</tr>
<tr>
<td>1990 – 1999</td>
<td>10 (17%)</td>
<td>8 (14%)</td>
<td>16 (27%)</td>
</tr>
<tr>
<td>NR</td>
<td>1 (2%)</td>
<td>2 (3%)</td>
<td></td>
</tr>
</tbody>
</table>

In the randomised group of GPs the gender ratios were similar in the guideline and small group arm, favouring male GPs, but the gender ratio was almost equal in the Internet arm. The Internet arm also tended to have more GPs who had graduated later than the small group and guideline study arms. These GPs may be older and less familiar with using the Internet as an educational modality. As the randomisation was at practice level, it is more likely that there will be imbalances.

The sex and length of time of study GPs were compared with national figures. Data for graduation was used as a proxy for length of time in practice which may skew the data slightly. As the table below shows there was a significant difference in the length of time in practice between the study GPs and the national sample.
The recruited GPs showed no difference in proportion of Fellowship status compared with the national sample (p = 0.5) but the GPs who participated were slightly more likely to be a Fellow of the College (p=0.045).

Table 5.1.f. Comparative data of vocational training.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Fellow</td>
<td>60.5%</td>
<td>60.1%</td>
<td>62.6%</td>
<td>57.9%</td>
<td>73.6%</td>
</tr>
</tbody>
</table>

5.1.3 Patients recruited into the study

The following tables divide the study population (practices, GPs and patients) by intervention arm and present the data by practice size (small, medium, largeb). The tables show how many practices, GPs and patients participated by practice size. (More detailed tables which display the number of patients per GP are in appendix 5A.) Differences in numbers of HF patients may be due to the characteristics of the GPs and the patient list they manage.

Table 5.1.g. Practices, GPs and patients in the guideline study arm

<table>
<thead>
<tr>
<th>Practice size</th>
<th>Participated</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
<td>GPs</td>
</tr>
<tr>
<td>Small</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Medium</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Large</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>TOTALS</td>
<td>8</td>
<td>18</td>
</tr>
</tbody>
</table>

b The practice sizes were defined as small with one GP, medium with two to three GPs and large as four or more GPs.
Table 5.1.h. Practices, GPs and patients in the small group study arm

<table>
<thead>
<tr>
<th>Practice size</th>
<th>Participated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
</tr>
<tr>
<td>Small</td>
<td>1</td>
</tr>
<tr>
<td>Medium</td>
<td>3</td>
</tr>
<tr>
<td>Large</td>
<td>3</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>7</strong></td>
</tr>
</tbody>
</table>

Table 5.1.i. Practices, GPs and patients in the Internet study arm

<table>
<thead>
<tr>
<th>Practice size</th>
<th>Participated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
</tr>
<tr>
<td>Small</td>
<td>2</td>
</tr>
<tr>
<td>Medium</td>
<td>2</td>
</tr>
<tr>
<td>Large</td>
<td>5</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>9</strong></td>
</tr>
</tbody>
</table>

There was considerable variation in the numbers of eligible HF patients per GP. The range of numbers of patients per GP was from one patient to 22 patients. This gives a mean number of approximately 7 HF patients per GP. Patient response rates for this study were high and ranged from 50% to 100%. Average response rate was 73%.

5.1.4 Contribution of GP / practice variables to analysis

The generalised linear mixed model used to investigate if the change over time in outcome (echocardiography, ACEi, β-blocker, and spironolactone) differed for patients across the three study arms included practice and GP variables as these could influence outcomes. (See Chapter 4 for further details.)

The GP variables included in the model are the sex of the GP and years since qualification as a GP. The practice variable used in the model was practice size.

The differences between male and female GPs would be (a) that male GPs would be more likely to treat older, male patients than would female GPs, and (b) that male GPs would be more likely to have been in practice longer, and as such have an older patient
As the practice dynamics change with practice size, this may also have an effect on the way GPs prescribe and also change their prescribing behaviour.

5.2 *Patient variables*

The patient variables consist of information collected on the data collection sheets titled ‘Patient Demographics’ and pre- and post-intervention ‘Patient Demographics’ and ‘Electrolytes’. The patient demographics illustrate the patient population. Information regarding patient age, sex, electrolytes and comorbidities was required for inclusion in the modelling for analyses of the outcomes. Comparison of the patients in the three intervention arms also indicated whether there were any differences between the groups that needed to be accounted for in the analyses or would need to be explained in the Discussion chapter. More details are given in Chapter 4.

The patient data are presented in this section with Standard Deviations (SD) expressed in units most appropriate for the particular measurement. Baseline characteristics for the patients are presented in tables 5.2.a, 5.2.b, 5.2.c and table 5.2.d. Table 5.2.a displays the results for age, sex and ethnicity, table 5.2.b displays results for systolic blood pressure, diastolic blood pressure and heart rate for both pre- and post-intervention, table 5.2.c displays results of electrolyte measurements (creatinine, sodium and potassium) for both pre- and post-intervention, and table 5.2.d displays results of recorded comorbidities.

5.2.1 *Age*

The ages for each group were compared and the differences were not statistically significant (P = 0.94).

5.2.2 *Sex*

In one instance this was not recorded, so it was inferred by checking the name of the patient. The proportions were almost equal overall and in each of the study arms (see table 5.2.a) and there was no significant difference (P = 0.30) between the groups. Sex was used as one of the variables in the model.
5.2.3 Ethnicity
Ethnicity data were collected from patients’ medical records. 19 patients did not have ethnicity data recorded. 5 patients had ‘Other’ recorded. When ethnicity was not recorded or left as ‘Other’, it was obtained from the ethnicity data supplied by patients on their consent forms. Only 5 patients did not have any ethnicity data in either medical records or consent forms.

The predominant ethnicity was ‘European’. The next most common ethnicity counted for only 4% of the total patient sample and as such the ethnicities are displayed as European and Other. The proportion of ‘Others’ found in the Guideline (control) arm was more than double that in the small group and Internet arms (see table 5.2.a). The ethnicity proportions were compared across groups. The differences were statistically significant (P = 0.0095). However the numbers of patients who were of non-European ethnicity were considered too small to have any effect on the study. The breakdown of the ethnicities listed above can be found in Appendix 5B.
Table 5.2.a. Baseline data (demographics, clinical characteristics) for entire patient sample, then by intervention arm for age, sex and ethnicity

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Patients N = 359</th>
<th>Guideline (control)a N = 137</th>
<th>Small-group N = 98</th>
<th>Internet N = 124</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years (SD)</td>
<td>76.3 (10.3)</td>
<td>76.1 (9.8)</td>
<td>76.8 (11.3)</td>
<td>75.9 (10.2)</td>
</tr>
<tr>
<td>(range, years)</td>
<td>(44.8 – 97.4)</td>
<td>(44.8 – 91.3)</td>
<td>(44.8 – 97.4)</td>
<td>(45.7 – 92.9)</td>
</tr>
<tr>
<td>Female n (%)</td>
<td>182 (51 %)</td>
<td>76 (56 %)</td>
<td>49 (50 %)</td>
<td>57 (46 %)</td>
</tr>
<tr>
<td>Ethnicity n (%):</td>
<td>European</td>
<td>315 (88 %)</td>
<td>111 (81 %)</td>
<td>90 (92 %)</td>
</tr>
<tr>
<td></td>
<td>All others</td>
<td>44 (12 %)</td>
<td>26 (19 %)</td>
<td>8 (8 %)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a The group of GPs who received the guideline only is referred to as the control group as they did not receive any ‘active’ intervention. In tables they will be referred to as Guideline to ensure clarity over which study arm is being discussed.
5.2.4 Blood Pressure
The median of the averaged BPs is presented here (see table 5.2.b). The SAS MIXED procedure (mixed linear models) was used to compare BPs by intervention, over time, intervention by time, sex and age (at first intervention).

Systolic blood pressure (SBP) decreased over time (p<0.001); there was no effect of intervention by time (p=0.12) but sex had an effect with women who had higher SBP (p=0.0009). Age at intervention was almost significant (p=0.057), suggesting that SBP may increase with age. The overall effect of intervention groups themselves was not significant (p=0.73). The values of SBP recorded for the guideline study arm were compared with small-group (p=0.11) and also with Internet (p=0.65) for the two time periods that BP had been collected.

Diastolic blood pressure (DBP) decreased over time (p<0.0001); intervention by time had no effects (p=0.93); sex had an effect with DBP in females being higher (p=0.015) and age at intervention had an effect with DBP decreasing with age (p=0.0048). The overall effect of the intervention groups themselves was not significant (p=0.12). No differences were seen between the groups over time (guideline vs. small-group p=0.96; guideline vs. Internet p=0.74).

The inclusion of BP as a variable in later models (GLIMMIX) destabilised the models to such a degree that BP was removed. The lack of interaction seen in the analysis above is reassuring that BP would not have had any major effect on the results of the later models.

5.2.5 Heart Rate
The median of the averaged HRs is presented in table 5.2.b. The SAS MIXED procedure (mixed linear models) was used to compare HR by intervention, over time, intervention by time, gender and age (at first intervention).

The overall effect of the intervention group was not significant (p=0.82). No differences were seen between the groups over time (guideline vs. small-group p=0.21; guideline vs. Internet p=0.1). No associations were seen with the variables and HR tested although sex did come close to significance (p=0.07).
Table 5.2.b. Pre- and post-intervention blood pressures (systolic and diastolic) and heart rates

<table>
<thead>
<tr>
<th>BP and HR</th>
<th>All Patients (N = 359)</th>
<th>Guideline (N = 137)</th>
<th>Small-group (N = 98)</th>
<th>Internet (N = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-Intervention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP mmHg (± SD) (range)</td>
<td>136 (18) (95 – 205)</td>
<td>135 (18) (95 – 205)</td>
<td>139 (19) (95 – 196)</td>
<td>135 (16) (95 – 180)</td>
</tr>
<tr>
<td>Diastolic BP mmHg (± SD) (range)</td>
<td>78 (9) (45 – 110)</td>
<td>79 (9) (55 – 110)</td>
<td>75 (10) (45 – 105)</td>
<td>78 (9) (55 – 104)</td>
</tr>
<tr>
<td>Missing data a</td>
<td>9 patients</td>
<td>1 patient</td>
<td>5 patients</td>
<td>3 patients</td>
</tr>
<tr>
<td>Heart Rate bpm (± SD) (range)</td>
<td>72 (11) (40 – 112)</td>
<td>75 (11) (40 – 108)</td>
<td>72 (11) (48 – 112)</td>
<td>70 (11) (50 – 110)</td>
</tr>
<tr>
<td>Missing data</td>
<td>97 patients</td>
<td>32 patients</td>
<td>41 patients</td>
<td>24 patients</td>
</tr>
<tr>
<td><strong>Post-Intervention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP mmHg (± SD) (range)</td>
<td>133 (18) (88 – 183)</td>
<td>132 (18) (98 – 183)</td>
<td>130 (18) (90 – 170)</td>
<td>135 (18) (88 – 181)</td>
</tr>
<tr>
<td>Diastolic BP mmHg (± SD) (range)</td>
<td>75 (9) (45 – 110)</td>
<td>76 (9) (57 – 110)</td>
<td>75 (10) (45 – 100)</td>
<td>75 (9) (49 – 100)</td>
</tr>
<tr>
<td>Missing data</td>
<td>2 patients</td>
<td>0 patients</td>
<td>1 patient</td>
<td>1 patient</td>
</tr>
<tr>
<td>Heart Rate bpm (± SD) (range)</td>
<td>72 (11) (51 – 130)</td>
<td>74 (9) (52 – 100)</td>
<td>72 (11) (59 – 108)</td>
<td>72 (13) (51 – 130)</td>
</tr>
<tr>
<td>Missing data</td>
<td>120 patients</td>
<td>39 patients</td>
<td>40 patients</td>
<td>41 patients</td>
</tr>
</tbody>
</table>

a Missing data refers to patients who had no blood pressures recorded or no heart rates recorded.
The inclusion of HR as a variable in later models (GLIMMIX) destabilised the models to such a degree that HR was removed. A lack of interaction seen in the analysis described above is reassuring because it indicates that HR would not have had any major effect on the results of the later models.

5.2.6 Electrolytes: Creatinine, Sodium, Potassium

The data are displayed in table 5.2.c.

5.2.6.1 Creatinine

Some outliers skewed the data so prior to analysis the log of Creatinine (Cr) was taken to better satisfy the assumptions of normality. There was an association with time (p=0.002) with Cr increasing over time but not by intervention (p=0.09). Gender was significant with Cr lower in women (p<0.001) as was age at intervention (p=0.04) with Cr levels increasing as age increased.

The overall effect of intervention groups was not significant (p=0.84). There was a difference between guideline and small group values over time (p=0.03) but not between the guideline and Internet values (p=0.69).

In the pre-intervention period, 114 patients in the guidelines arm had Cr measured as did 83 in the small-group arm and 107 in the Internet arm. No patients had Cr <0.05mmol/l. In the guideline group 22% of patients had Cr >0.12mmol/l, with 19% in the small-group arm and 20% of patients in the Internet arm. If the patients who had a Cr measurement 0.1 to 0.12mmol/l are included, the numbers of patients doubled in each study arm.

In the post-intervention period Cr was measured in 126 guideline patients, 93 small-group patients and in 106 Internet patients. Of these no patients had Cr measurement below the lower reference value but more patients had Cr >0.12mmol/l, with 23% in the guidelines group, 32% in the small-group, and 25% in the Internet group. The proportions of patients who had Cr in the 0.1 to 0.12mmol/l range were less than those >0.12mmol/l, but when these were added together, the percentages of patients who had high Cr ranged from 45% to 52% for patients in the three study arms.
5.2.6.2 Sodium
Laboratory-reported Na\(^+\) reference ranges showed only slight variation from 134 to 148mmol/l to 135 to 145mmol/l. In the pre- and post-intervention period no patients had values >148mmol/l and only a few patients had values less than 134 mmol/l.

The only association seen for Na\(^+\) values was time (p=0.0014) with Na\(^+\) increasing over time. There were no differences between the intervention groups. No associations were seen comparing different intervention arms over time.

5.2.6.3 Potassium
Laboratory-reported reference intervals ranged from 3.5 to 5.0mmol/l to 3.5 to 5.3mmol/l. In the pre-intervention study period only one patient had a K\(^+\) value below 3.5mmol/l and a total of 13 patients had values >5.3mmol/l but by the post-intervention period five patients had a K\(^+\) value >5.3mmol/l and four patients had values recorded <3.5mmol/l.

The only association seen with K\(^+\) was gender (p=0.04) with K\(^+\) lower in females, and there was no difference in the comparison of intervention groups over time or in the intervention groups themselves.
Table 5.2.c. Median values for electrolytes measured pre-intervention and post-intervention

<table>
<thead>
<tr>
<th>Electrolytes</th>
<th>All Patients</th>
<th>Guideline</th>
<th>Small-group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 359</td>
<td>N = 137</td>
<td>N = 98</td>
<td>N = 124</td>
</tr>
<tr>
<td><strong>Pre-Intervention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine mmol/L (± SD)</td>
<td>0.098 (0.036)</td>
<td>0.099 (0.039)</td>
<td>0.098 (0.035)</td>
<td>0.095 (0.032)</td>
</tr>
<tr>
<td>(range)</td>
<td>(0.050 – 0.333)</td>
<td>(0.050 – 0.333)</td>
<td>(0.055 – 0.316)</td>
<td>(0.056 – 0.250)</td>
</tr>
<tr>
<td>Missing data</td>
<td>55 patients</td>
<td>23 patients</td>
<td>15 patients</td>
<td>17 patients</td>
</tr>
<tr>
<td>Sodium mmol/L (± SD)</td>
<td>141 (3)</td>
<td>141 (3)</td>
<td>141 (3)</td>
<td>141 (3)</td>
</tr>
<tr>
<td>(range)</td>
<td>(130 – 147)</td>
<td>(133 – 147)</td>
<td>(131 – 145)</td>
<td>(130 – 147)</td>
</tr>
<tr>
<td>Missing data</td>
<td>62 patients</td>
<td>26 patients</td>
<td>17 patients</td>
<td>19 patients</td>
</tr>
<tr>
<td>Potassium mmol/L (± SD)</td>
<td>4.40 (0.50)</td>
<td>4.60 (0.47)</td>
<td>4.40 (0.55)</td>
<td>4.30 (0.49)</td>
</tr>
<tr>
<td>(range)</td>
<td>(3.40 – 6.00)</td>
<td>(3.60 – 5.80)</td>
<td>(3.40 – 5.70)</td>
<td>(3.60 – 6.00)</td>
</tr>
<tr>
<td>Missing data</td>
<td>60 patients</td>
<td>27 patients</td>
<td>16 patients</td>
<td>17 patients</td>
</tr>
<tr>
<td><strong>Post-Intervention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine mmol/L (± SD)</td>
<td>0.099 (0.050)</td>
<td>0.097 (0.069)</td>
<td>0.104 (0.032)</td>
<td>0.099 (0.035)</td>
</tr>
<tr>
<td>(range)</td>
<td>(0.050 – 0.692)</td>
<td>(0.050 – 0.692)</td>
<td>(0.058 – 0.210)</td>
<td>(0.058 – 0.267)</td>
</tr>
<tr>
<td>Missing data</td>
<td>34 patients</td>
<td>11 patients</td>
<td>5 patients</td>
<td>18 patients</td>
</tr>
<tr>
<td>Sodium mmol/L (± SD)</td>
<td>141 (3)</td>
<td>141 (3)</td>
<td>140 (3)</td>
<td>140 (3)</td>
</tr>
<tr>
<td>Missing data</td>
<td>38 patients</td>
<td>14 patients</td>
<td>5 patients</td>
<td>19 patients</td>
</tr>
<tr>
<td>Potassium mmol/L (± SD)</td>
<td>4.40 (0.46)</td>
<td>4.50 (0.44)</td>
<td>4.40 (0.47)</td>
<td>4.50 (0.46)</td>
</tr>
<tr>
<td>(range)</td>
<td>(2.90 – 5.80)</td>
<td>(3.60 – 5.70)</td>
<td>(2.90 – 5.30)</td>
<td>(3.30 – 5.80)</td>
</tr>
<tr>
<td>Missing data</td>
<td>38 patients</td>
<td>14 patients</td>
<td>6 patients</td>
<td>18 patients</td>
</tr>
</tbody>
</table>
5.2.7 Comorbidities

Additional diagnoses were recorded from the Problem List in medical records (either electronic or paper) and are presented in table 5.2.d. The diagnoses’ group headings are listed in appendix 4O as well as a breakdown of the free-text used to describe other diagnoses. Dates were noted when they occurred. However, as with HF diagnosis dates, there is no guarantee that this is the date when the problem was first diagnosed. 1996 was chosen as the starting date as this was approximately 10 years before the ‘Current Management’ data and five years prior to the earliest data collection.

The major diagnoses recorded in medical records showed that approximately two-thirds of patients had hypertension (HTN) listed, the majority of these had a start date listed as after 1996. Missing diagnosis date was observed in 8% of the guideline and the Internet groups, and almost a third of diagnoses of HTN in the small-group study arm did not have diagnosis date listed.

For diabetes, the guideline group and the small group patients were diagnosed in almost equal numbers before and after 1996. 23% and 36% had no diagnosis date. The Internet group had 7% of diabetics without a diagnosis date with about 80% of diabetics diagnosed in 1996 or later.

The majority of patients with atrial fibrillation (AF) were diagnosed in 1996 or later (76 to 85% across the study arms), and a similar pattern was seen with ischaemic heart disease (IHD) where 61 to 74% of patients were diagnosed in 1996 or later.

Few patients with HF had had this diagnosed prior to 1996 (11.5% guideline, 3% small-group, 4% Internet), with the majority of diagnoses between 1996 and 2004 (68%, 79%, 85%) although a few were diagnosed in 2005 and later (9%, 6%, 6%). The proportion of patients who had HF listed but no diagnosis date given ranged across the three study arms from 11.5%, 12% to 4%.

There was a variation between the study arms in the proportions of patients reported with previous myocardial infarction (MI). Only a few ever experienced multiple MIs (three guidelines arm patients, two in the small-group arm, and five in the Internet arm). Approximately a quarter of MIs were experienced by guideline patients and small group patients before 1996, but 46% of MIs occurred in the Internet group in this period. From 1996 onwards most MIs occurred in the guideline group (56%) and small-group arm (62%). Almost a quarter of recorded MIs in the guideline group had no date given, and approximately 14% of the small-group and of the Internet arm MIs had no date recorded.

P-values were calculated for comorbidities found in >10% of the patients (see table 5.2.d).
Table 5.2.d. Comorbidities recorded at the time of data collection

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>All Patients n (%)</th>
<th>Guideline n (%)</th>
<th>Small-group n (%)</th>
<th>Internet n (%)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>227 (63%)</td>
<td>86 (63%)</td>
<td>61 (62%)</td>
<td>80 (65%)</td>
<td>0.93</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>100 (28%)</td>
<td>31 (23%)</td>
<td>25 (26%)</td>
<td>44 (35%)</td>
<td>0.06</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>135 (38%)</td>
<td>51 (37%)</td>
<td>38 (39%)</td>
<td>46 (37%)</td>
<td>0.96</td>
</tr>
<tr>
<td>Ischaemic Heart Disease</td>
<td>203 (57%)</td>
<td>78 (57%)</td>
<td>56 (57%)</td>
<td>69 (56%)</td>
<td>0.97</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>257 (72%)</td>
<td>96 (70%)</td>
<td>65 (66%)</td>
<td>96 (77%)</td>
<td>0.18</td>
</tr>
<tr>
<td>Myocardial Infarction</td>
<td>92 (26%)</td>
<td>18 (13%)</td>
<td>37 (38%)</td>
<td>37 (30%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Other Diagnoses(^b)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CORD</td>
<td>87 (24%)</td>
<td>23 (17%)</td>
<td>28 (29%)</td>
<td>35 (28%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Asthma</td>
<td>54 (15%)</td>
<td>23 (17%)</td>
<td>13 (13%)</td>
<td>18 (15%)</td>
<td>0.74</td>
</tr>
<tr>
<td>Lipid Disorder</td>
<td>67 (19%)</td>
<td>28 (20%)</td>
<td>15 (15%)</td>
<td>24 (19%)</td>
<td>0.59</td>
</tr>
<tr>
<td>Renal Impairment</td>
<td>41 (11%)</td>
<td>18 (13%)</td>
<td>8 (8%)</td>
<td>15 (12%)</td>
<td>0.48</td>
</tr>
<tr>
<td>Smoker (current or previous)</td>
<td>28 (8%)</td>
<td>10 (7%)</td>
<td>6 (6%)</td>
<td>12 (10%)</td>
<td></td>
</tr>
<tr>
<td>Rhythm Disturbance</td>
<td>24 (7%)</td>
<td>11 (8%)</td>
<td>4 (4%)</td>
<td>9 (7%)</td>
<td></td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>16 (5%)</td>
<td>7 (5%)</td>
<td>5 (5%)</td>
<td>4 (3%)</td>
<td></td>
</tr>
<tr>
<td>Diastolic Dysfunction</td>
<td>5 (1.4%)</td>
<td>1 (0.7%)</td>
<td>0</td>
<td>4 (3%)</td>
<td></td>
</tr>
<tr>
<td>Blood / Endocrine Disorders</td>
<td>7 (2%)</td>
<td>3 (2%)</td>
<td>2 (2%)</td>
<td>2 (1.6%)</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>31 (9%)</td>
<td>14 (10%)</td>
<td>8 (8%)</td>
<td>9 (7%)</td>
<td></td>
</tr>
<tr>
<td>Alcohol Misuse</td>
<td>6 (2%)</td>
<td>2 (1.5%)</td>
<td>0</td>
<td>4 (3%)</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) These co-morbidities were specified in the Demographics data collection sheet.

\(^b\) Suggestions were given in the notes for the Demographic data collection sheet of co-morbidities that could affect HF. Differences in the ways these were recorded by the GPs and interpreted by the research assistants may cause this data set to be less accurate than the prior data set where the actual diagnosis was specified. See appendix 4O for examples of the free-text collected for ‘Other’ diagnoses.
5.2.8 Contribution of patient variables to model

Important patient variables that could have an effect on the management of HF were used in the model. Information that was used in the model was patient age at the date of the first intervention, gender, MI, CORD, asthma, and renal impairment. These were included because of their potential effect on the prescription of medication and because of the wide range in proportions of reported co-morbidities. SBP was added to the model, but addition of DBP and HR destabilised the model i.e. it would not run properly and these variables were removed. Earlier comparisons showed no differences between groups, which potentially could have resulted in GPs treating patients differently.

The above patient variables were included in the models of analysis for the following reasons. A younger patient could potentially be treated more aggressively than an older patient. A female patient may have less obvious signs of HF and therefore be treated more tentatively. Patients who had already experienced an MI may also be treated more aggressively, with a hospital or specialist initiating medication that a GP may be reluctant to alter. Patients with CORD or asthma may be less likely to receive a β-blocker, and patients with renal impairment may be less likely to receive an ACEi.

5.3 Primary outcomes – echocardiography and medications

**DIAGNOSIS AND ECHOCARDIOGRAPHY:** This section will focus on who first made the diagnosis of HF and when the diagnosis had been made. This will lead into analysis of the first study outcome, echocardiography, testing for a pattern of change over time and for an interaction between time period and study arm.

**MEDICATIONS OF INTEREST:** The prescription of the medications of interest (ACEi plus Angiotensin II Receptor blockers, β-blockers approved for use in heart failure, and spironolactone), was recorded in the pre-intervention and post-intervention periods and at 3 years after the intervention (Current Management). The pre-intervention data collection time period was the 18 months before the GP participated in the educational intervention and this 18 month time period spanned from mid-2001 to early 2003. The post-intervention data collection time period was in the 18 months after the GP participated in the educational intervention and this spanned from mid-2004 to late 2005. The data for current management were collected from medical records at 3 years after the educational intervention was given (± a few months if all relevant
prescriptions were not given at the same visit) and this time period was from late 2005 to almost mid-2006. There were 359 patients in the study. Data were collected from 359 patients in the pre- and post-intervention time periods but only from 357 patients for current management as records for two patients could not be located. The guideline study arm consisted of 137 patients, the small group study arm consisted of 98 patients (two of whom are missing from the current management time period) and the Internet study arm consisted of 124 patients.

The analysis in this section investigated whether the implementation strategies were effective in changing the study outcomes over time. The modelling performed on the prescription data looked at binary outcomes – whether the drug was given or not. The model also investigated if the change over time in outcome differed for patients in the three study arms. If the interaction between time period and study arm is significant this indicates that the pattern of change over time is different for the three implementation strategies.

The tables for each therapeutic class are displayed as the data for the entire patient sample, then by study arm, for rates of prescription over the study period.

### 5.3.1 Rates of Echocardiography

These figures of echocardiography received are expressed in the table below as numbers and proportions of all patients and then by study arm.

Table 5.3.a. Patients receiving first-time echocardiogram over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>All patients (N = 359)</th>
<th>Guideline (n = 137)</th>
<th>Small group (n = 98)</th>
<th>Internet (n = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>185 (51.5%)</td>
<td>65 (47%)</td>
<td>52 (53%)</td>
<td>68 (55%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>15 (4%)</td>
<td>4 (3%)</td>
<td>3 (3%)</td>
<td>8 (6%)</td>
</tr>
<tr>
<td>Current Management</td>
<td>9 (2.5%)</td>
<td>4 (3%)</td>
<td>4 (4%)</td>
<td>1 (1%)</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>209 (58%)</strong></td>
<td><strong>73 (53%)</strong></td>
<td><strong>59 (60%)</strong></td>
<td><strong>77 (62%)</strong></td>
</tr>
</tbody>
</table>

The analysis of binary outcomes (had an Echocardiography or not) found no significant interaction between time and group, indicating no difference in the pattern or change over time in rate of echocardiography in the three study arms (see table 5.3.a).
The only significant associations seen were with time (P < 0.0001), age at first intervention (P = 0.0002) where as age increased the chance of having an echocardiogram decreased, and a greater chance of receiving echocardiography if the patient had experienced an MI (P = 0.015).

The model outputs indicated no effect due to study arm (P = 0.90). No effect was seen in the interaction of study group by time period (P = 0.57). The effects of the remaining patient variables of gender (P = 0.98), renal impairment (P = 0.86), CORD (P = 0.65) and asthma (P = 0.95) were not significant. The rate of echocardiography was not significant for the GP or practice variables of practice size (P = 0.81), GP gender (P = 0.63) and years since qualification (P = 0.68).

The majority of echocardiographs were performed before the educational intervention, and by the end of the study HF remained unconfirmed in almost half the study population.

The echocardiography reports should simplify the diagnosis of HF and this is generally the case. However there is a lack of a consistent standard EF that signifies compromised function and is indicative of systolic HF. The reports that do not state EF but instead give a qualitative or descriptive report of the heart’s function are very difficult to interpret. The following table indicates the number of patients at different measured EFs and any other information that was reported.

Table 5.3.b. Reported results for the 209 first-time echocardiograms

<table>
<thead>
<tr>
<th>Ejection fraction</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 60 %</td>
<td>21</td>
</tr>
<tr>
<td>59 – 50 %</td>
<td>22</td>
</tr>
<tr>
<td>49 – 40 %</td>
<td>26</td>
</tr>
<tr>
<td>39 – 30 %</td>
<td>24</td>
</tr>
<tr>
<td>29 – 20 %</td>
<td>19</td>
</tr>
<tr>
<td>&lt; 20 %</td>
<td>6</td>
</tr>
<tr>
<td>‘N’ (normal not further defined)</td>
<td>42</td>
</tr>
<tr>
<td>NR (i.e. no EF stated)</td>
<td>20</td>
</tr>
<tr>
<td>Descriptive</td>
<td>29</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>209</strong></td>
</tr>
</tbody>
</table>
Out of the 20 patients who did not have any EF recorded, 12 of these had Fractional Shortening (FS) noted, although only four patients had an FS \( \leq 28\% \) which is generally indicative of HF.

The descriptive echocardiography reports were sometimes useful in that “very severe impairment of LV function” is clear. Other grades of impairment ranged from “severe” to “severe to moderate”, “moderate”, “mild to moderate” to “mild” to “low normal”. Other descriptions were simply “impaired”, “reasonable function”, and “LVH and LVD”. Without any accompanying reference of what these descriptions could equate to in terms of heart function, the diagnostic process for the GP becomes more difficult as do any future comparisons of echocardiograms.

5.3.1.1 Other diagnostic tests: NT-ProBNP

NT-ProBNP was used sparingly by GPs. Only 23 GPs in the study used BNP and 50 patients had at least one BNP test. It is not known what effect the consistent warnings (see example below) from LabPLUS had on GPs’ use of BNP.

<table>
<thead>
<tr>
<th>NT-ProBNP: 129 pmol/L ( &lt;35 ) H</th>
</tr>
</thead>
<tbody>
<tr>
<td>Please refer to the LabPLUS Electronic Handbook for interpretive information.</td>
</tr>
<tr>
<td>The cost of a BNP assay is $50.</td>
</tr>
<tr>
<td>Labplus currently spends more on BNP than any other single assay......</td>
</tr>
<tr>
<td>Please restrict requests for this test to patients in whom it is likely to provide critical information.</td>
</tr>
</tbody>
</table>

Figure 5.3.a. Note regarding cost of BNP tests, recorded on test results from late 2005

One patient had a BNP test in 2000 which would suggest that the patient was involved in early BNP trials. All other patients had BNP tests done from 2003 onwards which is when BNP testing became available for use.

In the results that were returned, three patients had ‘N’ recorded and one patient had a BNP test done but no value recorded. Seven patients had BNP values <40 pmol/L, 24 patients had values that fell in between normal and indicative of HF and 15 patients recorded values indicative of HF, three of these with values >1000 pmol/L. Apart from the test that did not have a value recorded, all these test outcomes provide critical

---

*a The reference range changed over the course of the study with a very early test listing normal as 0 – 50 pmol/L and HF > 150 pmol/L. In early 2003 these values changed to < 40 pmol/L being normal and > 220 pmol/L indicative of HF. This then changed to only the lower value being given as a reference, which then changed to < 35 pmol/L being normal and > 212 pmol/L indicative of HF. This still leaves a grey zone above the normal value and below the value which is considered to be indicative of HF.
information as they indicate dilation of and stress in the heart which cannot be determined clinically. A numerical value rather than ‘N’ may be more helpful.

5.3.2 Angiotensin inhibition

This section refers to both ACE inhibitors and Angiotensin II blockers (ARBs). ARBs were recommended as substitute drugs for patients who were intolerant of ACE inhibitors. Their combined prescription was not recommended. All tables are expressed as n (%) unless otherwise stated.

5.3.2.1 ACE inhibitors

The baseline proportion of patients receiving ACE inhibitors was reasonable at 73%.

Table 5.3.c. Overall ACE inhibitor prescription over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>N (%) of patients</th>
<th>All patients (n = 359)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>261 (73 %)</td>
<td></td>
</tr>
<tr>
<td>Post-intervention</td>
<td>250 (70 %)</td>
<td></td>
</tr>
<tr>
<td>Current Management</td>
<td>223 (63 %)</td>
<td></td>
</tr>
</tbody>
</table>

Over the study period (see table 5.3.c.), there was an overall decrease in ACE inhibitor prescribing of 10 percentage points. The difference between pre-intervention and post-intervention was -3%, and between post-intervention and current management -7%.

When the data were split into study arm (table 5.3.d.) the pattern of relatively high rates of ACEi prescription at the start of the study which decreased over time remained for all three study arms.

Table 5.3.d. ACE inhibitor prescription over time for each study arm

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline (n = 137)</th>
<th>Small group (n = 98)</th>
<th>Internet (n = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>97 (71 %)</td>
<td>82 (84 %)</td>
<td>82 (66 %)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>94 (69 %)</td>
<td>74 (76 %)</td>
<td>82 (66 %)</td>
</tr>
<tr>
<td>Current Management</td>
<td>87 (64 %)</td>
<td>64 (67 %)</td>
<td>72 (58 %)</td>
</tr>
</tbody>
</table>
Across the three study arms, the baseline rate of ACE inhibitor prescription differed between groups by 18 percentage points. When modelled, the effect of the intervention groups themselves was not significant (P = 0.28). The change in prescription of ACEi over time period was highly significant (P < 0.0001), but the interaction between time period and group was not significant (P = 0.13), indicating that there was no difference in the pattern of change over time in the three groups (see Table 5.3.d.).

The outcomes of modelling the patient variables found that there was a significant effect due to sex with higher prescription in males (80%) than in females (71%, P = 0.025). There was also an effect due to age with ACEi prescription reducing with increasing age at first intervention (P = 0.024). The prescription of ACEi for MI had a positive association but this was not significant (P =0.73). Renal impairment did not reach statistical significance (P = 0.087) however the association was negative for patients without renal impairment. The association of ACEi prescription was negative for patients without CORD but not significant (P = 0.69) but negative for patients without asthma, but not significant (P = 0.22).

None of the GP or practice variables that were tested reached significance but the trends were as follows. The association of ACEi prescription with increasing practice size was negative (P = 0.39). Prescription of ACEi was seen to be higher for female GPs (P = 0.9). The prescription of ACEi was negatively associated with increasing length of time since qualification (P = 0.32).

The changes over time between the three study arms seemed quite divergent. The decrease over time was 7% (guideline/control), 17% (small group), and 8% (Internet).

Table 5.3.e. Changes in ACEi prescription between time periods for each group

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre- to post-intervention</td>
<td>-2 %</td>
<td>-8 %</td>
<td>0 %</td>
</tr>
<tr>
<td>Post-intervention to current management</td>
<td>-5 %</td>
<td>-9 %</td>
<td>-8 %</td>
</tr>
</tbody>
</table>

When considering the change by time period, the small group showed the greatest decrease between the pre- and post-intervention period. All groups showed a decrease in ACEi prescription from post-intervention to current management. However the across group comparison of change over time did not show any significant difference.
The change over time in each of the two active interventions was compared with the change over time in the control arm. In the pre- to post-intervention time period, the change in small group compared with guideline group was significant (P = 0.0375) but the change in Internet group compared with control was not (P = 0.8295). Neither of the comparisons in the post-intervention to current management time period (control vs. small group P = 0.2630; control vs. Internet P = 0.5272) were significant.

5.3.2.2  **Angiotensin II receptor blockers**

Few patients were prescribed angiotensin II receptor blockers (ARBs). The numbers of patients prescribed ARBs are displayed below.

<table>
<thead>
<tr>
<th>Table 5.3.f</th>
<th>ARB prescription over time</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time period</strong></td>
<td><strong>N (%) of patients</strong></td>
</tr>
<tr>
<td></td>
<td><strong>All patients</strong></td>
</tr>
<tr>
<td></td>
<td>N = 359</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>25 (7%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>30 (8%)</td>
</tr>
<tr>
<td>Current Management</td>
<td>28 (8%)</td>
</tr>
</tbody>
</table>

The overall prescription of ARBs remained static over the study period. The pattern in each study arm was similar with only small increases and decreases in prescription over time. The number of ARB prescriptions was too small to allow meaningful interpretation so these patients were combined with patients prescribed ACE inhibitors.

5.3.2.3  **Combined Angiotensin inhibition**

The combined numbers of ACEi and ARB prescriptions became the standard group for analysis of angiotensin inhibition since they were prescribed for the same indication and analysis of the small numbers prescribed ARBs was not possible. The combined prescriptions show a baseline of 80% of patients prescribed some form of angiotensin blockade\(^1\). Table 5.3.g shows prescription of angiotensin inhibition over time.

---

\(^1\) A few patients are represented in both ACEi and ARB figures since some patients would have had their prescription for an ACEi stopped and a prescription for an ARB initiated during a time period. It is not possible to determine whether a patient was switched from an ACEi to an ARB as data were not collected about the sequence of medications within a time period and it may not have been possible to determine
A similar pattern of decreasing prescription over time was seen in each study arm.

Table 5.3.g. Combined ACEi and ARB prescription over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>N (%) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All patients (n = 359)</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>286 (80 %)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>280 (78 %)</td>
</tr>
<tr>
<td>Current Management</td>
<td>251 (70 %)</td>
</tr>
</tbody>
</table>

The differences between the intervention groups were not significant (P = 0.1016). The reduction in prescription over time was still significant (P = 0.0015), but there was no significance in the interaction between time period and intervention group (P = 0.1343).<sup>c</sup>

Table 5.3.h. Prescription of combined Angiotensin inhibition for each study group over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>N (%) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Guideline (n = 137)</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>106 (77%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>105 (77%)</td>
</tr>
<tr>
<td>Current Management</td>
<td>99 (72%)</td>
</tr>
</tbody>
</table>

The differences between the intervention groups were not significant (P = 0.0091) and prescription of angiotensin inhibiting agents reducing with increasing age (P = 0.0010). While the association between experiencing an MI and ACEi/ARB prescription was positive it was not significant (P = 0.73). However the effect of renal this. It is probably unlikely that a patient was switched from an ARB to an ACEi. Some patients may have been prescribed an ACEi and ARB blocker simultaneously. The numbers of patients who may be counted twice (by the data collection not capturing change from one to another during one time period) will be small and have negligible effect on the outcomes.<sup>c</sup>

The adjusted means for ACEi/ARB prescription show less difference across baseline and over time.

<table>
<thead>
<tr>
<th>Adjusted means</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>84 %</td>
<td>91 %</td>
<td>75 %</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>84 %</td>
<td>88 %</td>
<td>78 %</td>
</tr>
<tr>
<td>Current management</td>
<td>82 %</td>
<td>80 %</td>
<td>71 %</td>
</tr>
</tbody>
</table>
impairment did become significant ($P = 0.0227$) with higher prescription seen in patients with renal impairment. There was a positive but not significant association with CORD ($P = 0.66$) and a negative but not significant effect of asthma ($P = 0.25$). The GP and practice variables did not run with this model.

Tracking the change in prescription over time in the raw data showed large differences between the study arms, however the adjusted data show markedly less variation. (See also table in footnote.)

Table 5.3.i. Changes in prescription of angiotensin inhibitors between time periods (raw and adjusted data)

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th></th>
<th>Small group</th>
<th></th>
<th>Internet</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Raw</td>
<td>Adj</td>
<td>Raw</td>
<td>Adj</td>
<td>Raw</td>
<td>Adj</td>
</tr>
<tr>
<td>Pre- to post-intervention</td>
<td>0%</td>
<td>0%</td>
<td>-6%</td>
<td>-3%</td>
<td>+1%</td>
<td>+3%</td>
</tr>
<tr>
<td>Post-intervention to current management</td>
<td>-5%</td>
<td>-2%</td>
<td>-15%</td>
<td>-8%</td>
<td>-8%</td>
<td>-7%</td>
</tr>
</tbody>
</table>

Comparison of changes over time (adjusted data) showed borderline significant difference in pre- to post-intervention change for the comparison of guideline vs. small-group ($P = 0.052$) but no significance for guideline vs. Internet ($P = 0.84$). There were no significant differences in comparisons of post-intervention to current management (guideline vs. small-group $P = 0.088$, guideline vs. Internet $P = 0.28$).

### 5.3.3 β-blockers

The number of HF patients who at baseline were prescribed either metoprolol succinate or carvedilol was low at 21%. These data are from prescriptions written between mid-2001 and early 2003. The proportion did rise by 12 percentage points over time, but was still low by the end of the study (data collected for late 2005 to early 2006).

Table 5.3.j. Overall β-blocker prescription over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>N (%) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All patients (n = 359)</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>76 (21 %)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>91 (25 %)</td>
</tr>
<tr>
<td>Current Management</td>
<td>117 (33 %)</td>
</tr>
</tbody>
</table>
The increase in prescription of β-blockers over time was also seen in each of the three study arms (table 5.3.k).

Table 5.3.k. β-blocker prescription over time for each study arm

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline (n = 137)</th>
<th>Small group (n = 98)</th>
<th>Internet (n = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>(18%)</td>
<td>(16%)</td>
<td>(27%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>(21%)</td>
<td>(25%)</td>
<td>38 (31%)</td>
</tr>
<tr>
<td>Current Management</td>
<td>(30%)</td>
<td>(30%)</td>
<td>(38%)</td>
</tr>
</tbody>
</table>

The β-blocker prescriptions by study arm and time (table 5.3.k) indicate large differences between the groups, however when modelled, the effect of intervention group was not significant (P = 0.2488). The effect of time period alone was significant (P < 0.0001). The interaction between time and group was not found to be significant, with no difference in the pattern of change over time between the three groups (P = 0.507). The overall trend for β-blocker prescription was an increase across all groups and all time periods.

Analysis of the patient variables found a negative but not significant association between β-blocker prescription and female patients (P = 0.67). There was a significant effect of age at first intervention (P = 0.0033) i.e. increasing age was associated with decreasing prescription of β-blocker. Statistical data suggested that prescription of β-blockers was lower in patients without an MI but this finding did not prove to be significant (P = 0.19). The effect of renal impairment proved significant (P = 0.031) with lower prescription of β-blockers in patients without renal impairment. There was a positive but not significant association between β-blocker prescription and patients without CORD (P = 0.13). There was a positive and significant association between β-blocker prescription and patients without asthma (P = 0.0007).

None of the GP or practice variables reached significance.
The overall increase in β-blocker prescription over time was similar across the groups (11 to 14%). There was a large positive change in small group β-blocker prescription over the pre- and post-intervention period, whereas the guideline and Internet arms showed a larger change between the post-intervention and current management period of the study.

Table 5.3.1. Changes in β-blocker prescription over time for each group

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Raw</td>
<td>Adj</td>
<td>Raw</td>
</tr>
<tr>
<td>Pre- to post-intervention</td>
<td>+3%</td>
<td>+3%</td>
<td>+9%</td>
</tr>
<tr>
<td>Post-intervention to current management</td>
<td>+9%</td>
<td>+8%</td>
<td>+5%</td>
</tr>
</tbody>
</table>

Comparison of changes over time showed no significant differences between the groups for the pre- to post-intervention and the post-intervention to current management time periods.

### 5.3.4 Spironolactone

Spironolactone was not a widely prescribed drug at baseline or at the end of the study and prescription changed very little during the course of the study.

Table 5.3.m. Spironolactone prescription for all groups over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>N (% ) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All patients (n = 359)</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>68 (19%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>73 (20%)</td>
</tr>
<tr>
<td>Current Management</td>
<td>70 (19.5 %)</td>
</tr>
</tbody>
</table>

Unlike the ACE inhibitor and β-blocker prescriptions, no one trend was seen across the different study arms (see table 5.3.n). Only the small group arm showed a consistent increase in prescription of spironolactone over time.

---

\(^d\) The adjusted means for β-blocker prescription show less difference across baseline and over time.
Table 5.3.n. Spironolactone prescription rates for each arm over time

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline (n = 137)</th>
<th>Small group (n = 98)</th>
<th>Internet (n = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>24 (18%)</td>
<td>19 (19%)</td>
<td>25 (20%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>24 (18%)</td>
<td>22 (22%)</td>
<td>27 (22%)</td>
</tr>
<tr>
<td>Current Management</td>
<td>21 (15%)</td>
<td>25 (26%)</td>
<td>24 (19%)</td>
</tr>
</tbody>
</table>

The model showed no differences due to intervention arm (P = 0.52). There was no difference in time period for spironolactone prescription (P = 0.65). There was no interaction by group and time period (P = 0.58).

The trend of lower prescribing in female patients was not significant for spironolactone (P = 0.072). Associations were seen with age at intervention (P = 0.0038) which followed the same pattern as ACEi/ARB and β-blockers. There was a slight negative association between patients who had not experienced an MI and spironolactone prescription but this was not significant (P = 0.48). A significant difference was seen with renal impairment (P = 0.0092), with lower prescription seen in patients without renal impairment. Patients without a diagnosis of CORD or asthma were less likely to be prescribed spironolactone but the effect of neither of these variables was significant (CORD, P = 0.62; asthma, P = 0.46).

The comparisons of changes between time periods and intervention group did not indicate any significant differences (table not shown).

5.4 Secondary outcomes – doses of interest

The following tables illustrate the doses of ACEi, ARBs, combined ACEi and ARB prescriptions, β-blockers and spironolactone given by time period for the entire patient sample and then by study arm for each time period. Tables containing the numbers of patients at each dose code at the three time periods and also by each of the study arms can be found in Appendices 5F, G, H, K and M. The coding schedule can be found in appendix 4P.
5.4.1 Prescribed doses of ACE inhibitors and Angiotensin Receptor blockers

The data for ACEi and ARBs are presented first for each drug group and then combined, as was done for the prescription of these medications.

5.4.1.1 ACE inhibitors

The dose data for the entire patient sample are reported in table 5.4.a and the dose data for each study arm are reported in table 5.4.b.

When the model analysed intervention alone, the differences between the study arms were almost significant (P = 0.051). No significant difference was seen over time (p = 0.53). However when intervention over time was analysed there was no significant difference (P = 0.96). Gender had no effect on dose given (P = 0.08) although the relationship indicated that higher doses were less likely to be prescribed to female patients. Age at intervention was seen to be significant (P = 0.0008) with the direction the same as for prescription i.e. as age increased dose decreased.

There were no statistically significant effects seen with the other patient variables of MI (P = 0.83), renal impairment (P = 0.46), CORD (P = 0.28) or asthma (P = 0.81). The direction of the relationship suggested that higher doses of ACEi were more likely in patients without MI, patients with renal impairment, patients without CORD and patients with asthma. One other variable became significant in this analysis which was practice size (P = 0.036), and this had a positive relationship with dose. GP gender had no significant effect on dose prescribed (P = 0.12).

Comparisons of changes between time periods for guideline vs. small group and guideline vs. Internet were not significant.

Adjusted means (+ 95% CIs) for proportions of patients on maximum dose and above for each study arm and time periods can be found in appendix 5Eiv.

5.4.1.2 Angiotensin Receptor blockers

The numbers of patients on ARBs were too small to provide meaningful information and so were combined with ACEi as the therapeutic rationale for these medications is the same. The tables displaying the numbers and percentages of patients for the dose groupings are table 5.4.c for all patients, and for the three study arms table 5.4.d.
Table 5.4.a. Comparison of ACE inhibitor doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>n (% of patients)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-intervention</td>
<td>Post-intervention</td>
<td>Current management</td>
<td></td>
</tr>
<tr>
<td>Recommended dose or greater</td>
<td>115 (44 %)</td>
<td>114 (46 %)</td>
<td>100 (45 %)</td>
<td></td>
</tr>
<tr>
<td>Less than recommended</td>
<td>144 (56 %)</td>
<td>136 (54 %)</td>
<td>123 (55 %)</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>259</td>
<td>250</td>
<td>223</td>
<td></td>
</tr>
</tbody>
</table>

Table 5.4.b. Comparison by study arm of ACE inhibitor doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline n (%)</th>
<th>Small group n (%)</th>
<th>Internet n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>Recommended dose or greater</td>
<td>42 (43 %)</td>
<td>41 (44 %)</td>
<td>37 (43 %)</td>
</tr>
<tr>
<td>Less than recommended</td>
<td>55 (57 %)</td>
<td>53 (56 %)</td>
<td>50 (57 %)</td>
</tr>
<tr>
<td>N</td>
<td>97</td>
<td>94</td>
<td>87</td>
</tr>
</tbody>
</table>

\(a\) Total number of patients in that group or sub-group being prescribed ACE inhibitors for whom actual dose information was available.

\(b\) There were two patients who received ACE inhibitors but for whom no information was recorded regarding doses.
<table>
<thead>
<tr>
<th>Dose scale</th>
<th>n (%) of patients</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-intervention</td>
<td>Post-intervention</td>
<td>Current management</td>
<td></td>
</tr>
<tr>
<td>Recommended dose or greater</td>
<td>5 (20 %)</td>
<td>8 (27 %)</td>
<td>5 (18 %)</td>
<td></td>
</tr>
<tr>
<td>Less than recommended</td>
<td>20 (80 %)</td>
<td>22 (73 %)</td>
<td>23 (82 %)</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>25</td>
<td>30</td>
<td>28</td>
<td></td>
</tr>
</tbody>
</table>

Table 5.4.d. Comparison by study arm of ARB doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline n (%)</th>
<th>Small group n (%)</th>
<th>Internet n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>Recommended dose or greater</td>
<td>1 (11 %)</td>
<td>2 (18 %)</td>
<td>2 (17 %)</td>
</tr>
<tr>
<td>Less than recommended</td>
<td>8 (89 %)</td>
<td>9 (82 %)</td>
<td>10 (83 %)</td>
</tr>
<tr>
<td>N</td>
<td>9</td>
<td>11</td>
<td>12</td>
</tr>
</tbody>
</table>

\[^1\] Total number of patients in that group or sub-group being prescribed ARBs for whom actual dose information was available.
5.4.1.3  Combined Angiotensin inhibition

The numbers and percentages of patients who were taking the recommended dose or greater of ACEi or ARB and patients who were prescribed less than the recommended dose for the three time periods can be seen in table 5.4.e. The values for the 3 study arms over time are in table 5.4.f.

When the model analysed intervention alone, the differences between the study arms were not significant (P = 0.15). No significant difference was seen over time (p = 0.37). However when intervention over time was analysed there was no significant difference (P = 0.78).

Gender had no effect on dose given (P = 0.79) although the relationship indicated that higher doses were less likely to be prescribed to female patients. Age at intervention was seen to be significant (P = 0.0027) with the direction the same as seen for ACEi doses i.e. as age increased dose decreased.

There were no statistically significant effects seen with the other patient variables of MI (P = 0.77), renal impairment (P = 0.71), CORD (P = 0.37) or asthma (P = 0.40). The direction of the relationships for these patient variables was the same as seen with ACEi (higher doses of ACEi were more likely in patients without MI, patients with renal impairment, patients without CORD and patients with asthma).

Practice size, which had been statistically significant for ACEi dose prescription was no longer significant when ACEi and ARBs were combined (P = 0.065), although the positive relationship of larger practice size with higher dose was maintained. GP gender had no significant effect on dose prescribed (P = 0.22).

Comparison of change between time periods for the intervention groups showed that there were no significant differences.

Adjusted means and 95 % CIs can be found in appendix 5Ev.
Table 5.4.e. Comparison of ACE inhibitor and ARB doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention n (%)</th>
<th>Post-intervention n (%)</th>
<th>Current management n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommended dose or greater</td>
<td>120 (42%)</td>
<td>122 (44%)</td>
<td>105 (42%)</td>
</tr>
<tr>
<td>Less than recommended</td>
<td>164 (58%)</td>
<td>158 (56%)</td>
<td>146 (58%)</td>
</tr>
<tr>
<td><strong>N</strong></td>
<td><strong>284</strong></td>
<td><strong>280</strong></td>
<td><strong>251</strong></td>
</tr>
</tbody>
</table>

Table 5.4.f. Comparison by study arm of ACE inhibitor and ARB doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th></th>
<th>Guideline (control) n (%)</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>Recommended dose or greater</td>
<td>43 (41%)</td>
<td>43 (41%)</td>
<td>39 (39%)</td>
</tr>
<tr>
<td>Less than recommended</td>
<td>63 (59%)</td>
<td>62 (59%)</td>
<td>60 (61%)</td>
</tr>
<tr>
<td><strong>N</strong></td>
<td>106</td>
<td>105</td>
<td>99</td>
</tr>
</tbody>
</table>

*Total number of patients in that group or sub-group being prescribed ACE I or ARBs for whom actual dose information was available.*
5.4.2 Prescribed doses of β-blockers

The numbers and percentages of patients who were taking half or greater of the recommended dose of β-blocker and patients who were prescribed less than half the recommended dose for the three time periods can be seen in table 5.4.g. The values for the 3 study arms over time are in table 5.4.h.

The analysis of the effect of the intervention alone was not significant (P = 0.32). No significant difference was seen over time (p = 0.76). When intervention arms over time were analysed there was no significant difference (P = 0.21).

Gender had no effect on dose given (P = 0.90) although the relationship indicated that higher doses were less likely to be prescribed to female patients. The analysis of the effect of age on dose prescribed for β-blockers showed that while the effect of age was not statistically significant (P = 0.93) the direction of the relationship suggested that higher doses were more likely with increased age.

There were no statistically significant effects seen with the other patient variables of MI (P = 0.88), renal impairment (P = 0.13), CORD (P = 0.19) or asthma (P = 0.24). The direction of the relationships for these patient variables suggested that higher doses of β-blockers were more likely in patients without MI, patients without renal impairment, patients without CORD and in patients with asthma).

Practice size was not significant for β-blockers (P = 0.12), although the positive relationship of larger practice size with higher dose was maintained. GP gender had no significant effect on dose prescribed (P = 0.32).

Adjusted means and 95 % CIs can be found in appendix 5Fii.

5.4.3 Prescribed doses of Spironolactone

The numbers and percentages of patients who were taking the recommended dose of spironolactone and patients who were prescribed other doses for the three time periods can be seen in table 5.4.i. The values for the 3 study arms over time are in table 5.4.j.

The numbers of patients taking spironolactone were too small to be analysed.
Table 5.4.g. Comparison of β-blocker doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention n (%)</th>
<th>Post-intervention n (%)</th>
<th>Current management n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommended dose or half</td>
<td>26 (34%)</td>
<td>30 (33%)</td>
<td>40 (34%)</td>
</tr>
<tr>
<td>Less than half recommended</td>
<td>50 (66%)</td>
<td>61 (67%)</td>
<td>77 (66%)</td>
</tr>
<tr>
<td>N</td>
<td>76</td>
<td>91</td>
<td>117</td>
</tr>
</tbody>
</table>

Table 5.4.h. Comparison by study arm of β-blocker doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Guideline n (%)</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Recommended dose or half</td>
<td>7 (28%)</td>
<td>7 (24%)</td>
</tr>
<tr>
<td>Less than half recommended</td>
<td>18 (72%)</td>
<td>22 (76%)</td>
</tr>
<tr>
<td>N</td>
<td>25</td>
<td>29</td>
</tr>
</tbody>
</table>

*Total number of patients in that group or sub-group being prescribed β-blockers for whom actual dose information was available.
Table 5.4.i. Comparison of spironolactone doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention n (%)</th>
<th>Post-intervention n (%)</th>
<th>Current management n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommended dose</td>
<td>59 (87 %)</td>
<td>59 (81 %)</td>
<td>60 (86 %)</td>
</tr>
<tr>
<td>All other doses</td>
<td>9 (13 %)</td>
<td>14 (19 %)</td>
<td>10 (14 %)</td>
</tr>
<tr>
<td>prescribed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N^a</td>
<td>68</td>
<td>73</td>
<td>70</td>
</tr>
</tbody>
</table>

Table 5.4.j. Comparison by study arm of spironolactone doses prescribed in pre-intervention, post-intervention and at 3 years after intervention

<table>
<thead>
<tr>
<th></th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>Recommended dose</td>
<td>18 (75 %)</td>
<td>15 (62.5 %)</td>
<td>17 (81 %)</td>
</tr>
<tr>
<td>All other doses</td>
<td>6 (25 %)</td>
<td>9 (37.5 %)</td>
<td>4 (19 %)</td>
</tr>
<tr>
<td>prescribed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>24</td>
<td>24</td>
<td>21</td>
</tr>
</tbody>
</table>

^a Total number of patients in that group or sub-group being prescribed spironolactone for whom actual dose information was available.
5.5 Tertiary outcomes – diagnosis, initiation of new medications and electrolyte monitoring, specialist input

The previous section looked at whether a patient was prescribed a medication or not. This included medications that had been continued from before the time periods described in the study. One of the original questions posed by the study was whether the educational interventions would have an effect on the initiation of medications. A comparison of the number of study medications initiated during the pre-intervention and post-intervention periods would indicate whether a change had occurred.

During data collection, once a medication had been identified as being prescribed, the next question was to identify if it had been ‘continued’ i.e. started before that data collection period or whether it had been ‘initiated’ i.e. started within that 18 month time period. (See Appendix 4M for data collection sheets and instructions.)

The following tables in this section present the medication outcomes figures for the pre- and post-intervention time periods split into initiated prescriptions and continued prescriptions for each study arm. The initiated prescriptions tables present the numbers of new prescriptions as a percentage of the total prescriptions for that period. The total number of prescriptions for each medication outcome is also presented.

This section will also assess who made the diagnosis of HF. It is not possible to accurately describe how the GPs diagnosed HF in their practices as the criteria used to diagnose HF for the purposes of the study were those listed on the Boston score. It was not always possible to measure the criteria the GPs used to diagnose HF or whether the timing of the symptoms used by the GPs is the same as those used to determine the Boston Score. The occasions when dates concurred were on letters requesting admission to hospital or when the date of the visit that symptoms occurred was recorded in the text of the patient’s medical record or in the Problem List.

5.5.1 Responsibility for diagnosis

The responsibility for the diagnosis was derived from the ‘Source’ line of the Boston Score (see appendix 4O and also Chapter 4 section 3.2). The diagnosis was attributed to whomever first made it / wherever it first appeared. There were four possible attributions: problem list, text note, hospital, or specialist. It was determined that:
• One hundred and eighteen patients had HF listed in their problem list as the place where it was first listed.¹ Thirty five of these diagnoses were also listed elsewhere. Out of the 118 patients with problem list HF, 51 had not received an echocardiogram.

• Text notes of diagnosis were found for 129 patients. Only 23 text note diagnoses were transferred to the problem list. Five of these patients had objective evidence through CXR and six had objective evidence through echocardiography at about the time the diagnosis was recorded, four patients (of the 11) had both. For the other patients, it seems that diagnosis was made on clinical grounds alone.

• One hundred and nine diagnoses came from the hospital. Eighteen of these were transferred to the problem list at the time of diagnosis and 13 also appeared as text notes.

• Seventeen diagnoses came from specialists (i.e. independent of the hospital).

• Thirty seven patients had no diagnosis listed in any of the above categories. Eighteen of these patients had received an echocardiogram.

The greater number of initial HF diagnoses seem to have been made by the GP rather than the hospital. On many occasions the diagnosis remained in the text of the medical records and in letters / discharge summaries from the hospitals, making searches of problem lists as the only method for identifying HF patients less than successful. Even free text searches of medical records would need to take into account all the possible combinations of words to describe HF and would only be profitable if medical records for that time period were electronic (many in this study were not). The thorough search of medical records and accompanying documents that was undertaken in this study is justified given the number of patients that would otherwise have been missed.

¹ This differs from the total number of patients recorded as having HF in their problem list (in the demographics data) as the diagnosis may have been noted in the text of the medical records or by a specialist at an earlier date than the date recorded in the problem list. The Boston Score data only collected the first time and place the diagnosis of HF occurred. The demographic data recorded whether there was HF in the problem list or not, regardless of time. Also, the date in the problem list may have occurred after the date when the information for the Boston Score was collected i.e. added after the time the diagnostic data were collected and before the electrolytes and medication data were collected.
5.5.2 Initiation of new medications

5.5.2.1 Initiation of ACEi and Angiotensin Receptor Blockers

A total of 261 patients were prescribed ACEi during the 18 months of the pre-intervention period. Of these, 216 patients had a prescription for ACEi initiated prior to 18 months before the educational intervention had been given. The remainder of this total (n = 45 or 17% of the total of all ACEi prescriptions) had their prescription started during the pre-intervention period. These data are presented in table 5.5.a.

There were 25 patients taking an ARB, and of these 20 had their prescription initiated in the pre-intervention period. These data are presented in table 5.5.b.

The post-intervention data collection period was 18 months which started the day after the GP participated in the educational intervention. In the post-intervention period, a total of 250 patients were taking ACEi, and of these, 15 prescriptions were initiated in this period. These data are presented in table 5.5.a.

There were a total of 30 ARB prescriptions with 9 of these new prescriptions. These data are presented in table 5.5.b.

The combined data for ACEi and ARBs are presented in table 5.5.c. Data in all three tables are presented for each study arm by time period for all prescriptions (new and continued) and then new prescriptions (n and % of all prescriptions).

Table 5.5.a. New and total prescriptions for ACEi pre- and post-intervention

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>New</td>
<td>All</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>97</td>
<td>14 (14%)</td>
<td>82</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>94</td>
<td>6 (6%)</td>
<td>74</td>
</tr>
</tbody>
</table>

Table 5.5.b. New and total prescriptions for ARBs pre- and post-intervention

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>New</td>
<td>All</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>9</td>
<td>5 (56 %)</td>
<td>9</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>11</td>
<td>4 (36 %)</td>
<td>11</td>
</tr>
</tbody>
</table>
When the figures for ACEi and ARBs were combined, a total of 286 patients were taking some form of angiotensin inhibition in the pre-intervention period, with 65 of these (23%) new prescriptions. In the post-intervention period 280 patients were being prescribed angiotensin inhibiting drugs, with 24 of these new prescriptions (9%).

Table 5.5.c. New and total prescriptions for ACEi and ARBs pre- and post-intervention

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th></th>
<th></th>
<th>Small group</th>
<th></th>
<th></th>
<th>Internet</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>New</td>
<td>All</td>
<td>New</td>
<td>All</td>
<td>New</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>106</td>
<td>19 (18 %)</td>
<td>91</td>
<td>29 (32 %)</td>
<td>89</td>
<td>17 (19 %)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-intervention</td>
<td>105</td>
<td>10 (10 %)</td>
<td>85</td>
<td>6 (7 %)</td>
<td>90</td>
<td>8 (9 %)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The combination of ACEi and ARBs prescriptions demonstrated the same pattern of more initiations occurring in the pre-intervention time period than in the post-intervention time period. Overall numbers of patients who had new prescriptions for angiotensin inhibition still remained small.

### 5.5.2.2 Initiation of β-blockers

The pre-intervention period was from the date the GP participated in their educational intervention –18 months, which meant that the earliest date that for data collection could have been April 2001. The latest date for this time period, i.e. date of educational intervention, was April 2003. Although the time period that these dates encompass is fairly early in the publication of information regarding β-blocker use in HF, 76 patients (21%) were being prescribed either carvedilol or metoprolol succinate during this period. Of these, 44 prescriptions (58%) were initiated in the pre-intervention period.

The post-intervention data collection period was 18 months which started the day after the GP participated in the educational intervention. There were 91 patients who had β-blocker prescribed during the post-intervention period, although the proportion of new prescriptions was less, at 24% (22 patients).

β-blocker prescription data are presented in table 5.5.d. Data are presented for each study arm by time period for all prescriptions (new and continued) and then new prescriptions (n and % of all prescriptions).
Table 5.5.d. New and total prescriptions for β-blockers pre- and post-intervention

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th></th>
<th>Small group</th>
<th></th>
<th>Internet</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>New</td>
<td>All</td>
<td>New</td>
<td>All</td>
<td>New</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>25</td>
<td>18 (72%)</td>
<td>16</td>
<td>11 (73%)</td>
<td>35</td>
<td>15 (43%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>29</td>
<td>7 (26%)</td>
<td>24</td>
<td>10 (42%)</td>
<td>38</td>
<td>5 (13%)</td>
</tr>
</tbody>
</table>

5.5.2.3  \textit{Initiation of spironolactone}

There were 68 spironolactone prescriptions in the pre-intervention period, and 46 of these (68%) were new. In the post-intervention period, 73 patients were prescribed spironolactone and 23 prescriptions (32%) were new.

Data for spironolactone (table 5.5.e) are presented for each study arm by time period for all prescriptions (new and continued) and then new prescriptions (n and % of all prescriptions).

Table 5.5.e. New and total prescriptions for spironolactone pre- and post-intervention

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline</th>
<th></th>
<th>Small group</th>
<th></th>
<th>Internet</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>New</td>
<td>All</td>
<td>New</td>
<td>All</td>
<td>New</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>24</td>
<td>16 (67%)</td>
<td>19</td>
<td>15 (79%)</td>
<td>25</td>
<td>15 (60%)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>24</td>
<td>7 (30%)</td>
<td>22</td>
<td>7 (32%)</td>
<td>27</td>
<td>9 (33%)</td>
</tr>
</tbody>
</table>

When split into study groups (see table 5.5.e), the numbers were small but the proportions showed a wide range for pre-intervention (when the majority of initiations took place) but not for post-intervention.

5.5.2.4  \textit{Electrolyte testing and initiation of spironolactone}

\textit{PRE-INTERVENTION INITIATION OF SPIRONOLACTONE:} A total of 46 patients had a spironolactone prescription initiated in the pre-intervention period (guideline n = 16, small-group n = 15, and Internet n =15). Eighteen patients did not have any test results prior to initiation of spironolactone. For the patients who did have electrolytes measured before spironolactone was prescribed there was a wide variation in the timing of the measurements.
The NZ HF guideline states that the contraindications for spironolactone are Cr >0.25mmol/l or K+ >5.0mmol/l. No patients had Cr levels near this and only one patient had a higher K+ value (5.1mmol/l) prior to initiation.

Once spironolactone had been initiated, there were 10 patients who had no electrolytes measured. Six patients had no electrolytes tested before or after spironolactone was initiated. For the remaining patients, the first date of laboratory testing after the spironolactone prescription date was assumed to be a check of Cr and K+ levels in accordance with the NZ HF guideline. The NZ HF guideline recommends checking these electrolytes at 3 to 4 days post-initiation, then at 1 week, 1 month, then as indicated by renal function (6 monthly in stable patients). There was a wide variation in the timing of the electrolyte tests.

- < 3 days: 5 patients
- 3 – 4 days: 1 patient
- 5 days – 1 week: 10 patients
- 8 days – 1 month: 9 patients
- > 1 month: 11 patients.

Some patients’ test results came just at the start of the 5 days to 1 week period and the 8 days to 1 month period, which may suggest that the patients may not have accessed their diagnostic laboratory within the required time period. Approximately a third of patients were tested for Cr and K+ levels within a week of initiation. No patients had Cr >0.25mmol/l, and six patients had K+ >5mmol/l.

Ten patients who had spironolactone prescription initiated in this period had it stopped but only three of these had K+ 5.0mmol/l.

---

b This was the date in the medical records when prescription started or the date given in the discharge summary.

c This group was patients whose test dates were the same as the date of prescription of spironolactone.
**POST-INTERVENTION INITIATION OF SPIRONOLACTONE:** In the post-intervention period 23 patients had a prescription of spironolactone initiated (guideline n = 7, small-group n = 7, and Internet n = 9). Eight patients did not have any test results prior to initiation of spironolactone. The electrolyte tests performed before spironolactone was prescribed were spread over a broad period.

- 1–2 days prior to initiation date or on the apparent initiation date: 3 patients
- 3–7 days: 3 patients
- >1 week – <1 month: 6 patients
- >1 month: 3 patients.

No patients had Cr levels near the level at which spironolactone is contraindicated (Cr >0.25mmol/l) and no patients had K+ >5.0mmol/l.

After initiation of spironolactone, five patients did not have electrolytes measured. Four patients did not have electrolytes tested before or after spironolactone was initiated. For the remaining patients, the first date of laboratory testing after the spironolactone prescription date was assumed to be a check of Cr and K+ levels in accordance with the NZ HF guideline. The electrolyte tests were performed over a wide time period.

- < 3 days: 1 patient
- 3–4 days: 1 patient
- 5 days – 1 week: 2 patients
- 8 days – 1 month: 6 patients
- > 1 month: 8 patients.

Some patients’ test results came just at the start of the 5 days to 1 week period and the 8 days to 1 month period, which may suggest that the patients may not have accessed their diagnostic laboratory within the required time period. Less than 20% of patients were tested for Cr and K+ levels within a week of initiation. No patients had Cr > 0.25mmol/l, and four patients had K+ > 5mmol/l.

Four patients who had spironolactone prescription initiated in this period had it stopped but only one of these had K+ 5.0mmol/l.

None of the patients who had spironolactone initiated in the post-intervention period were re-challenges from the pre-intervention period.
5.5.3 Practitioners responsible for initiating new medications

5.5.3.1 ACE inhibitors

Forty-five ACEi prescriptions were initiated pre-intervention and 15 were initiated post-intervention. The source of the initiation is displayed in table 5.5.f.

Table 5.5.f. Number of doctors who initiated prescriptions of ACE inhibitors

<table>
<thead>
<tr>
<th>Source of initiation</th>
<th>Time period</th>
<th>GP</th>
<th>Hospital</th>
<th>Specialist</th>
<th>NR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-intervention</td>
<td>15</td>
<td>23</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Post-intervention</td>
<td>5</td>
<td>5</td>
<td>-</td>
<td>5</td>
</tr>
</tbody>
</table>

5.5.3.2 Angiotensin Receptor Blockers

There were 20 new prescriptions for ARBs pre-intervention and 9 during the post-intervention time period. The figures are displayed in table 5.5.g.

Table 5.5.g. Number of doctors who initiated prescriptions of ARBs

<table>
<thead>
<tr>
<th>Source of initiation</th>
<th>Time period</th>
<th>GP</th>
<th>Hospital</th>
<th>Specialist</th>
<th>NR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-intervention</td>
<td>9</td>
<td>9</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Post-intervention</td>
<td>5</td>
<td>3</td>
<td>-</td>
<td>1</td>
</tr>
</tbody>
</table>

5.5.3.3 β-blockers

Forty-four β-blocker prescriptions were initiated in the pre-intervention period and half this number (n = 22) in the post-intervention time period. The prescription number by initiating doctor is displayed in table 5.5.h.

Table 5.5.h. Number of doctors who initiated prescriptions of β-blockers

<table>
<thead>
<tr>
<th>Source of initiation</th>
<th>Time period</th>
<th>GP</th>
<th>Hospital</th>
<th>Specialist</th>
<th>NR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-intervention</td>
<td>12</td>
<td>26</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Post-intervention</td>
<td>7</td>
<td>10</td>
<td>-</td>
<td>5</td>
</tr>
</tbody>
</table>
5.5.3.4  **Spironolactone**

Forty six spironolactone prescriptions were initiated in the pre-intervention time period and 23 spironolactone prescriptions were initiated in the post-intervention time period. These results are displayed in table 5.5.i.

Table 5.5.i. Number of doctors who initiated prescriptions of spironolactone

<table>
<thead>
<tr>
<th>Time period</th>
<th>Source of initiation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GP</td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>10</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>12</td>
</tr>
</tbody>
</table>

5.5.4  **Specialist input into management**

The following examples are taken from letters sent to GPs directly after a hospital admission, a follow-up outpatient clinic or a visit to a specialist. The exploration of these letters was an attempt to understand the level of prescribing that occurred in secondary care and the influence over primary care management of HF. These examples illustrate differences in the quality of information conveyed to GPs about the management of their HF patients. The letters also indicate the different levels of responsibility that cardiologists take in managing HF patients. While it is not certain how representative the letters are of those received by GPs about their HF patients, there is wide variation. Specialist input ranges from the vague suggestions which assume a certain level of knowledge, to very detailed patient information and dose recommendations with titration hints. Another unknown is what proportion of letters offer helpful information and what proportion give no practical assistance to the GP. This section concludes with an insight into the deliberations of one GP around initiating a β-blocker in a patient.

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\[d\] These letters were copied from patients’ files late in the study on the two occasions when VA collected data and then reviewed selected data already collected. There was no pre-determined selection process. Letters from specialists or hospitals were copied from patients whose medical records were checked at these times.
5.5.4.1 Examples of letters from specialists

The letter for this patient (17/06/2002) clearly states at the start of the letter the target dose for Accupril in the medications list. There are titration instructions for a β-blocker, the target dose is given but no indication as to potential adverse events.

<table>
<thead>
<tr>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accupril 20mg bid (increased)</td>
</tr>
<tr>
<td>Lipitor 10mg daily</td>
</tr>
<tr>
<td>Diltiazem 240mg daily</td>
</tr>
<tr>
<td>Spironolactone 25mg daily</td>
</tr>
<tr>
<td>Frusemide 80mg mane</td>
</tr>
<tr>
<td>Aspirin 100mg daily</td>
</tr>
</tbody>
</table>

At this stage, I would like to increase his Accupril to 20mg bid both for his blood pressure and to maximise his failure treatment. He currently is not on Beta-Blocker and does not recall being on one recently, although this is mentioned in the discharge summary of September last year. Ideally, he should be on metoprolol 190mg daily and I would be grateful if you could initiate this, beginning at 23.75mg daily and doubling the dose every 2-4 weeks as tolerated.

I have not adjusted his Frusemide dose, though I suspect he is not particularly volume overloaded currently.

Figure 5.5.a. Example from patient 001003 regarding ACE inhibitor and β-blocker

The following letter extract is from a review at a cardiology clinic (31/10/2002). The patient was taking multiple cardiovascular medications which were listed in a sentence within a paragraph (and in this context is easy to misread). In this list of medications was 5mg Enalapril. The cardiologist was keen to up-titrate the dose of Enalapril despite the patient’s seemingly low blood pressure. The cardiologist states the target dose and highlights potential adverse effects. The message here is that the GP should pay less attention to the actual blood pressure reading and more attention to how the patient reports that they feel in the context of reducing BP.

His blood pressure was 95/55 and I asked him to increase to dose of Enalapril to 10mg daily and we could progressively increase the dose of Enalapril to 20mg daily provided he does not become symptomatically hypotensive and renal function does not deteriorate.

Figure 5.5.b. Example from patient 135003 regarding ACEi dose and blood pressure
The letter below is about the same patient, from the next cardiology clinic review (16/01/2003). The cardiologist is reinforcing the ACE inhibitor dose and the timing of the doses. There is also reiteration of messages about renal function testing pre – post spironolactone initiation although no time frames are given. There is a reminder of the overall medication regimen for “significant impairment of LV systolic function” and the need for β-blockade, and advises caution in this instance but no further instruction is given on initiation although the option for a review of the patient is left open.

Heart rate today was 70bpm and blood pressure 115/60.

We would like to progressively increase the dose of Enalapril to 20mg daily and hence I have asked him to start taking 5mg of Enalapril at night time in addition to the 10mg he takes in the morning. I would be grateful if you would increase the dose further. You will know that we should check his renal function to make sure this is not deteriorating, and after this start him on Spironolactone 25mg daily, once again making sure that the addition of Spironolactone does not cause hyperkalaemia or worsening renal function.

We are suggesting these changes because patients with significant impairment of LV systolic function should be on high dose ACE inhibitor therapy as well as Spironolactone and if possible on a beta blocker. Beta blockers need to be introduced very cautiously. It would seem that they may have a contra-indication to beta blocker therapy, he tells me he is taking a steroid inhaler at the moment and hence I presume it is thought that he has some bronchodilator response. I have not arranged routine review in Cardiology Clinic but please let us know if you feel this is indicated.

Figure 5.5.c. Patient 135003 further adjustments to medications

In this next extract from a cardiology clinic review (08/08/2002), there is a rare reference to the NYHA classification. There is emphasis on electrolyte testing after spironolactone initiation and combination with ACEi. There is also reinforcement of the use of β-blocker and its benefits. A target dose is given for the ACEi. The GP has marked the final paragraph.

**Impression:**

has a cardiomyopathy and I am uncertain as to possible etiology. At present, heart failure seems reasonably well optimised and her effort tolerance is in keeping with NYHFC II.

I have booked a Dobutamine stress echocardiogram to document LV size and function and also to assess for ischaemia and viability. Today I started Spironolactone 25 mg daily. I would be grateful if you would check urea and electrolytes within the next week due to the combination of an ACE inhibitor and Spironolactone. I will review her situation following echocardiogram at which stage I will consider starting a β-blocker which as you know have been shown to be very beneficial in heart failure. Importantly, the Inhibace dose should be optimised to maximum which is 5 mg daily.

Figure 5.5.d. From patient 057006. Electrolyte testing advised after spironolactone and benefits of β-blocker emphasised
The information given in a review 6 months later (10/02/2003) is less clear. The reference to effort tolerance is ambiguous. While metoprolol has been added by the cardiologist and suggestions are made for up-titration and a goal heart rate, the formulation is not defined, nor are the potential side effects.

At present remains well-optimised. Her effort tolerance is normal.

**Medication at present:** Spironolactone 25mg/day, Frusemide 40mg/day, Atacand

I started her on a small dose of Metoprolol, and I would be grateful if the Metoprolol could be increased gradually (12.5mg fortnightly) aiming for a heart rate of 60 b.p.m.

Figure 5.5.e. Less informative letter regarding management of patient 057006

While this next specialist’s letter does not list details of medications or problems, it does discuss trial evidence or lack of it for the medication changes. This letter was written at the end of 2001 and is early in promoting β-blocker evidence. There is also discussion of NYHA classes. The specialist is taking the lead in dose titration. The GP has marked passages in the letter.

I have stopped the Atenolol and instead have prescribed him Betaloc CR 23.75mg daily (Metoprolol is one of the beta-blockers which have been well studied in heart failure trials) and I have also started him on a small dose of Cilazapril 1.25mg daily (which has not been used in large HF trials). I think we should try and increase the dose of his Metoprolol and ACE inhibitor over a period of time and I have suggested that I would see him again in about 2 weeks time in order to up-titrate his doses. In retrospect, I think a diuretic would also be advisable – such as Bendrofluazide 2.5mg daily.

Now that his grafts have been shown to be patent and normal, the main aim of his treatment would be to prevent further remodelling of his left ventricle and initiate drug therapy which has been shown to improve survival in the context of LV dysfunction. The main drugs here are ACE inhibitors and β-blockers. It is interesting that he remains in NYHA Class 1 and one could argue that neither group of drugs have been shown to be especially beneficial in this situation but I think in his case the NYHA Class system does not adequately reflect the severity of his LV dysfunction.

Figure 5.5.f. Detailed letter to GP of patient 101005 at the end of 2001

The above letter was followed two weeks later with another detailed letter about up-titration, evidence-base, caveats and discussion of patient education. Again, the cardiologist is in charge of moderating the patient’s medication.

At this visit, I have simply increased his Metoprolol to 47.5mg and Cilazapril to 2.5mg, in addition to his Cartia and Liptor 40mg. I have also suggested that he start Frusemide 20mg three days a week in view of his very high LVEDP at the time of his catheter study on 4/12/01 (which showed patent grafts but important impairment of left ventricular contractility). I discussed these matters with him, including the rationale for the choice of drugs, and plan to see him again towards the end of January for further up-titration of his Betaloc and Cilazapril – if tolerated. As you know, the trials for Metoprolol (and certain other β-blockers) have shown substantial benefit in those with impaired left ventricular function although the drug needs to be introduced cautiously and progressively over a period of time.

Figure 5.5.g. Detailed follow-up letter regarding patient 101005
One month later, the patient’s GP receives another very detailed letter about the patient’s intolerance to the increase in medications. The letter relates the symptoms that the patient experienced and the steps that the cardiologist took to manage these. The cardiologist has also stressed patient education.

I was very pleased to see you again on 21/1/02 although was concerned to hear that his symptoms had returned since his last visit before Christmas. I am not sure whether this follows an increase in the dose of his Betaloc and Cilazaoril. He was finding it difficult to do certain things because of breathlessness.

He had also noticed a persistent cough following Christmas which disturbed his sleep and I wondered whether this reflected a mild degree of pulmonary oedema. He had not had any orthopnoea or PND.

On examination, he had a mild sinus bradycardia, probably due to the Betaloc, and systolic blood pressure was reduced at 95mmHg.

I think his symptoms reflect a degree of heart failure, possibly aggravated by his Betaloc. I explained to him the reasons for taking the Cilazaoril and Metoprolol, based on their benefit in people with left ventricular dysfunction, especially in those with more severe myocardial impairment, but it is possible that the doses have not been well-tolerated in his case. I have therefore suggested that he reduce his Cilazaoril to 1.25mg daily (half a 2.5mg tablet) and to reduce the Betaloc CR to 23.75mg daily (half a 47.5mg tablet). I have also indicated that he should increase the Frusemide to 40mg 3 days a week, or even daily, and take Slow-K 1 daily as well.

He will let me know in a few days whether his symptoms have improved and I will see him again in about 1 month’s time to check his progress. He was taking the lower dose of Metoprolol and Cilazaoril at his previous visit before Christmas, when he was largely free of symptoms.

Figure 5.5.h. Details of patient’s symptoms, possible causes and the steps the cardiologist took, including reinforcing patient education, and re-iteration of evidence

There are also examples of confused communication. The next extract (from May 2003) suggests that the cardiologist will be taking charge of the medication management.

Your patient is going to have an echo and then on the basis of his left ventricular function, I will adjust his medication. I would be aiming to optimise the ACE inhibitor, introduce Spironolactone and possibly Carvedilol. You will be kept informed of developments.

Figure 5.5.i. Cardiologist indicating the steps that they will take to manage the patient (054003)

Two months later, the responsibility for managing this patient seems to have been shifted to the GP. Management is indicated but no ACEi dose is recommended, nor is a medications list given. The stability of the condition prior to initiating a β-blocker is mentioned but not tips on initiation dose, titration steps or timing, or potential adverse effects. No reminders are given about checking electrolytes prior to and after initiation of spironolactone.
Your patient’s problem, therefore, is heart failure. I would be grateful if you could optimise the dose of ACE inhibitor, and then start Spironolactone 25mg daily. When he is stable on this combination, Carvedilol should be added, titrating the dose gradually upwards.

I am returning him to your care but would be happy to review him should you be concerned.

Figure 5.5.j. Vague management recommendations from the hospital cardiologist

The same hospital cardiologist together with a cardiac nurse specialist offer the following instructions for a different patient (057004) who is already taking 10mg Accupril daily. No information is given regarding electrolytes. The GP has marked the spironolactone.

Plan:
Commence cartia aspirin 100mg once daily.
Commence spironolactone 25mg once daily.

Figure 5.5.k. Instruction to GP to commence spironolactone

In a further letter from the cardiologist and another nurse specialist regarding the above patient, a fit 88 year old woman, an interesting point is raised regarding the evidence for use of β-blockers in the elderly. While the MERIT-HF (21) and MERIT-HF in the elderly (published in 2004) (413) did include patients only up to the age of 80 years, all trial evidence in patients younger than this points to significant benefits after the introduction of β-blockers.

Plan:
We will not be introducing betablockers at this stage as we are unsure of how favourable they are in patients of this age.

Figure 5.5.l. Reasoning for not introducing β-blocker in an ‘elderly’ patient (057004)

While the following clinical summary for the GP does mention monitoring of renal function for this patient (135003) who was discharged on 5mg of enalapril, there is no up-titration target dose or instructions. There is a vague reference to BP which may indicate that this house surgeon’s belief that BP is a guide to ACEi dose rather than patient-reported symptoms.

Figure 5.5.m. Instructions given to GP on increasing ACEi dose
5.5.4.2  **GP deliberation over initiating a β-blocker**

As can be seen in the extract below from the medical records of one patient (figure 5.5.n), the message for the need to initiate β-blockers and that this should be done once a patient is stable and euvoelaemic, was taken seriously. In January 2003, about three months after participating in the small-group educational intervention, the GP mentions that the patient still has oedema, indicates increasing Frusemide, and repeats a comment not to start Bentaloc CR until CHF is stable. The GP even suggests a starting dose, all of which indicates thorough knowledge of the issues discussed during the educational intervention.

![Figure 5.5.n. GP’s notes considering the initiation of β-blocker in a patient](image)

Three months later the GP is still considering Bentaloc but finds the patient’s current condition not suitable (see figure 5.5.o).

![Figure 5.5.o. Initiating a β-blocker still in the forefront of management for this patient](image)
The GP started spironolactone a short while later but did not get the opportunity to initiate a β-blocker as the patient moved overseas. The above notes illustrate an example of the legibility of some GPs’ handwriting and the difficulty involved in extracting information from medical records.

5.6 Effect of listed diagnosis

1. “Early” diagnosis. Diagnosis date is given in problem list and is prior to the date the GP participated in their educational intervention (i.e. listed during the pre-intervention time period or before). There were 185 patients in this group.

2. “Mid-point” diagnosis. A diagnosis made in the 18 months after the educational intervention (i.e. the post-intervention time period). There were 28 patients in this time period.

3. “Late” or “no” diagnosis. A diagnosis date which was later than 18 months after the educational intervention (n = 21) or patients where no HF diagnosis was listed (n = 102). The “no” diagnosis patients did however have evidence in their medical records that was strongly suggestive of HF, and this was collected in the Boston Score sheet.

4. No date given. Patients who did have HF listed in their problem list but without any date listed were not included in the analysis (n = 23). The HF could have been diagnosed at any stage and could therefore not be categorised.

Patients who did not have a dated diagnosis of HF were excluded. This left 336 patients who were included in the analysis of HF diagnosis and management. It must be reiterated that all patients had strong evidence of HF before the GP’s educational intervention date according to data collected on the Boston Score and supplementary diagnostic data that were collected.

The table below (5.6.a) displays the date of diagnosis for all patients and then by study arm the values displayed are n (%). Approximately half of the patients received a diagnosis before the educational intervention.
Table 5.6.a. Timing of listed HF diagnosis by intervention study arm

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>All patients (N = 359)</th>
<th>Guideline (n = 137)</th>
<th>Small group (n = 98)</th>
<th>Internet (n = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>185 (52%)</td>
<td>68 (50%)</td>
<td>48 (49%)</td>
<td>69 (56%)</td>
</tr>
<tr>
<td>Mid</td>
<td>28 (8%)</td>
<td>7 (5%)</td>
<td>5 (5%)</td>
<td>16 (13%)</td>
</tr>
<tr>
<td>Late</td>
<td>21 (6%)</td>
<td>10 (7%)</td>
<td>4 (4%)</td>
<td>7 (6%)</td>
</tr>
<tr>
<td>No diagnosis</td>
<td>102 (28%)</td>
<td>41 (30%)</td>
<td>33 (34%)</td>
<td>28 (23%)</td>
</tr>
<tr>
<td>No date</td>
<td>23 (6%)</td>
<td>11 (8%)</td>
<td>8 (8%)</td>
<td>4 (3%)</td>
</tr>
</tbody>
</table>

5.6.1 Echocardiography and annotated HF diagnosis

The rates of echocardiography for each HF diagnosis group were compared. The variables of intervention, gender, age at intervention, MI, CORD and asthma were tested prior to testing the effect of the HF groups in the analysis of effect of listed diagnosis on outcomes. The table below displays the adjusted rates of echocardiography for each of the HF diagnosis groups.

Table 5.6.b. Rates of echocardiography (adjusted) by HF group

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>Echocardiography</th>
<th>95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>63 %</td>
<td>50 to 74 %</td>
</tr>
<tr>
<td>Mid</td>
<td>72 %</td>
<td>49 to 87 %</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>65 %</td>
<td>51 to 77 %</td>
</tr>
</tbody>
</table>

No effect was attributable to intervention, gender, CORD or asthma, or renal impairment (see table below). However a strong effect was seen with age (likelihood of receiving an echocardiogram decreased with increasing age) and patients who had experienced an MI. There was a significant difference in the ORs for patients with and without an MI. There was no significant effect of HF group on echocardiography although there were slight differences in ORs; the 95% CIs were wide. The proportion of patients who received an echocardiogram was high even in patients who had late or no diagnosis listed (see table 5.6.c).
Table 5.6.c. Analysis of variables for echocardiography and HF group

<table>
<thead>
<tr>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Variable</strong></td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
</tr>
<tr>
<td>Guideline</td>
</tr>
<tr>
<td>Small group</td>
</tr>
<tr>
<td>Internet</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td><strong>Age</strong></td>
</tr>
<tr>
<td>0.93</td>
</tr>
<tr>
<td><strong>No MI</strong></td>
</tr>
<tr>
<td><strong>No CORD</strong></td>
</tr>
<tr>
<td><strong>No Asthma</strong></td>
</tr>
<tr>
<td><strong>Timing of diagnosis</strong></td>
</tr>
<tr>
<td>Early</td>
</tr>
<tr>
<td>Mid</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
</tr>
</tbody>
</table>

5.6.2 Angiotensin inhibition and annotated HF diagnosis
The ACEi and ARB prescriptions were combined to give an overall proportion of prescriptions for the RAAS as there were too few ARBs prescribed for these to be analysed as a separate group. The combination of ACEi and ARBs will be referred to as angiotensin inhibition. The prescription rates of these drugs were compared for the three HF diagnosis groups. The adjusted means for these groups were:

Table 5.6.d. Angiotensin inhibition prescription (adjusted) by HF group

<table>
<thead>
<tr>
<th><strong>Timing of diagnosis</strong></th>
<th><strong>ACEi / ARB</strong></th>
<th><strong>95 % CI</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>83 %</td>
<td>77 – 87 %</td>
</tr>
<tr>
<td>Mid</td>
<td>79 %</td>
<td>63 – 89 %</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>65 %</td>
<td>56 – 73 %</td>
</tr>
</tbody>
</table>
Looking at the other variables that were tested prior to HF groups in the analysis model, intervention was not significant. Time period showed differences that were significant (prescribing indicating a decrease over time). Gender was not significant. Age at intervention was highly significant and the direction of the association was negative (increasing age reduced the chance of any angiotensin inhibiting agent being prescribed). Even after testing for the effects of intervention, time, gender and age at intervention, the differences between HF groups (see table above) were still highly significant (see table 5.6.e).

Table 5.6.e. Analysis of variables for angiotensin inhibition and HF group

<table>
<thead>
<tr>
<th>Results</th>
<th>Variable</th>
<th>OR</th>
<th>95 % CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intervention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guideline</td>
<td></td>
<td>1.64</td>
<td>0.82 to 3.25</td>
<td></td>
</tr>
<tr>
<td>Small group</td>
<td></td>
<td>1.98</td>
<td>0.97 to 4.04</td>
<td></td>
</tr>
<tr>
<td>Internet</td>
<td></td>
<td>1</td>
<td>-</td>
<td>0.14</td>
</tr>
<tr>
<td><strong>Study time period</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-intervention</td>
<td></td>
<td>1.42</td>
<td>1.05 to 1.84</td>
<td></td>
</tr>
<tr>
<td>Post-intervention</td>
<td></td>
<td>1.39</td>
<td>1.14 to 1.71</td>
<td></td>
</tr>
<tr>
<td>Current management</td>
<td></td>
<td>1</td>
<td>-</td>
<td>0.0041</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td>0.66</td>
<td>0.42 to 1.03</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>1</td>
<td>-</td>
<td>0.069</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td>0.96</td>
<td>0.94 to 0.98</td>
<td>0.0007</td>
</tr>
<tr>
<td><strong>Timing of diagnosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early</td>
<td></td>
<td>2.57</td>
<td>1.63 to 4.05</td>
<td></td>
</tr>
<tr>
<td>Mid</td>
<td></td>
<td>2.01</td>
<td>0.87 to 4.65</td>
<td></td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td></td>
<td>1</td>
<td>-</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

5.6.3 β-blockers and annotated HF diagnosis

The prescription rates of the HF-specific β-blockers were compared for the three HF diagnosis groups. The model tested for significance of differences between the HF diagnosis groups. The adjusted means for these groups were:
Table 5.6.f. β-blocker prescription (adjusted) by HF group

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blockers</th>
<th>95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>29 %</td>
<td>23 – 36 %</td>
</tr>
<tr>
<td>Mid</td>
<td>33 %</td>
<td>19 – 50 %</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>20 %</td>
<td>14 – 27 %</td>
</tr>
</tbody>
</table>

Table 5.6.g. Analysis of variables for β-blocker prescription and HF group

<table>
<thead>
<tr>
<th>Results</th>
<th>Variable</th>
<th>OR</th>
<th>95 % CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervention</td>
<td>Guideline</td>
<td>0.69</td>
<td>0.37 to 1.26</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Small group</td>
<td>0.65</td>
<td>0.35 to 1.23</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internet</td>
<td>1</td>
<td>-</td>
<td>0.31</td>
</tr>
<tr>
<td>Study time period</td>
<td>Pre-intervention</td>
<td>0.53</td>
<td>0.41 to 0.64</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post-intervention</td>
<td>0.68</td>
<td>0.70 to 0.82</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Current management</td>
<td>1</td>
<td>-</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Sex</td>
<td>Female</td>
<td>1.1</td>
<td>0.70 to 1.72</td>
<td>0.69</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>1</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.97</td>
<td>0.95 to 0.99</td>
<td>0.001</td>
</tr>
<tr>
<td>Timing of diagnosis</td>
<td>Early</td>
<td>1.67</td>
<td>1.03 to 2.69</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mid</td>
<td>2.01</td>
<td>0.88 to 4.58</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Late or no diagnosis</td>
<td>1</td>
<td>-</td>
<td>0.073</td>
</tr>
</tbody>
</table>

5.6.4 Spironolactone and annotated HF diagnosis

The prescription of spironolactone was tested for significant differences between the three HF diagnosis groups. There were few patients in each of these groups, especially the mid-point diagnosis group (see Appendix 5E). The adjusted means for spironolactone can be seen in table 5.6.h.
Table 5.6.h. Spironolactone prescription (adjusted) by HF group

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>Spironolactone</th>
<th>95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>26 %</td>
<td>21 – 32 %</td>
</tr>
<tr>
<td>Mid</td>
<td>15 %</td>
<td>6 – 32 %</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>12 %</td>
<td>8 – 18 %</td>
</tr>
</tbody>
</table>

Intervention and time did not have a significant effect on spironolactone and nor did the sex of the patient. Despite an OR for age which was just under 1 the CIs were very small and the effect was highly significant. The direction of this effect was the same as had been observed with echocardiography, ACEi and β-blocker, with a negative association between prescription and age. The differences between the HF diagnosis groups were highly significant. (Refer to table 5.6.i)

Table 5.6.i. Analysis of variables for spironolactone prescription and HF group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Results</th>
<th>95 % CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intervention</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guideline</td>
<td>0.94</td>
<td>0.51 to 1.72</td>
<td></td>
</tr>
<tr>
<td>Small group</td>
<td>1.34</td>
<td>0.73 to 2.49</td>
<td></td>
</tr>
<tr>
<td>Internet</td>
<td>1</td>
<td>-</td>
<td>0.47</td>
</tr>
<tr>
<td><strong>Study time period</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>0.96</td>
<td>0.71 to 1.29</td>
<td></td>
</tr>
<tr>
<td>Post-intervention</td>
<td>1.08</td>
<td>0.86 to 1.36</td>
<td></td>
</tr>
<tr>
<td>Current management</td>
<td>1</td>
<td>-</td>
<td>0.52</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>0.65</td>
<td>0.39 to 1.07</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1</td>
<td>-</td>
<td>0.09</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.97</td>
<td>0.94 to 0.99</td>
<td>0.003</td>
</tr>
<tr>
<td><strong>Timing of diagnosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early</td>
<td>2.58</td>
<td>1.48 to 4.50</td>
<td></td>
</tr>
<tr>
<td>Mid</td>
<td>1.29</td>
<td>0.43 to 3.85</td>
<td></td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>1</td>
<td>-</td>
<td>0.003</td>
</tr>
</tbody>
</table>
6. Discussion

This chapter summarises the main thesis findings (primary, secondary and tertiary outcomes, post-hoc analyses), critiques more recent studies of Internet delivered CME, of HF management in primary care and other methods of changing practice, suggests explanations of how the interventions worked (or not), discusses the strengths and limitations of the study, and proposes recommendations.

Section one discusses the primary outcomes (echocardiography use and medication prescription), comparing the study results with other research. Findings of the pre- and post-intervention surveys are included here as they may provide context for the outcomes of the record review. Survey data are presented in the Discussion chapter as the items were unvalidated and the rigour of the methods lay in between qualitative and quantitative methodologies. The response rates between survey rounds also differed. The discursive nature of the analysis was deemed to be more suitable in the Discussion chapter where the survey results would aid insight into the thesis findings and provide rationale for other features of the study not directly measured.

Section two describes the secondary outcomes (change of dose over time) and again includes survey results to provide a richer in-depth context for the outcomes. The tertiary outcomes (responsibility for diagnosis, initiation of new medications and electrolyte monitoring, and specialist input into management) are presented in Section three. As the specialist letters gathered for this thesis may not be representative, an example is given of a study that evaluated the management advice given in consecutive letters for HF patients discharged back to their GPs. Almost no survey data were collected on these outcomes, apart from specialist input which is described in section 6.7.4 ‘Other influences on HF management’.

Section four examines the post-hoc analyses – the effect of the diagnosis date on the four primary outcomes. Research has suggested that the presence/absence of a diagnosis in a patient’s medical record problem list effects clinical management.

The practice, GP and patient populations are discussed in Section five. These data were presented at the beginning of the Results chapter as it was important to describe these populations and to identify any variables that would be necessary to include in analysis. Whereas in this chapter the purpose is discussion.
Section six provides critical analysis of more recent studies of Internet-delivered interventions and of studies that have attempted to improve the management of HF in primary care. Some studies reflect aspects of the Chronic Care Model, which is also described in this section, as well as how this works in practice and gives a New Zealand example. This section also looks at methods (of varying complexity) of using computerised tools to assist in HF management. Survey data presented here suggests that prompts or reminders were seldom used by GPs participating in this study.

Section seven investigates other aspects of the study that could have had an effect on outcomes. This section first described how a conventional RCT and educational intervention RCT differ by comparing domains and applies these domains to this thesis study, and highlights aspects of the study – expectations and assumptions, and potential solutions regarding the outcomes. This section describes aspects and the extent of practice re-organisation that GPs engaged in following the educational intervention, as articulated by the GPs themselves. This is followed by further survey data which describe other influences on management (specialist input into care and other sources of information). This section also presents questionnaire data from each intervention arm about their experiences (likes, dislikes and possible improvements to the educational arm in which the GPs participated), and a study that suggests that learning and cognitive preferences and instructional methods may not have any effect on knowledge gained.

The final part of this section deals with barriers to change: from inertia, lack of clarity over responsibility for management, and lack of patient participation in care (see also 6.6.3), and whether any solutions have been identified for these barriers.

Section eight provides an in-depth assessment of the strengths and weaknesses of this study and relates these to other research. There is also discussion of aspects of the study that could have been improved on.

The final section of this chapter (section 9) presents recommendations. These are in three groups.

1. Practice (primary and secondary care);
2. Policy (national level and local – District Health Board), and
3. Research (including the need for greater insight into the effect of HF on the indigenous population of NZ).

Recommendations are given in italics, and are accompanied by observations from this study and evidence from the research literature.
6.1 Primary outcomes – echocardiography and medications

This section presents broad overviews of the results of the four primary outcomes (echocardiography, prescription of ACEi/ARB, β-blockers and spironolactone) and related GPs’ responses about their knowledge of HF management.

6.1.1 Echocardiography

The gold standard diagnostic test for HF – echocardiography – was received by less than 60% of patients. This figure is high for a community-based HF population. Other studies indicate 30 to 40%. (6-9, 261, 263, 268, 274, 300) Most of the tests were carried out prior to the educational intervention (51.5% vs. 6.5% after). The effect of time was statistically significant as were patient age and previous MI. Younger patients were more likely to receive an echocardiogram as were patients who had experienced an MI. There was no effect due to intervention over time.

The echocardiography rates may also be due to beliefs held by the study GPs. These were expressed in the study survey of HF management. Pre-intervention survey figures indicated that less than 20% of GPs thought echocardiography should be used to confirm diagnosis or in all suspected cases of HF, and only 60% mentioned echocardiography at all. There was a slight increase in the post-intervention survey with 71% of GPs mentioning echocardiography, but only a quarter stipulated this test in all suspected cases or for confirmation of diagnosis. This finding is consistent with studies described in chapter 3, section 2.6.2 which reported that many GPs have limited understanding of the value of echocardiography in the diagnostic process. (9-12, 20, 34, 304, 305) The uncertainty surrounding diagnosis for suspected HF patients without echocardiography reports results in more cautious prescription of HF medications as stated by some GPs. Lack of diagnostic certainty and consequent reluctance to prescribe has also been observed in overseas studies. (9, 11, 12, 260)

All patients had clinical evidence of HF before the educational interventions were delivered. It was outside the remit of this thesis to determine any differences in HF between male and female patients. However, the sexes may report different signs and symptoms, which may be interpreted as conditions other than HF. (273-276, 309) No differences were found in echocardiography rates between men and women. Almost half the patients who received an echocardiogram had no numerical EF recorded in the report. Qualitative reporting does not convey heart function with the same clarity as a report that primarily reports quantitative findings. While there is no unified definition
of EF cut-off points (14, 15, 25, 29, 259, 264, 286, 297, 311-315) that signify systolic HF vs. Preserved EF, there is no doubt over lower values.

One aim of the educational intervention was to encourage GPs to refer possible cases of HF for echocardiography but the small number of patients who received first-time echocardiogram after the date of education suggests that this did not occur. The data that were collected did not indicate whether the referral was initiated by the GP or by the hospital.

6.1.2 Angiotensin inhibition (ACEi and ARB)

At baseline the proportion of patients receiving ACEi (73%) was high in comparison with studies (32, 34, 35, 252) reporting data from around that time (2001 to 2002). The baseline prescription of ARBs and ACEi (80% or 93% seen in small group) suggested a ceiling effect – the level of angiotensin inhibition at the start of the study may have been the maximum level achievable with this patient population in general practice and any educational intervention might not have increased the baseline level. The use of ACEi in HF has been standard practice since the early 1990s which may account for the relatively high prescription levels and may also account for only 46% of GPs who mentioned ACEi as a change in HF management within the past three years. Only 6% indicated ACEi as first line or early use, or routine use (in HF) or use of ACEi wherever possible. 4% of GPs mentioned that ARBs could be used in ACEi-intolerant cases.

Unexpectedly during the study ACEi prescription fell to 63%, a statistically significant drop, and seen across all three study arms. This finding cannot be explained although greater attention to patients (if this did occur) may have led to detection of other problems and a heightened awareness of potential adverse effects as seen in structured care. (308) However 70% of GPs mentioned ACEi in the repeat survey and some GPs responded that they would use ACEi early or be more likely to use ACEi.

Over the study period there was no effect of the interventions. But female patients and older patients were less likely to receive ACEi, as demonstrated in other studies of primary care HF management. (31, 252, 359, 361) ACEi/ARBs prescriptions also showed a significant reduction over time (from 80% to 70%) and the associations with age and sex were still evident (males and younger patients being more likely to receive ACEi/ARB prescription).
An unexpected finding indicated that patients with renal impairment (in their problem list) were more likely to be prescribed an angiotensin inhibiting agent. The differences in change over time between the groups (adjusted: -2% to -11%) could not be explained.

6.1.3 β-blockers
There was a statistically significant increase in prescription of β-blockers over time but there was no difference in change over time in β-blocker prescription across the three study arms. Pre-intervention survey data indicated that over 80% of GPs were aware of β-blocker use in HF. In the post-intervention survey 95% of GPs were aware of β-blocker use in HF. GPs were possibly more confident and comfortable in initiating β-blockers by the time of the second round of the survey.

Older patients were less likely to receive β-blockers, as seen overseas in primary care. (252) Patients with renal impairment were more likely to receive β-blockers as were patients without asthma. There are no adverse effects with short term administration of cardioselective β-blockers in mild to moderate reversible airways disease or COPD. (376) A further meta-analysis of cardioselective β-blockers in COPD, including severe obstruction, also concluded that administration did not produce adverse respiratory effects. (375) Both analyses concluded that cardioselective β-blockers should not be withheld from these patients. (375, 376) No GP or practice variables had a significant effect on β-blocker prescription in HF.

Prior to the educational interventions, study GPs expressed confusion about which patients should receive β-blockers and GPs were reluctant to initiate these medicines, preferring the hospital to do this. One GP did refer to good results and outcomes, and two referred to use in volume-stable patients. The side effects of β-blockers as well as intolerance and worsening HF were mentioned as concerns. GPs had continuing concerns about worsening HF, hypotension or general intolerance of β-blockers, seen in the post-intervention survey. One GP mentioned that two patients had died suddenly after use of β-blockers, but no association can be determined. The overall caution voiced by GPs reflects overseas studies of GPs’ attitudes towards β-blockers in HF. (10, 12, 24, 34)

6.1.4 Spironolactone
The prescription of spironolactone remained relatively static over time, and no effect was seen for any of the interventions over time. The use of spironolactone was not
recognised by all GPs as a change in the management of HF as 64% of GPs mentioned it pre-intervention. In the second round of the survey 76% of GPs mentioned spironolactone as a new change in HF management. It was still not clear that GPs understood the new indications for spironolactone.

Older patients were less likely to receive spironolactone, as also suggested in an overseas study. (252) But patients with renal impairment were more likely to receive spironolactone. Renal impairment has greater potential for adverse events. K+ monitoring and dose adjustment become even more important in this group. (See 6.2.4 for more information regarding electrolytes and spironolactone.)

In the pre-intervention survey, concerns with spironolactone therapy were electrolyte imbalance, over-diuresis, and deterioration in renal function. In the second round of the survey electrolyte imbalance and decreased renal function were again mentioned.

### 6.1.5 Summary

Echocardiography use did not significantly increase after the educational interventions. There was no significant interaction of study arm and time. Younger age and previous MI were statistically significant for receipt of echocardiography. Overall, the proportion of suspected HF patients receiving echocardiography was high for a community-based population.

The prescription of ACEi decreased significantly over time but the interaction of time and study arm was not significant. Other variables that had a significant effect were sex (higher prescription in males) and age (lower prescription in older patients). Analysis of ARBs was not possible due to small patient numbers. These were combined with ACEi. The reduction in prescription during the study period remained statistically significant for the combined ACEi/ARBs. Reduced prescription in women and older patients was statistically significant. Patients with renal impairment had a statistically significant higher prescription of ACEi/ARBs.

The prescription of β-blockers increased significantly during the study. Age and renal function were statistically significant variables, with older patients and those without renal impairment being less likely to be prescribed a β-blocker. Patients without asthma had higher prescription of β-blockers.
Spironolactone prescription remained static over the study period. Women and older patients were less likely to receive spironolactone. Patients with renal impairment were more likely to receive spironolactone.

There was no effect of any of the three interventions over time on echocardiography or the study medications. All three study arms showed similar patterns of change for each of the medications of interest and for echocardiography.

While all patients had sufficient clinical or objective evidence to either suspect or confirm HF, the appropriateness of the prescriptions was not able to be determined. One example is NYHA class, which is a prescribing guide for β-blockers and spironolactone, but rarely documented in medical records. Another point of uncertainty was whether the GPs or specialists had primary responsibility for the management of these patients or whether this responsibility was shared. This was not usually clearly stated. GPs and specialists have significantly different approaches to HF management. The effect of specialist care could not be directly determined, although some data (see 6.3) were collected regarding the responsibility for initiation of medications.

6.2 Secondary outcomes – doses of interest

The secondary outcomes compared the change in doses over time for ACEi/ARBs, β-blockers and spironolactone.

6.2.1 Angiotensin inhibition (ACEi and ARB) dose

The educational intervention emphasised the use of maximum tolerated ACEi dose. At the start of the study just under 50% of patients were prescribed the recommended dose or greater. This did not change for the duration of the study. The only significant findings were that as patients aged they were less likely to be prescribed higher doses and larger practice size also resulted in higher doses being prescribed. For combined ACEi/ARBs, only the effect of age was significant.

The data in the three study arms indicated differences between them in the proportion of patients taking high dose ACEi, however the comparison of change over time in the intervention arms did not indicate any differences.

The pre-intervention survey found that only 13% of GPs mentioned the maximal tolerated dose of ACEi. However these GPs backed up their statements with good
quality evidence, with one mentioning a survival advantage associated with use of ACEi and spironolactone. In the post-intervention survey, maximal dose ACEi was mentioned slightly more frequently (29% of GPs, although these may represent more motivated participants). In both surveys reported concerns centred around hypotension or hypotension during up-titration, deterioration in renal function, electrolyte imbalance, cough on ACEi (some GPs were aware of the option of switching to an ARB, although they did not mention trialling another ACEi or checking that the patient was euvoalaemic), the effects of age and polypharmacy, patient resistance and the work involved in up-titration. The survey responses of doses and concerns of adverse effects, and the reports of good quality evidence do not differ from other studies of GPs and ACEi use. (9-12, 20, 35, 203)

6.2.2 β-blocker dose
A third of patients were prescribed ≥50% of the recommended β-blocker dose throughout the study. There was no effect of the intervention over time for β-blocker doses and none of the variables tested reached statistical significance. Over 80% of GPs were aware of β-blocker use in HF in the pre-intervention survey but only 3% mentioned maximal dose. In the post-intervention survey 95% of GPs were aware of β-blocker use in HF, but use of maximal β-blockade was indicated by only 8%.

Study data (carvedilol newly introduced) found that half of the patients were initiated at 6.25mg, which rose to and stabilised at ≥6.25 to <50mg for the remainder of the observation period (up to 3 years). (416)

NZ GPs held similar concerns to overseas GPs about worsening HF, hypotension or general intolerance of β-blockers. (10, 12)

6.2.3 Spironolactone dose
The small numbers of patients precluded analysis of the spironolactone doses. A large majority of patients were taking the recommended daily dose of 25 mg.

The new indication recommended use at 25mg instead of 100mg and as an aldosterone blocker not a K⁺-sparing diuretic, with evidence from a large study to support it. (25) A few GPs did correctly identify the class of patient it should be used in, the survival advantage and the new recommended dosage. By the second survey round it was still not clear that GPs understood the new indications for spironolactone.
6.2.4 Summary
The evaluation of proportion of patients prescribed ACEi at recommended dose and higher, compared with lower ACEi doses, found no effect of intervention, time or intervention over time. Age was still seen to have a significant effect – as age increased, dose decreased. Larger practice size increased the chance of receiving a higher dose.

The combined ACEi/ARB analysis also found no significant change in the study arms over time. Patient age had a significant effect, as observed with the ACEi alone. The effect of practice size and larger dose was no longer significant but the direction of the relationship was still positive.

The maximum recommended dose did not seem likely to be obtained in primary care patients for β-blockers. Literature around dosages achieved in trials and in community-based HF patients was checked so that it could be applied to the CME and HF management study. Given the nature of clinical trials as described in chapter 3 section 3.2, the doses achieved in clinical trials of β-blockers would most likely be higher than those achieved in primary care. Analysis of ≥50% of maximum dose of β-blocker compared with lower doses found no change over time or significant effects due to any other variable.

The analysis of the recommended dose of spironolactone was not completed due to insufficient sample size.

Overall, the results of the analyses found that the maximum doses prescribed did not change over time nor did the interventions have any effect on them.

Whether the treatment regimens initiated in hospital remained without up-titration due to inertia, fear of side effects, patient disagreement or contraindications cannot be determined.

6.3 Tertiary outcomes – diagnosis, initiation of new medication and electrolyte monitoring, specialist input
This broad set of outcomes identifies who made the HF diagnosis, how many drugs were initiated and by whom, the electrolyte monitoring prior to and subsequent to spironolactone initiation, and the information that specialists provided to primary care.
For all three medications of interest, most initiations of these medications occurred before the educational intervention took place. This pattern occurred in all study arms.

6.3.1 Responsibility for diagnosis

Reviews of patients’ medical records found that 62% of HF diagnoses were made by GPs, however not all diagnoses were recorded in the problem list. A hospital diagnosis did not ensure that this would then be recorded in the problem list. The lack of a diagnosis in the problem list makes it difficult to identify and manage patients especially if they are being managed by more than one GP or outside of primary care.

6.3.2 Initiation of new medications and electrolyte monitoring

Uncertainty over diagnosis may then cause GPs to hesitate over treatment. However the NZ HF guideline’s diagnostic algorithm advises treatment if it is likely that an echocardiogram will be delayed. This important element of the guideline did not receive enough attention. This instruction seems to encourage treatment without objective evidence and may encourage GPs to continue an established pattern of treating suspected HF, not only for that patient but in general. The treatment regimen may then become ingrained without any rationale. However the need for symptomatic treatment cannot be negated. The guideline itself also mentions that recommendations for specialist referral should not delay the initiation of appropriate treatment. There is still some lack of confidence in primary care in using ACEi or a lack of understanding of its benefits. (10-12, 20, 417) The introduction of β-blockers into accepted management of HF was contrary to all medical school and hospital teaching up until the early 2000s. (10, 12) This change in indication for a medication reflects a dramatic shift in knowledge. Initiating a β-blocker could be further undermined by early bad experiences. The rationale for the re-introduction of spironolactone to the HF treatment schedule may be poorly understood and potential benefits may be missed. (342, 344, 418) Less attention may be paid to monitoring adverse effects of an established drug compared with a new drug. All three of these drug classes require careful monitoring, which can be time-consuming, and poorly monitored adverse effects could have serious consequences for the patient.

Thus, adding a new drug requires careful scrutiny of the existing medications for any potential interactions as well as discussions with patients regarding any other products they may consume. Managing a complex medication regimen requires a good comprehension of pharmacology.
Reluctance to treat may also be due to perceived lack of applicability of the recommendations, where clinical trial patients are dissimilar to those found in primary care and where the trials have been conducted in different settings. (54, 59, 60, 62-64, 96, 99) This would include for example the evidence on which the recommendations are based.

6.3.2.1 Angiotensin inhibition
New prescriptions for ACEi and ARBs were more common pre-intervention (23%) than post-intervention (9%). Most new prescriptions occurred in the small group arm before the intervention but all groups were similar post-intervention.

6.3.2.2 β-blockers
Most new prescriptions took place before the educational intervention was delivered and were initiated in secondary care. Most pre-intervention initiations occurred in the guideline and small group study arms. It is evident that more patients in the Internet arm were on ‘continued’ prescriptions (started more than 18 months before the intervention). This may also be a function of the dates of the educational interventions. In general, the Internet arm tended to be later than either the guidelines or small group, giving more time for patients in the Internet arm to have a prescription for a β-blocker initiated before data were collected for the pre-intervention time period.

6.3.2.3 Spironolactone
Most initiations occurred before the educational intervention and in hospital, but after the intervention more occurred in primary care than in hospital.

6.3.2.4 Spironolactone and electrolyte testing
The recommended testing of electrolytes before and after initiation was adhered to loosely. Some patients were not tested before or after initiation or tests occurred several weeks before or after spironolactone prescription in both pre- and post-intervention periods. Attempts were made to define reasons for stopping spironolactone but only occasionally was this due to rising K+ levels. More patients had spironolactone stopped than had recorded electrolyte levels above those stated in the NZ HF guideline. Concerns around electrolyte imbalance, over-diuresis, and deterioration in renal function were mentioned by GPs in both survey rounds.
Since spironolactone is a familiar drug, there may be less adherence to guidelines and a reversion to customary (potentially inappropriate) prescribing. (26) This could be hazardous to patients. Higher numbers of adverse events (hospitalisations related to hyperkalaemia and more hospital deaths) with spironolactone have been reported in studies subsequent to the RALES trial. (386) In addition to the ACEi/ARB associated reduction in $K^+$ excretion, $\beta$-blockers can also interfere with the RAAS and $K^+$ uptake, further potentiating adverse effects. (419, 420) Non-steroidal anti-inflammatory drugs (NSAIDs), either prescribed or bought over the counter, as well as herbal or ‘natural’ remedies can also disturb electrolyte balance. (419)

Medical records rarely give reasons for medications being stopped so this information was not collected. However studies into the duration of therapy with spironolactone and ACEi may give some indications. One study found a high proportion of patients discontinuing spironolactone, ACEi or both. (27) The patient population was older than in the RALES trial (72.6 years) and almost 50% of the patients were taking a starting dose of 50mg (study conducted before the RALES trial). (27) Another study found that the patients prescribed spironolactone were taking higher ACEi doses and higher spironolactone doses, and more patients had renal insufficiency compared with the RALES trial (31% vs. ‘excluded’). (26) More patients experienced hyperkalaemia (12% severe vs. 2%) and discontinued spironolactone (21% vs 8%) than in the RALES trial. This may suggest that the familiarity of spironolactone, results in less adherence to guidelines and a reversion to customary prescribing. (26)

6.3.2.5 Summary
Overall, the initiation of medication, especially in the post-intervention period, was sparse. The numbers in each study arm were too small to compare. Most initiations were implemented in the pre-intervention time period, i.e. before the GPs participated in the educational interventions. For ARBs, $\beta$-blockers and spironolactone the number of initiations in the post-intervention time period was 50% of those pre-intervention. For ACEi the initiation dropped to a third in the post-intervention time period.
6.3.3 Practitioner initiating new medications
Medications were started in primary care, the hospital, or by a specialist (private practice).

6.3.3.1 Angiotensin inhibition
ACE INHIBITORS: In the pre-intervention period the majority of the new prescriptions for ACEi originated from the hospital, while the few initiations that occurred post-intervention were distributed equally between hospitals and GPs.

ANGIOTENSIN RECEPTOR BLOCKERS: GPs seemed to be more confident in prescribing ARBs than ACEi. Almost half of these prescriptions were initiated by GPs in the pre-intervention time period and during the post-intervention period. GPs may initiate more ARBs as they are more likely to see patients who have ACEi induced cough\(^a\) and then change medication to an ARB to alleviate side effects.

6.3.3.2 β-blockers
Most new prescriptions took place before the educational intervention was delivered and were initiated in secondary care. A large difference was seen between the numbers of initiations by GPs and hospitals in the pre-intervention period. Almost 60% of pre-intervention prescriptions were started by the hospital. While there were fewer initiations in the post-intervention period, the difference between hospital and GP initiations was not nearly as great. An early study of β-blockers (all carvedilol prescriptions dispensed for HF late 1999 to mid-2001) found that hospital physicians were responsible for 85% of initiations. (416) The context of prescription was that initiation and up-titration of carvedilol should be under the supervision of a hospital physician.

6.3.3.3 Spironolactone
Most initiations occurred before the educational intervention and in hospital, but after the intervention more occurred in primary care than in hospital. In the pre-intervention time period the hospital took the lead in initiating spironolactone prescriptions. In the post-intervention period the majority of initiations were made by GPs.

\(^{a}\) The theory that ACEi cause cough may not be as accurate as believed or reported. The cough may be due to increased congestion and as such an increase in diuretics may resolve the cough.
6.3.3.4 Summary

Hospitals initiated more new prescriptions than GPs. In the pre-intervention time period, apart from ARBs (for which the numbers of new prescriptions were equal between GPs and hospitals), hospitals initiated medication 1.5 to 3 times more often than GPs. During the post-intervention period GPs initiated the same number of ACEi, slightly more ARBs, slightly fewer β-blockers and twice the number of spironolactone than hospitals.

In the pre-intervention period, the GPs initiated β-blockers and spironolactone less frequently than the hospital. The apparent trend to hospital prescribing of β-blockers may have been due to fear over initiating β-blockers in primary care. This leads to a question about whether patients who had been admitted to hospital for what was presumably an exacerbation of HF were suitable candidates for the initiation of β-blockers i.e. were experiencing mild to moderate symptoms and were clinically stable as stated as requirements by the NZ HF guidelines. (29) Hospitalised patients may be in sufficiently severe HF to warrant spironolactone prescription rather than a β-blocker. Perhaps it is better for specialists to take the opportunity to initiate β-blockers in a well supported environment. However this may result in confusion for GPs about the ideal circumstances to start β-blockers. The change in the source of the majority of spironolactone initiations in the post-intervention period may be due to GPs responding to the NZ HF guideline which recommends spironolactone for patients who have severe HF but this includes patients who have been in class IV within the last 6 months. From the data collected regarding initiation of HF medications, it seems that changes in the medication management of HF patients were started by the hospitals once they became aware of study data, and before the educational intervention was provided to the participating GPs.

6.3.4 Specialist input

The examples presented of letters written by specialists (and by HF nurses) suggest a wide range in the quality of information provided to GPs to assist them with follow-up care of HF patients after hospitalisation. While some specialists decide to manage the patient themselves, the remainder provide GPs with letters whose content varies from extremely precise titration guides and potential adverse effects, to uninformative letters that assume an in-depth knowledge of HF management. Repeating the same
appropriate messages to GPs will help them to get this information being put into practice. The evidence-base of some of the advice given in the letters is variable.

The letters presented in this thesis were selected to illustrate different styles of letter writing and as such may not accurately represent the overall standard of advice given. Other studies have evaluated consecutive letters written from a particular hospital over a time period and quantifying the advice given to GPs. Few instructions are given to assist the GP in optimising management as described in the following example.

Discharge recommendations were evaluated in a study of 212 HF patients, discharged back to the care of their GPs with no specialist HF programme follow up during 2005. (235) Patients were relatively old (mean age 78.4 years), with multiple conditions (mean number 5.4 co-morbidities) and 50% were male. (235)

A relatively high proportion of patients was prescribed ACEi (74%, n=156). Of these patients prescribed ACEi 35% were on a ‘high’ dose (≥50% maximum dose). For patients taking a ‘low’ dose (<50% maximum dose) an accompanying titration schedule or stated titration would be expected. 5% of patients had up-titration instructions (8/156): the remainder had none. (235) This left 56 patients without any ACEi, which could be expected to be due to contraindication or no opportunity to commence ACEi. No explanation for lack of prescription was offered for 41%; 23% had a documented contraindication, and the remainder were unknown as no discharge letter was received by the GP. (235)

A similar pattern was seen for β-blockers, prescribed to 44% of all patients. High dose was received by a third of patients, over half received a low dose with no up-titration recommendations and <10% received low dose with up-titration recommendations. (235)

In the remaining patients who were not prescribed β-blockers (n = 93), almost 60% had no explanation, a third had a documented contraindication, but 8% had a recommendation for future prescription. (235) This 8% represents a very small number of patients (n=7) but may indicate that some specialists are encouraging GPs to commence β-blockers. This leaves a large number of unexplained prescription omissions, even though β-blockers were established HF therapy by the time study was conducted. The information contained (or omitted) may be due to relegating the discharge summaries to interns. (235) A junior doctor may not have full understanding of the importance of detailed management recommendations. (235)
If, as discussed, GPs tend not to alter secondary care management plans, patients will remain on the doses that they were discharged on. GPs may also view the initiation of β-blockers as a hospital-only intervention (10, 12, 20) and could be left confused if no reason is given for non-prescription, which will not encourage GPs to initiate β-blockers in their care. Hospital admission has been described as an opportunity to improve management (365, 370) and the discharge letter an opportunity for educating GPs (see section 2.4.8) and influencing the management of HF patients in primary care. As most discharge letters used in this thesis were written by cardiologists (unless otherwise stated), there is still a gap between what information is provided and what could be provided to assist GPs in their management of HF.

There is perhaps a need for form letters that structure information given to GPs about the continued care. (See also Hospital Correspondence in 6.9.1 Practice Recommendations and Secondary – primary care communication in 6.9.3 Research Recommendations.)

**GP β-BLOCKER NOTES:** The notes written by a GP in a patient’s medical record (see section 5.5.4.2) are a rare example of a GP recording the “thinking aloud” carried out when considering medication options. This extract demonstrates many of the aspects that the GP has to consider prior to initiating a β-blocker.

### 6.4 Post-hoc analyses – effect of listed HF diagnosis

The proportions of diagnosis times were approximately the same for all groups; however the intervention arm was included in the analysis model to account for any differences and any potential confounding effect.

All patients had evidence of definite or possible HF before GPs participated in the educational intervention. This evidence would give sufficient grounds for suspected HF and for referring patients for echocardiography.

What also needs to be considered is the tendency of GPs to treat HF suspected on clinical grounds alone and also the suggestion in the NZ HF guideline’s diagnostic algorithm to treat empirically if there is a delay in accessing echocardiograms. Thus all patients had an equal chance of being treated for HF.

The effect of time is itself covered in the HF groups and there is a “chicken and egg” effect of whether the diagnosis preceded the echocardiogram or vice-versa. This would
only be able to be determined by a meticulous analysis of actual dates which have not usually been detailed in the medical records e.g. some diagnoses have only years recorded or months-years or days-month-year.

6.4.1 HF in problem list and echocardiography
Most first-time echocardiograms were performed before the educational intervention. However the presence of echocardiography results did not automatically mean that a diagnosis of HF was entered into the patient’s medical record. For the analysis of the effect of a listed diagnosis of HF on the study outcomes, the problem list was used to define patient groups. Any patients left out of this analysis were patients who had a diagnosis listed but no date given. Patients were compared on the basis of the timing of the diagnosis during the study.

The timing of the HF diagnosis did not affect when an echocardiogram was obtained. The variables that were strongly significant (i.e. whether the patient received an echocardiogram) in this analysis were age (older patients less likely to receive echocardiography) and MI (patients with an MI more likely to receive an echocardiogram). This possibly reflects the problems around the currency of problem lists and the responsibility to enter data (see Recommendation section 6.9.1.1). There may also be aspects of uncertainty regarding the meaning of the echocardiogram. Some examples of echocardiography reports were cryptic, giving the findings of the test but no interpretation (see Recommendation section 6.9.1.2).

The responsibility for obtaining echocardiograms for patients in this study is not known so it cannot be confirmed whether a number of GPs did not believe that echocardiography was required. The lower echocardiography rate seen in older patients is consistent with GP surveys which suggest rationing of echocardiography use in older patients and patients who GPs perceive may not ‘benefit’ from the findings of echocardiography. Patients who have experienced an MI are more likely to receive an echocardiogram as part of the management process of the MI.

**SUMMARY:** For echocardiography, the length of duration of the HF diagnosis had no effect on whether the patient had received an echocardiogram, but a history of MI had a strong effect on whether echocardiography had been performed as did the patient’s age. Since the majority of echocardiograms were performed in the pre-intervention time period, it seems from the results above that receiving an echocardiogram does not affect whether a diagnosis is written in the patient’s medical record.
6.4.2 HF in problem list and angiotensin inhibition

Analysis of the prescription of ACEi/ARB found that a patient with a HF diagnosis made early in the study or prior to the study was more likely than a patient with a mid-point diagnosis to receive ACEi/ARB, and a patient with a diagnosis made at a mid-point in the study was more likely to receive an echocardiogram than a late or no diagnosis patient. These differences were statistically significant after testing for study time period (earlier time period was associated with greater ACEi/ARB prescription), sex (males more likely than females to receive a prescription) and age (younger patients more likely to receive a prescription). Patients with an early diagnosis would have been diagnosed when the evidence of the benefits of ACEi was available. Even with a diagnosis of HF, GPs were reluctant to prescribe to older patients despite the clear evidence of benefit of ACEi in older patients and little evidence to suggest increased adverse effects, even at high doses. (16, 18, 362, 381, 421) A diagnosis of HF should give GPs confidence to initiate and up-titrate ACEi in all patients, provided there are no contraindications. Clinical judgement is important as is the understanding that age is a continuum, not a cut-off point, and that treatment could potentially benefit older patients more than younger patients.

**SUMMARY:** The data showed a decrease in angiotensin inhibition prescription over time and also that the age of the patient was significant in accounting for whether any angiotensin inhibition was prescribed. Even once these variables had been analysed, there was still an obvious effect of length of duration of HF diagnosis on prescription of angiotensin inhibiting agents.

6.4.3 HF in problem list and β-blocker

The results of β-blocker analysis found a significant effect of time (patients were most likely to receive β-blockade in the last study time period) and of age (younger patients more likely to receive β-blockers). Patients who had a mid-point diagnosis were more likely to be prescribed a β-blocker than patients who received a diagnosis early in the study or a late or no diagnosis, but the difference (due to the length of time with a HF diagnosis) was not significant. Patients who received a diagnosis mid-way through the study may have received the diagnosis when information about β-blockers was being disseminated and would have been more likely to be remembered as having a diagnosis of HF (given that it was recent and easier to recall) and therefore started on a β-blocker.
The results indicate that this medication was not being prescribed to older patients as frequently as to younger patients.

The outcomes of the analysis showed no significant differences between the study arms. However the time was highly significant with prescription of β-blocker more likely to occur in the current management period (three years after the educational intervention). There was no significant difference due to sex. Age did show a significant effect in that prescription of β-blockers decreased as age increased. Once all of these variables had been tested, the differences between the prescriptions of β-blockers in the HF groups were tested and these were found not to be significant.

**SUMMARY:** Once the significant effects of time (the effect of the evidence of β-blocker use becoming more normalised by the end of the study) and of age had been taken into account, no significant effect of HF group (length of diagnosis) was seen.

### 6.4.4 HF in problem list and spironolactone

The effect of age was also seen in the spironolactone and HF diagnosis analysis. Patients who had an early diagnosis of HF were more likely to be prescribed spironolactone than patients with mid-point or late/no diagnosis, and patients with mid-point diagnosis were more likely to receive spironolactone than patients with late/no diagnosis. Patients diagnosed a few years after the RALES study was published may have been tried on spironolactone because of the trial evidence or may have long standing HF that deteriorated into NYHA class IV and been prescribed spironolactone due to the severity of the HF.

**SUMMARY:** A significant effect of length of time of diagnosis (expressed as HF group) was seen on the prescription of spironolactone after the other variables had been analysed. Age was also seen to have a significant effect on spironolactone prescription.

### 6.4.5 Summary

The effect of the timing of the listed HF diagnosis in relation to the four study outcomes was tested. The effects of age were statistically significant on the relative odds of receiving an echocardiogram (more likely in younger patients) as was experience of MI but there was no effect due to the timing of diagnosis of HF.
The effect of timing of HF diagnosis on ACEi prescription was analysed and after study time period and age were found to be significant, the timing of diagnosis was also found to be a highly significant predictor of whether a patient was prescribed an ACEi, this being 2½ times more likely in a patient with an early diagnosis. The same comparisons with β-blockers found that study time period was highly significant (although in the opposite direction to ACEi/ARBs). The effect of age on prescription of β-blockers was also significant. The effect of timing of diagnosis did not reach statistical significance. For spironolactone prescription, age was again significant (odds of receiving spironolactone reduced with age) and the timing of diagnosis was significant with odds increasing as length of time with diagnosis increased.

In all the outcomes tested for effect of timing of HF diagnosis, age had a significant effect on each of the outcomes but the timing of HF diagnosis only had a statistically significant effect on the prescription of ACEi/ARBs and of spironolactone.

The proportion of patients receiving ACEi/ARBs was higher than the proportion of patients who had received an echocardiogram, suggesting that some patients were being treated on clinical grounds alone or were prescribed ACEi for hypertension (by coincidence). All of the patients included in the analysis of the effect of a listed diagnosis had sufficient information in their medical records to indicate suspected HF before the GPs participated in the educational intervention. This however does not seem sufficient grounds to prescribe for HF. As seen in this analysis, the presence of a diagnosis in the problem list determines whether a patient receives medication.

6.5 Practice, GP and patient populations

The practice, GP and patient variables were presented at the start of the Results chapter as it was important to establish the demographics (i.e. for generalisability) of the populations and any differences between them prior to describing results of the educational intervention.

6.5.1 GP and practice variables

The overall practice and GP participation rate was disappointingly low with less than a third of practices agreeing to participate by supplying patients. However the participation rate across the groups was similar. A cluster design was suitable for this project since the interventions were given at practice level (cluster) and the outcomes

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were measured at patient level. Clustering is appropriate where groups may exhibit similar characteristics. This was evident at two levels in this study: patients of one GP, and GPs at a practice. The consequence of clustering means that the effective sample size is reduced. The practices had been stratified prior to randomisation in order to reduce the likelihood of an imbalance of practice sizes in the study arms. The stratified randomisation was successful. The drop outs after randomisation left more large group practices in the Internet study arm. The practice size was accounted for in the analysis. Within the GPs there was an imbalance between the groups in the sex ratios and in the length of time since they had graduated with their medical degrees. The drop outs after randomisation balanced the sex ratio. They resulted in greater differences between the study arms for graduation but these proved to be not statistically significant. Sex of the GP and qualification year were included (where possible) in the analyses. Comparison of the participating GPs with the NZ GP population showed no difference in proportion of male and female GPs. The participating study GPs were more likely to be Fellows of the Royal New Zealand College of General Practitioners and to have been in practice longer than NZ GPs.

### 6.5.2 Patient variables

There was a wide range in the number of eligible patients from each GP/practice. The patient population in this thesis had a median age of 76.3 years. The sex ratio was similar across study arms and overall 51% of the study participants were female. The patients were similar to those reported in other community studies. The proportion of European participants in the HF management implementation study seemed high and the difference in ethnic groups between the study arms can only be explained by chance. BP, HR, creatinine and electrolyte values were all within expected population ranges. BP decreased over time. Many patients had co-morbidities; 57% of patients had CHD/IHD, 63% had HTN, 28% had DM, 24% had COPD and asthma 15%. The wide variation (25%) in MI across the study arms seems unusual. There is no obvious explanation for this and it is most likely due to chance. There were high rates of DM in the Internet arm, which had a high number of large practices, so there may have been protocols around updating problem lists which can account for this higher proportion. However, this hypothesis does not fit with the higher rate of MI in the small group arm. The accuracy of the figures depends on how well records were kept. The date of diagnosis of these conditions was not always recorded. When HF diagnosis was
examined information from hospital correspondence did not always appear in the problem list.

Both GP and patient populations are likely to be generalisable to other countries with similar health care systems.

6.6 Update on HF management in primary care and Internet implementation research

This section looks at two aspects of HF management: general strategies to improve HF management in primary care, and Internet delivered interventions.\(^b\) Other methods to improve HF management; the Chronic Care Model and computerised aides from generic prompts to tailored decision support are also described.

**STUDY QUALITY:** Many of the studies of HF management and of Internet education have methodological issues. These include no control group for observational studies (to determine if any effect is due to the intervention), before-and-after tests of knowledge with no assessment of practical application (i.e. audit to evaluate change in management, patient outcomes), and RCTs that do not account for clustering. In those that are clustered, the ICC is often not given. In addition, many of the studies have small sample sizes, and short follow-up times that are insufficient to determine important patient outcomes. Some studies disclose few details of their educational interventions. This creates difficulties in determining factors that could influence change in practice and consequently adding little if anything to the development of theories regarding which domains and constructs are critical in causing behaviour change in primary care HF management.

Despite these flaws there are some positives. Several studies used community-based patient populations, and there was interaction between primary and secondary care regarding patient management. (422, 423) Some studies provided clear titration instructions and information about managing adverse events which acts to explain and reinforce practice (nature of behaviours, beliefs about capabilities) and allay beliefs about consequences and emotions particularly for β-blocker prescription. (422)

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\(^b\) This section was updated by running simple searches in Medline and EMBASE on ‘primary care’ and ‘Internet’ and ‘heart failure’ and ‘primary care’. A search was also carried out using the phrase ‘chronic care model’.
The artificial nature of trials with highly restrictive inclusion/exclusion selection criteria also raises concerns. For example, if patients are excluded from a trial because of contraindications (correctly or incorrectly), the consultation scenario loses some realism as the decision to treat or not to treat (or refer) these patients is made for the GP (study not generalisable or pragmatic). So while the patient population may be community-based, it is highly selected and not comparable to that which is usual in primary care. The number of patients excluded due to contraindications usually is not reported either. Unless the differences in populations are acknowledged the results may be unobtainable in general practice. This is similar to the efficacy vs. effectiveness debate of drug RCTs and generalisability (discussed in Chapter 3, section 3.2 and section 3.3.3).

While excluding those with obvious contraindications may make audit more accurate, especially since contraindications may not be listed in medical records, there are other more subtle reasons for not prescribing a drug, so again, the full extent of the patient’s quality of care is unable to be determined. Even if audit does not completely illuminate the complexities of HF management, it is more informative than studies that do not attempt any form of record review before and after delivering an educational intervention.

The following sections will describe some more recent studies that attempted to change aspects of HF management. The first section will examine Internet-delivered interventions and the second will describe other forms of interventions in primary care. The third and fourth sections discuss the Chronic Care Model (CCM) and various types of computerised support.

### 6.6.1 Internet-delivered interventions

Only one relevant study was found that described such an intervention. The mode of delivery of CME was basic: viewing a seminar on-line. The seminar used in the example below is described as a “grand round” format with real-life images, illustrations, charts, graphs with text and narration. (424) This description sounds like a didactic lecture but without any interaction with either the presenter or peers.

The stated objectives of this web seminar were similar to the areas covered in the CME delivered for this thesis: participants would be able to describe stages of HF, rationale for certain classes of drugs, goals of treatment and stage-dependent treatment. The seminar described the American 4 stage classification system (not the NYHA classes),
how to evaluate HF through history and examination, and treatment by HF stage. The rationale for pharmacologic and non-pharmacologic treatments was also given. (424) This American study does not state the year it was conducted. There was no discussion of observed barriers to care or how the intervention would improve care.

The comparison group consisted of GPs randomised to view a seminar on diabetes mellitus (DM). Both groups answered vignette questions online for DM and HF. (424) There was no control group (i.e. who did not view a seminar) and no assessment of management prior to the seminar. There was no comparison of HF management in actual patients before the seminar was viewed or afterwards.

Quality of care was defined for each vignette. For the first HF case the management decisions were: check lipid levels, provide smoking cessation counselling, and initiate ACEi/ARB. For the second HF case: measure LV function, start Coumadin (presume AF), start β-blocker. Vignette questions were open-ended. (424) This differed from the Internet CME for this thesis. The online quiz questions were multi-choice. This was done to indicate immediately after the question whether it was correct or not and provide reasoning for all the answers.

The majority of doctors were internal medicine specialists, the remainder were family practice, with a small proportion of ‘other’ specialities. (424) There was no discussion of clustering or if participating doctors were from the same hospitals or practices. There were no sample size calculations. 113 doctors participated, 49 in HF and 64 in DM, from a total of 6129 in the study area (2% participation). (424) There was no demographic comparison with non-participants. The reason given for not asking any management questions at baseline was to avoid the Hawthorne effect. (424)

The responses to the first HF vignette were not significantly different between the HF and DM groups. HF participants did have significantly more correct answers for each question in the second vignette and a significantly better overall score. (424) Despite this, 47% of HF seminar participants were unsure if they would modify their clinical practice. 29% did intend to change patient management but 24% said they did not intend to change practice. (424) There was no follow up to determine whether these intentions were undertaken or changed.

Participants were overwhelmingly positive about the course – course content, learning and knowledge and the effect on their clinical skills, and the ease of participation. (424) Overall, the course was rated “excellent” or “very good” by 88% of participants. (424)
The doctors appreciated that they could participate in the course at a time that suited them, complete it at their own pace, and at home or in the office. (424) There was no reporting of what the GPs did not like or felt could be improved. Generally, the aspects of Internet CME that are not popular (depending on how the CME is delivered) are the lack of interactivity, the discipline required to complete the modules, and the deficit of social contact (see 2.4.4). (132, 133, 211, 212)

6.6.2 Improving HF management in primary care

The studies here have taken a number of different approaches to deliver interventions to improve HF management in primary care. Some interventions are highly specific (focus only on β-blocker use) and others are more broad.

**INTENSIVE TRAINING – ALL MEDICATIONS:** The effect of an intensive didactic intervention and feedback on guideline adherence for ACEi/ARBs, β-blockers, and aldosterone antagonists had a similar problem at baseline as faced in this thesis – a high prescription rate of an outcome medication. (423) A small German cRT (37 GP participants of 750 invited, 5% participation) with data from late 2004 to 2005 found baseline prescription rates in intervention and control groups of 90% for ACEi/ARBs and 79% for β-blockers, but only 29% for aldosterone antagonists (AA). (423) This high level of prescribing, or ‘ceiling effect’, where the maximum of appropriate prescribing has occurred, and any educational intervention will not result in any change. (44, 58)

The study outcomes were change in prescription rates and doses over time. These are similar to this thesis, except the study time frame was much shorter. Study patients were clearly defined as having HF with EF ≤40%, NYHA class II to IV, but who were stable. (423) Mean age was 68.8 years, range 44 to 90 years, and 70% were male. (423) It is unclear how the patients were selected into the study. The intervention and control group educational interventions are described in the table below (423) but this is not entirely clear about the delivery timeline and the various components involved. The table below may seem detailed but it is not apparent which barriers are being targeted or what types of behaviours or theoretical domains. This confusion will make it difficult to identify which factor/s are effective if any change is observed.
Table 6.6.a. Comparison of intervention and control education for guideline adherence

<table>
<thead>
<tr>
<th>Control</th>
<th>Train-the-Trainer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td></td>
</tr>
<tr>
<td>3 hours</td>
<td>16 hours</td>
</tr>
<tr>
<td>Format</td>
<td></td>
</tr>
<tr>
<td>“State of the art” lecture by a senior cardiologist with didactic expertise</td>
<td>Didactic (using different didactic formats), repeated (four sessions) and interdisciplinary (GP, Cardiologist and Psychosomatic Specialist) educational intervention</td>
</tr>
<tr>
<td>Additional material</td>
<td></td>
</tr>
<tr>
<td>Clinical practice guideline</td>
<td>Individual level pharmacotherapy feedback of participating patients (from data of the baseline documentation). Second session: case audit Fourth session: pharmacotherapy feedback and case audit</td>
</tr>
<tr>
<td>Attendance at education</td>
<td></td>
</tr>
<tr>
<td>Article suggests 100%</td>
<td>Article suggests 100%</td>
</tr>
</tbody>
</table>

Follow up was at seven months and patient follow up was two months after the final session. (423) But again this timeline is unclear and imprecise. There were 91 patients in the intervention arm and 77 in the control arm, but this had dropped to 80 and 66 by the end of the study, although a few patients were excluded as they had absolute contraindications. (423) Analysis was per protocol and not by ITT.

In another parallel finding with figures found in this thesis, ACEi/ARB prescription rates decreased over time although in this study there was a 2% drop in intervention and an 8% drop in control (no comparison). (423) No difference was seen between groups for β-blocker prescription, and there was no change over time. (423) Intervention group patients were more likely to be prescribed AAs at 7 months (43% vs. 23%, OR 3.5, 1.1 to 11.1; p=0.04). (423)

Patients in the intervention arm were also more likely to receive target ACEi/ARB dose (27.5% vs. 15.2%, OR 3.3, 1.0 to 10.2; p=0.04) but no difference was seen in β-blocker doses (13.8% vs. 12.1%). (423) Similarly, the intervention group achieved significantly higher mean percentages of daily target doses for ACEi/ARBs over seven months (42%
to 52\% vs. 43\% to 42\%, mean difference 10.3\%, 0.8 to 19.8\%; \(p=0.03\)), but no significant difference was seen between groups for β-blockers. (423)

COPD and DM were relatively prevalent (19.6\% and 36.5\%) but were not associated with non-prescription of β-blockers (423) which is unusual. As has been demonstrated in other studies (33, 252, 352), age was associated with non-prescription of β-blockers. (423)

There was an imbalance between the study arms in median practice size at allocation (intervention median practice size = 5.5 vs control median = 3) which diverged further at analysis (intervention median = 6 vs. control median = 2). The practice was included in the model (random effect nested in group) but this is for ICC within each practice due to clustering from randomisation at practice level (423), rather than the potential effect of practice size.

**INTENSIVE TRAINING – β-BLOCKERS:** The reported low use of β-blockers in their country by the Euro-HF, IMPROVEMENT and SHAPE studies, triggered research in Spain, in patients recruited between early 2004 and early 2006, into the feasibility and tolerability of up-titrating β-blockers in primary care patients with stable HF. (422) The premise was that primary health care physicians can safely do this in appropriate patients. (422) This approach was encouraged by the cardiologist who participated in the small group CME of this thesis, 12 to 18 months before this Spanish study started recruiting.

There was a clear titration protocol, including procedures to manage adverse events and a definition of maximum tolerated dose. GPs were able to consult with a cardiologist about β-blocker selection and prescription. The intervention involved four hours of theoretical training on HF and practical workshops on β-blocker dosing. (422) Unfortunately the article does not elaborate on the content of the educational intervention. It is not possible to determine the facilitators/influences provided by the training and the practical components of the intervention.

The sample size was calculated (\(n = 88\) patients) and the assumed treatment tolerance was 89\%. (422) Even in a carefully selected patient population this seems at the upper end of guideline adherence. (44, 58) The study was able to detect differences >7.5\%, which is in line with the expected effect sizes expressed by Grimshaw et al. 2004. (182, 422)
The patient exclusions (e.g. signs or symptoms of overload, cardiovascular exacerbation or instability three months prior to the study, 2\textsuperscript{nd} or 3\textsuperscript{rd} degree AV block, SBP $<80\text{mmHg}$, or HR$<50\text{bpm}$) (422) were reasonable except perhaps for the patients asthma or severe COPD. These conditions should not be considered contraindications (375, 376) especially in this context where prescribing decisions were discussed with a cardiologist. COPD patients who participated were significantly more likely to be prescribed a non-cardioselective $\beta$-blocker (carvedilol) than a cardioselective $\beta$-blocker (bisoprolol) (422), although this is pharmacologically counterintuitive.

Obese patients were more likely to be prescribed bisoprolol which makes sense since for patients $>85\text{kg}$, the daily dose of carvedilol is doubled, increasing the up-titration process.

The strategy was successful – treatment at six months was maintained in 97\% of patients, 75\% reached target dose, 22\% achieved lower than target dose. (422) However this is a very short time frame and it would be useful to know how fluctuations in HF would affect the use of $\beta$-blockers. This time frame does not assist in answering the anecdotal observation of medication withdrawal in HF patients over a much extended length of time. In this Spanish study 70\% of patients experienced adverse effects, most common between weeks 2 and 4, but only three patients discontinued treatment (in the first week) due to adverse effects requiring hospitalisation. (422)

This study did not have a parallel control group of usual care. Sampling was consecutive but the study does not supply further information, for example on how many patients declined participation. The prescription rates seem very high, on par with the ‘mid-way clinic’ model (279) described in Chapter 3 of this thesis. The Spanish study patients were ‘young’ (mean age 65.6 yrs, SD 8.5yrs) and the sex ratio favoured males 3:1, which is unusual in primary care studies, including this thesis, as the male:female ratio is usually 1:1. (6, 286, 300) The patients were recruited from primary care which is a good starting point for a study of community-based management. The titration instructions and management options for adverse events were detailed. This and the encouragement to use specialist advice on a less formal basis than a referral seems aligned with the Decision Support dimension of the Chronic Care Model (as discussed in the next section). (425-427) Given all the exclusion criteria, this study has set up a somewhat artificial environment. These patients would normally be seen by a GP in NZ. Their GP would have to use their clinical judgement of the patients’
circumstances and suitability for treatment (48-51, 65) to determine whether it would be appropriate to prescribe. In this situation there was a close relationship between primary and secondary care and the discussion of more complex patients would have provided an opportune learning experience. The domains of implementation of evidence-based practice that could have been supported are Knowledge, Skills, Beliefs about capabilities, Beliefs about consequences. (166) The role of the practice nurse in patient management was not mentioned in this study, and there is scope for their assistance in β-blocker titration and monitoring.

The study underlines the need for enthused and motivated participants (422), analogous to parts of the Organisation of the health care delivery system dimension of the Chronic Care Model. (425-427) The detailed dosing protocol was undoubtedly useful but there is no indication of whether this was paper-based or whether it was contained in a computer prompt or as a computerised decision support system (see section 6.6.4).

A larger study discharged patients (n = 627, no date given for data collection) from 53 Spanish hospitals, with a principal diagnosis of chronic HF, back to the care of GPs (in 292 health care centres) who were either trained in the optimisation of β-blockers or ‘usual care’ GPs. (428) Only ‘elderly’ patients were included (≥70yrs), mean age 78±5 years and 42% were female. (428) Patients who were excluded had (amongst other usual exclusion criteria) bronchial hyper-reactivity, who may have been potential participants, and those who required close follow up in specialist or HF clinic. (428)

The intervention included written material based on recommendations from the ESC and AHA HF guidelines and an interactive meeting between the lead researcher and the primary care physicians at which time practical aspects of β-blocker therapy were discussed and the training material was presented. (428) This description of the intervention lacks detail, and as such it is difficult to determine the mechanisms of change.

Randomisation was at practitioner level and not at practice level. There was no adjustment for clustering. Patients were not randomised to ‘trained’ or usual care – the first 7 patients at each centre saw the ‘trained’ doctor. Analysis was per-protocol and not ITT.

β-blocker use was extremely high at baseline (90% intervention, 85% control) and at 3 months (92.5% and 88%). (428) The mean doses at baseline are not supplied. No differences in drug doses prescribed were seen at baseline or at final visit. (428) No
between group differences were seen for outcomes (total cardiovascular events including readmission for HF or in days hospitalised). (428) No between group differences were seen in withdrawals of β-blocker therapy (7%) or in severe adverse events (10%). (428)

Similar proportions of patients in each group achieved maximum β-blocker dose (25% vs. 19%, p = NS), but significantly more intervention patients achieved maximum dose/maximum tolerated dose at 3 months after discharge (48% vs. 38%, p=0.014). (428)

Analysis of patients who received maximum dose/maximum tolerated dose indicated they had less HTN/HTN aetiology and more previous MI. (428) Significant predictors of dose were training group and initial β-blocker dose. (428) Other factors which may have been relevant (close to non-significance) were ischaemic aetiology, lower EF and higher BNP. (428)

The short follow up period may not have allowed up-titration or had any effect on morbidity/mortality outcomes.

6.6.3 Back to basics: the Chronic Care Model

As can be seen from the examples above, study quality is variable as are the interventions and outcomes, creating difficulties in determining which aspects of the study are effective and should be explored further. Given the complexity of some aspects of clinical management and the uncertainty regarding where the responsibility lies (primary or secondary care), it may be more logical to turn attention to primary care, where HF patients are seen most often, and put strategies in place to manage chronic diseases well, and only once this has been done, to focus on on-going inappropriate care and only then specific implementation strategies and the theories that may assist the change in management.

The attractive aspects of the CCM are that it provides concrete examples of how to improve practice rather than offering only abstract theories. (426) Theories are still being widely debated, with no one theory reaching prominence. There is also the challenge of integrating theoretical perspectives into empirical evaluations.

One problem with primary care is the ‘tyranny of the urgent’ which has been proposed as a reason for the shortcomings in chronic disease care wherein acute symptoms and
patient concerns take precedence over the need to optimise management for chronic disease. (426) CCM proposes that effective care needs appropriately organised delivery systems, linked with complementary community resources available outside the organisation. (429)

The chronic care model uses six organisational level dimensions (425-427)

1. **Organization of the health care delivery system**: structured to focus on chronic illness care, and practice leaders prioritise improving this care. There should be support for improvement at all levels, and promotion of effective improvement strategies for comprehensive system change. (425-427)

2. **Community linkages**: with community-based resources that assist patients with chronic illness (e.g. self-help groups, exercise programmes, or hospitals offering patient education classes or home care). There would be referral systems to such agencies. Encourage patients to participate in effective courses and groups. (425-427)

3. **Self-management support for patients**: encourage patients to play an active role in their own health by providing programmes that encourage lifestyle changes and develop competency in illness management. Emphasise that lifestyle aspects such as diet, exercise, self management and medication use are under patient control and include family members. Use strategies such as collaborative goal setting, action planning, problem solving and follow up. (425-427)

4. **Decision support for primary health care staff**: i.e. evidence-based guidelines for treating chronically ill patients, integrate guidelines into patient care by using reminders/prompts in the electronic medical record. Provide primary care physicians with easy and timely access to specialist expertise. Include all relevant primary healthcare professionals in education to reinforce guidelines using ‘champions’ and effective methods. (425-427)

5. **Delivery system design**: provides continuity between primary and specialty care, and allows for team-based, non-urgent chronic care visits with regular follow up. Division of tasks within the practice needs to be defined. Culturally appropriate and comprehensible care should be provided. (425-427)

6. **Clinical information systems**: should include a patient registry. Individual- and population-level information on chronically ill patients will give primary healthcare professionals performance feedback and care reminders, enabling individual patient care plans. The registry will also assist in determining sub-populations (i.e. high risk) who would benefit from proactive care. (425-427)

As described above, an engaged patient is an important component of the model. Patients are informed – understand their disease process; are activated; and are empowered to self-manage their care. (426, 427) This needs to be matched to a

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Encouraging patients to take responsibility for managing aspects of their condition is not straightforward as there can quite often be a lack of dialogue between patient and care provider about HF. This is covered in section 6.8.2 under ‘Responsibilities for management’
prepared and proactive primary care team that has the right information, decision support, equipment and personnel at the time of the visit, to result in productive interaction. (426, 427)

Clinical care is customised as stepped protocols (427) as illustrated below (426, 429):

- Level 1 indicates that patients have reasonable control over their condition, and receive care from the primary care team.
- Level 2 patients have poorly controlled conditions, and are referred to disease-specific care managers who could be from a range of health professions and who are mentored by disease-specific physician champions. Care managers are responsible for a list of patients. They work intensively with these patients for 6 to 15 months with the aim of improving the patients’ control of their illness to level 1 quality.
- Level 3 patients have complex multiple diagnoses, are high use patients or both, who receive case management from registered nurses or medical social workers within the primary care team. Case management at this level is not disease-specific due to the multiple co-morbidities.

**CCM IN PRACTICE:** The extent of implementation of these processes and systems was assessed (early 2000s) in large medical groups that offer high quality care (e.g. Mayo clinic, Permanente, Puget Sound and six others). (429)

Data were collected on care management processes (e.g. practice guidelines, population disease management, case management, health promotion/disease prevention for asthma, CHF, depression and DM) and clinical information systems that support chronic disease management (electronic medical record, recording of medical history, tests and procedures, diagnoses and prescriptions, computerised record of drug prescription, automated reminders and electronic information exchange with patients) and incorporate a number of practices of the chronic care model. (429)

While the use of practice guidelines by these medical groups was routine for almost all of the chronic conditions, implementation of other care management processes was less consistent. Similarly, not all organisations utilised all clinical information systems. (429)
Unexpectedly, no relation was seen between the level of clinical information systems and level of implementation of care management processes. (429) Other factors such as supportive clinical and administrative leadership, group culture promoting quality improvements, and other incentives to improve quality influenced the implementation of processes for chronic disease management. (429)

The most frequently mentioned barriers, as assessed by senior staff members (not doctors or practice nurses), to implementing CCM processes were lack of financial and staff resources (37%), lack of an adequate clinical information system (34%) followed by too busy doctors (22%), lack of additional payment for providing high quality care (17%) and doctor resistance (17%). (429) Facilitators were described as: an organisational culture that supports quality improvement, existing EMR or information system, supportive managerial and medical leadership (all 39%), organisation’s strategic plan, and support from external organisations (each 22%). (429) Studies based in the American health system have different push-pull factors than New Zealand, particularly financial and legal. Finances feature in this study of American health groups; however other organisational culture and technological factors assist in the implementation of CCM. (429) This study also illustrates the difficulties in implementing all aspects of CCM even for large well-funded medical centres.

In a 2008 American study related to chronic care management, doctors’ use of ‘care management tools’ for chronic conditions (asthma, DM, CHF and depression) was low. (430) In general practice/family practice, nurse management for all or some of the four conditions was low at 27% and only 10% of primary care physicians used nurse managers for all four chronic conditions. (430) The incidence of patient registries (to identify patients with specific chronic conditions) was better but still low at 43%, but 68% used reports on the quality of chronic care which could help improve care. (430) Out of 7 care management tools, general practice used an average of 2.8. (430) Only 29% of physicians reported that they had a fully electronic medical record. (430) An EMR can facilitate management of chronic conditions. Overall, smaller practices were less likely to utilise care management tools. (430)

Work around CCM has started in New Zealand in one of the District Health Boards that has high rates of poorly controlled chronic conditions. Counties Manukau DHB has implemented a computerised Chronic Care Management (CCM) system. (431) The focus is on high-risk patients with DM, CHF, COPD, cardiovascular disease and
depression, and enhances primary care for these patients by reminders, decision support and case management. (431) This system also assimilates aspects of Integrated Care (IC) e.g., preventive care, social care and care at support in the home, using the patient’s perspective as the ‘organising principle of service delivery’. (431)

The philosophy of CCM and IC is to implement systems of structured care that deliver current treatments and technologies to appropriate patients at the appropriate time. (431)

Additional funding is provided as well as training and support (also for IT – computerised template and decision support) since the CCM programme requires more time spent with patients to proactively manage their conditions, and according to the latest guidelines. (431, 432) Patients receive 4 free GP visits/year, 6 hours nursing time/year, structured notes, extended support from community health teams network, and a patient-held wellness plan (information about their condition and how to best manage it). (432) The proposed savings for HF were NZ$447/person/year. (433)

In 2007, only 240 of an estimated 3000 HF patients were enrolled. (431) This low proportion may indicate the uncertainty of diagnosis seen in HF patients. And, in similarity to the NSF (434), patients require an echocardiogram before entering into the HF programme. (431)

Key performance indicators for the Counties Manukau DHB CCM programme for HF are (435):

- Echocardiography-documentated ventricular failure (% patients)
- Systolic dysfunction on ACEi or documented contraindication
- ACEi >50% target dose or dose increased from baseline
- β-blocker use in NYHA Class II or III and systolic dysfunction or documented contraindication
- Spironolactone prescribed in Class III or IV symptoms and systolic dysfunction or documented contraindication
- Current smokers.

Patients will need to report a good understanding of HF, the role of their medications, their responsibilities to improve their condition, their role in managing their condition and an understanding of their Action Plan (of which they hold a copy). Patients also have a wellness plan – lifestyle changes and goal setting. Patients also need to report whether they are satisfied with their healthcare. Additional patient outcomes include
not smoking, reduction in alcohol intake, daily weight monitoring, cardioprotective/low salt diet, 30 minutes/day physical activity (if possible), annual influenza vaccination, and medication compliance. (435)

The programme is still being evaluated, but data indicate that savings in admissions and hospital bed days exceed those forecast. (432) More recent data (2010) indicate a decrease in smoking in enrolled HF patients, decrease in average NYHA class and 92% holding wellness plans. (436) 81% of HF patients are prescribed ACEi, 59% β-blockers and 39% spironolactone. (436)

Individual components of the CCM, such as Decision support for primary health care staff and Clinical information systems have also been tested as stand-alone items. These are covered in the next section.

6.6.4 Forward to the future: reminders to CDSS

The GPs who participated in the educational interventions for this thesis were asked if they had used any form of computerised prompts. The responses included statements that indicated they did not know how or were not able to set these up although a few GPs searched patient lists for HF diagnoses in order to recall patients. The computerisation and medical record software programmes in general practice can offer more sophisticated database management than this.

Reminders – generic prompts – may not be relevant to the patient who is before the GPs. Of the prompts for HF (from the Veterans’ Affairs medical centre’s own guidelines), 41% were agreed with by health care staff, but there were major disagreements with 12% of reminders and minor disagreements with 22%. Staff asked for reminders not to be displayed again in 7% of cases. (437) (Response was unknown for 18%.) The reminders that were disagreed with were thought to be wrong or unnecessary in 45% of cases or it was believed that the suggested medication would not be tolerated due to past experience or co-morbidity (33%). (437) The reminder system used was slightly more advanced than a generic prompt as it used patient symptoms, although these had been completed in a questionnaire prior to the clinic. (437) This is not a sustainable option for general or long term use. The reminder programme had no interface with other patient history e.g. co-morbidities, past medications, physical examination findings, past medications and adverse events. (437) The system needs to be more sophisticated to appear more relevant to the end users.
While computerised reminder systems are popular and have some evidence to support their use, the ultimate computerised aide is the decision support system. (438) Computerised decision support systems (CDSS) were developed in response to the 1999 IOM report that demonstrated inconsistencies in the delivery of quality health care in America. (439) The CDSS also provides standards-based care delivery and provides patient- and situation-specific advice by comparing patient characteristics with a knowledge base. (438)

CDSS are used for a number of care processes: prevention/screening, drug dosing, medical management of acute diagnoses and chronic disease management. (439) Studies on these processes including systematic reviews found that, while no harm resulted from the intervention, outcomes were either positive or neutral. (438, 439) There was great variability in implementation, design, type of CDSS (e.g. some systems required acknowledgement of each prompt and others allowed prompts to be skipped), completeness of CDSS, type of activity and outcome measured. (438, 439) Time frames of the studies were also relatively short. (438, 439) Overall, there was a positive effect on outcomes but further research is required with a more concise definition of CDSS that is fully computerised and not a computer-paper hybrid. (439)

Current general practice may not have the necessary framework, hardware or software to support CDSS. (438) If a system could be developed that integrated recommendations from multiple guidelines for patients with several co-morbidities, this would be a considerable improvement on individual guidelines that do not cross-reference.

A CDSS specific to the diagnosis and treatment of HF (small study with 5 GPs, reassessing 48 of their own patients) was based on three guidelines. (440) The GPs filled in templates (independent for diagnosis and treatment) to trigger the programme, with definitions of all terms that pop up when the mouse is held over them. (440) The diagnosis template recorded HF symptoms, clinical signs present at the visit, and echocardiogram result (42% impaired heart function, 27% normal echocardiogram, 31% no echocardiography). (440) The CDSS responded with diagnosis suggestions (see bullet points below) and suggestions on further investigations (31% echocardiography, 17% BNP). (440)
Suggestions on diagnosis:

- ‘CHF is present’ occurred when both symptoms and objective evidence were present
- ‘asymptomatic dysfunction’ occurred when objective evidence was present without any symptoms
- ‘suspect CHF’ occurred when symptoms were present but echocardiography was not performed
- ‘CHF is not present’ occurred when the result of the echocardiogram was normal
- ‘not possible to calculate’ occurred when there were no symptoms and echocardiography was not performed. (440)

GPs did not change confidence in 75% of their diagnoses, and confidence was increased in 12.5% and decreased in 12.5%. (440) Echocardiography was ordered for fourteen of the fifteen patients who had not originally had an echocardiogram. (440)

The treatment screen recorded patients’ NYHA classification, number of HF drugs, drugs recorded and related medical conditions. (440) The CDSS used this information to suggest additional HF medications. (440) The next table illustrates prescribed drugs, CDSS suggestions and the GPs’ responses:

Table 6.6.b. A comparison of drugs entered into template, CDSS suggestions and GP changes

<table>
<thead>
<tr>
<th>Drug</th>
<th>Prescribed</th>
<th>CDSS</th>
<th>GP Addition</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi</td>
<td>21 (44%)</td>
<td>11 (23%)</td>
<td>5</td>
</tr>
<tr>
<td>ARB</td>
<td>4 (8%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-blocker</td>
<td>28 (58%)</td>
<td>4 (8%)</td>
<td></td>
</tr>
</tbody>
</table>

Blank cells indicate no suggestion made by the CDSS or no change made by GP

One fault with this study is that the findings of the CDSS and the GPs’ responses were not evaluated by a cardiologist (as a gold standard) and that GPs were not given vignettes to compare inter-rater reliability. The CDSS as it stands does not seem to advise on drug doses. These need to be included given the tendency for low doses to be prescribed and/or for GPs to be reluctant to alter hospital prescriptions. (6, 36, 260, 355) There was the added ambiguity of hospital discharge letters that do not address up titration. (233, 235)

The benefits of a CDSS are that the template clearly indicates which investigations have been carried out and the suggested investigations are firstly from evidence-based
sources and secondly presented to the GP without the need for the guideline to be accessible at that moment. There is still the opportunity for the GP to exercise their clinical expertise and judgement, which has to be balanced against GPs’ reluctance to change hospital discharge instructions.

6.6.5 At a fork in the road
A challenge to implementing evidence is to determine what barriers exist, and which facilitators will help to overcome them. Numerous theories endeavour to explain how to influence change but are they the correct approaches to take for chronic disease? The CCM model proposes six areas of organisational-level modifications that could improve the management of chronic conditions. They seem logical aspects of care that should be embedded in primary health care clinics. There is a focus on team work and division of labour within a practice. These were slightly evidenced in practices that participated in this thesis, but could be better organised with specific protocols. The CCM also calls for champions to prioritise improving care. If these healthcare professionals are made visible the critical mass will be reached to propel the late adopters to change as well. Both immediate decision support (e.g. CDSS) to assist in answering queries as they arise or even before (instead of spending time hunting for a paper copy of a guideline or searching online), and easy access to and continuity with secondary care are needed and cannot be argued against; especially for a condition such as HF where secondary care can play an important management role and provide brief educational opportunities for the GP and other practice staff.

Perhaps then it is time to step back from multi-faceted theories and ensure that the ground work has been done.

6.7 Effects of implementation strategies in HF management
The implementation strategies tested for effectiveness in changing HF management in primary care were one-off small group CME, Internet course and self-directed learning (guideline only control group).

The first section describes the domains of an RCT (drug vs. educational intervention) and gives the details of these domains in the current study and some contextual aspects
of the study. The second section summarises the study outcomes and suggests some solutions. Section three investigates the extent and types of practice reorganisation reported by GPs in response to the educational intervention. Section 4 discusses additional influences on management behaviours (specialists, hospital admissions, other sources of information). The fifth section describes the learning experiences and preferences of GPs in each of the educational intervention groups. The final section discusses barriers to change in particular inertia, confusion over responsibility for management, and issues that prevent patients participating in their own care.

6.7.1 Introduction – the different faces of randomised controlled trials

The medical education curriculum has been described as:

“not like a drug, which can be given at standard doses, but instead contains many components, delivered with variable quality by different teachers.” (240)

In the example of a RCT, there is low variance (homogeneity) in the domains of context (situation, setting or organisation where the intervention takes place), content (nature or characteristics of the intervention), application (the delivery of the intervention) and outcomes. (441) The variance can also be controlled for in patient selection or in analysis. But in the delivery of education, as the degree of variance increases, generalisability becomes less likely. The impact of an intervention delivered with certain context, content and application variables, may not be a sound basis from which to draw conclusions and predict how the same intervention may work if the context, content and application were different. (441)

While in this study the context was primary care, there are reported differences in the way that male and female GPs – part-time and full-time, older and younger, single handed and group practices – practise medicine. (124, 126, 136, 140, 141, 143, 145, 146) Any differences between the study groups were shown to be not significant; but there were probably other organisational differences between the practices. The context could also refer to how the intervention was delivered, and of course the interventions chosen for this thesis were on the basis that they were dissimilar in order to test for differences between the two.

The content of the two active arms was very similar, but once the intervention had been delivered, it is not possible to determine how the practices tailored the information in their contexts, or what information the control GPs acted on. GPs may have initiated practice-specific changes in response to the educational intervention. At one small-
group session there was discussion between GPs from the same practice about how they should start recording NYHA classification in the medical records and how they should identify possible HF patients. However this is anecdotal and cannot be confirmed.

The application process may vary depending on the skill and experience of those involved, and the response or behaviour of the recipients. (441) There should have been little difference within each study arm regarding skill and experience. However, the response or behaviour of the recipients both immediately and later on could have differed. This process is relevant to this study once the GPs returned to their practices to try to implement the HF recommendations. The skill and experience of the participating GPs would have been critical: although GPs were unlikely to have much experience of recommending β-blockers to patients with HF. Application also refers to the response or behaviour of recipients (441)

The outcomes stipulated in this project were those that could be found in an empirical study – dichotomous for echocardiography and prescription, and comparison of change in proportions over time – but as discussed, these are not necessarily representative of the quality of care delivered (48-51, 64, 442) and in studies with high variance, there can be many outcomes which are not straightforward to measure (learning, development or behavioural changes at individual and/or organisational level). (441)

The question of whether something works has, for implementation research, been superseded by the ‘when’, ‘how’ and ‘why’ and the unravelling of the ties between context, content, application and outcomes. (441)

**THIS THESIS IN CONTEXT:** The study for this thesis began with more of an incline towards the former type of RCT; testing whether a CME event would change behaviour by measuring dichotomous outcomes. But without any formal conceptualisation, the beliefs held were that the social nature of the small group CME, with the influence of an academic GP and an expert (viewed either as a specialist or an opinion leader, depending on each GP) and the interactive session (media rich – described in 2.3.3), would have a greater positive effect on GP behaviour than either the Internet arm or the guideline arm.

This thesis was testing usual practice – a one-off education session. It would then be up to the GP and/or practice staff to determine how to use this knowledge and whether any further learning was required. The medical knowledge required to stay current falls into two categories: what any doctor in the particular field should know, and what
information is needed to do one’s specific job well. (153) The assumption was that GPs would assess the gaps in their knowledge – their estimate of what they ought to know or do and their actual knowledge and performance – and move to fill these gaps. The learning need is described as being associated with anxiety and the drive to reduce this is the motivation to learn and change. (147)

While single education sessions are considered normal for CME, this did not preclude GPs attending further CME from different providers, or seeking their own information from other literature, peers and specialists. The extent to which they did this is described in section 6.7.4. GPs seemed willing to rely on specialists for assistance with HF management. In contrast, GPs expressed varied views about the quality of care received by patients in secondary care and communication of this at discharge.

In the context of this study, it was thought appropriate for GPs to manage the translation of evidence into practice, develop protocols and determine responsibilities within the practice for various HF management tasks. The results of these issues are presented in section 6.7.3.

6.7.2 Study outcomes
This section summarises the study outcomes and offers some brief but evident solutions to some of the problems encountered.

The Cabana et al. review of barriers to guideline adherence (113, 175) reported that the particularly important barriers to adopting guidelines were lack of awareness and motivation, as well as perceived external factors. The GPs in our study were made aware of the guidelines, and the only barrier beyond their control was the waiting list for echocardiography. However the instructions conveyed in the education were to refer for echocardiography regardless of what was communicated by the local DHB regarding waiting times. There remains a question around motivation which again is a multipart construct. To motivate professionals there is a need to understand their needs, character, learning processes, environment, behaviour and behavioural influences. (171) An extensive list of theories can govern motivation, and these have been grouped into content, cognitive, psychoanalytic and environmental theories as explained in this cited reference. (171)
6.7.2.1 Echocardiography, prescriptions, doses, initiation and monitoring

ECHOCARDIOGRAPHY: The lack of referrals for the remaining 40% of patients who did not receive echocardiography is unsatisfactory. These patients had clinical diagnoses of HF. There is no substitute for the objective testing offered by echocardiography. Long waiting lists or patient age cannot excuse not referring patients as these excuses have the potential to adversely affect patient health. The proportion of patients receiving angiotensin inhibiting drugs may suggest that GPs are following the recommendation given in the NZ HF guideline diagnostic algorithm to treat empirically should there be delays in assessment. But patients still need to be referred and there is an onus on echocardiography services to write reports that are comprehensible and that suggest strategies for further management. Similarly, secondary care could ensure that no patient who is diagnosed with clinical HF is discharged without an echocardiogram. Planning should include spaces left in the echocardiography schedule for the expected number of inpatients. Instructions on how to initiate and up-titrate medications need to accompany the echocardiography report. This would require consultation with a specialist and not simply a report on heart function being dispatched to the GP who may know little about the nuances of echocardiography findings.

ACEI: While different overseas studies at different time periods indicate an increasing trend to prescribe ACEi over time, (31, 32, 36, 260, 355) the opposite trend was seen in this study of the effects of implementation strategies on the management of HF in primary care. This finding was unexpected and the reasons for it are unclear. Without knowing the reasons behind the slide in prescriptions it may be difficult to halt and reverse. The proportions of patients prescribed high doses did not change over the course of the study. Certainly the evidence for the benefits of ACEi and of prescribing maximally tolerated doses is unequivocal. (13-16, 18, 331, 332)

β-BLOCKERS: The initial impetus for β-blocker prescription came from the hospital (admitted patients) in the pre-intervention period, but this did not seem to translate into primary care in the post-intervention period. By three years after the educational intervention (current management) β-blocker prescription had risen, but no data were collected about the doctor responsible for initiations. There is room for improvement and while GPs may be reluctant to initiate β-blockers, they could refer patients to cardiologists or cardiology outpatient clinics.
SPIRONOLACTONE: The prescription rates of spironolactone remained static throughout the study. The reasons are not clear but could include the lack of use of the NYHA classification system to determine whether patients were sufficiently unwell; that patients did not deteriorate to classes III and IV; or that GPs were not aware of the benefits of prescribing spironolactone. As hospital admissions during the study period were not monitored in depth, it is not known what role the hospitals played in initiating spironolactone. While the NYHA system has been criticised as being subjective (between and within observers), recording NYHA scores at each visit could be useful to monitor a patient’s status.

The role of the guideline is to “help clinicians make better decisions” (53) and without detailed knowledge of the patients in this study and their circumstances, preferences, contraindications, it is not possible to state with certainty that the decisions not to prescribe were not better decisions.

6.7.2.2 HF practice register and prescriptions

The effects of a HF diagnosis listed in the patient’s problem list were both expected and unexpected. The actual effects of the educational intervention on this analysis are certain – there were no overall effects. Except in one practice where several patients had HF diagnosis dates listed as a few days after the GP participated in the Internet CME.

The odds of receiving echocardiography were significantly higher for younger patients and those who experienced MI than older and those without an MI, but the timing of the diagnosis had no effect. Angiotensin inhibiting agents and spironolactone were more likely to be prescribed to those who had HF the longest and for patients who were younger, and ACEi/ARBs more likely at the start of the study. β-blocker prescription was not affected by the length of diagnosis. Patients were more likely to be prescribed them by the end of the study and if they were younger.

6.7.3 Practice reorganisation following educational intervention

The following data were obtained from the surveys that GPs completed prior to data collection commencing within their practices. As mentioned in 6.7.2, the study team expected that GPs would take responsibility for translating their knowledge gained from
the educational intervention into their own practice management and structure. This expectation was only reinforced on hearing a few discussions between GPs at the small group sessions and witnessing their enthusiasm to implement change. Other GPs were more reserved about potential changes to management. Some discussions were about using the NHYA classification system in their medical records, or ensuring that all suspected HF patients were referred for echocardiography regardless of the waiting time. It may be that these things were done by GPs who did not return their surveys but from the answers given, GPs were very candid about what was not done.

**GENERAL AND PATIENT FINDING:** Generally, GP follow up of HF patients after the educational intervention was approached passively – either when the patient came in for routine follow-up (3 monthly) or other opportunistic occasions. Very few GPs actively searched their medical records database to list all HF patients or to code the diagnosis of all HF patients. The work required to develop a query builder, checking all patients’ management and recalling inadequately managed patients was seen as too onerous when this approach was mentioned. Also many GPs do not know how to conduct such queries with their patient management systems. A few GPs recalled patients when they were due for a repeat prescription, to review medications rather than writing scripts without patient contact.

One GP made an effort to ensure all suspected HF patients received echocardiography. A couple of GPs referred to hospital discharge reports and intimated they continued the treatment as implemented by the hospital. Some GPs lamented that they did not audit their patients and therefore patients were not targeted and reviews were only opportunistic. Inertia to promote change was also seen in a highly motivated and interested group of GPs. (443)

**RESPONSIBILITY FOR RE-ORGANISATION:** A minority of GPs collaborated with colleagues, mostly through informal discussions. These discussions ranged from making others aware of the current guidelines and algorithms, to organising a meeting to discuss the guidelines in order to promote change through practice. However consensus about practice changes was not reached.

The lack of practice routine to discuss guidelines and manage change for common problems, making it difficult to translate guidelines into practice was also seen in a cRT of tailored interventions for guideline implementation. (443)
**COMPUTER-AIDED ORGANISATION:** Many GPs did not know how to add prompts to their electronic medical records, or were not aware that they could, or said that the computer system did not support this, or their records were not computerised or it was not within their authority. A few GPs did disclose that prompts could have been useful. A small number of GPs thought that prompts were not necessary. Computerised reminders and the more sophisticated decision support system have been discussed in 6.6.4.

**NURSES WITHIN THE PRACTICE:** Less than half the GPs had nurse involvement and this tended to be associated with BP checks, blood tests, smoking checks, education, weight monitoring, telephone monitoring for compliance or general patient follow up. One GP mentioned nurse involvement in Care Plus and one GP worked with practice nurses to identify patients with CHF to add them to the Care Plus programme. It was disappointing that more GPs did not utilise their practice nurses more fully e.g. to titrate or monitor increased β-blocker or ACEi dose, or organise practice-wide strategies or management decisions.

**OVERALL:** GPs tended to be unaware of how to develop their practice management software’s capability to assist in managing their HF patients. Collaboration between GPs within a practice was rare. Few GPs gave their practice nurses greater responsibility in managing HF patients.

### 6.7.4 Other influences on HF management

GPs were surveyed (pre- and post-intervention) to identify their information sources for changes in HF management. The first round was completed by 125 GPs. The second round responses may not be reliable due to the very small sample (n = 38).

**RELIANCE ON SPECIALIST INPUT:** Before the study (mid-2002), GPs identified specialists / hospital discharge letters as their third most common information source for changes in HF management. Professional contact in relation to clinical information sources and decision making (prescribing) is quite often highly ranked. (107, 120, 123, 125, 137)

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4 Care Plus is a primary health care initiative targeted at patients with chronic conditions and provides additional funding for more frequent visits or longer periods of care from members of the primary health care team including individualised care plans.  
A fifth of GPs (26 of 125) indicated that they relied on specialist or hospital management of their HF patients. This ranged from relying on hospitals to initiate β-blockers, only continuing discharge medication and not implementing any themselves, to not implementing any of the new changes in HF management without referral to a specialist. Only 7% of GPs said that they had implemented none of the new changes in management because of hospital / specialist lead in care.

84% of GPs said that the hospital made changes to HF management. The most common was β-blockers (mentioned by 69% of GPs), followed by spironolactone (41%), ACEi (22%) and echocardiography (16%).

Reactions to hospital management were quite mixed. Comments ranged from just ‘fine tuning’ of management that the GP would carry out anyway, excessive doses by the hospital, given a management plan about up-titration and addition of metoprolol and spironolactone, to not being provided with a clear and concise management plan.

By the time the post-intervention survey was completed (GPs = 38), specialist/hospital discharge letters were the least likely source of information (5th equal with colleagues). This is a considerable change from the first survey round, but may either be due to the respondents themselves or possibly their growing confidence in prescribing.

A few GPs indicated that they did not always manage their patients single-handedly. They said that they would leave severe cases of HF to a specialist, or that they relied on specialist advice when initiating or changing therapy, or that some of their patients were seeing specialists (presumably this meant that the specialists were directing treatment decisions).

By far the most likely change to management was β-blockers (81%), followed by spironolactone (35%), echocardiography (29%) and ACEi (26%).

Some GPs were quite critical of the way their patients were managed during their admission and at discharge, with aggressive work-up of doses and insufficient monitoring resulting in side effects. Management appeared variable depending on the service the patient was under and/or the clinician involved. Some GPs thought that the hospitals and cardiologists provided a good service. There was faster processing of patients, regular outpatient clinic reviews, and good advice regarding management given on discharge letters (described as ‘invaluable’ assistance in modifying practice).
These issues raise a valid point about the necessity for patients to be stabilised in the case of initiating β-blockers, i.e. patients need to be stable for two weeks prior to initiation. (29) Since patients would be admitted to hospital in an acute state, this raises a point of difference between secondary care and primary care management of HF. Perhaps the benefit of starting β-blockers in hospital on average outweighs the harms. It is not known if there was an internal hospital guideline that was being adhered to. The reported apparent variation in management links to the specialist input into management described in Chapter 5 which demonstrated the large variation in the usefulness of advice given to GPs in discharge summaries.

The small number of GPs who responded post-intervention raises problems with reliability, making it difficult to determine trends over time. GPs may have become more confident in managing HF and thus been less reliant on specialist advice. It is clear that some GPs still display a lack of confidence in HF management. As indicated by the initiation data (Chapter 5, section 5.3.3) hospitals take the lead in initiating β-blockers.

**SOURCES OF INFORMATION:** In the first survey (prior to randomisation) CME events and peer group meetings were the most common source of information (identified by 71% of respondents), followed closely by journals (51%). Generally these were not identified apart from New Ethicals, the New Zealand Medical journal, National Heart Foundation bulletins, information from the College (RNZCGP) and other weekly magazine-type circulars which may have had drug advertising. No major international journals were identified. Specialists and hospital discharge summaries were as frequently referred to as sources of information as drug company representatives (31%, 26%). Colleagues were not used frequently as sources of information (15%). ‘Other’ sources (14%) included the NZ HF guidelines, the ESC HF guidelines, Internet sites (Medscape, GP magazines but generally not specified).

Second round survey responses reported the same first two information sources as previously; CME events or peer group meetings (74%) and journals (55%). Colleagues and discharge letters were not often identified as sources of information about changes in HF management (each 21%). Drug representatives were more frequent sources of information (34%) but a number GPs (45%) identified ‘Other’ sources (NZ HF guidelines, Internet sites not specified apart from one GP in the Internet group who said the Goodfellow Unit site).
Two-thirds of GPs who responded post-intervention had further discussions around HF management – generally with colleagues, a few with specialists – and all bar one found these discussions helpful. The discussions tended to be a sharing of difficulties, learning new information and reinforcement of learning or a confirmation of practice. These discussions offered combined experience and gave the opportunity of discussing difficult to manage patients. The effect of the discussion tended to give GPs confidence in decision making, understanding that individual patients have different needs, and seeing how their management practices fitted with other GPs.

6.7.5 Participation in the study arms and effect of intervention type

GPs’ experiences of and reactions to the implementation strategies were reported in the study questionnaire.

CONTROL EXPERIENCES: The time taken for self-directed learning ranged from one hour to six hours. GPs liked that the intervention was convenient – they could do it in their own time and stop and start as they wished – it was comprehensive, it would result in better outcomes for patients, and the guidelines were always available for reference, except when they were buried in all the information that GPs received. GPs thought that their learning could be tailored to their needs (it was not determined how these needs were established). But many GPs said that the self-directed learning required discipline.

CONTROL PREFERENCES: GPs suggested they would prefer an interactive learning method that allowed social contact and the opportunity to ask questions and that small group peer CME discussions would be more effective to support their learning needs. Other proposals were more prompts to ask how it was going, and if the CME could be available on the Internet with updates whenever they became available.

SMALL GROUP EXPERIENCES: The small group sessions lasted for approximately two hours. The themes that emerged about GPs’ perceptions were that a ‘small’ group of colleagues made it easy to exchange ideas, with the size of the group removing barriers to participation. It was informal and it was with peers, which enabled them to share experiences and concerns openly, e.g. the uncertainties of diagnosis in primary care and treatment. Participants were reassured about ‘accepted practice’ and how to
implement management changes. The GPs enjoyed the interactive nature of the group and the free-flow of ideas, the open ended format and the opportunity to ask questions of the specialist and academic GP to clarify the key ‘take home points’. These findings reinforce other studies discussed in 2.4.7. (134, 225, 227, 228) These aspects of small group fit closely with a number of the theoretical domains proposed by Michie et al. 2005 (166) for example, skills, knowledge, professional role, beliefs about capabilities and beliefs about consequences, social influence and possibly even motivation and goals/behavioural regulation.

A range of dislikes were expressed: that the CME was time consuming; that ‘as always’ there was a need to travel and work at night; and that the facilitator was too directive and acted as an authority figure, with too much time spent indicating content and not enough on group discussion of quiz solutions.

It is possible that this GP had different expectations from the CME event. The objective was for the academic GP to take a lead and direct the session, not for a peer group meeting. There was also the need to ensure that all participants understood the information they had been given. It may be that this GP was already well read and understood the new evidence-base for HF management. The resources were not distributed before the meeting but handed out to GPs as they arrived and time was given for the GPs to settle in and browse through the folder. There would have been significant costs to mail or courier the folder to the participating GPs, plus additional folders would have been needed to give to the GPs who did not bring their copy. If the folders had been distributed in advance, there was no guarantee that GPs would have found time to read through them. Advance distribution may also have created a greater disparity in familiarity with the folder once the meeting started, which could have made those GPs who had read through the folder more impatient. Since GPs might not have been familiar with the NZ HF guideline and not wishing to delay the meeting, the facilitator pointed out from guideline the relevant pages that were needed to answer the question and GPs were left to consider and discuss possible answers.

**SMALL GROUP PREFERENCES:** One suggestion was a follow up session in a few months to check on progress. Another preference was for ongoing review (including a practice audit and review, follow up CME and audit at one year with introduction of a software clinical guide, and a formal search of patients with CHF in order to bring them in for review as opposed to opportunistic care). While the study stipulated that there
would be a record review at 18 months, in the PRA⁶ that GPs filled out they were encouraged to review medical records, identify any patients needing further investigation, and check key management issues. Survey results suggested that GPs did not self-audit and there was a lack of active case-finding.

**INTERNET EXPERIENCES:** Most GPs completed the tasks within one to two hours. GPs liked that they could stop and start or return to it if they had patient queries. They were able to do the CME when it suited them, not needing to go out for evening meetings or to arrange for a babysitter. Some liked the opportunity to work independently and without time-wasting peers. This implementation strategy was judged as convenient, which is similar to other studies’ findings. (211-213)

GPs could skip areas they felt comfortable and competent with, again as seen in other studies. (211-213) This suggests a need for Internet courses that all questions are answered before credits are given for completing a course, as feeling comfortable and competent with a subject does not necessarily equate to correct practice.

Dislikes of the Internet intervention included simply not liking to learn from a computer, or being unused to working on a computer-based course. Just over half the GPs missed the interactive nature of peer groups, and missed being able to ask questions and hear questions that others asked. A couple of GPs missed having a hard copy of the guidelines. The responses reported in this paragraph again echo other studies. (1, 4, 132, 133, 210-212)

While a number of GPs liked the freedom the Internet course gave them to complete it when they wished, discipline was required to make time to do it.

**INTERNET PREFERENCES:** Some of the GPs in the Internet study arm seemed to prefer the interactive nature of small group meetings The comment about the need for an opportunity to ask questions could only be solved by some form of simultaneous chat room or a message board where questions could be posted but these forms of education would need to be moderated by a cardiologist. The Internet site attempted to anticipate potential questions from questions raised in the piloted version of the CME and in the early small-group sessions. While some of the GPs in the Internet study arm did seem

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⁶ PRA. Practice Review Activity. GPs filled this out in order to qualify for reaccreditation points from the RNZCGP.
to prefer the interactive nature of small group meetings they acknowledged the many convenient aspects of the Internet course.

**Learning Preferences:** It may be thought that forcing GPs to participate in a non-preferred type of CME could have a negative effect on outcomes. This could be the case for learning styles (e.g. active vs. reflective) and cognitive styles (holist vs. analytic). One small study of year 3 and 4 medical students and internal and family medicine residents found this not to be true. (444) Learners with different learning styles, active and reflective were matched and mis-matched (randomised and cross-over) to two Internet-based modules, case series with a review activity (active: Multi-choice questions with feedback, participants required to answer questions until they were correct; reflective: open-ended questions, where participants were encouraged to view the information from different viewpoints, contrast cases and no answers or feedback were provided). (444) No significant differences were found between matched and mis-matched post-test scores or between active and reflective questions. The review activity consisted of reviewing a provided table of summarised module content or the participant developing a summary table. No difference was seen in post-test scores for modules that included tables or in preferences. The cognitive styles were not found to have an effect on post-test scores, or to have a preference for review activity. (444) While developing educational interventions that are tailored to differing learning styles and/or preferences is thought to enhance learning, this study suggests that the styles do not affect outcomes and nor do instructional methods.

### 6.7.6 Barriers to change

One systematic review described three different barriers. Knowledge – limiting adherence through a cognitive component, attitude – an affective component, behaviour – restriction of physician ability. (175) While it is possible to change behaviour without a change in knowledge or attitude, modifying knowledge and attitude probably produces a more sustainable behaviour change. (175)

The inertia of previous practice (or lack of motivation to change) requires a ‘priming’ phase and an initial force for change which could be one or a combination professional, personal and social. (175) One systematic review found respondents reported inertia of previous practice twice as frequently as other barriers to adherence (e.g. lack of
familiarity with guidelines, lack of awareness, agreement, self-efficacy, outcome expectancy, external barriers). (175)

The concept of lack of transferability between settings for strategies to target barriers was mooted in the late 1990s. (175) This same review also stated that the effectiveness of interventions [to improve adherence] will depend on the intervention and on the existence and intensity of baseline barriers. (175) They suggest a framework that could be useful to standardise the reporting of these barriers. (175)

However it may be the wording of guideline recommendations that needs to change. Recommendations that are more “behaviourally specific”, i.e. describe what, who, when, where and how, are clearer regarding what actions are needed, and determining whether these have been achieved. (168) These steps were unclear regarding β-blockers, and specialists also gave inconsistent advice in hospital discharge letters. Specifying behaviour may also allow observation of how the behaviour is linked to what occurs before and after. (168) Behaviour can be facilitated or obstructed by these ‘Antecedents’ and ‘Consequences’. (168)

**INERTIA:** One cluster randomised trial of tailored interventions for guideline implementation found little or no change in the outcomes. (443) Participants reported that the project was relevant to their practice⁷, but while there was intention to agree on common routines, many practices had had not managed to organise themselves to initiate a change process. (443) Respondents also conceded how difficult it was to change routine practice, and although there are many interesting topics, GPs ‘end up doing things as we have been used to doing them’. (443) Again this is despite enthusiasm and positive disposition towards what was seen to be a useful project. (443)

Furthermore the project had close to 100% response rate to the initial questionnaire, and relatively little disagreement with the guidelines, but some participants were simply not interested in the subject matter or project. (443) This study illustrates issues with inertia. Despite the interest in the project and agreement with the guidelines, which are important to achieve to overcome implementation barriers, the GPs themselves stated it

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⁷ There were two foci: most sore throats do not need antibiotics, clinical examination and laboratory tests are usually unnecessary, patients can be given advice by phone; and uncomplicated UTI, with typical symptoms, in non-pregnant women aged 16 to 55yrs can be treated with antibiotics without testing, women who have experienced previous UTI can be offered treatment over the phone. (443) The main components included a short summary of the main recommendations (electronic and laminated), patient educational material (electronic and printed), computer-based decision support and reminders, fee increase for telephone consultation for the above diagnoses, printed material to facilitate practice discussions about routine and change, interactive courses for GPs and assistants. (443)
was difficult to change patterns of practice. (443) Inertia is also an important barrier to the implementation of research evidence, and has been demonstrated in other studies. (158, 175)

**RESPONSIBILITY FOR MANAGEMENT:** The NZ HF management guideline (29) seems to suggest that primary care manages HF, possibly excepting β-blockers which should be started “by clinicians experienced with their use in HF or in specialist clinics”. The criteria for specialist referral also indicate that specialist referral may be considered “when β-blocker treatment is being considered”, or in “those who have inadequate response to treatment”. (29) Since patients may be managed across sectors, then some type of formal shared care arrangement may be appropriate.

Shared care may improve management of chronic disease. This model has the benefits of specialist intervention, and continuity of care from the GP who also manages co-morbidities and assumes responsibility for all other aspects of care. (445) This meta-analysis included chronic conditions such as DM, HTN, COPD/asthma, mental health and one study of HF. (445) Types of care reviewed\(^6\) included liaison (meeting between specialist and primary care team to discuss and plan ongoing management), shared care record card (usually held by the patient that includes an agreed dataset and more formal arrangement for information sharing), computer-assisted shared care and e-mail (agreed dataset collected in primary and secondary care and exchanged between sectors). (445) Aspects of shared care such as physical health outcomes, mental health outcomes and treatment satisfaction were no better than control but appropriate medications and medication use were significantly improved in the shared care models. (445)

Clinical service organisation (or disease management interventions) may also have an effect on HF outcomes. Patients in one meta-analysis (15 different interventions) had at least one hospital admission with HF. (446) Clinical service interventions could be in- or out-patient or community-based delivery. These had to deliver enhanced or novel service provision rather than prescription or administration of medications. (446) Case management\(^h\) and clinic model\(^i\) approaches\(^j\) were compared with usual care. Only high

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\(^6\) The design and quality of the studies were not consistent and follow up times were short. Cost data were not widely available and not easy to interpret. (445).
\(^h\) Case management: following discharge the patient is intensely monitored by a nurse (usually) through home visits and/or phone calls. (446)
\(^i\) Clinic models: outpatient clinics, run by cardiologists (special interest in HF) or specialist nurses using agreed protocols to manage medications. (446)
\(^j\) The intervention components, intensity and duration varied across studies. Study sample sizes were small, and studies were generally single-centre. (446)
quality studies demonstrated a significant reduction in mortality for case management compared with usual care. (446) The case-management OR for hospital readmission for HF was almost halved compared with usual care. (446) However critical aspects such as content and duration of the interventions varied. (446) Clinic care did not show any mortality benefit over usual care (hospital readmission not compared). (446) It was difficult to determine benefits of case management or clinic care for outcomes such as all cause deaths, HF-related mortality, event-free survival, all cause readmission, days spent in hospital, length of time between admissions, health related QoL, and cost analyses. (446) The information from the studies was considered insufficiently robust to form recommendations.

**BARRIERS TO PATIENT PARTICIPATION IN CARE:** This thesis did not collect individual patient feedback and so cannot comment on the study patients’ participation in the management of their condition. Barriers to patient participation in care relates to the use of the Chronic Care Model, or hand-held plan, or any model of care that actively involves patient responsibility. The ‘secrecy’ and guarding the patient from the knowledge of their condition will need to be resolved. (447) In response to a hypothetical vignette, GPs were significantly more likely to use euphemisms than the words “heart failure” to describe HF to a patient (e.g. “you have fluid on your lungs as your heart is not pumping hard enough”, followed by “your heart is a bit weaker than it used to be” and “your heart is not pumping properly”). (448) Although some GPs lapsed into complex terminology in order to “protect” their patients. (447, 448) GPs were significantly more likely to record “heart failure” in the medical record than communicate this to the patient. (448) The unexpected disclosure of their diagnosis to a patient caused confusion, anxiety and shock. (447, 449) Patients’ responses to the term “heart failure” compared with the GPs’ preferred euphemisms indicated they thought the condition had significantly more serious consequences and would last for longer, be more variable over time and cause more anxiety and depression. (447, 448) Patients who are unaware of their condition are unable to participate as fully as necessary in the decision-making processes regarding their condition and this could also hinder their self-management. While patient-centred management is desirable, the decision of whether this is appropriate for the patient needs to be made on an individual basis.
The effects of this failure of communication were seen in a nurse-led HF clinic where some patients failed to attend as they were unaware of their diagnosis. (450) Studies indicate that patients did not understand the implications of their condition as it had been discussed in a way they had not understood. (447, 449, 450) The delivery of chronic disease management services could not proceed effectively until the patient’s understanding had been assessed and remedied. (450)

6.8 Study strengths and weaknesses

This section discusses the strengths and weaknesses of the study and some aspects that could be done differently in future studies.

6.8.1 Strengths of the study

STUDY DESIGN: The cluster randomisation was used to try to avoid contamination of the different implementation strategies. The advantages of using cluster randomisation have been covered extensively in Chapter 4. Stratification of the practice sizes attempted to equally distribute the variables associated with different practice sizes across the study arms. Allocation was concealed. The different practice sizes were equally represented in each arm, by stratification prior to randomisation, and by chance at the end of the study. The numbers of patients for each GP varied widely which may have resulted in the management practices of one or two GPs dominating each study arm. However, the information about each study arm, GP and the number of patients of each GP was provided for analysis. Multi-level modelling was used to account for the nested effects of the data and the many variables that could have affected the outcomes.

This study design is superior to numerous other studies of HF management. Previous studies have used observational designs with inherent issues of selection bias, potential uncontrolled confounders and lack of blinding. Many studies use short follow up times, so even if the study is effective in the short term, long term effectiveness is unknown. However the opposite can be possible – a short term study may show no effects but the intervention may be beneficial in the long term. The study for this thesis followed patients for almost five years and gained a long-term perspective on HF management. Other studies use before and after tests of knowledge to determine the improvement in HF management. This thesis used medical record audit and did not rely on GP recall of
prescribing data or give GPs the responsibility for collecting data. Attempts were made to measure the effects of the intervention on whole practice HF populations. This was done to the best of our ability, except when GPs within group practices declined to participate or withdrew during the study or had no HF patients in the study timeframe.

The patient response rates were high at almost 75%. Considerable effort was required to obtain this figure as Ethics Committee requirements are that individual patients need to be consented for research (i.e. not an audit).

**STANDARDISATION OF DIAGNOSIS & DATA COLLECTION:** If the diagnosis of HF had not been transferred from paper to electronic record, the electronic prescription data searches gave an indication of which patients might have HF, which was where the Boston Score was used as a proxy for a diagnosis. An audit of medical records was necessary to retrieve all the information required for this study since GP recall of salient points of their management of HF is prone to error.

All patients were assessed with the same diagnostic tool (where information regarding HF signs, symptoms and tests was available) leading to a standardisation of patient data amongst those collecting the data, thus avoiding selection bias and adding rigour to the ‘diagnostic’ process. GPs were not asked to identify patients due to the amount of work required and also to avoid biases. The Euro-HF study is a prime example – the gap between assumed prescribing and actual prescribing of ACEi ranged from about 10% to 36%. (35)

To avoid detection bias research assistants were not told the study arm of the practices they visited. To further minimise bias, search strategies for potential patients followed the same pattern (where the practice management software allowed) and the diagnostic algorithm and data collection sheets were carefully written and had accompanying information sheets. Variation in interpretation of medical record data was also minimised by the small research team.

**TIME FRAME:** Besides the use of cRT for this study, another unique aspect is the long time frame. As mentioned above, many studies use cross-sectional data, or studies with very short follow up times, some of which even inadvertently exclude patients as there is insufficient time for them to be recalled for a check up. This was demonstrated in a
study (Netherlands, 2001, 16 peer review groups) in which only 53% of HF patients had a contact with their GP between the intervention and follow-up measurement which was a six month period. (451) The study for this thesis was designed to be longitudinal, initially up to 18 months after the educational intervention was delivered but was extended to 3 years. This purposefully long study would capture even a newly diagnosed patient from initiation and up-titration of ACEi with sufficient time to then monitor the subsequent initiation and up-titration of a β-blocker.

**INTERNAL VALIDITY:** This has been described as the ‘extent to which systematic error (bias) is minimised in clinical trials’. (452) Selection bias was negligible as practices were randomised – GPs were not allowed to select their intervention modality. Performance bias may have occurred depending on how much additional care was provided by other sources but this should have been equal across groups. Detection bias would have been avoided at every stage of outcome assessment as data collection sheets with clear instructions were used by all who collected data. Attrition bias could have been a large problem in this study, however despite the large drop-out rate, this was equal across study arms. The Hawthorne effect is unlikely given the lack of contact between the study team and the participating GPs after the educational intervention and also because the data collection was carried out at the end of the study – data relevant to the study had already been recorded. Even if the Hawthorne effect was operating, it would have been similar for all groups. But this may explain the lack of effect.

**EXTERNAL VALIDITY:** This has been described as the ‘extent to which results of trials provide a correct basis for generalisation to other circumstances’. (452) The areas covered include patients, treatment, settings and modalities of outcomes. (452) The patient demographics were well described including co-morbidity and risk factors, and represent an urban population, but the patient sample was less likely to be representative of lower socioeconomic areas. The treatment i.e. the method of implementation of the educational intervention (small group, Internet, guideline mail-out) was described. The setting was primary care – but there were also influences from secondary care, which is normal practice for HF management. The modalities of outcomes were clearly defined as was the follow up.
The external validity should also include practitioner variables. Compared with NZ GPs, participating study GP group had a similar proportion of male and female GPs. The participating study GPs had spent significantly longer in practice than NZ GPs and were more likely to be Fellows of the RNZCGP. GPs who have practised longer may have more familiarity with managing HF patients, and may be treating an older patient population (141). GPs with more experience tend to possess greater clinical intuition. However this tendency must be weighed up against the less positive attitudes to guidelines that older GPs tend to hold, since they are also less likely to believe in the potential of guidelines to improve quality of care. (59, 61, 104) GPs who trained more recently will have learnt about the utility of echocardiography, and may have been taught about using β-blockers in HF. The higher proportion of Fellows indicates that the study GPs will have higher levels of skills, knowledge and competence. (453)

**ASSUMED LEARNING MODEL:** The principles of adult learning are applicable (most) to the small group education and also to the Internet education. Adults are said to be motivated by learning that seems relevant, is based on and extends previous experiences, is active and participatory, is problem-focussed, allows responsibility for learning, can be applied in practice immediately, involves cycles of action and reflection, and is based on mutual trust and respect. (454) Mutual trust is an aspect of small-group CME, as is the active and participatory nature. Small group sessions also allowed experiences to be shared and questions asked. The Internet CME was also active and problem focussed and allowed responsibility for self learning. The Internet CME would have been respected as it was delivered by the Goodfellow Unit. The self-directed or control arm also had aspects of adult learning – it allowed responsibility, immediate application, and cycles of action and reflection.

Deep learning (an “active search for understanding”) is supported when participants are motivated by the need to know, active learning and small-group exploratory work, with well-structured knowledge base. (454)

### 6.8.2 Weaknesses of the study

The attrition rate was high but equal in all three study arms. Testing showed no statistically significant differences between the GPs who did not complete the study and those who did. The drop outs in the Internet arm resulted in a GP sex ratio that was
equal to the two other study arms, however the drop outs in the small group arm left more GPs who had been qualified for longer. The Internet arm (participating GPs) had a higher proportion of more recently qualified GPs. This distribution of length of time since graduation should ideally have been avoided since younger GPs may be more familiar with the Internet. However, since randomisation was clustered at practice level, the characteristics of the GPs that may have influenced outcomes were not the variables of randomisation. Randomisation by GP was not feasible due to contamination of implementation strategies within each practice, which means that the only option was to randomise by practice. The GP variables that may be thought to influence outcomes can be accounted for in analysis.

Performance bias is unlikely in this study since for this to occur, all patients/GPs/practices in one (or more) study arm would need to have been influenced. GPs in all study arms attended additional CME events or did some general reading on HF.

The fact that the randomisation was single-blinded was unavoidable. It would not have been possible for the principal investigator (VA) to run the study without being aware of the implementation method to which the practices had been allocated.

**INTERPRETATION OF MEDICAL RECORDS:** In a majority of practices that participated in this study, determining whether a patient had HF meant that both electronic and paper medical records were needed. This study had the difficult task of assessing and combining these two types of records. Paper medical records had to be read through in order to collect data and the standard of handwriting in some cases meant that data may have been missed because the writing could not be interpreted. Searching of electronic medical records was limited by the limited amount of data entered.

While attempts were made to reduce the variation in interpretation of the data in the medical records, the variation in data recorded by the GPs could not be controlled for. The amount of data ranged from sparse to detailed and the quality of the handwritten notes ranged from barely legible to easy to read. With the advent of computerised practices one problem that is increasingly less common is the illegibility of doctors’ handwriting. Differences in the ways that practices filed current information (e.g.
letters, laboratory results) and archive material may also have affected the quality of the data collected.

**TIME FRAME:** The time taken to collect patient data may have resulted in differences in patient health between the study arms. All patient data were collected at 30 to 42 months after the educational interventions had been delivered. Only patients who were alive at the time of the two first phases of data collection (patient identification and diagnostic evaluation) were asked to give their consent for information to be gathered from their medical records. Practices that had data collected towards the end of the study (more likely to be Internet or guideline participant) may have had healthier patients included (i.e. they had stayed alive for longer) than patients who had been identified at the start of the data collection, which may have influenced how GPs prescribed. The number of patients who died during the study period was not recorded and perhaps should have been. Practices were not approached at the end of the study to enquire how many patients had died since data collection. It is therefore unknown whether the death rate differed across the study arms.

**SAMPLE SIZE:** While the sample size of the original calculation was not achieved, the analysis of the four outcomes by study arm indicated that the change over time in each of the implementation strategies was similar, suggesting that the same influences were acting on all three groups. A larger sample size may have clarified some smaller scale outcomes e.g. spironolactone dose and initiation of medications. However the sample size was sufficiently large to demonstrate significant effects on HF management due to patient age and sex, and time.

The effect sizes were based on prescriptions achieved in a small study of local data. (30) These data indicated few HF patients were on maximal ACEi dose and few patients were prescribed β-blockers or spironolactone and so indicated that large improvements could be made, hence the estimated effect sizes. There was also the consideration as to what amount of change was worth committing resources to investigate i.e. would implementing a study that was estimated to change practice by 5% be worth the time and effort? But the sample size and effect size calculations were carried out prior to the Grimshaw et al. systematic review (182) that indicated the maximum change in practice achieved by implementation strategies is approximately 10%.
The high baseline level of ACEi prescription (overall 73%; guideline 71%; small group 84%; Internet 66%) had not been anticipated since the local and international literature suggested the opposite. For the primary outcome of ACEi prescription, there was little room for improvement, especially in the small group arm which had 84% of patients prescribed ACEi. This group had quite clearly reached a ‘ceiling’ of prescription, with a maximum adherence for recommendations of approximately 80 to 90%. (44)

Had the full sample size been achieved (i.e. all GPs participated) this would have created an insurmountable problem. It was difficult to recruit research assistants and the funding for the research assistants who were employed was spent before all the data were collected. Many grant applications were unsuccessful. Funding was very good at the beginning of the trial but when it became a problem it was too late to stop the study.

Practices and GPs lost interest in the study over time, although compared with some studies cited in 6.6, the GP and practice participation rates of 30 and 35% seem considerable. The study team thought that the payment for participating in the CME events (usual practice) and the provision of PRA/CQI\textsuperscript{b} points (for very little work on the GP’s part), which are generally hard to obtain as the audit activity needs to be endorsed by the RNZCGP, would be adequate to interest the GPs. Over time, the receipt of payment was probably forgotten and GPs participated in other audit activities to gain their MoPS\textsuperscript{1} points (for their triennial cycle). A few practices declined to participate simply because of the lack of space in their practice i.e. there was no spare room or computer for data collection.

It may simply have been too difficult to keep GPs interested in the project over a three year time frame. GPs were not sent any update material or contacted between the educational intervention (i.e. a ‘low intensity’ intervention) in order to preserve what would naturally occur after participation in a CME event. Any additional contact from the study team could have caused them to behave differently and the study question was to find out what happened to HF management after a ‘one-off’ educational intervention. From this perspective it would not have been possible to run this study differently. Perhaps this study indicates that this model of CME is not sufficient to cause change and sustain change, but attending more CME events takes time – either from practice time or after hours, neither of which is seen as desirable. While the interventions were brief, the GPs were free to attend additional HF CME events, talk to specialists and

\textsuperscript{b} Practice Review Activity / Continuous Quality Improvement
\textsuperscript{1} Maintenance of Professional Standards
colleagues and to do further reading. The responsibility was with the GPs to determine how to alter their systems to achieve change in practice. Software support is readily available and GPs could have sought assistance to add reminders/prompts that were relevant. Instead this seems to have been put in the ‘too hard basket’.

**AUDIT:** We cannot say whether the referrals or prescriptions indicate the quality of care. Aspects of care such as prescription or referrals are measureable, and guideline recommendations indicate thresholds of standards of care, but the importance of dimensions that are not captured also need to be given value. (455) An audit can measure whether a particular item of care has been completed or not and as such, an argument could be made for Evidence-Determined Practice; that is for patients, and a relevant set of evidence, the medical treatment should be equal, regardless of who the doctor is (i.e. evidence can or should determine practice). (51) Thus it should be possible to reconstruct the decision made, and if practice is standardised, this generates equality and security for both doctor and patient. (51) Although there may be theoretical merit in this approach, it is not realistic. The heterogeneity of patients in primary care suggests it would be inappropriate to follow this methodology. (51) Evidence-informed practice is more flexible, but does not take into account the patient perspective. A model of evidence-informed patient-centred practice takes into account the doctor’s history with the patient and their knowledge of the patient’s health status, potential benefits from treatment, and preferences. (51)

Additional aspects that need to be considered to determine quality of care include acceptability, accessibility, effectiveness, efficiency, equity, legitimacy, and patient-centredness. (456)

When prescribing, the doctor needs to take the recommendation and apply it to the individual patient in the consultation using their clinical expertise, judgement and experience and the patient’s individual characteristics, preferences, and local availability of services. (457) If these circumstances are not recorded and not known to researchers, then a dichotomous yes/no for prescription of drug or referral to a service will not fully explain reasonable and rational decisions by the patient and the GP.
6.8.3 Lessons learned

This study was initially planned to focus on β-blocker prescription in primary care since it was a new development in HF management. A funding body indicated that they preferred to fund a larger study. In response, the use of echocardiography, ACEi and spironolactone prescription were also added to the educational intervention. In retrospect, this was possibly a mistake. A smaller study, implementing one recommendation, would have been more manageable and more focussed, allowing in-depth evaluation of the barriers and facilitators to prescribing. More patient data could have been collected to determine QoL, quality of care, and patient experiences.

Regarding the current study, the objective was to test the effect of a “one-off” CME intervention in a randomised trial over a longer time than other identified studies of Internet CME and/or HF management. In this respect it would have been difficult to run the study differently. The significant drop out was probably unavoidable given the study.

This study may have been helped by more precisely defined practitioner and patient exclusions, with these tabulated. This information would have enhanced external validity. Additional data that could have been collected include the number of patients who died during the study period, although there may be complex Ethics committee requirements for that type of data collection.

Power calculations would have taken into account the degree of change observed in other studies. For example the Grimshaw et al. 2004 HTA report, although published after this study started, indicates that the anticipated change was optimistic to say the least. However this does raise the question of whether statistically significant change is clinically significant and vice versa. Furthermore, at what level is clinically significant change measured?

Clearly, the CME payment and the CQI points offered to GPs in return for their participation in the study were insufficient to motivate most GPs throughout the study period. This would be important to know for any future long-term study in primary care. Financial incentives are not always possible given the straitened research funding in NZ.
6.9 **Recommendations**

The recommendations from this thesis take three forms. Practice recommendations for both primary and secondary care, policy recommendations at national level and at local level – District Health Board (DHB), and finally research recommendations.

6.9.1 **Practice recommendations**

The following are aspects of practice, discovered during this study, which could be improved upon.

6.9.1.1 **Medical records**

1. Delegate responsibility to one or several practice staff members to maintain electronic medical records on a daily basis and identify when diagnoses are made outside of the practice.

There may be a role for a ‘medical records summariser’ to compare computerised records against paper notes (especially important when a patient moves practice), specialists’ letters, and to check whether diagnoses are confirmed/provisional/excluded. (458) For HF the tasks would include entering results such as echocardiography, systolic or diastolic function. Ideally the medical records summariser would be clinically trained, and have sufficient time to read the material. (458) From my experience in collecting data in general practice, this seems like a very worthwhile addition to any practice. Updated and accurate problem lists can be expected to improve the quality of patient care. The CCM emphasises that one member of the primary care team needs to be responsible for maintaining the HF registry. (426) This responsibility appears sound but depending on the time and skill required to fulfil it, another staff member may need to be employed. The additional cost of this and how it would be financed and organised would need to be worked out.

2. **Promote a system of incoming and outbound communication in electronic form whenever possible.** Paper communication should be scanned and appended to the electronic file. Request that all incoming correspondence be received electronically. The entire medical record should convert to paperless form from a set date.
Paper records where still used in practices, need to be filed chronologically or by medical speciality rather than being inserted randomly into the patient’s file as was the case in some practices involved in this thesis. More respect needs to be given to these critical records of a patient’s medical history. A well maintained file will expedite the location of past documents. Medical records that blend paper and electronic records are impracticable. All laboratory results and other relevant correspondence can be received electronically or be scanned and then recorded. Likewise, all outbound correspondence can be feasibly written into the computer and saved, with a back-up system external to the practice.

3. Implement the recording template used in the Swedish CDSS study (440) or some form of prompt that appears for each HF-related visit.

GPs’ notes from clinical consultations need to clearly express reasons for discontinuing medication and not up-titrating, and record intentions for future medication changes. This will facilitate the coordination of care. Other GPs within a practice can then continue with a management plan at an opportune time. A rare but illuminating example of how helpful this can be was noted in 5.5.4.2, with respect to the GP’s clinical findings and reasoning for not introducing a β-blocker. There may be a place for indicators for the appropriateness of long term prescribing. (459) If they could be developed, indicators could usefully include the following criteria (459) in HF:

Table 6.9.a. Examples of prescribing indicators that could be useful for HF

<table>
<thead>
<tr>
<th>Dimensions of appropriateness of prescribing (459)</th>
<th>Examples for primary care HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reasons for prescribing the drug are:</td>
<td>NYHA classification to ensure prescription to appropriate patients.</td>
</tr>
<tr>
<td>• Stated in the medical record</td>
<td></td>
</tr>
<tr>
<td>• Recommendations / expert opinion</td>
<td></td>
</tr>
<tr>
<td>Drugs are not prescribed when they should be, according to:</td>
<td>E.g. for β-blockers would act as an additional challenge to prescribe or to identify contraindications.</td>
</tr>
<tr>
<td>• Evidence-based guidelines or similar</td>
<td></td>
</tr>
<tr>
<td>The expected clinical benefits are realised in the patient</td>
<td>Assess whether patients are benefitting from the medications. If there is no improvement investigate why (e.g. dose too low to give benefit). For HF these may be medium- and long-term rather than short-term.</td>
</tr>
</tbody>
</table>
Unexpected drug reactions:
- Are recorded
- Result in the drug being discontinued in that patient, or
- If continued, the patient is monitored closely

Necessary to assess whether risks outweigh benefits, for other practice staff to be aware of any further adverse events, and to avoid a re-challenge with a drug that is not tolerated.

The dosing schedule is made as simple as possible

This is particularly important as patients are likely to have multiple co-morbidities, and may be prescribed different drugs from the same class which then may be rationalised.

The patient is given information about why and how to use the treatment

As above, and also gives patients an active role in self-management which may be above and beyond adjusting frusemide doses for weight fluctuations.

There is a summary in the medical record of:
- All prescribed drugs
- And regular OTC drugs

All a patient’s medications are not necessarily prescribed at the same time. One clearly displayed list will help to identify potential drug-drug interactions in the current regimen and prior to prescribing any new medication. OTC medications can interact with prescribed medications to cause adverse events, reduced effect or potentiated effect. This list should also include supplements.

The patient on long term medication is reviewed:
- At least once a year/in accordance with evidence-based guidelines/expert opinion

Provides the opportunity to review all of the above.

The table above describes several aspects that prevented this thesis from determining whether prescribing was appropriate e.g. lack of NYHA, inconsistent ‘front page’ (paper or electronic) of all current medications, incomplete recording of contraindications or failed medication challenges. Including this sort of information in a medical record can only improve continuity of care and communication with secondary care regarding HF patients.

Basic measurements such as BP, HR and weight should be made at regular intervals. These variables are also useful in shared care within a practice. HR and weight were seen to be especially lacking in the data collected for this thesis. In the pre-intervention data collection period 27% of patients had no HR recorded, rising to 33% post-intervention. The HR is important for β-blocker initiation and monitoring and weight is
particularly important for monitoring potential fluid gain and deterioration in HF status. So few study patients had weight or BMI recorded that it was not reported by this study.

4. Improve communication between primary care staff and patients regarding their condition so that patients can assume more control over their condition.

Patient responsibility for aspects of their condition is encouraged in the CCM model and also in the Counties-Manukau DHB version of Chronic Care/Integrated Care. We respect the personhood and moral agency of patients by accepting their frequent capacity to self-care. For example, they can monitor their daily weight, adjust their frusemide dose as necessary and stop spironolactone if they experience dehydration (diarrhoea or vomiting). The CCM and the Counties-Manukau DHB model are discussed in 6.6.3. Barriers to patient participation in care are covered in 6.7.6.

6.9.1.2 Hospital correspondence

Given the high likelihood that patients with suspected or confirmed HF will be hospitalised, the hospital admission provides an opportunity to refer the patient for diagnostic tests not immediately available to the GP and to scrutinise and revise medication regimens. Feedback which reiterates important recommendations will help the GP to remember them and apply them to other patients. The presence of the hospital diagnosis and procedural data has also been shown to improve the quality of data in primary care. (231)

5. Echocardiography: Request that hospitals standardise EF values and qualitative descriptions of HF, and provide clear explanations of the findings.

6. Medication instructions and template: Introduce a standardised template for discharge letters, including recommendations for future management. (235)

Chapter 3, section 2.6.5 illustrated the differences in EF that are used to define HF, and the difficulties experienced in primary care interpreting echocardiography reports. These differences underpin the need for hospitals to standardise their use of definitions of HF. Outgoing correspondence should clearly and concisely explain to the GPs the meaning of test results such as echocardiograms.
Similarly, any advice given on medications needs to be appropriate, and consistent with guideline recommendations to reinforce this knowledge for the GP. Details of up-titration steps, potential adverse effects and target drug doses need to be described. Information about monitoring should be included and discharge summaries or outpatient clinic letters need to arrive at the GP’s practice in a timely manner. (232, 234, 236, 460) These needs can be feasibly met. Agreement has been reported between primary and secondary care regarding the key details to include. (232) Important aspects of consultants’ replies to GP referral letters were thought to be ‘appraisal of problem including diagnosis’ and ‘management plan’ by over 97% of GPs and consultants. (232) Introducing a template to structure this hospital correspondence would improve the quality of data in primary care. (231) This is made salient by the common lack of detail in the letters sent for patients who need regular monitoring of their medication after an admission or outpatient visit. (234) Templates, if adhered to, would help to ensure that full and complete information is conveyed to GPs, minimising differences in recorded information between hand-written and electronic discharge summaries. (461)

6.9.2 Policy recommendations

7. Set clinical indicators relevant to patients who have the greatest potential health gains accompanied by appropriate motivators.

The one strategy that has been shown to cause a large change in behaviour is the financial incentive of the UK’s GMS. (462-464) An example is the improvement in ACEi prescribing for HF patients over a two year period – from just under half to over 80%. (303) The change in practice seems too large to be attributable to other changes that may have occurred in primary care or the NHS in general. The increase in ACEi prescribing in HF showed an extremely high health gain with up to 308 lives saved/100000/yr\textsuperscript{m} but the maximum payment for a typical practice/yr was £2400. (465) This contrasts with statins in IHD, and HTN. (465) Care therefore needs to be taken to incentivise activities that will produce the greatest population gain.

However the resources that developed and are funding the NHS framework are not available in all other countries including NZ. The concept of a national quality and outcomes framework is appealing – for example in NZ, where GPs operate under the

\textsuperscript{m} Newer analysis of the data may be able to express lives saved as QALYs which would be a more accurate definition. (465)
PHO Performance Management Programme which has recently introduced its second phase Clinical Performance Indicators. (466) But debate is needed about the ethics of additional payment for providing a standard of care that should already be provided. In NZ, performance payments for achieving the Indicators are calculated at PHO level, are paid to the PHO and make up only a small portion of primary care income (431, 466, 467). They are unlikely therefore to be effective as a motivator at the provider level. (467) The question now is how to blend appropriate extrinsic motivators with the innate satisfaction that professionals ought to derive from doing a good job.

8. Allow unrestricted access to BNP as a first option for all suspected HF patients (apart from acute cases). Discussions regarding budgets need to take place between the test provider and the Auckland DHBs providing echocardiography.

The cost of BNP testing in Auckland seems to be a significant issue for the provider. It is counterintuitive to ask GPs to request tests only for patients for whom it will provide critical information. The recent HTA report (288) recommendations regarding use of BNP in the diagnostic pathway are unequivocal, based on an individual patient data meta-analysis. Regardless of the actual value of NT-proBNP, all results provide the GP with valuable advice about the next step to take in the diagnostic process.

In light of the message from LabPlus regarding the cost of BNP (see Chapter 5, section 3.2.1), it would be interesting to estimate the “value” of BNP (see 3.2.7) where a negative result may rule out HF, avoid the need for echocardiography and save costs.

6.9.3 Research recommendations
The research recommendations cover a wide range of topics.

6.9.3.1 Secondary – primary care communication

9. Ascertain the extent to which the lack of information recorded in discharge letters can be attributed to the doctor responsible for composing them.

The lack of detail and critical information for ongoing management in discharge summaries has been attributed to the junior position of the hospital doctor who wrote them. (234, 235) However, the contribution of the inexperience of junior doctors requires formal study rather than speculation, because letters written by cardiologists to
GPs participating in this thesis were also lacking in detail (thesis section 5.5.4). Comparing the use of templates for discharge summaries between senior and junior doctors may indicate how well junior doctors understand the importance of this communication with primary care.

6.9.3.2 Maori and other at risk populations

10. Assess reasons for poorer outcomes experienced by Maori, beyond SES, co-morbidities, lifestyle factors.

11. Further investigate Maori case management clinic provision of care and outcomes, or ensure that primary care practices have in place Delivery system design with culturally appropriate and comprehensible care (425-427) if a ‘by Maori for Maori’ provider (468) is not available. Strengthen Community linkages of the CCM.

12. Investigate impact of drug metabolising pathways on health outcomes for Maori.

Cultural and social responsibility is set down in the Ethics Committee’s form completion guidelines for study requirements. Ethics forms incorporate a focus on reducing health inequalities, the relevance of the proposed research to Maori and the effect on Maori health outcomes. Analysis of the effect of implementation strategies on Maori was not possible in this thesis owing to the small number of Maori patients (see section 5.2.3 and Appendix 5B). However, it is known already that Maori have worse HF outcomes at all levels of deprivation. (469) Overall hospitalisation rates for Maori with HF were more than twice those for non-Maori. (470) Maori with a primary diagnosis of HF experience higher HF mortality and hospital admissions. (471) Mortality from HF is nine times higher for Maori men and Maori women aged 45 to 64 years, and three and a half times more likely for older Maori compared with non-Maori. (471) Maori:non-Maori rate ratios for hospital admissions with a primary diagnosis of HF were nine and ten times higher for men and for women aged 45 to 64 and four times higher for older men and women. (471) Potential confounders have not been factored in, and ethnicity and diagnostic coding errors could be expected to underestimate effect size rather than overestimate. (471)

There may also be a case for assessing drug PK/PD performance in Maori (472) and other ethnicities. Pharmacogenomic studies suggest ethnic differences in some CYP450
allele frequencies. (473) These alleles are common metabolisers of ARBs and β-blockers. (473) This may change the likelihood of adverse events occurring or the success of treatments. (473) There may need to be an initial determination of ethnic vs. genetic Maori to identify if genetic characteristics exist that affect drug performance.

6.9.3.3 Prognosis and survival time

13. Review more recent HF populations to determine survival time and QoL after diagnosis.

A recent population cohort study, of patients discharged after a first episode of HF, has indicated an improvement from 1986 to 2003 in 30-day case fatality rates, and 1-year survival and from 1986 to 1999 for 5-year survival. (474) A number of studies suggest that survival after a diagnosis is short, as described in a 2005 Cochrane review of clinical service organisation in HF. (475) On closer scrutiny the studies used to support this assertion of short survival time and low quality of life (476-480), used patient data sets from 1984, 1991, 1994 to 1997, 1995 to 1996, and 1990 to 1999. HF-appropriate management, i.e. which acts on the neurohormonal system rather than providing symptomatic relief, has improved markedly in the last two decades. Therefore it is possible that available data on HF prognosis are no longer accurate. Indeed, during the data collection, which included only patients who were alive three years or more after the educational intervention, there seemed to be few patients who had died.
7. Conclusion

The main objective of this study was to test the effectiveness of an Internet delivered educational intervention against small-group session and self-directed learning in NZ primary care. In this cluster RCT, HF patients in the randomised practices were followed for almost five years with a before and after comparison of echocardiography use and HF medications. GPs were surveyed about their knowledge of HF management and their experiences of the implementation method.

Four recommendations from the NZ National Heart Foundation HF guideline were selected for implementation to improve primary care HF management. The evidence for the medication recommendations is strong and would reduce deaths and hospital admissions. None of the study arms proved superior in improving the application of these recommendations. There was still a reluctance to refer patients with signs and symptoms of HF for confirmation by echocardiography. The decrease in ACEi prescription over time is an interesting finding but one for which no solid explanation was able to be produced. There was no concurrent increase in the prescription of ARBs. The prescription of β-blockers was hospital driven and mainly occurred before the GPs participated in their educational intervention. Overall prescription of spironolactone showed little change. The doses of these drugs remained static over the duration of the study.

The reasons for trying to implement only four recommendations were the established gaps in GP knowledge and practice and the desire to avoid overwhelming the GPs with too many messages. Results from the surveys found that even after the intervention, understanding of the importance of these recommendations was still incomplete.

These findings are discouraging as GPs tend to hold positive attitudes towards EBM and guidelines. They report believing that the use of EBM and guidelines improves the quality of patient care, and are an accessible guide to current best practice. However these attitudes may be moderated by age, practice setting, clinical demand, inertia or habit and personal characteristics, and may be filtered through past experiences, personal characteristics and unconscious knowledge. Subsequent management should also be individualised to each patient presenting for care, and take the values and preferences of these patients into account.
The patient variables that proved statistically significant in many of the analyses were age and sex. If there is a conscious or unconscious bias towards optimally managing younger male patients, then there will need to be extensive education of providers in order to change this attitude and achieve equity in the delivery of care. The effect of patient age on how patients are managed is well recognised, however the effect of being female has been observed in cross-sectional studies rather than in longitudinal studies. If women are less likely to present with ‘typical’ HF signs and symptoms then the meaning and diagnostic significance of those symptoms will need to be established.

Additional to this, a standardised diagnostic table that could be used in electronic form may assist GPs in determining who might have suspected HF. This table would also need to recommend when to refer patients for echocardiography. Informative and evidence-based communications from specialists e.g. advice on up-titrations would improve the management of HF patients. Responsibility for updating problem lists as new information is received by the practice would also need to be assigned. An intensive audit of medical records (such as conducted for this study) may need to be carried out to identify all patients whose condition has been overlooked. There is no simple way of finding heart failure patients for audit or research purposes when high quality coding is absent. There was little or no high quality coding and little that distinguished normal ejection fraction from low ejection fraction. The implementation of the National Services Framework / General Medical Services in the UK, based on the Quality and Outcomes Framework, has been highly successful in the management of HF (objective diagnosis and appropriate medication) and consideration should perhaps be given to implementing such a strategy in NZ. The financial viability of such a scheme would require discussions at State level but the concept of minimum quality standards has merit but will not be realised without adequate funding.

GPs need to spend time to understand the mechanism of action of drugs and the indications for their use, and combine this knowledge with their clinical judgement. The cited ‘lack of time’ to up-titrates and monitor medications such as ACEi and β-blockers is a poor excuse for compromising a patient’s quality and quantity of life. The benefits gained from prescribing these medications, will in turn benefit the GPs themselves and reduce the patient’s demands on other parts of the health system.

The results of this study suggest that Internet-based education does not perform any better or worse than education delivered to a small-group or through self-directed
learning. In the time since this study was conducted, the use of the Internet has burgeoned, making it more accessible and acceptable as an education tool. How this might have changed the findings of this study is uncertain.

Current psychological theories of behaviour change, such as targeting the type of implementation to the characteristics of the GP, continue to be debated and require thorough knowledge of each individual GP. Empirically testing these theories remains challenging. The best option available appears to be to offer educational interventions in different formats and to measure how far the education has penetrated into practice. This would be done in conjunction with the GP to determine how best to address any gaps.

GPs also need to learn how to help themselves. Audit can be a complex and time-consuming activity which must be carried out systematically. However, learning the stages of the audit cycle and using them for self- or practice-wide assessment will estimate the quality of care more accurately than will recall.

HF is a difficult condition to diagnose and to treat. There are no markers or obvious improvement (once the patient is euvoletic) that can be observed in some indicators, so the management is purely academic. Greater certainty over diagnosis and more constant reminders about medication use could assist in optimising HF management. The most effective implementation strategy to achieve this is as yet undetermined.


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Appendices
Appendices for Chapter 3. Heart Failure
3A. Heart failure defined by other guidelines

The 1994 AHCPR guideline provides a description of HF as follows.

“Heart failure is a clinical syndrome or condition characterized by (1) signs and symptoms of intravascular and interstitial volume overload, including shortness of breath, rales, and edema, or (2) manifestations of inadequate tissue perfusion, such as fatigue or poor exercise tolerance. These signs and symptoms result when the heart is unable to generate a cardiac output sufficient to meet the body’s demands. This guideline uses the term heart failure in preference to the commonly used “congestive heart failure” because many patients with heart failure do not manifest pulmonary or systemic congestion.” (70)

This definition seems to be the basis for the following guidelines e.g. the ACTION-HF:

“... a complex clinical syndrome that can result from any cardiac disorder that impairs the ability of the ventricle to eject blood. The cardinal manifestations of heart failure are dyspnea and fatigue (which may limit exercise tolerance) and fluid retention (which may lead to pulmonary and peripheral edema).” (86)

The 1999 SIGN guideline splits its HF description into detailed pathophysiology and clinical features.

“PATHOPHYSIOLOGY. Heart failure may be defined as an inability of the heart to deliver blood and, therefore, oxygen at a rate commensurate with the requirements of the metabolising tissues despite normal or increased cardiac filling pressures. This abnormality of cardiac function may be acute or chronic and may arise as a consequence of a myocardial, valvular pericardial, endocardial or electrical problem (or some combination of these).

CLINICAL DEFINITION AND FEATURES: Clinically, heart failure is a syndrome that consists of breathlessness, fatigue and fluid retention (peripheral oedema and an elevated jugular venous pressure) resulting from cardiac dysfunction.” (71)

The 2003 NICE guideline for the management of chronic heart failure in adults in primary and secondary care includes a brief definition of HF;

“Heart failure is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to function as a pump to support a physiological circulation. The syndrome of heart failure is characterised by symptoms such as breathlessness and fatigue, and signs such as fluid retention.” (72)

The European Society of Cardiology (ESC) definition has been referred to several times in this thesis but is included here for completeness. There is a short definition based on three criteria and a longer, text description of HF which expresses the difficulties in defining what HF is. The text definition from the 2001 version of the guidelines (76)
and the 1995 version (249) are very similar and runs to several paragraphs. The short definition is given below, followed by the longer definition:

“Definition of heart failure. Criteria 1 and 2 should be fulfilled in all cases.

1. Symptoms of heart failure (at rest or during exercise), and

2. Objective evidence of cardiac dysfunction (at rest), and (in cases where the diagnosis is in doubt)

3. Response to treatment directed towards heart failure.” (76, 249)

The longer definition reads:

“Many definitions of chronic heart failure exist but highlight only selective features of this complex syndrome. None is entirely satisfactory and one commonly used definition is: heart failure is a pathophysiological state in which an abnormality of cardiac function is responsible for the failure of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues.

A simple objective definition of chronic heart failure is currently impossible as there is no cut-off valve or cardiac or ventricular dysfunction or change in flow, pressure, dimension or volume that can be used reliably to identify patients with heart failure. The diagnosis of heart failure relies on clinical judgement based on a history, physical examination and appropriate investigations.

The Task Force considers the essential components of heart failure to be a syndrome where the patients should have the following features; symptoms of heart failure, typically breathlessness or fatigue, either at rest or during exertion, or ankle swelling and objective evidence of cardiac dysfunction at rest. A clinical response to treatment directed at heart failure alone is not sufficient for diagnosis, although the patient should generally demonstrate some improvement in symptoms and/or signs in response to those treatments where a relatively fast symptomatic improvement could be anticipated e.g. diuretic or nitrate administration. It should also be recognized that treatment may obscure a diagnosis of heart failure by relieving the patient’s symptoms. Therapy should not usually be initiated until a diagnosis of chronic heart failure has been established with reasonable certainty.” pp1528-9, (76)

The 2006 Australian HF guideline, gives a definition of CHF similar to AHCPR and ACTION-HF, but refers to ‘congestive’ and also the response to treatment indicator;

“A complex clinical syndrome with typical symptoms (e.g. dyspnoea, fatigue) that can occur at rest or on effort that is characterised by objective evidence of an underlying structural abnormality OR cardiac dysfunction that impairs the ability of the ventricle to fill with or eject blood (particularly during exercise). A diagnosis of CHF may be further strengthened by a beneficial clinical response to treatment(s) directed towards amelioration of symptoms associated with this condition.” (247)
The ESC definition encapsulates the problems associated with defining HF and brings together the three critical aspects that are needed to make a diagnosis. Even as early as 1995 the ESC were outlining the difficulties with defining HF.
3B.  Heart failure defined by major trials

*Cooperative North Scandinavian Enalapril Survival Study*

*CONSENSUS*

The clinical criteria for the diagnosis of CHF were a history of heart disease with symptoms of dyspnoea or fatigue or both, together with signs of fluid retention and no evidence of primary pulmonary disease. Patients were NYHA Class IV (symptomatic at rest). Heart size was measured by CXR. For men the heart size had to be >600ml/m$^2$ of body surface area (normal <550). For women heart size had to be >550ml/m$^2$ (normal <500). The Methods section states that ‘measurements of myocardial function were not required’. (13)

*Studies of Left Ventricular Dysfunction*

*SOLVD*

Participants in the SOLVD trials were required to have CHF and LV dysfunction which was defined as resting EF ≤35%. Patients with a history of overt HF participated in the treatment trial. Those without overt failure were entered in the prevention trial. (14, 15)

*Randomised Aldactone Evaluation Study*

*RALES*

Participants were eligible if they had NYHA Class IV six months before enrolment and were in NYHA Class III or IV at the time of enrolment, had been given a diagnosis of HF at least six weeks before enrolment, were being treated with an ACEI and loop diuretic, and had a LVEF ≤35% six months before enrolment. (25)

*The Metoprolol CR/XL Randomised Intervention Trial in Congestive Heart Failure*

*MERIT-HF*

Patients needed to have asymptomatic HF for three months or more that were NYHA Class II to IV, and a LVEF ≤40%. (21, 313)
3C. **Heart failure defined by other trials**

In one study of prevalence of HF (261) a patient had HF if one of the following were present:

- Pulmonary oedema confirmed radiologically;
- Peripheral oedema and a raised jugular venous pressure (JVP) on clinical examination;
- Evidence of heart disease (clinical, ECG, or echocardiographic) where symptoms of dyspnoea improved on taking diuretic drugs and relapsed on discontinuing treatment. (261)

Another study of prevalence (263) based their definition of HF on the one quoted above, and was almost the same word for word.

A study of incidence of newly diagnosed HF in general practice in the UK (266) used the definitions reported in (261) and (263), and in their study a patient was considered as a case of HF when the GP reported, at presentation, dyspnoea together with at least one of the following criteria:

- pulmonary oedema, confirmed clinically or radiographically;
- peripheral oedema and raised JVP on clinical examination; or
- evidence of heart disease (by clinical exam, ECG or echocardiogram).

Some diagnoses of HF made by the GP were accepted without any information of symptoms. (266)

Another study looking at GP diagnosis and incidence of HF (267) defined cases as:

- **Definite HF:** Where written diagnosis and clinical description in the database unequivocally indicated an intention to make a diagnosis of HF. Examples are phrases such as congestive cardiac failure, ventricular failure, and pulmonary oedema.
- **Possible HF:** Mainly had descriptions of signs and symptoms suggestive of HF or had related cardiac abnormalities such as heart enlargement or ventricular hypertrophy. This includes the possibility of non-cardiac pathology or heart disease in absence of the features of HF.
- **“Other”**: First prescription of diuretics without concomitant and / or previous diagnosis of HF (neither definite nor possible).
The Rotterdam Study assessed patients for shortness of breath on exertion or at rest, ankle oedema, pulmonary crepitations and patients needed at least two of these symptoms / signs plus evidence of cardiovascular disease, with shortness of breath not attributable to COPD to be considered as having HF. These features were used to determine prevalence of HF as well as medications (diuretics, glycosides or ACEi) which indicated possible HF. Medication use was also considered as HF patients who are stabilised on medication may exhibit less obvious signs and symptoms. Out of 5540 participants assessed for HF, 2823 participants had LVSF measured by echocardiography. (264)

The Hillingdon HF study based diagnosis on clinical criteria (e.g. shortness of breath, effort intolerance, fluid retention), radiographic and echocardiographic evidence, (268) used criteria of the ESC (or similar). The criteria included symptoms (shortness of breath, fatigue, fluid retention or any combination of these symptoms) in the presence of an underlying abnormality of cardiac structure and function and if doubt remained, a beneficial response to therapy for HF was required to confirm the diagnosis (“a brisk diuresis accompanied by substantial improvement in breathlessness”). (268)

The ECHoES study (312) used clinical criteria and echocardiographic evidence (EF <40%), but split patients into ‘definite’ and ‘probable’ HF. Definite HF was defined, in accordance with the ESC criteria as patients who were dyspnoeic (NYHA II or more) and with objective evidence of cardiac dysfunction (LVEF <40%, or AF or moderate to severe valve disease, or any combination). Probable HF was defined as patients with symptoms and borderline LVEF, or patients with definitely impaired EF who previously had symptoms but now were symptom free as a result of appropriate therapy (ACEi or diuretic). (312)

The MONICA (Glasgow) study used clinical criteria and echocardiographic evidence (EF ≤30%).
Appendices for Chapter 4. Methods.
4A. Initial letter of invitation to general practitioners

There were two letters sent out. One formatted for GPs who were members of the Comprehensive Health IPA and one formatted for members of IPCS IPA.
Dear Dr

Comprehensive Health is collaborating with the Department of General Practice and Primary Health Care in a study evaluating different forms of continuing medical education (CME) on the management of heart failure.

We would like to take this opportunity to invite you and the other doctors in your practice to participate. The educational intervention itself will take about two hours. You will be asked to complete a brief survey (enclosed with a freepost return envelope) and to fill out a short questionnaire of your experiences of the education you received.

In approximately 18 months a researcher will visit your practice to assess which patients could be considered to be definite or possible heart failure patients. These patients will be sent information about the study and a signed consent form for them to fill out and return. This will be done by the research team and will require minimum input on your part. Once the consent forms have been returned, the researcher will gather treatment data from patients’ medical records.

The benefits of participating are that you will receive an advance copy of the new Heart Failure Guidelines. This project has been endorsed by the RNZCGP QA Unit as a Practice Review Activity for three years and this means you will gain 30 MoPs points.

In a few days a member of the study team will call you to answer any questions you may have and to see if you and the other doctors at your practice would like to participate.

This study has received ethical approval from the Auckland Ethics Committee.

Yours sincerely

Bruce Arroll  
Associate Professor, General Practice

Ross McCormick  
Professor, Director of GFU

Victoria Andersen  
PhD student

Clinical Projects Manager, Comprehensive Health
Dear Dr

IPCS is collaborating with the Department of General Practice and Primary Health Care in a study evaluating different forms of continuing medical education (CME) on the management of heart failure.

We would like to take this opportunity to invite you and the other doctors in your practice to participate. The educational intervention itself will take about two hours. You will be asked to complete a brief survey (enclosed with a freepost return envelope) and to fill out a short questionnaire of your experiences of the education you received.

In approximately 18 months a researcher will visit your practice to assess which patients could be considered to be definite or possible heart failure patients. These patients will be sent information about the study and a signed consent form for them to fill out and return. This will be done by the research team and will require minimal input on your part. Once the consent forms have been returned, the researcher will gather treatment data from patients’ medical records.

One of the benefits of participating is that since the RNZCGP QA Unit has endorsed this project for three years as a Practice Review Activity, you will gain 30 MoPS points. You will also receive a copy of the Heart Failure Guidelines and Algorithms.

In a few days a member of the study team will call you to answer any questions you may have and to see if you and the other doctors at your practice would like to participate.

This study has received ethical approval from the Auckland Ethics Committee.
    Committee Y, 5 December 2001, Project # 2001/280

Yours sincerely

Bruce Arroll  Ross McCormick  Victoria Andersen
Associate Professor, General Practice  Professor, Director of GFU  PhD student

Alan Greenslade
General Manager, IPCS
4B. **Survey of HF management**

The survey of HF management was sent out to GPs with the first letter sent out regarding the study.
Survey: Heart Failure Management.

1. What changes in heart failure management have you heard of in the past 3 years? (e.g. Investigations, Medications, Other management)

If you HAVE answered QUESTION 1 go to QUESTION 3.

If you have NOT answered QUESTION 1 go to QUESTION 2 & 2a.

2. In the past 3 years, have you made any changes in the way you manage your heart failure patients? (Please circle) YES NO

If YES, what sort of changes?

2a. Has the hospital made any changes in the management of your heart failure patients? (Please circle) YES NO

If YES, what sort of changes?

IF you have answered Question 2 & 2a, finish the questionnaire here and return it.
3. Where did you hear about these changes?
(Note: Consider the points described in Question 1.) (Consider the following sources e.g. colleagues, IPA meetings/cell groups, journal articles, e-journals, Internet sites, drug companies.)

4. Which of these changes have you implemented?
(Note: Consider the points described in Questions 1 & 3.)

5. What proportion of your heart failure patients have you changed the management of?

6. Have you had any problems or concerns with these patients during these changes in management? YES NO
If YES, what sort of problems have you encountered?
7. Has the hospital made any changes in the management of your heart failure patients? (Please circle) YES NO

If YES, what sort of changes?

Additional Information. (Please number question.)

Thank you.

Please return the survey in the free post envelope provided, or if it is missing to:

FREEPOST 122259, Victoria Andersen, Department of General Practice, Private Bag 92019, AUCKLAND.
4C. Sample size calculations

The calculations are based on data on HF management in central Auckland and data on practices in North and West Auckland. The sample population consists of 81 practices that range in size from 1 to 7 GPs. A small survey suggests there may be 7.5 HF patients per doctor. From earlier data, the intra-cluster correlation between patients from the same GP is of the order of 0.01. If the practice is used as the sampling unit, the average cluster size is about 17. The design effect is therefore 1.2. The expected total number of patients is 1320 (440 per study arm).

The study arms will be randomised by practice. This will reduce contamination, effects of patient “sharing”, and the way patients may be filed “by practice”. The numbers of solo vs. group practices are fairly evenly scattered by randomisation without stratifying. Information on individual doctors will still be collected.

Data on the central Auckland audit (30), showed that 60% of patients had undergone echocardiography. Of the patients on ACE inhibitor, 20% were on maximal dose, 9% of patients were on a β-blocker and a similar percentage were taking a K⁺-sparking diuretic. Even though these figures are from a few years ago, it is doubtful that there has been a large increase in the prescription of either of these to HF patients. These figures may also over-represent the true picture as only the class of drug was recorded, not specific drug names.

The following three tables indicate sample size calculations. There are three estimates for each calculation. β-blocker and spironolactone values were similar at baseline (central Auckland audit) so these are presented as one table. The column headings for the tables are described below:

- “Effect” refers to the change in behaviour of the intervention/s.
- “Baseline” values are from the central Auckland audit (percentage of patients in each ‘outcome’).
- “Final” value is our estimate of the proportion of patients in that outcome measure at the end of the study.
- “Phi (\( \phi \))” is the correlation of measurements within the same person.
- “Sample” refers to an assumed simple random sample.
- “Adjusted” is the sample multiplied by the cluster effect.
- “Total” is “Adjusted” multiplied by 3 (i.e. the number of study arms).
ECHOCARDIOGRAPHY

<table>
<thead>
<tr>
<th>Effect</th>
<th>Baseline</th>
<th>Final</th>
<th>Power</th>
<th>α</th>
<th>φ</th>
<th>Sample</th>
<th>Adjusted</th>
<th>Total</th>
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<td>60%</td>
<td>66%</td>
<td>0.8</td>
<td>0.05</td>
<td>0.7</td>
<td>310</td>
<td>372</td>
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<tr>
<td>Medium</td>
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<td>75%</td>
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<td>0.05</td>
<td>0.6</td>
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</table>

MAXIMAL ACE-INHIBITOR DOSE

<table>
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<tr>
<th>Effect</th>
<th>Baseline</th>
<th>Final</th>
<th>Power</th>
<th>α</th>
<th>φ</th>
<th>Sample</th>
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<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>20%</td>
<td>25%</td>
<td>0.8</td>
<td>0.05</td>
<td>0.7</td>
<td>340</td>
<td>408</td>
<td>1230</td>
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<tr>
<td>Medium</td>
<td>20%</td>
<td>50%</td>
<td>0.8</td>
<td>0.05</td>
<td>0.3</td>
<td>26</td>
<td>32</td>
<td>96</td>
</tr>
<tr>
<td>High</td>
<td>20%</td>
<td>80%</td>
<td>0.8</td>
<td>0.05</td>
<td>0.3</td>
<td>16</td>
<td>20</td>
<td>60</td>
</tr>
</tbody>
</table>

β-BLOCKER / SPIRONOLACTONE

<table>
<thead>
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<th>Baseline</th>
<th>Final</th>
<th>Power</th>
<th>α</th>
<th>φ</th>
<th>Sample</th>
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<th>Total</th>
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</thead>
<tbody>
<tr>
<td>Low</td>
<td>9%</td>
<td>13.5%</td>
<td>0.8</td>
<td>0.05</td>
<td>0.7</td>
<td>250</td>
<td>300</td>
<td>900</td>
</tr>
<tr>
<td>Medium</td>
<td>9%</td>
<td>35%</td>
<td>0.8</td>
<td>0.05</td>
<td>0.4</td>
<td>35</td>
<td>42</td>
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<tr>
<td>High</td>
<td>9%</td>
<td>50%</td>
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<td>0.05</td>
<td>0.3</td>
<td>20</td>
<td>24</td>
<td>72</td>
</tr>
</tbody>
</table>

Small group education is most likely to achieve a High level of Effect (change in behaviour). To demonstrate this against control we would use figures such as those estimated in the High Effect row. Internet-based intervention will probably cause a change similar to the Medium Effect row. To demonstrate this against control we could use the Medium Effect figures. To compare the effectiveness of small group education against the Internet-based education, sample sizes nearer the Low Effect estimates would need to be used. The sample sizes for the Low Effect are feasible with the estimated total number of heart failure patients in the Waitemata area.
4D. Details of randomisation and participation

The following tables show data within each study arm on the number of practices (and GPs) that were randomised for each arm of the study by practice size\(^a\) and by the IPA / PHO affiliation, compared with the number of practices and GPs who eventually participated (i.e. in data collection from patient medical records).

**Guideline study arm**

Table 4D.i. CHS/Comprehensive GPs and practices randomised and participating

<table>
<thead>
<tr>
<th>Practice size</th>
<th>Randomised</th>
<th>Participated</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
<td>GPs</td>
</tr>
<tr>
<td>Small</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Medium</td>
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<td>12</td>
</tr>
<tr>
<td>Large</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>TOTALS</td>
<td>13</td>
<td>29</td>
</tr>
</tbody>
</table>

Table 4D.ii. IPCS/HealthWest GPs and practices randomised and participating

<table>
<thead>
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<th>Practice size</th>
<th>Randomised</th>
<th>Participated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
<td>GPs</td>
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<tr>
<td>Small</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Medium</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Large</td>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td>TOTALS</td>
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<td>30</td>
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</table>

**Small group study arm**

Table 4D.iii. CHS/Comprehensive GPs and practices randomised and participating

<table>
<thead>
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<th>Practice size</th>
<th>Randomised</th>
<th>Participated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
<td>GPs</td>
</tr>
<tr>
<td>Small</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Medium</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>Large</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>TOTALS</td>
<td>12</td>
<td>32</td>
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</tbody>
</table>

\(^a\) Small = 1 GP, Medium = 2 to 3 GPs, Large = 4 or more GPs
Table 4D.iv. IPCS/HealthWest GPs and practices randomised and participating

<table>
<thead>
<tr>
<th>Practice size</th>
<th>Randomised</th>
<th>Participated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Practices</td>
<td>GPs</td>
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<tr>
<td>Small</td>
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<td>4</td>
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<tr>
<td>Medium</td>
<td>4</td>
<td>10</td>
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<tr>
<td>Large</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>TOTALS</td>
<td>11</td>
<td>27</td>
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</tbody>
</table>

**Internet**

Table 4D.v. CHS/Comprehensive GPs and practices randomised and participating

<table>
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<tr>
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<td>Large</td>
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<tr>
<td>TOTALS</td>
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Table 4D.vi. IPCS/Health West GPs and practices randomised and participating

<table>
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<td>9</td>
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<tr>
<td>Large</td>
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<td>20</td>
</tr>
<tr>
<td>TOTALS</td>
<td>12</td>
<td>33</td>
</tr>
</tbody>
</table>
4E. Educational intervention
There are four objectives for this CME on management of heart failure:

1.) Maximal dose of ACE inhibitor
2.) Place of echocardiogram
3.) Use of metoprolol succinate or carvedilol
4.) Use of spironolactone

In more detail, this includes:

1.) Use of highest tolerated dose of ACE inhibitor dosages for heart failure.
2a.) Ordering of Echocardiograms to confirm a clinical diagnosis of congestive heart failure and to clarify if it is systolic dysfunction rather than diastolic dysfunction. (There is limited evidence on the benefits of medication for diastolic dysfunction.) Echocardiograms are the only test that provides objective evidence of heart failure and are considered the “gold standard” diagnostic test.
2b.) Starting preventive medications (metoprolol succinate/carvedilol and spironolactone) before an Echocardiogram is obtained if getting the Echo is going to take a long time.
3.) Starting patients on low dose metoprolol succinate and slowly increasing it to 190 mg daily (where possible) for patients with New York Heart Association (NYHA) classification 2 and 3. Or starting patients on low dose carvedilol and slowly increasing to 25mg bid (where possible).
4.) Starting patients on 25 mg of spironolactone for patients with NYHA class 3 and 4.
Reading Material

Diagnostic and Treatment Algorithms
Heart Foundation Guidelines
Journal Abstracts
Key Management Issues & Pathophysiology Review
Diastolic Heart Failure Summary
Assessment of left ventricular function is an important part of the investigation. However, if this is delayed due to local resource constraints, then treatment should continue on an empirical basis.

**Diagnosis Algorithm**

**Suspected Heart Failure**
Dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, unexplained confusion or fatigue

**Clinical History**
- Age
- Onset of symptoms
- Previous heart disease
  - Myocardial infarction
  - Angina
  - Hypertension
  - Valvular heart disease, rheumatic fever
  - Palpitations
- Alcohol / tobacco use
- Medications

**Clinical History**
- Elevated JVP
- Third heart sound
- Pulse rate and rhythm
- Displaced apex beat
- Pulmonary rales
- Peripheral oedema
- Pulsus alternans
- Baseline weight

**Echocardiogram**

**Aetiology Determined?**

- No
- Yes

**Consider Specialist Referral**

**Proceed to Treatment Algorithm**

**Investigations**
- Full blood count
  - Anaemia
  - CHF due to decreased oxygen carrying capacity
- Serum creatinine
  - Renal failure
- Serum albumin
  - Oedema due to hypoalbuminaemia

**Thyroid function tests**
(indicated only with AF, age >65 yrs, evidence of thyroid disease)
- Abnormal T4, TSH
  - Hypo-/hyperthyroidism

**Urinalysis**
- Proteinuria
  - Nephrotic syndrome
- Red cells, casts
  - Glomerulonephritis

**Electrocardiogram**
- ST/T wave changes
  - Myocardial ischaemia
- Q waves
  - Previous MI
- AF, other tachyarrhythmia
  - Thyrotoxicosis, CHF due to rapid heart rate
- Bradycardia
  - Hypothyroid, CHF due to slow heart rate
- Left ventricular hypertrophy
  - Diastolic dysfunction

**Chest X-ray**
- Pulmonary congestion
  - Heart failure
- Pulmonary disease
  - CORD etc

**Echocardiogram**
- Confirm systolic dysfunction

*Assessment of left ventricular function is an important part of the investigation. However, if this is delayed due to local resource constraints, then treatment should continue on an empirical basis.*
**NEW ZEALAND GUIDELINE FOR THE MANAGEMENT OF CHRONIC HEART FAILURE**

### Non-Pharmacological Management
- General counselling (compliance, prognosis)
- Record weight daily (for diuretic titration)
- Avoid smoking
- Regular exercise
- Low-salt diet
- Limited alcohol

### Diuretics
- Titrate according to symptoms and dry weight
- Mild CHF – thiazide alone may suffice (e.g., bendrofluazide 2.5-5mg daily)
- Moderate-severe CHF – loop diuretic (e.g., initially frusemide 40mg daily)
- Monitor K⁺/creatinine weekly during titration, then 3 monthly
- K⁺ supplementation usually not required with concomitant ACE inhibitor
- Serious hyperkalaemia can arise with combination of high-dose K⁺-sparking diuretic and ACE inhibitors (see also spironolactone)
- In cases of resistant oedema, double the daily dose of diuretic, rather than give the same dose twice daily

### ACE inhibitors
- Start at low dose (e.g., captopril 6.25mg tds, enalapril 2.5mg daily)
- Titrate to target dose over 2-3 weeks (e.g., captopril 25-50mg tds, cilazapril 5mg daily, enalapril 10mg bid, quinapril 10-20mg bid)
- Risk of first-dose hypotension if SBP <90mmHg, or over-diuresis
- Consider lower dosages if elderly or renal impairment
- Monitor K⁺/creatinine/BP weekly while titrating
- Contraindications: K⁺>5.5mmol/l, creatinine >0.25mmol/l, symptomatic hypotension or SBP<80mmHg, angioedema

### Beta-blockers
- Consider for patients with chronic stable CHF and:
  - mild-moderate symptoms
  - minimal signs of congestion
  - stable for one month on adequate doses of ACE inhibitors and diuretics
- Contraindications: asthma, 2nd/3rd degree heart block, symptomatic hypotension, SBP <80mmHg, HR <50bpm
- Initiation and titration may require referral (see NHF HF Doctors Guide)

### Spironolactone
- Consider for patients with New York Heart Association class III/IV (moderate-severe) CHF symptoms
- Recommended dose = 25mg daily
- Hyperkalaemia/renal failure may arise if higher doses are used with ACE inhibitor
- Contraindications: K⁺>5mmol/l, creatinine >0.25mmol/l
- Monitor K⁺/creatinine 3-4 days after starting
- 10% of males may suffer breast pain or gynaecomastia

### Digoxin
- Consider for patients in AF or in sinus rhythm if CHF is severe and not controlled with ACE inhibitor and diuretic
- If normal renal function – start with 0.25mg daily and check levels in 1 week
- If elderly or renal impairment – start at 0.125 or 0.0625mg daily, check levels in 2-3 weeks
- Toxicity: confusion, anorexia, nausea, visual disturbance, arrhythmias
- Drugs which increase levels: antibiotics, amiodarone, diltiazem, verapamil, quinidine

---

Diuretics
- Fluid Overload?
  - Yes: Diuretic ACE inhibitor
  - No: ACE inhibitor

ACE inhibitors
- Titrate ACE inhibitor dose
  - Clinically stable with minimal congestion?
    - Yes: Consider: Beta-blocker
    - No: Consider: Spironolactone and/or digoxin
A GUIDELINE FOR THE MANAGEMENT OF HEART FAILURE

Doctor’s Guide

Heart Foundation

Ta Hotu Manawa Whanui

December 2001
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The Guideline Process

Scope
This guideline makes recommendations relating to the management of patients with an established diagnosis of congestive heart failure due to systolic ventricular dysfunction. Management of diastolic dysfunction is not included. There is commentary on diagnosis in order to define the population of patients to which this guideline refers.

Objectives
The aim of this guideline is to reduce morbidity and mortality from congestive heart failure. It is also hoped that patients’ understanding and satisfaction with their health care will be improved. Outcomes predicted are increased survival and reduced morbidity as represented by either functional scores or by hospital admission.

Process
The first version of this guideline was published by the NHF in 1999. The guideline team developed the guideline by adhering to the systematic approach developed by the New Zealand Guidelines Group and the Guidelines for Guidelines Trust.¹

1. A systematic search of the external literature was undertaken to identify explicitly developed evidence based guidelines on the management of heart failure. The following guidelines were reviewed:
   - The New Zealand Guidelines for the Management of Chronic Heart Failure²
   - The American College of Cardiology/American Heart Association (1996)
   - The Canadian Cardiovascular Society (1994)
   - The Task Force on Heart Failure by the European Society of Cardiologists (1996)
   - The Anglia and Oxford Regional Health Authority (1995), and
   - The Agency for Health Care Policy and Research (AHCPR)³
   Of these the AHCPR guideline was determined to be systematically developed from a review of external evidence, and for which the strength of evidence could be ascertained for each recommendation. The NHF guideline² served as the key domestic resource.

2. Areas requiring further review and evaluation of the external evidence were identified on the basis of the strength of the evidence backing current recommendations (recommendations with less strong evidence were selected) or areas where there was identifiable new evidence. The following topics were selected for further review of the external evidence:
   - The management of atrial fibrillation
   - The role of anticoagulation in heart failure
   - The role of amiodarone in heart failure
   - The role of beta blockers in heart failure
   - The role of digoxin in heart failure
   - The effectiveness and role of patient education
   - The effectiveness of interventions to improve patient compliance
   - The effectiveness and role of exercise in heart failure.

3. Each review included a systematic Med-line search of the literature by medical librarians.
4. Each paper was reviewed and critiqued by a member of the guideline team and strength of evidence assigned according to the following quality-rating scale. Final decisions regarding each paper and the recommendations of the guideline was established by consensus.

In April 2000 the Guideline Team selected topics for which there was consensus that significant new evidence was available. These were:

- The role of beta-blockers
- The optimal dose of ACEIs
- The role of angiotension II antagonists
- The role of spironolactone
- The effectiveness of patient held action plans

A systematic search of the literature to April 2000 was undertaken and evidence reviewed as the original guideline process. Two further large-scale RCTs of the effects of beta-blockers in patients with heart failure were published in 2001 and have been included because of the importance of these data.

The Strength of Evidence

The quality-rating scale, described by AHCPR was used. Seven levels were used:

- I Evidence from large, well conducted randomized controlled trials (RCTs)
- II Evidence from small, well conducted RCTs
- III Evidence from well-conducted cohort studies
- IV Evidence from well-conducted case-control studies
- V Evidence from uncontrolled or poorly controlled studies
- VI Conflicting evidence, but tending to favour the recommendation
- VII Expert opinion

The classification scheme for the guideline document was then simplified into a three-level system for strength of evidence:

- A Good evidence: Evidence from well conducted RCTs or cohort studies (Levels I-III).
- B Fair evidence: Evidence from other types of studies (Levels IV-VI).
- C Expert opinion: (Level VII).

5. The Draft Guideline was written by the Guideline Team and subjected to peer review by the Goodfellow Unit, Division of General Practice and Primary Health Care, University of Auckland, the Royal New Zealand College of General Practitioners, and members of the NHF Heart Failure Guideline Committee. The review confined itself to issues of format, presentation and utility, not issues of evidence.

Review

A Heart Foundation Committee will review these guidelines annually and decide whether updates are required.

Funding

A one off grant by Merck Sharp and Dohme sponsored the initial guideline development process. Expenditure of the grant was at the discretion of the guideline team, such expenditure being for the purposes of developing the guideline. Any funds remaining at the
end of the project were returned to the sponsor. No team member, except the MSD representative, received any remuneration from the sponsor. Printing of the revised guideline was supported by Merck Sharp & Dohme and Roche.

**Future Research**

The cost effectiveness of echocardiography in improving outcomes for patients with left ventricular failure and whether there are identifiable characteristics that will stratify patients that will most benefit from echocardiology.

**The Guideline Team**

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- The National Heart Foundation Heart Failure Guideline Committee: Professor Hamid Ikram (chair); Dr. Sandra Hicks; Professor Gary Nicholls; Professor Norman Sharpe; Dr. Larry Skiba; Professor Harvey White.
- National Heart Foundation staff: Dr Diana North, Dr Boyd Swinburn, Mrs Pip Mason, Dr Mary Seddon
Heart Failure Guidelines
These guidelines relate to the diagnosis and management of patients with congestive heart failure due to left ventricular systolic dysfunction.

DIAGNOSIS

Clinical Evaluation: Summary

- All patients, who complain of paroxysmal nocturnal dyspnoea, orthopnoea or new onset of shortness of breath on exertion, should undergo evaluation for heart failure unless history and physical examination clearly indicate a non-cardiac cause for their symptoms. (Strength of evidence = B)

- The physical examination can provide important information about the aetiology of patients' symptoms and about appropriate initial treatment. However, physical signs are not highly sensitive for detecting heart failure.

- Elevated jugular venous pressure, a third heart sound, and a laterally displaced apical impulse are the most specific and are virtually diagnostic in a patient with compatible symptoms. (Strength of evidence = B)

Symptoms of Heart failure (see Box 1)
When clinical heart failure develops, dyspnoea on exertion is often the earliest symptom followed by paroxysmal nocturnal dyspnoea oedema, cough and orthopnoea. Fatigue is an important symptom and may occur early in failure due to valvular disease. A history of hypertension, previous myocardial infarction, cardiac murmur or other heart disease in conjunction with the above symptoms points strongly toward a diagnosis of heart failure. It should be noted that many patients with impaired left ventricular function have no symptoms, eg 20% of patients with a left ventricular ejection fraction (LVEF) of less than 40% may have no clinical criteria for heart failure. Furthermore, the symptoms listed above are not always due to heart failure.

Box 1. Symptoms suggestive of heart failure:
<table>
<thead>
<tr>
<th>Paroxysmal nocturnal dyspnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthopnoea</td>
</tr>
<tr>
<td>Dyspnoea on exertion</td>
</tr>
<tr>
<td>Lower extremity oedema</td>
</tr>
<tr>
<td>Decreased exercise tolerance</td>
</tr>
<tr>
<td>Unexplained confusion or fatigue in elderly</td>
</tr>
<tr>
<td>Nausea or abdominal pain (ascites or hepatic engorgement)</td>
</tr>
</tbody>
</table>
Physical Examination

Box 2. Abnormal physical findings in heart failure include:

- Tachycardia, irregular pulse
- Elevated jugular venous pressure or positive hepato-jugular reflux
- A third heart sound
- Laterally displaced apical impulse
- Pulmonary rales that do not clear with coughing
- Peripheral oedema

In many patients with moderate-to-severe left ventricular systolic dysfunction or early symptoms of heart failure, there are few abnormal physical findings. A pathological third heart sound is the most sensitive physical sign, and is present in two-thirds of patients with ejection fractions below 30%. Rales and/or a displaced apical impulse are present in about a third of patients. Jugular venous distension and peripheral oedema appear to be less sensitive signs.

The specificity of physical findings are less well defined but an elevated jugular venous pressure and a third heart sound are probably the most specific clinical signs of heart failure. Lower extremity oedema is a relatively non-specific finding, common in older people, and usually due to chronic venous insufficiency.

Clinical Assessment of Functional Capacity

A well-established clinical schema for assessing functional capacity is the New York Heart Association (NYHA) Functional Classification. This is based on the degree of limitation of the patient's life-style (Box 3.) It is a useful shorthand method for recording functional status and is helpful for inter-patient comparisons and for monitoring response to therapy. However classifying heart failure on the basis of exercise intolerance examines only one facet of heart failure symptomatology. Many symptoms of heart failure (eg fatigue) are impossible to quantify with precision.

Box 3. New York Heart Association Functional Capacity

Class 1. Patients with cardiac disease but without resulting limitation in physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnoea or anginal pain.

Class 2. Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea or anginal pain.

Class 3. Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnoea or anginal pain.

Class 4. Patients with cardiac disease resulting in an inability to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency or of anginal pain are present at rest. If any physical activity is undertaken discomfort is increased.
AETIOLOGY OF HEART FAILURE

Heart failure should never be the final diagnosis. The aetiology of heart failure and the presence of exacerbating factors or other diseases that have important influence on management should be carefully considered. The extent to which the cause of heart failure should be pursued by further investigation will depend on the life expectancy of the patient, the resources available, and the likelihood that diagnosis will influence management.

Chronic heart failure may be due to several different underlying aetiological factors (Table 1). Myocardial dysfunction as a result of coronary artery disease (most commonly from myocardial infarcts) is the most common cause of heart failure under the age of 75-years, and clear abnormalities of systolic function are usually present. In the elderly, accurate diagnosis is more difficult and obscured by multiple other diagnoses. Hypertension, hypertrophy, and myocardial fibrosis may be more important causes of heart failure in the elderly and may occur in the presence of preserved systolic function. Often there is uncertainty over which factor dominates.

A. Causative Factors:
- Coronary Artery Disease
- Hypertension
- Valvular Heart Disease
- Infections
- Cardiomyopathies (including alcoholic and idiopathic)
- Endocrine disorders (especially thyrotoxicosis).
- Genetic Conditions
- Congenital Heart Disease
- Inflammatory/immunological
- Chronic arrhythmias eg complete heart block or incessant tachycardia

B. Precipitating or Exacerbating Factors:
It is important to identify and treat any reversible factors, which may be exacerbating the symptoms of heart failure.

These factors include:
- Anaemia
- Infection
- Arrhythmias, especially atrial fibrillation
- Drugs, eg non-steroidal anti-inflammatory drugs, calcium channel blockers, corticosteroids and liquorice
- Renal dysfunction / renal artery stenosis
- Pulmonary embolism
- Silent myocardial infarction
- Excess salt intake
Table 1. Recommended Tests for Patients With Suspected Heart Failure

<table>
<thead>
<tr>
<th>Test Recommendations</th>
<th>Finding</th>
<th>Suspected Diagnosis</th>
</tr>
</thead>
</table>
| Chest x-ray          | Cardiomegaly  
Pulmonary venous congestion  
Interstitial fluid  
Pulmonary disease | Heart failure  
Lung conditions |
| Electrocardiogram    | Acute ST-T wave changes  
Atrial fibrillation, other tachyarrhythmia  
Bradyarrhythmias  
Previous MI (eg Q waves)  
Left ventricular hypertrophy | Myocardial ischaemia  
Thyroid disease or heart failure due to rapid ventricular rate  
Heart failure due to slow heart rate  
Heart failure due to reduced left ventricular performance  
Diastolic dysfunction |
| Echocardiogram*      | LV systolic dysfunction  
LV hypertrophy  
LV diastolic dysfunction  
Valve disease | Severity of LV dysfunction and clues to potential aetiology of heart failure |
| Complete blood count | Anaemia | Heart failure due to or aggravated by decreased oxygen carrying capacity |
| Urinalysis           | Proteinuria  
Red blood cells or cellular casts | Nephrotic syndrome  
Glomerulonephritis |
| Serum creatinine     | Elevated | Volume overload due to renal failure |
| Serum albumin        | Decreased | Increased extravascular volume due to hypoalbuminemia |
| T4 and TSH (obtain only if atrial fibrillation, evidence of thyroid disease, or patient age >65) | Abnormal T4 or TSH | Heart failure due to or aggravated by hypo/hyperthyroidism |
| Brain natriuretic peptide# | Elevated BNP | Heart failure likely if BNP elevated |

* TSH = Thyroid-stimulating hormone, MI = myocardial infarction

**Note regarding echocardiography for assessment of suspected heart failure**

The Guideline Group recognise the important role of echocardiography for the assessment of patients with suspected heart failure. However, in many areas in New Zealand echocardiography is not widely available and there may be considerable delays in obtaining an echocardiogram. The Guideline Group have discussed this potential limitation in the assessment of suspected heart failure at length and the following points summarise the current position with regards to echocardiography:

- Imaging of the left ventricle is of paramount importance in the assessment of a patient with suspected heart failure (usually this will be with echocardiography)
- “Open-Access” echocardiography for primary care practitioners has been promoted in many areas. However, there is no randomised controlled evidence that the provision of this service alters outcome or is cost-effective.
Open access echocardiography is generally not available in New Zealand. Despite these limitations echocardiography, where available, is still recommended as part of the assessment of patients with suspected heart failure.

# Note regarding brain natriuretic peptide

Brain natriuretic peptide (BNP) is a protein released from the heart in response to changes in left ventricular wall stretch and is elevated in heart failure. BNP is a powerful neurohormonal predictor of left ventricular function and of prognosis in heart failure. BNP concentrations can help to discriminate between heart failure and other causes of breathlessness in patients admitted to hospital. In particular, a normal BNP result in a symptomatic patient makes the diagnosis of heart failure very unlikely. BNP testing is available in some areas in New Zealand and, with the availability of point-of-care meters, is likely to increase over the next few years. However, the usefulness of BNP in unselected patients in the community is uncertain, and further studies will address this further.

MANAGEMENT

A. Non-Pharmacological Management

Effective Patient Education

Educational interventions, including one to one patient counselling, improve patient compliance and outcomes. (Strength of Evidence = B)  

General Counselling (see table 2)

After a diagnosis of heart failure is established, patients and their families or caregivers should be counselled regarding the nature of heart failure, drug regimens, dietary restrictions, symptoms of worsening heart failure, what to do if these symptoms occur, and prognosis (see table 2). The impact of heart failure on a patient's life may be related as much to psychological adaptation to the disease as to impairment in physical functioning. Nursing interventions, family involvement, and support groups may all help patients cope with heart failure.

Smoking

Practitioners should emphasise the importance of not smoking or chewing tobacco.

Vaccination

Practitioners should recommend that patients receive vaccination against influenza and pneumococcal disease. (Strength of Evidence = C)

Discussion of Prognosis

Patient counselling with respect to prognosis should be guided by evidence from recent trials and the Framingham experience, which indicate that the average annual mortality rate for patients with heart failure is approximately 10% per year. Mortality increases with age, severity of disease, and the presence of angina. The one-year mortality for patients with severe heart failure (NYHA IV) is approximately 30-50%.

It is vital that patients receive accurate information concerning prognosis in order to make decisions and plans for the future. Practitioners should discuss patients’ desires regarding resuscitation. All patients should be encouraged to complete advance directives regarding their health care preferences.
### Table 2. Suggested Topics for Counselling and Education

1. **General Counselling**
   - Explanation of heart failure and reasons for symptoms
   - Cause of heart failure
   - Expected symptoms
   - Symptoms of worsening heart failure
   - What to do if symptoms worsen
   - Self-monitoring with daily weights
   - Explanation of treatment/care plan
   - Clarification of patient's responsibilities
   - Importance of cessation of tobacco use
   - Role of family members or other caregivers in the treatment/care plan
   - Importance of vaccinations against influenza and pneumococcal disease

2. **Prognosis**
   - Life expectancy
   - Advance directives regarding resuscitation
   - Advice for family members in the event of sudden death

3. **Activity Recommendations**
   - Recreation, leisure, and work activity
   - Exercise
   - Sex, sexual difficulties, and coping strategies

4. **Dietary Recommendations**
   - Sodium restriction
   - Avoidance of excessive fluid intake
   - Fluid restriction (if required)
   - Alcohol restriction

5. **Medications**
   - Effects of medications on quality of life and survival
   - Dosing
   - Likely side effects and what to do if they occur
   - Coping mechanisms for complicated medical regimens
   - Availability of lower cost medications or financial assistance

6. **Importance of Compliance With the Treatment / Care Plan**

### Activity Recommendations

Rehabilitative exercise training in patients with heart failure and moderate-to-severe left ventricular systolic dysfunction improves functional capacity and symptoms. (Strength of evidence = A)

Comments:
There have been a number of randomised controlled trials regarding the role of exercise training in patients with heart failure. These have shown significant improvements in functional capacity and symptoms.\(^{13-15}\) One RCT demonstrated that a combination of ACE inhibitor treatment and exercise training produced greater symptomatic improvement than ACE inhibitor therapy alone.\(^{16}\) It appears that this improvement is mediated through adaptations in the peripheral circulation and skeletal musculature.
rather than adaptations in cardiac musculature. Elderly patients are able to participate in exercise training programs and should be strongly encouraged to participate. A non-randomised controlled trial showed that patients older and younger than 70 years of age had comparable responses to exercise training, which was similar in men and women.\textsuperscript{17}

**Dietary Recommendations**

Dietary sodium should be restricted to as close to 2 grams per day as possible. In no case should sodium intake exceed 3 grams daily. (Strength of Evidence = C)

In simple terms the advice to patients should be to avoid adding salt to cooking, not to add extra salt at the table and to avoid foods which are very high in salt (2 gms of salt is equal to approximately half teaspoon).

Patients with heart failure should be advised to avoid excessive fluid intake. However, fluid restriction is not advisable unless patients develop hyponatremia. (Strength of Evidence = C)

**Alcohol:**

Alcohol use should be discouraged. Patients who drink alcohol should be advised to consume no more than one drink per day or, if suffering from alcohol related cardiomyopathy, abstain altogether. (Strength of Evidence = C)

**The Problem of Non-compliance**

Non-compliance is a major cause of morbidity and unnecessary hospital admissions in heart failure.

Practitioners should be attuned to the problem of non-compliance and its causes. They should discuss the importance of compliance at follow-up visits and assist patients in removing barriers to compliance (eg cost, side effects, or complexity of the medical treatment regimen). (Strength of Evidence = B)

Other risk factors for admission or early readmission to hospital include:
- previous admissions in the last 12 months
- uncontrolled hypertension
- myocardial infarction, and
- low socio-economic status and low life satisfaction score.

Patient held action plans. No data was found on the effectiveness of patient held (self-management) plans.
B. Pharmacological Treatment Guidelines

Angiotensin-converting enzyme (ACE) inhibitors

All patients with heart failure due to systolic dysfunction should be considered for treatment with an angiotensin converting enzyme inhibitor in appropriate doses. (Strength of evidence = A)\textsuperscript{18,19}

Key Points

- ACE inhibitors improve symptoms of heart failure, improve left ventricular function, decrease hospital admissions and improve survival.
- ACE inhibitor therapy can also prevent the progression to heart failure in patients with asymptomatic LV dysfunction and should thus be used early in the course of the disease.
- It is likely that the effects of ACE inhibitors in heart failure are a class effect and thus no specific ACE inhibitor is recommended.
- Consider low starting dose (e.g., captopril 6.25 mg, enalapril 2.5 mg) and titrate up to the doses used in the RCTs, i.e., recommended dose captopril 50 mg tds, enalapril 10 mg bd, cilazapril 5 mg daily, quinapril 10 mg bd. Higher doses may be indicated for some patients (e.g., if coexisting hypertension).
- Hypotension may occur after the first dose especially if there is pre-existing hypotension, hyponatraemia, or over-diuresis.
- Monitor blood pressure, K\textsuperscript+ and renal function (at least weekly initially)
- Contraindications to ACE inhibitors:
  - prior ACE inhibitor intolerance; symptomatic hypotension; angioedema; K\textsuperscript+ >5.5 mmol/l; creatinine >0.25 mmol/l (some patients with renal failure may tolerate an ACE inhibitor but specialist referral is recommended)
- Concomitant use of diuretics is usually required for management of fluid overload.

Comments.

The ACE-inhibitor enalapril has been shown to reduce mortality in patients with moderate and severe heart failure in the SOLVD\textsuperscript{19} and CONSENSUS\textsuperscript{18} trials respectively (see Evidence Tables 1 & 2). The relative risk reduction was 31% (absolute risk reduction of 16%) for those with severe heart failure over 12 months. While in those with mild heart failure the overall relative risk reduction was 16% (absolute risk reduction of 4.5%) The greatest risk reduction in these patients (23%) was seen at 12 months. The survival curves indicate that treatment with enalapril increases survival by approximately 6 months. The effect of captopril on survival in patients with overt heart failure has not been studied.

Both enalapril and captopril have been shown to improve functional status, in 40-80% of patients.\textsuperscript{18,20} The average improvement has been 0.5-1 NYHA functional class.\textsuperscript{3} The SOLVD trial showed a modest reduction (RRR = 9.5%) in hospitalisation for those with mild to moderate congestive failure.\textsuperscript{19}

Side-effects with ACE-inhibitors are common - experienced by 87% in the SOLVD trial (note, 82% taking placebo also reported side-effects). The most common side-effect is dizziness due to hypotension, and cough. The actual average changes in blood pressure were modest, a decrease of 5 mmHg systolic blood pressure. Symptomatic hypotension is more common in those who have been over-diuresed, or are hypotensive to begin with. In the CONSENSUS trial\textsuperscript{18} (severe heart failure), 5.5% were withdrawn because of symptomatic hypotension. In general, systolic blood pressure of > 90 mmHg, without postural hypotension, is acceptable.\textsuperscript{21}
Cough is common with ACE-inhibitors, but is also common in patients with heart failure. It was reported in 37% of those taking enalapril in the SOLVD trial, and in 31% taking placebo. A patient presenting with cough should be carefully assessed for signs of increasing congestion before the cough is attributed to ACE-inhibitor therapy. Many patients with a cough attributed to ACE inhibitor therapy can continue with the treatment if the cough is not severe and the benefits are explained.

Initiating ACE inhibitor therapy
ACE-inhibitors can be added after volume overload has been controlled with diuretics. Patients who are at high risk of hypotension i.e. those with severe left ventricular systolic dysfunction, systolic blood pressure <100mmHg, or serum sodium <135mmol/l, should be given a small dose of a short acting agent (eg captopril 6.25mg), and monitored closely for 2-hours.3,22

Initial dose of ACE inhibitor should be low and dose titrated over 2-3 weeks with monitoring of BP and renal function. It is recommended that for those who are not at risk of hypotension, low doses should be started (eg enalapril 2.5mg BID), and patients reviewed after a week to monitor blood pressure, renal function and serum potassium.3 Doses should be titrated up over 2-3 weeks aiming for the doses used in the large-scale trials, ie enalapril 10mg BD and captopril 50mg TID, cilazapril 5mg per day, quinapril 10mg BID.

Diuretics
Patients with heart failure and clinical signs of fluid overload should be started on a diuretic. (Strength of evidence = B)

Key Points
- Diuretics provide relief of symptoms of pulmonary and systemic venous congestion in patients with heart failure
- There are no data regarding the effects of loop or thiazide diuretics on mortality in patients with heart failure
- Target doses of diuretics depend on the identification of a “dry” (or target) body weight
- Diuretics cause activation of the renin-angiotensin-aldosterone system in patients with mild symptoms of heart failure and thus should be used in combination with an ACE inhibitor to counteract this neuro-hormonal activation

Comments
There are few studies of the optimal diuretic therapy for heart failure, and the dose requirements may vary depending on the patients’ needs. In mild heart failure a thiazide may be sufficient (eg bendrofluazide 2.5 - 5mg daily initially).23

In general a loop diuretic will be required in moderate or severe heart failure or if the patient has failed to respond to thiazide diuretics (eg frusemide 40mg daily initially). If the initial dose proves inadequate, greater diuresis will usually be achieved by doubling the dose rather than by giving the same dose twice daily.3 Diuretic use should be combined with careful clinical monitoring, usually with patients monitoring their own weight.

A thiazide may be used in combination with loop diuretics for resistant oedema but only with extreme caution as a profound diuresis may ensue.
It is essential to monitor potassium and creatinine levels during diuretic use, usually at least every 3 months, but more frequently during initiation of therapy, and as required.

**Diuretics and ACE Inhibitors**

- Volume depletion from over-diuresis may increase the risk of first-dose hypotension when starting ACE inhibitor therapy, therefore it is very important to avoid excessive diuresis prior to starting ACE inhibitor therapy.
- If an ACE inhibitor is used with a diuretic then usually potassium replacement will not be required.
- Serious hyperkalaemia can occur if potassium-sparing diuretics are used in combination with ACE inhibitors, this combination should only be used under careful supervision (see section regarding use of spironolactone).

**Beta-Blockers**

Beta-blockers should be considered for all patients with heart failure due to systolic dysfunction (low ejection fraction) who have mild to moderate symptoms and are clinically stable. The aim of treatment is to improve survival and reduce hospitalisations. (Strength of Evidence = A).\(^\text{24-28}\)

**Key Points:**

- To date, 14,776 patients with chronic heart failure have been entered into 28 randomised clinical trials of beta-blocker therapy\(^\text{24-28}\) (these clinical trial data are approximately double that which is available for ACE inhibitors in patients with heart failure).
- The trials have now shown conclusively that beta-blockers improve survival, decrease hospitalisations and improve left ventricular function in patients with chronic heart failure (see below)
- Effects on patients symptoms and exercise tolerance are less consistent, and probably should not be considered a main aim of therapy (at least in the short-term)
- The benefits of beta–blockers are *in addition* to the benefits gained with ACE inhibitor therapy.
- There is a potential for adverse effects of beta-blockers particularly during initiation of therapy. Patient selection, timing of starting therapy and careful dose titration are of key importance (see below)
- The role of beta-blockers is in the treatment of patients with chronic heart failure and there is no place for the use of beta-blockers in the treatment of acute pulmonary oedema.

**Benefits**

- The following data for survival benefits are from the total dataset combined in a meta-analysis:\(^\text{24-28}\)
  - Absolute risk reduction 4.5% (approx. annual mortality rate 17.4% in placebo treated patients vs 12.9% in beta-blocker treated patients)
  - Relative risk reduction 28% (SD 4%)
  - Number needed to treat 22 (to prevent one death during approximately 1 year of treatment)
Practical Points for Use of Beta-blockers in heart failure
Patients considered for beta-blocker therapy should be similar to those represented in the clinical trials. Patients should:

- have chronic stable heart failure
- have left ventricular systolic dysfunction (LVEF< approximately 45%)
- have mild to moderate symptoms (NYHA functional class II-III)
- be clinically stable on adequate doses of ACE inhibitor and diuretic
- in general, be stable for about 2 weeks (without major changes in diuretic regime)

Starting Patients with Heart Failure on Beta-blockers
Patients with heart failure should be started on beta-blockers by clinicians experienced with their use in heart failure or in specialist clinics. Contraindications to beta-blockade, such as asthma or heart block (in the absence of a permanent pacemaker) should be checked for prior to starting treatment.

Initiation of beta-blockers in patients with heart failure:
- Start at low dose, eg metoprolol CR 47.5 mg ¼ tablet, or carvedilol 3.125-6.25mg
- Give under supervision in out-patient setting
- Some patients may need observation of heart rate and BP for 2 hours
- In some case, beta-blockers may be initiated prior to hospital discharge provided that the patient does not have signs of overt congestion

Dose titration:
- Fortnightly visits to titrate dose of beta-blocker. Check specifically for signs of worsening congestion, hypotension or bradycardia at each visit
- Withhold the morning dose on the day of the visit
- Some patients may need observing for 2 hours after each dose increment (eg if relative hypotension)
- Doubling of the dose every two weeks is a reasonable titration regime. However, titration can occur slowly and sometimes may take several months to achieve the desired maintenance dose

Potential adverse effects of beta-blockers in heart failure patients:
- Dizziness (common with the vasodilating beta-blockers such as carvedilol, often decreases if persist with treatment)
- Hypotension – usually a sign of intolerance (decrease dose or stop)
- Worsening heart failure – mainly increasing congestion. Manage by increasing diuretics and continuing beta-blocker if possible
- Heart block

Target doses:
- Aim for metoprolol 150-200mg daily (exact dose depends on preparation of metoprolol used) or carvedilol 25mg bid
**Spironolactone**

Patients with severe heart failure (NYHA classification III or IV, and who have been class IV within the last 6 months) should be considered for the addition of spironolactone 25mg daily to existing therapy (including ACEIs). \(^{(\text{Strength of evidence } = \text{A})}\)

Until recently it has been assumed that the suppression of the renin-angiotensin system by an ACE inhibitor alone would suppress the formation of aldosterone. In addition, there has been concern that the concurrent use of an aldosterone-receptor blocker and an ACEI could lead to dangerous hyperkalaemia. The RALES trial\(^{29}\), a single yet large and well designed trial, found that the use of spironolactone in people with severe heart failure was not only safe but conferred substantial survival benefits.

In the RALES trial\(^{29}\) patients with severe heart failure (NYHA classification III or IV and LVEF< 35%) who had spironolactone 25mg daily added to usual therapy (including ACEIs) had reduced mortality, improved quality of life and reduced hospital admissions.

- **Survival benefit** –
  - Absolute risk reduction 11% (two year mortality rate 46% in placebo treated patients vs 35% in spironolactone treated patients)
  - Relative risk reduction 30%
  - Number needed to treat 9 (to prevent one death during average 2 years treatment)

- **Reduction of number of patients requiring hospital admission for cardiac causes:**
  - NNT=11 (2 years)
- **Improved symptoms of heart failure.**
- **10% of patients experienced gynaecomastia or breast pain (NNH=11, 2 years)**

**Dose:** Spironolactone 25mg od

Creatinine and electrolytes should be checked 3-4 days, one week and one month after initiation and then as indicated by renal function (6 monthly in stable patients).

**Contraindications:**
Serum creatinine > 0.25mmol/l, potassium >5.0 mmol/l.

**Digoxin**

**(a) Digoxin in patients with atrial fibrillation**

Digoxin should be considered for all patients with heart failure who are in atrial fibrillation. \(^{(\text{Strength of evidence } = \text{B})}\)

**Key Points**
- **Digoxin is useful for control of the ventricular rate in patients with heart failure and atrial fibrillation**
- **Digoxin alone may control the ventricular rate at rest but usually does not provide sufficient rate control with exercise**
- **Additional agents such as low dose diltiazem or amiodarone may be required to control the exercise heart rate. If a beta-blocker is to be used for the treatment of heart failure then this may provide additional rate control.**
(b) *Digoxin in patients with heart failure and sinus rhythm*

Digoxin should be considered for patients with heart failure who remain symptomatic despite treatment with ACE inhibitor and diuretics, with the aim of improving symptoms and preventing further clinical deterioration. (Strength of evidence = A)\(^{30,31}\)

**Key Points**
- Digoxin can improve symptoms of heart failure, reduce hospitalisation for worsening heart failure but has no overall effect on total mortality in patients with heart failure who are in sinus rhythm.\(^{31}\)
- The lack of effect on total mortality means that digoxin need not be used if patients are asymptomatic with diuretics and ACE inhibitors.
- Generally, digoxin should be considered if a patient has failed to respond to ACE inhibitors and diuretics.

**Comments**

The DIG Trial\(^{31}\) examined the role of digoxin in patients with heart failure who are in sinus rhythm (see evidence table 3). While this trial showed that overall mortality was not affected in those taking digoxin, both hospitalisation due to worsening heart failure, and the combined end-point of death or hospitalisations due to worsening heart failure were decreased. The absolute risk reduction (ARR) was approximately 7%. It had been previously shown that when digoxin was withdrawn from patients, that exercise tolerance, NYHA class and quality-of-life scores deteriorated.\(^{32}\) However, given that digoxin does not reduce mortality, patients who are asymptomatic after treatment with ACE inhibitors and diuretics, are unlikely to gain a benefit from the addition of digoxin.

Loading doses of digoxin are generally not required. In the presence of normal renal function a dose of 0.25mg daily may suffice. In the elderly or in those with renal impairment a reduced dose such as 0.125 or 0.0625mg daily is necessary. Digoxin levels should be checked after about 1 week in those with normal renal function, although steady state may take longer to be achieved in those with renal impairment.

Signs of digoxin toxicity include: confusion, nausea, anorexia, visual disturbance and either tachy- or bradyarrhythmias. Digoxin toxicity should be suspected in any patient presenting with any of the above symptoms or unusual symptoms, particularly in the elderly. Some drugs may increase plasma digoxin levels, for example: amiodarone, diltiazem, verapamil, antibiotics, quinidine.

**Angiotensin II (All) Antagonists**

All antagonists should be considered for patients intolerant of ACE inhibitors (Strength of evidence = C)

**Evidence summary:**

These drugs (eg losartan) block the angiotensin II type 1 receptors and thus block the renin-angiotensin-aldosterone system at a point beyond the angiotensin converting enzyme. The potential advantages include more complete renin-angiotensin-aldosterone blockade and reduced side-effects such as cough and angio-oedema. An initial pilot study in patients with heart failure (ELITE I)\(^{33}\) suggested that losartan may reduce mortality relative to captopril. However, a large-scale mortality trial (ELITE II\(^{34}\)) has not shown a superiority of losartan over captopril. Currently AT II blockers should be considered for patients intolerant of ACE inhibitors.
Anticoagulation

Routine anticoagulation is not recommended for all patients with heart failure.

Long-term anticoagulation with warfarin should be considered in patients with concurrent atrial fibrillation (INR 2.0 – 3.0). (Strength of evidence = A)\(^{35,36}\)

Anticoagulation with warfarin should be considered in patients with a history of systemic or pulmonary emboli, documented left ventricular thrombus. (optimal range for anticoagulation in these groups has not been ascertained, consider using INR 2.0 – 3.0). (Strength of evidence = C)

Key Points
- There are no controlled trials of the effects of routine anticoagulation in all patients with heart failure
- Left ventricular systolic dysfunction is a significant risk factor for stroke in patients with atrial fibrillation
- Consider referral to specialist with aim to restore sinus rhythm.

Comments.
Atrial fibrillation occurs in 15% to 30% of patients with heart failure.\(^{37}\) Furthermore, the risk of stroke is greater in patients with atrial fibrillation and concomitant heart failure, than those with isolated lone atrial fibrillation (annual risk of stroke 5-8% versus 1.3%).\(^{35,36}\) An analysis of pooled data from five randomised controlled trials, concluded that warfarin consistently decreased the risk of stroke in patients with atrial fibrillation (RRR of 68%), with virtually no increase in the frequency of major bleeding.\(^{35}\) The Stroke Prevention in Atrial Fibrillation (SPAF) III trial\(^{38}\) which compared conventional warfarin dosage (aiming for an INR of 2.0-3.0) with low dose warfarin (INR of 1.2 to 1.5) combined with aspirin, was terminated early, because of an excess of strokes in the low dose warfarin arm. It was concluded that conventional dose warfarin should be regarded as optimal treatment for the majority of patients with atrial fibrillation. An INR of 2.0-3.0 provides a reasonable balance between reducing the risk of thromboembolism and minimising potential for bleeding complications. Patients who are at higher risk of serious bleeding include those susceptible to falls, those with a previous history of gastro-intestinal haemorrhage, those with impaired liver function, and those who are unable to participate in the monitoring required. Monitoring will be required for the duration of the anticoagulation with regular INRs.

Aspirin

Patients with underlying coronary artery disease or concomitant peripheral vascular or cerebrovascular disease should be treated with low-dose aspirin (eg 75-150mg daily) to prevent further vascular events. (Strength of evidence = A)

Key Points
- Aspirin reduces vascular events when used as secondary prevention in patients with coronary artery, peripheral vascular or cerebrovascular disease
- There is some concern that aspirin reduces the survival benefit of ACE inhibitors in patients with heart failure. However, this is still unclear and further clinical trials are awaited to clarify this situation.
**Co-prescribing**

Certain drugs interact adversely with the primary therapeutic agents for congestive heart failure or are poorly tolerated. Vigilance should be exercised in all prescribing. The following groups of drugs should be used cautiously or avoided altogether:

- NSAIDS
- Calcium channel blockers (with the exception of amlodipine and felodipine)
- Corticosteroids
- Tricyclic Antidepressants
- Carbenoxolone
- Urinary alkalinisers (high sodium content)

**Concomitant conditions:**

**Atrial fibrillation**

The following points should be considered when managing a patient with heart failure who is in atrial fibrillation:

- Should restoration of sinus rhythm be attempted?
- Is AF the cause or consequence of heart failure?
- Does the patient have underlying mitral valve disease?
- Does the patient have thyrotoxicosis?
- Are there contraindications to warfarin therapy?

Consideration of these points helps to identify those in whom intervention may be required, rather than just aiming for rate control.

**Recommendation:**

**In patients with AF anticoagulate with warfarin to prevent thromboembolism (INR 2.0-3.0).** (Strength of evidence = A)

**Consider the need for cardioversion (will require specialist referral for cardioversion)**

Medical cardioversion may be achieved by amiodarone: 200mg tds for 2 weeks, 200mg bid for 2 weeks then 200mg daily (Strength of evidence = B). Anticoagulation with warfarin is required whether cardioversion is undertaken electrically or chemically. Cardioversion is recommended after 4 weeks if still in AF (success is much higher if the history of AF is less than 1 year or the left atrial diameter is less than 50mm) (Strength of evidence = C).

**Continue anticoagulation for a further 6-12 months while monitoring for recurrence. If AF persists consider long-term therapy with amiodarone.** (Strength of evidence = C)
Clinical Notes
Digoxin alone will usually not adequately control the ventricular rate in atrial fibrillation. The increasing use of beta-blockers in patients with heart failure will allow these agents to be used for rate control. Diltiazem or amiodarone may be required in some cases to achieve adequate rate control.

Ischaemic heart disease
Patients with congestive heart failure and ischaemic heart disease, and who do not have contraindications to bypass surgery, should have the risks and benefits of coronary artery surgery considered. This will usually require a specialist cardiology assessment.

CABG surgery improves survival in patients with moderate (LVEF 35 to 50%) heart failure due to ischaemic heart disease. (Level of evidence = A)⁴⁰

CABG surgery improves survival, NYHA class and angina in selected patients with severe (LVEF < 30%) heart failure. (Level of evidence = B)

Comments
Ischaemic heart disease (or coronary heart disease) is a common cause of heart failure.

There are several ways in which ischaemia may present as heart failure, including one or more myocardial infarctions culminating in heart failure, primary presentation as congestive heart failure without clinically overt antecedent infarction (“ischaemic cardiomyopathy” which is most commonly a diffuse fibrosis), a large full thickness infarct resulting in a left ventricular aneurysm, or transient global ischaemia resulting in acute pulmonary oedema.

The aim of coronary artery bypass grafting (CABG) is to prevent further ischaemic myocardial damage and to reverse myocardial hibernation. Hibernating myocardium occurs in some patients with congestive heart failure due to underlying chronic ischaemia. It is characterised by areas of hypocontractile myocardium that are potentially reversible if adequate coronary perfusion is restored by revascularisation.⁴¹ Hibernating myocardium may be identified using radionucleotide ventriculography or dobutamine stress echocardiography.

Most of the large randomised controlled trials of medical versus CABG surgery excluded patients with heart failure and severe left ventricular impairment. The Coronary Artery Surgery Study (CASS) did examine a subset of 160 patients with LVEF between 35 and 50%. After seven years follow-up, survival in the surgical group was 84% compared with 70% in the medical group.⁴⁰ The CASS Registry data on patients with LVEF <26%, showed a 5-year survival of 63% in those undergoing CABG surgery, compared with 43% in the medical cohort.⁴² The benefits of CABG surgery have not been confined to improved survival. Several cohort studies⁴³,⁴⁴ have shown improved LV function after surgery (in patients with baseline LVEF < 30%), and corresponding improvement in functional status. Most report that patients improve by 1 to 1.5 NYHA classes. This together with improvements in angina class could reasonably be expected to improve patients’ quality of life.
CRITERIA FOR SPECIALIST REFERRAL

Many patients with heart failure are elderly and have multiple concomitant medical conditions in whom extensive investigation may not be appropriate. Recommendations regarding the criteria for specialist referral cannot be based on evidence from randomised controlled trials as the interventions evaluated in such trials are usually refer to subsets of patients with established diagnoses. Consequently, the recommendations for referral outlined below are based on consensus from this guidelines group (with outside consultation). Clinicians should rely on their clinical judgement and when in doubt should err on the side of referral.

There are certain patients who may benefit from consideration of further investigation. Of particular note are:

- The onset of heart failure in younger patients (in whom transplantation may be considered)
- Those whose history suggests severe ischaemia or significant valvular disease where further investigation and intervention (such as angioplasty or surgery) may be indicated.

In these cases specialist referral is recommended.

Specialist referral may also be considered in the following situations where:

- The diagnosis is uncertain
- The aetiology is uncertain
- Arrhythmia (either supra-ventricular, ventricular or at times atrial fibrillation) are apparent
- In those with sudden onset of heart failure
- When beta-blocker treatment is being considered
- Those who have an inadequate response to treatment
- When the indication for anticoagulation is uncertain

Clinical Notes
The recommendations for specialist referral should not delay initiation of appropriate treatment for patients with symptomatic heart failure.
References


**APPENDIX I Evidence Tables**

**Evidence table 1: Clinical Area: ACE-inhibitor treatment of severe CHF.**


<table>
<thead>
<tr>
<th>Study type/grade</th>
<th>Randomised-controlled trial. Grade 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Outcomes</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>· primary - 6-month mortality; cause of death</td>
</tr>
<tr>
<td></td>
<td>· secondary -12-month mortality</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td></td>
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<tr>
<td></td>
<td>· n= 253; 127 enalapril, 126 placebo; with severe CHF (NYHA class IV)</td>
</tr>
<tr>
<td></td>
<td>· Mean age 70 years; 29.5% female; mean ejection fraction not stated.</td>
</tr>
<tr>
<td></td>
<td>· exclusion criteria: Acute pulmonary oedema, haemodynamically important aortic or mitral valve stenosis; myocardial infarction within the previous 2 months; unstable angina, planned cardiac surgery; right heart failure due to pulmonary disease; serum creatinine &gt;300μmol/L.</td>
</tr>
<tr>
<td></td>
<td>· method of randomisation: Computer-generated allocation, stratified with regard to treatment with vasodilators.</td>
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<td></td>
<td>· intervention: Enalapril titrated to a maximum of 20mg/day. (mean dose 18.4mg)</td>
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<td></td>
<td>· blinding: Double-blinded for randomisation and end-point ascertainment.</td>
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<td></td>
<td>· length of follow-up: 12-months.</td>
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<td></td>
<td>· completeness of follow-up: 100%</td>
</tr>
<tr>
<td><strong>Validity</strong></td>
<td></td>
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<tr>
<td></td>
<td>· is the study type appropriate for the questions being asked? - Yes</td>
</tr>
<tr>
<td></td>
<td>· was the study population typical of patients with this disease? - Yes</td>
</tr>
<tr>
<td></td>
<td>· were the treatment/control groups comparable at baseline? - Yes</td>
</tr>
<tr>
<td></td>
<td>· was the intervention compared to placebo and/or best accepted intervention? - Yes</td>
</tr>
<tr>
<td></td>
<td>· was there compliance with the intervention? - Withdrawals were not significantly different in the two groups (14% placebo, 17% enalapril group). No specific mention of compliance in the remaining subjects.</td>
</tr>
<tr>
<td></td>
<td>· was there equal intensity of observation of study and control subjects? - Yes</td>
</tr>
<tr>
<td></td>
<td>· was the process of observation likely to effect the outcome? - No</td>
</tr>
<tr>
<td></td>
<td>· intention to treat analysis? - Yes</td>
</tr>
<tr>
<td></td>
<td>· did conclusions about safety take into account the limited size of the study? - Yes</td>
</tr>
<tr>
<td></td>
<td>· is effectiveness proven? - Yes</td>
</tr>
<tr>
<td></td>
<td>· <strong>Summary</strong>- Excellent study. Enrolment and follow-up terminated ahead of schedule because of consistent difference in favour of enalapril.</td>
</tr>
<tr>
<td>Results</td>
<td>Outcome</td>
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Note: Improvement in NYHA class occurred in 22% of placebo group and 42% of enalapril group. Heart size reduced by 3.2% in placebo group and 9.6% in enalapril group (p=0.02). Most common adverse effects were hypotension necessitating withdrawal of 7 cases in treatment group (notably when starting dose was 5mg,) and raised creatinine, 6 withdrawals.

**Authors’ conclusions**
That treatment with enalapril improves survival in those with severe CHF. Also noted improvement in NYHA class, heart size, and concomitant use in those in the enalapril group. The entire treatment effect was due to a reduction in mortality from the progression of heart failure, with no differences in rate of sudden death between the groups.

**Reviewers’ conclusions**
Agree with authors’ conclusions.
### Evidence table 2: Clinical Area: ACE-inhibitor treatment of CHF


| Design | ● n=2569 (39 924 screened): 1285 enalapril, 1284 placebo; with clinically stable CHF.  
● Mean age 60 years; 20% female; 90% NYHA Classes II - III; mean ejection fraction 24.85%. Groups comparable at baseline.  
● exclusion criteria: Age > 80-years; Haemodynamically serious valvular disease requiring surgery; unstable angina; myocardial infarction in the previous month; severe pulmonary disease; serum creatinine> 0.177mmol/L.  
● method of randomisation: Computer-generated allocation schedule, stratified by site.  
● intervention: Enalapril titrated to a maximum of 10mg twice daily.  
● blinding: Double-blind for randomisation and end-point ascertainment. If patient remained symptomatic despite maximal therapy, open-label treatment with an ACE-inhibitor was allowed, and blinded medication discontinued.  
● length of follow-up: Range 22 to 55 months, average 41.4.  
● completeness of follow-up: Almost complete: the vital status of one patient in each group was not known. |
| --- | --- |
| Validity | ● is the study type appropriate for the questions being asked? - Yes  
● was the study population typical of patients with this disease? - Yes, except those over 80-years old.  
● were the treatment/control groups comparable at baseline? - Yes  
● was the intervention compared to placebo and/or best accepted intervention? - Yes  
● was there compliance with the intervention? - Proportion of patients taking at least 75% of prescribed dose at: 1-year = 80% in treatment group, 77% in placebo; 2-years = 74% and 67%; 3-years = 69% and 60%. At the final visit 49.3% in the treatment group were taking 10mg/day, 49.1% of the placebo group were taking equivalent amount. Mean daily dose of enalapril was 11.2 mg.  
● was there equal intensity of observation of study and control subjects? - Yes  
● was the process of observation likely to effect the outcome? - No  
● intention to treat analysis? – Yes  
● did conclusions about safety take into account the limited size of the study? - Yes  
● is effectiveness proven? – Yes  
● **Summary**- Very good study. |
<table>
<thead>
<tr>
<th>Results</th>
<th>Outcome</th>
<th>Relative Risk Reduction % (95% CI)</th>
<th>One-sided p-value</th>
<th>Absolute risk reduction (over 48-months)</th>
<th>Number needed to treat (over 48 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Death # - cardiovascular deaths - progressive heart failure - myocardial infarction</td>
<td>16 (5 to 26) 18 (6 to 28) 22 (6 to 35) 28 (-8 to 52)</td>
<td>&lt;0.0036 &lt;0.002 &lt;0.0045 &gt;0.07</td>
<td>4.5 4.8 3.2 not statistically significant</td>
<td>22 21 31 not statistically significant</td>
</tr>
<tr>
<td></td>
<td>Hospitalisation for cardiovascular reasons</td>
<td>9.5</td>
<td>&lt;0.001</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>Deaths or hospitalisation for CHF</td>
<td>26 (18 to 34)</td>
<td>&lt;0.0001</td>
<td>9.6</td>
<td>10</td>
</tr>
</tbody>
</table>

* risk reduction calculated by the authors using a log-rank analysis, reflecting the risk over the entire follow-up period.

# Greatest reduction in mortality seen in the first 24 months (23% RRR)

Authors’ conclusions
That ACE-inhibitor therapy in the form of enalapril reduced overall mortality by 16%, and this was both clinically and statistically significant. Treating 1000 patients for 3-years, would prevent 50 premature deaths, and 350 hospitalisations. The greatest benefit of treatment was the decrease in mortality from progressive heart failure.

Reviewers’ conclusions
Good evidence of survival benefit in patients with mild to moderate heart failure, when given enalapril in addition to usual management.
**Evidence table 3: Clinical Area: Management of heart failure: the role of digoxin.**


<table>
<thead>
<tr>
<th>Study type/grade</th>
<th>Randomised-controlled trial. Grade 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outcomes</td>
<td>Primary - All cause mortality.</td>
</tr>
<tr>
<td></td>
<td>Secondary - Mortality from cardiovascular causes; mortality from worsening heart failure; hospitalisation from worsening heart failure.</td>
</tr>
<tr>
<td>Design</td>
<td>n= 6800; 3397 digoxin: 3403 placebo.</td>
</tr>
<tr>
<td></td>
<td>Patients with left ventricular ejection fraction (LVEF) ≤0.45. Predominantly mild to moderate heart failure, with only 2% in NYHA functional class IV. Mean age 63.5, 22.3% women. 98.4% taking either ACE-inhibitors or diuretics. (94.4% on ACE-inhibitors).</td>
</tr>
<tr>
<td></td>
<td>exclusion criteria: myocardial infarction, cardiac surgery or PTCA within previous 4 weeks; atrial fibrillation/flutter; II-III° AV block without pacemaker; unstable or refractory angina; hypertrophic cardiomyopathy.</td>
</tr>
<tr>
<td></td>
<td>method of randomisation: Telephone, stratified by site.</td>
</tr>
<tr>
<td></td>
<td>intervention: Digoxin, initial dose titrated according to an algorithm based on age, sex, weight and renal function.</td>
</tr>
<tr>
<td></td>
<td>blinding: Double-blinded.</td>
</tr>
<tr>
<td></td>
<td>length of follow-up: Average 37-months.</td>
</tr>
<tr>
<td></td>
<td>completeness of follow-up: 98.6%</td>
</tr>
<tr>
<td>Validity</td>
<td>is the study type appropriate for the questions being asked? - Yes</td>
</tr>
<tr>
<td></td>
<td>was the study population typical of patients with this disease? - Yes, with the exception that their were few with severe heart failure.</td>
</tr>
<tr>
<td></td>
<td>were the treatment/control groups comparable at baseline? - Yes</td>
</tr>
<tr>
<td></td>
<td>was the intervention compared to placebo and/or best accepted intervention? - Yes</td>
</tr>
<tr>
<td></td>
<td>was there compliance with the intervention? - Yes: at 1-year 85.6% of those in the digoxin group were compliant, 82.9% in the placebo group; at final visit the figures were 70.8% and 67.9% respectively.</td>
</tr>
<tr>
<td></td>
<td>was there equal intensity of observation of study and control subjects? - Yes</td>
</tr>
<tr>
<td></td>
<td>was the process of observation likely to effect the outcome? - No</td>
</tr>
<tr>
<td></td>
<td>intention to treat analysis? - Yes</td>
</tr>
<tr>
<td></td>
<td>did conclusions about safety take into account the limited size of the study? - Yes</td>
</tr>
<tr>
<td></td>
<td>is effectiveness proven? - Yes</td>
</tr>
<tr>
<td></td>
<td><strong>Summary</strong> - Valid study.</td>
</tr>
<tr>
<td>Results</td>
<td>Outcome</td>
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<td>---------</td>
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</tr>
<tr>
<td>- Death (all cause)</td>
<td></td>
</tr>
<tr>
<td>- Death from cardiovascular causes</td>
<td></td>
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<tr>
<td>- Death from worsening heart failure</td>
<td></td>
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<tr>
<td>- Hospitalisation due to worsening heart failure</td>
<td></td>
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<tr>
<td>- Death or hospitalisation due to worsening heart failure.</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Relative risk reduction (95% CI)</th>
<th>Absolute risk reduction</th>
<th>Numbers needed to treat</th>
</tr>
</thead>
<tbody>
<tr>
<td>1% (-0.07 to 9, p=0.80)</td>
<td>-0.3% (not significant)</td>
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<td>-0.01% (-0.10 to + 0.07, p=0.78)</td>
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<td>12% (-0.01 to 33%, p=0.06)</td>
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<td>28% (21 to 34, p&lt;0.001)</td>
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<td>25% (18 to 31)</td>
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Authors’ conclusions: Digoxin had no effect on overall mortality, though there were fewer deaths and hospitalisations due to worsening heart failure. This reduction was greatest in those that had LVEFs < 0.25, or NYHA functional class III/IV.

Reviewers’ conclusions: Adding digoxin to ACE-inhibitors and diuretics for the management of patients with heart failure who are in sinus rhythm, has no effect on overall mortality. Further the effect on death and hospitalisation due to worsening heart failure would appear to be confined to those with moderate to severe heart failure.
Comparative effects of low and high doses of the angiotensin-converting enzyme inhibitor, lisinopril, on morbidity and mortality in chronic heart failure. ATLAS Study Group.

Packer M, Poole-Wilson PA, Armstrong PW, Cleland JG, Horowitz JD, Massie BM, Ryden L, Thygesen K, Uretsky BF.
College of Physicians and Surgeons (M.P.), Columbia University, New York, NY 10032, USA.

Abstract
BACKGROUND: Angiotensin-converting enzyme (ACE) inhibitors are generally prescribed by physicians in doses lower than the large doses that have been shown to reduce morbidity and mortality in patients with heart failure. It is unclear, however, if low doses and high doses of ACE inhibitors have similar benefits.

METHODS AND RESULTS: We randomly assigned 3164 patients with New York Heart Association class II to IV heart failure and an ejection fraction < or = 30% to double-blind treatment with either low doses (2.5 to 5.0 mg daily, n=1566) or high doses (32.5 to 5 mg daily, n=1568) of the ACE inhibitor, lisinopril, for 39 to 58 months, while background therapy for heart failure was continued. When compared with the low-dose group, patients in the high-dose group had a nonsignificant 8% lower risk of death (P=0.128) but a significant 12% lower risk of death or hospitalization for any reason (P=0.002) and 24% fewer hospitalizations for heart failure (P=0.002). Dizziness and renal insufficiency was observed more frequently in the high-dose group, but the 2 groups were similar in the number of patients requiring discontinuation of the study medication. Conclusions: These findings indicate that patients with heart failure should not generally be maintained on very low doses of an ACE inhibitor (unless these are the only doses that can be tolerated) and suggest that the difference in efficacy between intermediate and high doses of an ACE inhibitor (if any) is likely to be very small.


Publication Types, MeSH Terms, Substances
Publication Types:
- Clinical Trial
- Comparative Study
- Randomized Controlled Trial
- Research Support, Non-U.S. Gov't

MeSH Terms:
- Aged
- Angiotensin-Converting Enzyme Inhibitors/administration & dosage
- Angiotensin-Converting Enzyme Inhibitors/adverse effects
- Death
- Double-Blind Method
- Female
- Heart Failure/drug therapy
- Heart Failure/mortality
- Hospitalization
- Humans
- Lisinopril/administration & dosage
- Lisinopril/adverse effects
- Male
- Middle Aged
- Morbidity
- Survival Analysis

Substances:
- Angiotensin-Converting Enzyme Inhibitors
- Lisinopril


Department of Cardiology, Sahlgrenska University Hospital, Gothenburg, Sweden.

CONTEXT: Results from recent studies on the effects of beta1-blockade in patients with heart failure demonstrated a 34% reduction in total mortality. However, the effect of beta1-blockade on the frequency of hospitalizations, symptoms, and quality of life in patients with heart failure has not been fully explored.


DESIGN: Randomized, double-blind controlled trial, preceded by a 2-week single-blind placebo run-in period, conducted from February 14, 1997, to October 31, 1998, with a mean follow-up of 1 year.

SETTING: Three hundred thirteen sites in 14 countries.

PARTICIPANTS: Patients (n = 3991) with chronic heart failure, New York Heart Association (NYHA) functional class II to IV, and ejection fraction of 0.40 or less who were stabilized with optimum standard therapy.

INTERVENTIONS: Patients were randomized to metoprolol CR/XL, 25 mg once per day (NYHA class II), or 12.5 mg once per day (NYHA class III or IV), titrated for 6 to 8 weeks up to a target dosage of 200 mg once per day (n = 1990); or matching placebo (n = 2001).

MAIN OUTCOME MEASURES: Total mortality or any hospitalization (time to first event), number of hospitalizations for worsening heart failure, and change in NYHA class, by intervention group; quality of life was assessed in a substudy of 741 patients.

RESULTS: The incidence of all predefined endpoints was lower in the metoprolol CR/XL group than in the placebo group, including total mortality or all-cause hospitalizations (the prespecified second primary end point; 641 vs 767 events; risk reduction, 19%; 95% confidence interval [CI], 10% to 27%; P < .001); total mortality or hospitalizations due to worsening heart failure (311 vs 439 events; risk reduction, 31%; 95% CI, 20% to 40%; P < .001), number of hospitalizations due to worsening heart failure (317 vs 451; P < .001), and number of days in hospital due to worsening heart failure (3401 vs 5303 days; P < .001). NYHA functional class, assessed by physicians, and McMaster Overall Treatment Evaluation score, assessed by patients, both improved in the metoprolol CR/XL group compared with the placebo group (P = .003 and P = .009, respectively).

CONCLUSIONS: In this study of patients with symptomatic heart failure, metoprolol CR/XL improved survival, reduced the need for hospitalizations due to worsening heart failure, improved NYHA functional class, and had beneficial effects on patient well-being.


Department of Internal Medicine, Division of Cardiology, University of Michigan, Ann Arbor, USA.

BACKGROUND AND METHODS: Aldosterone is important in the pathophysiology of heart failure. In a doubleblind study, we enrolled 1663 patients who had severe heart failure and a left ventricular ejection fraction of no more than 35 percent and who were being treated with an angiotensin-converting-enzyme inhibitor, a loop diuretic, and in most cases digoxin. A total of 822 patients were randomly assigned to receive 25 mg of spironolactone daily, and 841 to receive placebo. The primary end point was death from all causes. RESULTS: The trial was discontinued early, after a mean follow-up period of 24 months, because an interim analysis determined that spironolactone was efficacious. There were 386 deaths in the placebo group (46 percent) and 284 in the spironolactone group (35 percent, relative risk of death, 0.70; 95 percent confidence interval, 0.60 to 0.82; P<0.001). This 30 percent reduction in the risk of death among patients in the spironolactone group was attributed to a lower risk of both death from progressive heart failure and sudden death from cardiac causes. The frequency of hospitalization for worsening heart failure was 35 percent lower in the spironolactone group than in the placebo group (relative risk of hospitalization, 0.65; 95 percent confidence interval, 0.54 to 0.77; P<0.001). In addition, patients who received spironolactone had a significant improvement in the symptoms of heart failure, as assessed on the basis of the New York Heart Association functional class (P<0.001). Gynecomastia or breast pain was reported in 10 percent of men who were treated with spironolactone, as compared with 1 percent of men in the placebo group (P<0.001). The incidence of serious hyperkalemia was minimal in both groups of patients. CONCLUSIONS: Blockade of aldosterone receptors by spironolactone, in addition to standard therapy, substantially reduces the risk of both morbidity and death among patients with severe heart failure.

Publication Types:

- Clinical Trial
- Multicenter Study
- Randomized Controlled Trial

PMID: 10471456 [PubMed - indexed for MEDLINE]
The Guts of the Guideline.

Four Key Issues

1.) Need for an Echocardiogram.
It is helpful to have an echo that shows left ventricular dysfunction as you can then be confident in pushing the medication. There are no other objective tests of systolic dysfunction, so do order an echo for patients with suspected heart failure. If the echo takes time to get, START the ACE-inhibitor and β-blockers/spironolactone as advised.

2.) High dose ACE-Inhibitors.
ACE inhibitors need to be at the maximum tolerated dose. Be guided by patient reports of symptoms and side effects rather than blood pressure values when up-titrating. The maximum dose of ACE-inhibitor for heart failure may be different from that used in hypertension.

3.) β-blockers.
Start metoprolol C.R. low and build up to 190 mg per day or start carvedilol low dose and titrate up to 25mg bid. Be guided by patient reports of symptoms and side effects rather than blood pressure values when up-titrating.

4.) Spironolactone.
Consider starting spironolactone 25mg and watch the potassium and renal function.
Heart Failure – Brief Pathophysiology Review.

What is heart failure?

A useful pathophysiological definition of heart failure is in the WHO Heart Failure Guideline:

“. . . is an inability of the heart to deliver blood (and therefore oxygen) at a rate commensurate with the requirements of the metabolising tissues at rest or during light exercise. This leads to the characteristic systemic pathophysiological responses (neural, hormonal, renal and others), symptoms and signs.”

This leads on to a clinical definition of heart failure (from the Scottish Intercollegial Guidelines Network aka ‘SIGN’):

“. . . a syndrome that consists of breathlessness, fatigue and fluid retention (peripheral oedema and an elevated jugular venous pressure) resulting from cardiac dysfunction.”

Systolic function is accompanied by dilated LV with reduced wall motion which means reduced LV contraction and emptying. Diastolic dysfunction is covered in a separate summary. The terms ‘Right Heart Failure’ (meaning congestion of the systemic veins) and ‘Left Heart Failure’ (congestion of the pulmonary veins) are terms that are sometimes also used.

As a side issue, it must be noted that there is not necessarily a correlation between the degree of dysfunction and symptoms of heart failure, and that there are patients without the classical symptoms of heart failure, but who are extremely unlikely to be diagnosed except through a chance investigation.

Warning flags (for heart failure) in a patient’s history include previous MI, hypertension, angina, valvular disease/rheumatic fever, irregular heart rhythm, or alcohol abuse. Any disorders that could precipitate or exacerbate heart failure should be identified and treated.
**Why do we use the drugs we do?**

At some stage prior to heart failure becoming overt, neurohormonal systems and the SNS have become over-induced. The drugs that are used in heart failure either block the over-active neurohormonal systems or their effects, or reduce the symptoms that the neurohormones have caused.

The activation of the renin-angiotensin-aldosterone system results in the following:

**Renin:**
- Converts angiotensin to angiotensin I (which is converted to angiotensin II by angiotensin converting enzyme (ACE)).

**Angiotensin II:**
- Potent vasoconstrictor on vascular smooth muscle, increasing systemic resistance
- Increases blood vessel permeability thereby increasing extracellular fluid
- Stimulates NA release
- Acts as a growth factor → cardiac hypertrophy
- Acts on thirst and drinking behaviour (→ vasopressin release [anti-diuretic]).

**Aldosterone:**
- Increases Na⁺ and H₂O retention
- Increases K⁺ loss

Triggered by falling cardiac output (and the RAAS) the activation of the SNS is a compensatory mechanism to provide inotropic support and maintain cardiac output.

**Sustained SNS activation:**
- Activates RAAS
- Increases plasma NA → Na⁺ and H₂O retention
- Cardiac cell death, hypertrophy
- Acts on juxtaglomerular cells → increase in renin release
- Baroreceptor dysfunction
- Down-regulation to myocardial β-receptors.

The drugs that are used in heart failure either block receptors, disrupt hormonal system cascades, or reduce the resultant effects of RAAS/SNS over stimulation:
Diuretics:
Used to reduce the symptoms of congestion. May be possible to down-titrate once ACE-inhibitors are established.

ACE-Inhibitors:
Inhibit the conversion of Angiotensin I to Angiotensin II and the deleterious effects of angiotensin II. Blockade of ACE stops the breakdown of bradykinin into inactive fragments, enabling greater vasodilation.

Angiotensin II Antagonists:
Antagonise the effects of angiotensin II (used especially in patients who are unable to tolerate any ACE inhibitor).

β-blockers:
Inhibit renin production (and the RAAS), reduce the effects of NA on α- and β-receptors, regulates heart rate to some extent.

Spironolactone:
Acts as a competitive antagonist to aldosterone, reduces K⁺ loss.

Digoxin
Acts to increase stroke work and cardiac output. This reduces the activation of the sympathetic system. Spironolactone may increase levels of digoxin.

References:
Diastolic dysfunction

Diastolic dysfunction is characterised by slowed ventricular relaxation and/or myocardial stiffness, typical symptoms of heart failure and normal or slightly reduced ejection fraction. It is more likely to occur in the elderly and in female patients.

Current diagnostic tests are not able to define diastolic dysfunction with great accuracy so diagnosis may be one of chance or exclusion.

It has been estimated that up to 50% of patients with signs and symptoms of heart failure may have preserved systolic function. Patients have a reduced mortality rate when compared with patients with systolic dysfunction. Rates of hospitalisation and length of stay are similar for both patient populations.

No large-scale randomised-controlled trials have been conducted on therapy for diastolic dysfunction, so treatment should focus on:

1.) Controlling symptoms
2.) Treating underlying/precipitating factors
3.) Improving survival
4.) Reducing adverse outcomes.

Diuretics reduce pulmonary congestion (central blood volume) although these must be used cautiously to avoid severe hypotension.

ACE inhibitors (especially lipophilic) and Angiotensin II antagonists reduce blood pressure and LV hypertrophy.

β-blockers and CCBs reduce blood pressure, ischaemia and tachycardia (and possibly reduce left ventricular hypertrophy). Slowing heart rate prolongs filling time.

Digoxin should be used with caution in atrial fibrillation. Electrical conversion is suggested to restore sinus rhythm. β-blockers (especially sotalol) are recommended to control recurrence but the use of amiodarone is controversial.

References:
Testimonial – β-blocker Use
Clinical Experience with Metoprolol and Spironolactone for Patients with Congestive Heart Failure

By Bruce Arroll, Trust Health Care, Manurewa. Ph: 2676883

I had been aware for some time that β-blockers were helpful for patients with congestive heart failure. My understanding was that it was considered necessary to start patients on this while in hospital. I was also aware that my patients who had been hospitalised with congestive heart failure were not being put on β-blockers either at the time of discharge (understandably as they would have been symptomatic) nor on follow up.

I read an article by Dr Rob Doughty in the New Zealand Medical Journal 9th Feb 2001 volume 114 pages 36-8 where he suggested commencing β-blocker therapy in an outpatient clinic with an experienced physician.

On discussing this with some cardiologists it seemed a feasible activity to undertake in general practice with patients sitting in the waiting room for 2-3 hours so that they could be monitored and have their symptoms pulse and blood pressure checked before going home. This consisted of asking the patients to come to the clinic at 8.30 am without having taken their medication. They were then given their medication (either a higher dose of ACE inhibitor, small dose of metoprolol succinate 12 mg (i.e. half a 23.75 mg tablet) or the 25 mg tablet of spironolactone. They were checked after 2-3 hours and then went home. Subsequently they increased their dose at home and came in to get their symptoms, pulse and blood pressure checked.

One of the patients who was started on metoprolol had an echocardiogram performed after she had started on metoprolol. The echo showed diastolic dysfunction (rather then systolic dysfunction) which is where the heart is contracting too fast to enable the ventricle to be filled with blood. One of the treatments for this ironically is a β-blocker although it is not known if this prolongs life. Thus my patient was already on appropriate therapy.

contin . . /
Results.
None of the 7 patients had any problems in the waiting room. The major limiting factor was a low systolic blood pressure and if they were getting symptomatic or their systolic pressure was getting near 100mmHg I ceased increasing their medication. One patient (who had been on atenolol) subsequently developed bronchospasm and had her metoprolol stopped. One patient on spironolactone had an elevation in her serum potassium and had to have her spironolactone stopped. None of the patients felt worse by this treatment and I felt professionally satisfied that I was providing optimal therapy for my patients.

Conclusion.
Metoprolol, spironolactone, and high dose ACE inhibitors, represent an enormous benefit to our patients with congestive heart failure in terms of the ability to improve quality and length of life. These are therapies that can readily be commenced on most patients with congestive heart failure in general practice. I would be happy to discuss this with any GP who wished to call me.
QUESTIONS: Management of congestive heart failure

This quiz is mainly based on A Guideline for the Management of Heart Failure: Doctor’s Guide, by the New Zealand Heart Foundation, December 2001, with which you have been provided. A few of the answers come from the original studies or other sources and these references will be highlighted.

Question 1.
Mr Gregory is a 75 year old retired engineer who has been under your care for several years. He is has a history of emphysema and still smokes about 10 cigarettes a day. He enjoys a gin and tonic before dinner plus the occasional stout. He otherwise keeps good health, plays bowls regularly and manages to do the gardening. He lives with his 70 year old wife who is fit and well.

Mr Gregory comes to see you on Monday morning with a history of recent weight gain of about 7 or 8 kilograms, feeling increasingly tired and worsening short of breath on exertion. While he is able to walk from the living room to the kitchen and bathroom, he has been finding it increasingly difficult to climb the stairs to his bedroom and for the past week he has been sleeping sitting in an armchair in the living room.

On examination: weight 90 kg; height 185 cm (BMI = 26); BP 150/90; HS S3 present; P regular 80/min; basal crepitations in the chest; pitting oedema ankles.

Given that he has at least two symptoms or signs, you diagnose congestive heart failure (CHF). Please refer to the New York Heart Association (NYHA) Functional classification on page 7 of the Guidelines.

Mr Gregory’s (NYHA) classification is:

[a] Class I
[b] Class II
[c] Class III
[d] Class IV

Question 2.
You would like to admit Mr Gregory to sort out the management of his CHF but he insists that he does not want to go to hospital. You conduct an ECG which shows LVH but no signs of ischaemia and he is in sinus rhythm.

Your surgery has radiology and laboratory facilities next door. You order an immediate chest x-ray and laboratory investigations: Which of the following are recommended for patients with symptoms or signs of heart failure? (Please note: more than one answer may be correct.)

[a] Full blood count
[b] ESR
[c] Thyroid function tests (T4, TSH)
[d] Serum creatinine and electrolytes
[e] Liver function tests
[f] Serum albumin
Question 3.
With Mr Gregory’s consent, you invite his wife in from the waiting room and discuss his further management with them both. You explain that his symptoms are caused by heart failure and discuss his probable prognosis. You inform them that there are a number of things that can be done to improve his condition and his life expectancy. These include life-style changes he can make himself as well as drug treatment. You explain that it will take a little while to find the best combination and dose of drugs and that it is important that he follows your instructions carefully.

Life-style changes you advise include: (Please note: more than one answer may be correct.)

[a] Stopping smoking
[b] Spending the next week resting in his chair or in bed and not exerting himself.
[c] Daily weighing
[d] Increasing his alcohol intake, especially regular red wine, to improve his HDL levels.
[e] Start a low-salt diet and avoid excessive fluid intake.

Question 4.
You see Mr Gregory again that afternoon to review his investigation results. The chest x-ray shows an enlarged heart and increase in upper lobe vasculature consistent with your diagnosis of CHF. All the blood test results are normal. Which of the following investigations is/are also indicated? (Please note: more than one answer may be correct.)

[a] Exercise ECG
[b] Holter monitor
[c] Echocardiogram

Question 5.
You write a referral for Mr Gregory to have an echocardiogram at his local public hospital, enlist your practice nurse to reinforce your messages about daily weighing, low salt, stopping smoking and getting regular exercise, and tell him you will see him again to start drug treatment once you have the result of the echo. This is the correct management for Mr Gregory.

[a] Yes
[b] No

Question 6.
You tell Mr Gregory that you want to start drug treatment right away. Your first step is to prescribe:
[a] A loop diuretic such as frusemide (for example, Diurin) 40mg mane.
[b] A loop diuretic plus potassium supplement (for example, Slow-K): frusemide 40mg plus Slow-K one mane.
[c] An ACE Inhibitor such as enalapril (for example, Enahexal) 2.5 mg mane.
[d] Loop diuretic plus ACE inhibitor: frusemide 40 mg mane plus enalapril 2.5mg bd (twice daily).
[e] Loop diuretic plus ACE inhibitor plus potassium supplement: frusemide 40 mg, enalapril 2.5mg bd (twice daily) and Slow-K one mane.
**Question 7.**
You therefore start Mr Gregory on frusemide 40mg mane and enalapril 2.5mg bd. He has a home BP monitor and also scales to conduct daily weighing. You call him at home a couple of weeks later and he says he is feeling a little better. His exercise tolerance has slightly improved, he has lost 2 kg in weight and his BP is 140/85. You explain that you want to increase his ACE-I dose to the appropriate level for CHF treatment. Which of the following are safe options? (Please note: more than one answer may be correct.)

[a] Bring him into the surgery, give him a single dose of 5mg enalapril (in order to titrate up to 5mg bd), ask him to wait 2 to 3 hours in waiting room and check his BP and pulse.
[b] Allow him to take 5mg enalapril at home with instructions regarding self-monitoring of BP and pulse.
[c] Allow him to take 5mg enalapril at home with instructions to come back in the afternoon for your practice nurse to check his BP and pulse.
[d] Allow him to take 5mg enalapril at home, warning him that he may feel a little light-headed but not to worry about it.

**Question 8.**
Which of the following will need to be monitored regularly? (Please note: more than one answer may be correct.)

[a] Full blood count
[b] Thyroid function tests
[c] Renal function and electrolytes
[d] Blood pressure.

**Question 9.**
Is enalapril 5mg bd an adequate dose for maintenance of ACE inhibition in CHF with a blood pressure of 120/80 and if the patient feels fine?

[a] Yes
[b] No

**Question 10.**
Over the next three weeks you titre Mr Gregory’s enalapril up to 10mg bd. One month after starting his treatment he is clinically stable. His basal crepitations and pitting oedema have resolved and his weight is back down to 82kg. Although he has to rest after every two or three steps he can now make it up the stairs and is back sleeping in his upstairs bedroom and has daily exercise including a quiet game of bowls twice a week. You classify him as NYHA Grade II. You commend him on his complete cessation of smoking. You explain that the combination of diuretic plus ACE-I is the ideal treatment for his heart failure. He will need to have his blood pressure and potassium and creatinine levels monitored regularly. Are additional drugs indicated at this stage?

[a] Yes
[b] No
Question 11.
You add metoprolol succinate (Betaloc CR) 23.75mg ½ of a tablet and keep him in the waiting room for two hours, monitoring his blood pressure and heart rate. Which symptoms and signs do you need to be alert for? (Please note: more than one answer may be correct.)

[b] Heart block.
[c] Hypertension.
[d] Atrial fibrillation
[e] Bronchospasm

Question 12.
Mr Gregory visits fortnightly to allow the dose of metoprolol succinate to be up-titrated. He withholds his morning dose on each occasion. What observations or tests need to be done following each dose increment?

[a] Check for signs of worsening congestion.
[b] Perform an ECG.
[c] Check for hypotension.
[d] Check for bradycardia.

Question 13.
The target dose of metoprolol succinate to aim for is:

[a] 47.5 to 95 mg daily
[b] 95 to 142.5 mg daily
[c] 142.5 to 190mg daily

Question 14.
Mrs Williams is a 64 year old Māori patient who transfers to your care. She was diagnosed with heart failure several years ago and has had worsening symptoms over the last couple of months. She is now largely house-bound and is dyspnoeic even at rest. She is finding it a struggle to walk to the toilet. Her current medications are frusemide 40mg and quinapril (Accupril) 20mg bd.

Should you consider adding spironolactone (for example, Spirotone) 25mg daily to her existing therapy?

[a] Yes
[b] No
**Question 15.**
Before starting the spironolactone you check her creatinine and electrolytes. Her serum creatinine is 0.19mmol/l and her potassium is 4.0mmol/l. After prescribing the spironolactone you arrange for her to have a repeat creatinine and electrolytes in:

[a] 3 to 4 days  
[b] 2 weeks  
[c] one month

**Question 16.**
After 4 days her creatinine is 0.26mmol/l and her potassium is 5.5mmol/l. You therefore:

[a] Stop the frusemide.  
[b] Maintain the same dose of spironolactone and repeat the blood tests in 3 days.  
[c] Stop the spironolactone.

**Question 17.**
As either an alternative to, or in addition to, spironolactone, which of the following can be considered to be added to existing therapy for patients with heart failure in sinus rhythm who remain symptomatic despite treatment with ACE-I and diuretics? (Please note: more than one answer may be correct.)

[a] Angiotensin II receptor antagonist such as losartan (*Cozaar*).  
[b] Digoxin (*Lanoxin*).  
[c] Calcium channel blocker such as diltiazem (*Dilzem*).  
[d] Non-Steroidal Anti-Inflammatory Drug (excluding aspirin).

**Question 18.**
Mrs Williams is in NYHA Class IV for 2 weeks. She is on ACE-I, frusemide and spironolactone, her renal function and potassium are okay and she has no ankle or sacral oedema. You could consider her for carvedilol (a recently listed β-blocker).

[a] Yes  
[b] No
QUIZ ANSWERS
ANSWERS: Management of congestive heart failure quiz

Question 1.
The correct is [c].

Reasoning:-
[a] This answer is incorrect. In Class I there is no limitation of physical activity and ordinary activity does not cause undue fatigue or dyspnoea.
[b] This answer is incorrect. In Class II there is slight limitation of physical activity. The patient is comfortable at rest but experiences some fatigue and dyspnoea with ordinary activity (for example, climbing a flight of stairs).
[c] This answer is correct. In Class III there is marked limitation of physical activity. While comfortable at rest, less than ordinary activity causes fatigue or dyspnoea. In Mr Gregory’s case he can walk around the ground floor of their flat but has great difficulty climbing stairs.
[d] This answer is incorrect. In Class IV the patient is unable to carry out any physical activity without symptoms and gets symptoms even at rest. As a rule of thumb, Class I is no symptoms; Class IV is symptoms at rest, and Classes II and III are in between.

Question 2.
The correct answers are [a], [c], [d] and [f]. Also refer to p9 of the Guidelines

Reasoning:-
[a] This is correct. Heart failure may be due to or aggravated by anaemia form the decreased oxygen-carrying capacity.
[b] This is incorrect. ESR has no direct relevance in the diagnosis of heart failure.
[c] This is correct. Heart failure may be due to or aggravated by hypo or hyperthyroidism.
[d] This is correct. Volume overload may be due to renal failure. It is also important to have base-line electrolyte values prior to treatment.
[e] This is incorrect. Liver function has no direct relevance in the diagnosis of heart failure.
[f] This is correct. Hypoalbuminaemia may lead to increased extravascular volume.

Question 3.
The correct answers are [a], [c] and [e].

Reasoning:-
[a] This answer is correct. The importance of cessation of smoking should be stressed and help offered to achieve this.
[b] This answer is incorrect. Regular exercise is important within his tolerance limits. Rehabilitative exercise training in patients with heart failure and moderate-to-severe left ventricular systolic dysfunction improves functional capacity and reduces symptoms. Sexual activities and possible coping strategies to deal with difficulties could also be discussed.
[c] This answer is correct. Drug treatment will be titrated to dry weight (in his case, 82kg).
[d] This answer is incorrect. Limited alcohol is indicated although he should be allowed to enjoy his daily G & T. Alcoholic cardiomyopathy is one of the three main causes of heart failure. In this situation (i.e. heavy drinking), alcohol needs to be avoided completely.
[e] This answer is correct. Dietary sodium should be restricted to under 3 grams per day, and preferably 2 grams (about ½ a teaspoon). In simple terms adding salt in cooking and at the table should be avoided, as should high salt foods. Excessive fluid intake should be avoided but fluid restriction is not advised unless he develops hyponatraemia. A non-salt diet, however, may result in hyponatraemia.

Non-pharmacological management can be found on pp10-12 of the Guidelines.
**Question 4.**  
The correct answer is [c].

**Reasoning:**
[a] This answer is incorrect. Given that Mr Gregory has no chest pain or signs of ischaemia, this investigation is not warranted. If the patient is treated as best as possible and there are still questions about the causes of the heart failure, an exercise ECG may be warranted.
[b] This answer is incorrect. Given that Mr Gregory has no evidence of cardiac arrhythmia, this investigation is not warranted.
[c] This answer is correct. An echocardiogram will allow you to differentiate between systolic and diastolic dysfunction and hence guide the appropriate medications to use. Coupled with Doppler Flow studies, it can be determined whether the primary abnormality is pericardial, myocardial or endocardial. The management of systolic dysfunction is well established whereas it is less clear for diastolic dysfunction. (Anonymous (1999). “Consensus recommendations for the management of chronic heart failure. On behalf of the membership of the advisory council to improve outcomes nationwide in heart failure.” American Journal of Cardiology 83(2A): 1A-38A). While management of chronic heart failure due to left ventricular systolic dysfunction has been the subject of considerable research in the past five years, there are very few studies on the treatment of patients with diastolic dysfunction. In diastolic dysfunction the left ventricular chamber is usually of normal size and empties normally but has impaired filling due to its inability to relax. Also refer to Diastolic dysfunction summary.

**Question 5.**  
The correct answer is [b]

**Reasoning:**
[a] This answer is incorrect. It may take several months to get an echocardiogram and it is important that Mr Gregory is started on medication right away to treat his CHF.
[b] This answer is correct. See [a].

**Question 6.**  
The correct answer is [d].

**Reasoning:**
[a] This answer is incorrect. All patients with heart failure due to systolic dysfunction should be considered for treatment with an angiotensin converting enzyme (ACE) inhibitor which should be initiated at a low dose. See Guidelines pp13, 14 for contraindications. A patient with a systolic blood pressure below 100mmHg may be at risk of hypotension when initiating an ACE inhibitor. Concomitant use of diuretic is usually required for the management of fluid overload. If the patient has diastolic dysfunction then ACE inhibitors and diuretics are still acceptable management.
[b] This answer is incorrect. See [a]. The patient should receive an ACE inhibitor and potassium supplementation is not indicated.
[c] This answer is incorrect. See [a]. ACE-Is available in NZ as of February 2002 need to be given twice daily for heart failure and probably twice daily for hypertension but the doses may be different.
[d] This answer is correct. See [a]
[e] This answer is incorrect. See [a]. If an ACE inhibitor is used with a loop diuretic then usually potassium replacement is not required, which runs the risk of hyperkalaemia.
**Question 7.**
The correct answers are [a], [b] and [c].

**Reasoning:**
[a] This answer is **correct**. Side-effects with ACE-inhibitors are common, especially dizziness due to hypotension, and cough. It is therefore important to monitor his blood pressure when initiating ACE-inhibitors: the Guidelines (p14) suggest systolic blood pressure of <90mmHg as a level for concern. This situation is the most cautious approach. However, depending on circumstances, you may feel that returning for practice-nurse monitoring or conducting self-monitoring are safe options and proceed this way. Monitoring of the evening dose by the GP will not be possible so you would have to rely on the patient to report symptoms. An increase in ACE-I dose may facilitate a reduction or elimination of diuretic.

[b] This answer is **correct**. See [a].

[c] This answer is **correct**. See [a].

[d] This answer is **incorrect**. See [a].

**Question 8.**
The correct answers are [c] and [d].

**Reasoning:**
[a] This answer is **incorrect**. Mr Gregory was not anaemic and his drug treatment is not expected to cause changes to his full blood count.

[b] This answer is **incorrect**. Mr Gregory was norm-thyroid and his drug treatment is not expected to cause changes to his thyroid function.

[c] This answer is **correct**. It is essential to monitor potassium and creatinine levels during diuretic use. If the creatinine goes up by a concerning amount it is worth stopping the ACE-I in case the patient has renal artery stenosis. It may be appropriate to refer the patient for evaluation of renal artery stenosis.

[d] This answer is **correct**. Hypotension can occur from use of ACE-inhibitors and also from possible over-diuresis or hyponatraemia.

**Question 9.**
The correct answer is [b].

**Reasoning:**
[a] This answer is **incorrect**. Higher doses of ACE-I are needed in the treatment of heart failure than those used for hypertension. High dose lisinopril (30mg) has been shown to be more effective than low dose (5mg) for reducing combined mortality and cardiovascular events. *(Packer, M., P. A. Poole-Wilson, et al. (1999). “Comparative effects of low and high doses of the angiotensin-converting enzyme inhibitor, lisinopril, on morbidity and mortality in chronic heart failure. ATLAS Study Group.” Circulation 100(23): 2312-8).* See the abstract of this paper provided. The recommended dose of enalapril is 10mg bd. (See Guidelines pp 13, 14 for dose recommendations.)

[b] This answer is **correct**. See [a]
**Question 10.**

The correct answer is [a].

Reasoning:-
[a] This answer is **correct**. β-blockers should be considered for all patients with heart failure due to systolic dysfunction who have mild to moderate symptoms and are clinically stable. The aim of this treatment is to improve survival and reduce hospitalisation. *(Hjalmarson, A., S. Goldstein, et al. (2000). “Effects of controlled-release metoprolol on total mortality, hospitalizations, and well-being in patients with heart failure: the Metoprolol CR/XL Randomized Intervention Trial in congestive heart failure” JAMA 283(10): 1295-302).* See the abstract of this paper provided.

[b] This answer is incorrect – see [a]. Mr Gregory may get added benefit from β-blockers as his symptoms indicate that he is now an NYHA Class II. Refer to Guidelines pp15 & 16.

**Question 11.**

The correct answers are [a], [b] and [e].

Reasoning:-
[a] This answer is **correct**. Worsening heart failure can occur from increasing congestion. This can be managed by increasing the diuretics and continuing the β-blocker if possible. Reports from experienced clinicians suggest that this is very uncommon.

[b] This answer is **correct**. β-blockers should be stopped. If a baseline ECG has not been performed, this would be the opportunity to check if heart block exists. If heart block of any grade (1st, 2nd or 3rd) is found, reconsider the use of β-blockers.

[c] This answer is incorrect. β-blockers may cause hypotension. In such cases in heart failure patients the dose should be decreased or stopped, particularly if the systolic drops markedly or symptoms of postural hypotension occur.

[d] This answer is incorrect. Atrial fibrillation is not a side effect or a contraindication to β-blocker use.

[e] This answer is **correct**. If bronchospasm occurs the β-blocker should be withdrawn. If the patient already has a diagnosis of asthma then β-blockers are contraindicated.

**Question 12.**

The correct answers are [a], [c] and [d].

Rationale:-
[a] This answer is **correct**. Each time the dose of β-blocker is increased, Mr Gregory should be watched for signs of worsening congestion, hypotension or bradycardia. Be suspicious of heart block if pulse rate is <50 bpm and do not attempt any further increase in β-blocker dose. Some patients may need observing for two hours after each dose increment (for example if they have relative hypotension).

[b] This answer is incorrect. An ECG would only be required if there was evidence of bradycardia.

[c] This answer is **correct**. See [a]

[d] This answer is **correct**. See [a]
Question 13.
The correct answer is [c].

Reasoning:-
[a] This answer is incorrect. If tolerated, the target dose of metoprolol to aim for is 142.5 to 190mg daily.
[b] This answer is incorrect. See [a].
[c] This answer is correct. See [a].

Carvedilol is to be listed in the New Zealand Pharmaceutical Schedule on 1st April 2002 with the following indications:

a) Approved where patients are already on an ACE inhibitor or Angiotensin II Antagonist with;
   1. Symptomatic heart failure NYHA functional class II-III who have been treated with metoprolol and are intolerant to metoprolol or have demonstrated a sub-optimal response to metoprolol; OR
   2. Symptomatic heart failure NYHA functional class III-IV or left ventricular systolic dysfunction with an ejection fraction of less than 35%.

The recommended dose of carvedilol is 25mg bid. (See Guidelines p16.) Only metoprolol succinate and carvedilol are both licenced and recommended for CHF.

Question 14.
The correct answer is [a].

Reasoning:-
[a] This answer is correct. Patients with severe heart failure (Class III or Class IV within the past 6 months) should be considered for the addition of spironolactone 25mg daily to their existing therapy. Contraindications to the initiation of spironolactone are serum creatinine >0.25mmol/l, K+ >5.0mmol/l. (See Guidelines p17.) Spironolactone, in addition to standard therapy, has been shown to substantially reduce the risk of both morbidity and death among patients with severe heart failure. (Pitt, B., F. Zannad, et al. (1999). “The effect of spironolactone on morbidity and mortality in patients with severe heart failure. Randomized Aldactone Evaluation Study Investigators.” New England Journal of Medicine 341(10): 709-17). See the abstract of this paper provided. Treatment with spironolactone will show a marked improvement in 2-4 weeks. This may allow a reduction in frusemide dose.
[b] This answer is incorrect. See [a]. Refer to p17 of the Guidelines.

Question 15.
The correct answer is [a].

Reasoning:-
[a] This answer is correct. Creatinine and electrolytes should be checked at 3-4 days, one week and one month after initiation and then as indicated by renal function or 6 monthly in stable patients. After 1 year, tests should be done 6 monthly. (The 6 monthly testing is used in the study referenced above from the NEJM and is not mentioned specifically in the Guidelines.)
[b] This answer is incorrect – see [a].
[c] This answer is incorrect. – see [a].
Question 16.
The correct answer is [c].

Reasoning:-
[a] This answer is incorrect. Either a creatinine >0.25mmol/l or a potassium >5.0mmol/l is a contraindication to continuing the spironolactone at that level. If K+ goes up to a level close to 5.0mmol/l, spironolactone can be given every second day. (The last sentence is from the study referenced above from the NEJM and is not mentioned specifically in the Guidelines.)
[b] This answer is incorrect. See [a].
[c] This answer is correct. See [a].

Question 17.
The correct answer is [b].

Reasoning:-
[a] This answer is incorrect. Angiotensin II blockers could be considered for patients intolerant of ACE-I but should not be considered as an adjunct to ACE-I. See Guidelines p18.
[b] This answer is correct. Digoxin can be considered for patients with heart failure and in sinus rhythm who remain symptomatic despite treatment with ACE-I and diuretics, with the aim of improving symptoms and preventing further deterioration. Digoxin does not prevent death but does reduce hospitalisation when compared with placebo. See Guidelines p18.
[c] This answer is incorrect. Calcium antagonists are not recommended in severe heart failure, and possibly in any form of heart failure. If a CCB is seen to be the only possible treatment for e.g. angina, it may pay to refer the patient. See Guidelines pp8 & 20.
[d] This answer is incorrect. NSAIDs should be avoided or used with caution as they may exacerbate CHF. It is unclear whether aspirin interferes with ACE-I benefit, but if the patient has underlying vascular disease (coronary, cerebral or peripheral) then low dose aspirin (75mg – 150mg daily) is recommended. See Guidelines pp8, 19 & 20.

Question 18.
The correct answer is [a].

[a]. This answer is correct. The PHARMAC indications include a listing of carvedilol for patients with NYHA Class III-IV or LV systolic dysfunction with an EF < 35% (see also answer to Question 13). Metoprolol is used in patients with NYHA Class II-III. As GPs have little experience with this medication, and the indications differ between the Heart Foundation Guidelines and PHARMAC’s listing, you may feel more comfortable collaborating with or referring to a cardiologist to initiate this medication. See Guidelines pp15 & 16.
[b]. This answer is incorrect. Preliminary results of the COPERNICUS trial (carvedilol prospective randomised cumulative survival) in patients with severe chronic heart failure who were free from marked oedema showed a reduction in mortality, all-cause hospitalisation, hospitalisation for cardiovascular reasons or for heart failure and days in hospital for any reason were also reduced. (Louis A, JG Cleland et al. (2001). “Clinical Trials Update: CAPRICORN, COPERNICUS, MIRACLE, STAF, RITZ-2, RECOVER and RENAISSANCE and cachexia and cholesterol in heart failure. Highlights of the Scientific Sessions of the American College of Cardiology, 2001”. European Journal of Heart Failure. 3(3): 381-7.)
4F. Randomisation letters sent to GPs
Re:  CME & HF Management

Dear Dr «lastname»

Thank you for agreeing to participate in this project. You have been randomly allocated to the Guidelines arm of the study. Please find enclosed your copy of the Heart Foundation’s Heart Failure guidelines and algorithms.

We would encourage you to read through the Guidelines and consider which aspects are relevant to you and the patients in your practice. Towards the end of January we will send you the RNZCGP Practice Review Activity Cycle 1 Summary Sheet, with notes to assist you complete it.

Once we have carried out the full audit – in 12 to 18 months’ time – we will offer you one of the other two forms of CME that we are evaluating.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Associate Professor, General Practice

encl:  Guidelines
       Algorithms

This study has received ethical approval from the Auckland Ethics Committee.
3 March 2003

Dr «firstname» «lastname»
«practice»
«address_1»
«address_2»
«address_3»

Re: CME & Heart Failure Management

Dear Dr «lastname»

Towards the end of last year you would have received a copy of the Heart Foundation Heart Failure Guidelines and Algorithms from us as part of the evaluation of different types of CME. We hope that you have had the opportunity to read the Guidelines and consider which aspects are relevant to your practice.

Enclosed is the RNZCGP Practice Review Activity Cycle 1 Summary Sheet, with notes to assist you in completing it. Also enclosed is a freepost envelope so that you can return the PRA Summary to us – we are undertaking the administrative work of copying the Summary sheet and returning a copy to you and forwarding a copy to the RNZCGP. The PRA would be made easier if you could classify/code or develop a list of patients with heart failure or suspected heart failure. We would like to offer you a CME payment of $100 + GST for the work involved in considering and identifying issues in the Guidelines and categorising heart failure patients.

For your interest, a Special Authority form for Dilatrend (carvedilol) is also included. This can be prescribed by general practitioners for patients who fulfil one of the three prescribing criteria.

We hope that you will be able to implement the relevant issues that you identified in the Guidelines. In 12 to 18 months time we will carry out a retrospective audit as part of the second cycle of the PRA. Completing both cycles of the PRA means that you will earn 30 MoPS points.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Associate Professor, General Practice

encl: RNZCGP PRA Summary Sheet
      Notes on how to complete the PRA
      Freepost envelope
      Requirements for invoice
      Dilatrend (carvedilol) Special Authority Application Form
Checklist for CME & HF Management Study:

1. Read through Guidelines and Algorithms and identify issues that are relevant to you and the patients in your practice.
2. Complete Practice Review Activity Summary Sheet cycle 1 (white sheet and refer to “Notes to assist . . ” on blue sheet).
4. Return invoice and PRA Summary Sheet in the free-post envelope.
5. Copy of PRA Summary Sheet is returned to you.
6. Review medical records and implement “Actions” as described by you on the Summary Sheet.
7. Beginning of 2004, study team contacts you to arrange for audit.
8. Summary of information returned, second PRA Summary Sheet completed.

This study has received ethical approval from the Auckland Ethics Committee.
Dear Dr,

Thank you for agreeing to participate in this project. You have been randomly allocated to the small-group session arm of the study. The small-group sessions will be run by Associate Professor Bruce Arroll, and a cardiologist from North Shore Hospital. There will be a maximum of 10 participants per session and this will be on a “first in first served” basis. The dates, times and locations are listed below and all are open to members of both IPAs that are participating.

<table>
<thead>
<tr>
<th>DATE</th>
<th>TIME</th>
<th>LOCATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday 14\textsuperscript{th} October</td>
<td>7pm</td>
<td>Integrated Primary Care Services, Snelgar House, Waitakere Hospital, 55-75 Lincoln Rd, Henderson</td>
</tr>
<tr>
<td>Thursday 31\textsuperscript{st} October</td>
<td>7pm</td>
<td>Department of General Practice &amp; Primary Health Care, 52-54 Grafton Mews, Grafton</td>
</tr>
<tr>
<td>Monday 4\textsuperscript{th} November</td>
<td>7pm</td>
<td>Department of General Practice, Grafton</td>
</tr>
<tr>
<td>Tuesday 5\textsuperscript{th} November</td>
<td>7pm</td>
<td>Integrated Primary Care Services</td>
</tr>
<tr>
<td>Thursday 7\textsuperscript{th} November</td>
<td>7pm</td>
<td>Integrated Primary Care Services</td>
</tr>
<tr>
<td>Monday 11\textsuperscript{th} November</td>
<td>7:30pm</td>
<td>Comprehensive Health Services, 26 William Pickering Drive, North Harbour</td>
</tr>
<tr>
<td>Tuesday 12\textsuperscript{th} November</td>
<td>7pm</td>
<td>Integrated Primary Care Services</td>
</tr>
<tr>
<td>Wednesday 13\textsuperscript{th} November</td>
<td>7:30pm</td>
<td>Comprehensive Health Services</td>
</tr>
<tr>
<td>Thursday 28\textsuperscript{th} November</td>
<td>7:30pm</td>
<td>Comprehensive Health Services</td>
</tr>
</tbody>
</table>

You may reserve your place by phone (3737 599 ext. 4450) or by e-mail (v.andersen@auckland.ac.nz) and we will send you a confirmation letter with a map of the location. Please note that if there are insufficient numbers enrolled for a session we will have to reschedule you to another meeting or arrange additional sessions. If you are unable to attend your selected session, please let us know as soon as possible so that we can reschedule you to a different session.
The payment for attendance will be $100 + GST and the invoice should be addressed to Victoria Andersen, Department of General Practice etc, once you have attended your session.
All the material required for the small-group meeting will be provided at the start of the session. Drinks and nibbles will also be provided.

Yours sincerely

Victoria Andersen  
PhD student

Bruce Arroll  
Associate Professor, General Practice

This study has received ethical approval from the Auckland Ethics Committee.
Dear Dr 

We did not see you at any of the CME evenings in October or November. Feedback that we have had from GPs who have attended the CME sessions indicates that they have found the discussions stimulating and informative. We would like to urge you to attend one of the sessions listed below:

<table>
<thead>
<tr>
<th>DATE</th>
<th>TIME</th>
<th>LOCATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tuesday 10 December</td>
<td>7:30pm</td>
<td>Department of General Practice &amp; Primary Health Care, 52-54 Grafton Mews, Grafton</td>
</tr>
<tr>
<td>Thursday 12 December</td>
<td>7:30pm</td>
<td>Comprehensive Health Services, 26 William Pickering Drive, North Harbour</td>
</tr>
<tr>
<td><strong>NB. No cardiologist at this session.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monday 16 December</td>
<td>7:30pm</td>
<td>Comprehensive Health Services, 26 William Pickering Drive, North Harbour</td>
</tr>
</tbody>
</table>

You may reserve your place by phone (3737 599 ext. 84450) or by e-mail (v.andersen@auckland.ac.nz) or by return fax (3737 006) and we will send you a confirmation letter with a map of the location if required.
Please note that if there are insufficient numbers enrolled for a session we will have to reschedule you to another meeting or discuss possible times for sessions in the New Year.

There will be payment for attending this CME evening – $100 + GST – which you can invoice us for.

All the material required for the small-group meeting will be provided at the start of the session. Drinks and nibbles will also be provided.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Associate Professor, General Practice

This study has received ethical approval from the Auckland Ethics Committee.
Dear Dr

Thank you for agreeing to participate in this project which is evaluating the effectiveness of CME modalities. One of the benefits of participating in this study is that it has been endorsed by the RNZCGP as a Practice Review Activity (30 MoPs points!) – we will do almost all the audit work for you.

You have been randomly allocated to the small-group session arm of the study. The small-group sessions will be run by Associate Professor Bruce Arroll, and a cardiologist from North Shore Hospital and you may attend either session.

<table>
<thead>
<tr>
<th>DATE</th>
<th>TIME</th>
<th>LOCATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday 10 February</td>
<td>7:30pm</td>
<td>Comprehensive Health Services, 26 William Pickering Drive, North Harbour</td>
</tr>
<tr>
<td>Thursday 13 February</td>
<td>7:30pm</td>
<td>Integrated Primary Care Services, Snelgar Building, Waitakere Hospital, Lincoln Rd, Henderson</td>
</tr>
</tbody>
</table>

You may reserve your place by phone (3737 599 ext. 84450) or by e-mail (v.andersen@auckland.ac.nz) or by return fax (3737 006). We will send you a reminder fax a couple of days prior to the session.

The payment for attendance will be $100 + GST once you have attended your session. All the material required for the small-group meeting will be provided at the start of the session. Drinks and nibbles will also be provided.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Associate Professor, General Practice
Re: CME & HF Management

Dear Dr «lastname»

Thank you for participating in this study. You have been randomly allocated to the Internet arm of this project. This involves accessing a web-site specifically designed for this study, with reading material, links to other sites, the Guidelines and Algorithms and case studies to work through. The web site is being hosted by the Continuing and Distance Education Centre of the Faculty of Medical and Health Sciences and can be accessed at:

www.cdec.auckland.ac.nz

Then click on LOGON and follow the instructions.

Your user name is: «firstnamelastname»
Your password is: chf2002

We urge you to visit the web site sooner rather than later so that you may put into practice aspects of heart failure management that are relevant to you and your patients before the Practice Review which is scheduled for the end of 2003/beginning 2004.

If you are having problems viewing the Guidelines and Algorithms, please contact us and we will send a paper version out to you.

Once you have completed the case studies, we invite you to send in an invoice to claim your usual CME payment of $100 + GST. Please refer to the notes provided regarding the information required on the invoice.

Access to the web-site will be available to you throughout the study period in case there are features of the course you would like to review. There will however only be one payment for completing the case studies!
Enclosed is the RNZCGP Practice Review Activity Cycle 1 Summary Sheet with notes on how to complete it. Please fill this in once you have worked through the web-site and return in the freepost envelope provided. The study team will be forwarding the summary sheets to the RNZCGP so that they are aware you are participating in our research programme.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Associate Professor, General Practice

encl:
RNZCGP PRA Summary Sheet
Notes on how to complete the PRA
Tips on Internet use
Requirements for invoice

**Checklist for CME & HF Management Study:**

1. Access web-site and work through material (see “Tips on Internet Use” on lilac sheet).

2. Complete Practice Review Activity Summary Sheet cycle 1 (white sheet and refer to “Notes to assist . . ” on blue sheet).


4. Return invoice and PRA Summary Sheet in the free-post envelope.

5. Copy of PRA Summary Sheet is returned to you.

6. Review medical records and implement “Actions” as described by you on the Summary Sheet.

7. Beginning of 2004, study team contacts you to arrange for audit.

8. Summary of information returned, second PRA Summary Sheet completed.
CME & HF Management  Tips on Internet Use

- If you are not familiar with using web sites, we will happily walk you through how to use it (during the day or in the evening), either over the phone or in person – do not hesitate to contact either:
  
  Victoria  3737 599 ext 84450  v.andersen@auckland.ac.nz
  
  or

  Bruce  3737 599 ext 86978  b.arroll@auckland.ac.nz

- Traffic on the University Intranet is greatest during the day (particularly during the afternoon), but much lighter in the evenings and at weekends and during holidays.

- Consider installing the latest version of Internet browser on your computer if specifications allow.

- Adobe Acrobat Reader is needed to read some files (download is free from: www.adobe.com).

- If you are having problems viewing the Guidelines and Algorithms, contact us and we will send a paper version out to you.
4G. PRA and information sheet
Notes to assist you in completing the Practice Review Activity Summary Sheet.

The activity was designed by the Department of General Practice & Primary Health Care, University of Auckland and your IPA/PHO.

The topic was chosen to follow on from the release of the new Heart Failure Guidelines by the Heart Foundation.

Data.

Date of collection. September.

This is an “Organisation” activity – return the summary sheet to us and we will forward it to the RNZCGP. Certificates are not longer issued. However, we do encourage you to create an informal summary of data for your own reference – see below.

Check. Describe the issues brought up by your CME that are key management issues. Refer to the objectives of the project, and consider any other issues that are relevant to your practice. (Your analysis of the data collected would be reviewing medical records/medication of established heart failure patients and identifying any patients that may require further investigation.)

Action. Assessing patient records and checking against the key management issues you identified.

Monitor. Describe how the process is working – e.g. is it difficult identifying patients? The second cycle will be done in 12 – 18 months’ time. This will be a full audit with retrospective analysis.

NB. All Summary Sheets will be sent to the RNZCGP to be processed – please use your discretion in varying the above instructions for the Data section.

DO NOT RETURN YOUR P.R.A. SUMMARY SHEET TO THE RNZCGP – SEND IT BACK TO THE DEPARTMENT OF GENERAL PRACTICE IN THE FREEPOST ENVELOPE PROVIDED.
Summary Sheet for a Practice Review Activity

Doctor's name: ___________________________________________________________

The activity was designed by, please tick appropriate box:

☐ RNZCGP
☐ Organisation e.g. IPA (name of organisation) ________________________________
☐ Individual (self)

Topic: Describe why you chose this topic (relevance, needs assessment, etc)?

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

First cycle (10 credits)

Data: Information collected.

Date of data collection: ______________________________

Please attach:

• a summary of data collected and

• if this is an organisation activity also attach a certificate of participation

Check: Describe any areas targeted for improvement as a result of the data collected.

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Action: Describe how these improvements will be implemented

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Review: Describe how well the process is working. When will you undertake a second cycle?

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
Summary Sheet for a Practice Review Activity

Doctor's name: ________________________________________________________________

Topic: _________________________________________________________________

Second cycle (20 credits)

Data: Information collected.

Date of data collection: __________________________

Please attach:

- a summary of data collected and
- if this is an organisation activity also attach a certificate of participation

Check: Describe any areas targeted for improvement as a result of the data collected.

________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________

Action: Describe how these improvements will be implemented

________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________

Review: Describe how well the process is working. Will you undertake another cycle?

________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________

Additional comments:

________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________
4H. Carvedilol Special Authority application form
DILATREND® SPECIAL AUTHORITY APPLICATION FORM

Applications for Dilatrend® (carvedilol) 6.25mg, 12.5mg and 25mg tablets must be made on this form.

Before completing this form, you should consult the Special Authority Criteria in the Pharmaceutical Schedule or see below on this form. (PLEASE USE BLOCK CAPITALS OR A TYPEWRITER)

MEDICAL PRACTITIONER MAKING APPLICATION
Name: ____________________________
Address: __________________________________________________________

GENERAL PRACTITIONER (if not practitioner applying)
Name: ____________________________
Address: __________________________________________________________

PATIENT DETAILS
Surname: ____________________________
Firstname: __________________________
Address: __________________________________________________________
D.O.B: _______ / _______ / _______
NHI No: ____________________________
Sex: [ ] Male [ ] Female

SPECIAL AUTHORITY CRITERIA
If patient qualifies, please state the criteria under which application is made.

Special Authority – Retail Pharmacy
a) Approved where patients are already on an ACE inhibitor or Angiotensin II Antagonist and meet ONE or more of the following: (please tick)
   [ ] Symptomatic heart failure NYHA functional class II-III who have been treated with metoprolol and are intolerant to metoprolol or have demonstrated a suboptimal response to metoprolol; OR
   [ ] Symptomatic heart failure NYHA functional class III-IV; OR
   [ ] Left ventricular systolic dysfunction with an ejection fraction of less than 35%.

b) Applications can be made and prescriptions can be written either by a relevant specialist or general practitioner.
c) Approvals valid indefinitely;
d) Dispensed by retail pharmacy.

Note: where possible treatment should be initiated by or on the recommendation of a specialist.

I confirm that the above the details are correct and that in signing this form I understand I may be audited.

Signed: ____________________________ Registration No: ____________________________ Date: ____________________________

Failure to complete the form correctly will result in a delay in approval and the return of your application.
4I. **Re-contact letter, questionnaire and second round survey**

The re-contact letter was slightly different for each study arm (see examples).

The declaration had the study arm printed on the top left corner for easy identification.

The questionnaire had the intervention that the GP participated in pre-printed on the second page as a reminder. Each questionnaire was individually numbered with the GP’s study identification number.

The second round of the HF management survey was a different colour from the first for ease of identification. Each survey was individually numbered with the GP’s study identification number.
3 November 2005

Dr «firstname» «lastname»
«practice»
«address1»
«address2»
«address3»

Dear Dr «lastname»

Re: Heart Failure Management and CME

Approximately 2 ½ years ago you were invited to participate in self-directed-learning CME run by the Department of General Practice & Primary Health Care and the Goodfellow Unit. You would have received a copy of the Heart Foundation Heart Failure Guidelines and a copy of the RNZCGP Practice Review Activity Sheet and invited to fill out the PRA, detailing areas of heart failure management that you would review.

This was part 1 of a 30 MoPS points Practice Review Activity. We would now like to complete the second half of the PRA for you. If you did not fill in a first PRA, this will become Part 1.

This research, comparing the effectiveness of different types of delivery of education on the management of heart failure, will involve one of the research team contacting you in order to arrange times to visit the practice to help identify your heart failure patients. The identification of patients will require several visits. Eligible patients will then be sent information sheets and consent forms for their medical records to be accessed. Once the consent forms have been received, data will be collected from their medical records.

Enclosed with this letter are some items that need to be completed.
1. **Consent form (blue paper).**
This is a requirement by the Auckland Ethics Committee which has given this project Ethics approval (#2001/280). Only one needs to be filled out per practice, and the signatory needs to either be the senior partner or practice manager. This is to be returned to the University of Auckland. A freepost envelope is attached.

2. **Survey (pink paper).**
This is a repeat of the survey you completed at the start of the project. Please complete and return in the freepost envelope.

3. **Questionnaire (lilac paper).**
Please complete the questionnaire as best you can and return them in the enclosed freepost envelope.

If you have any questions, please contact Victoria on 3737 599 ext 84450 or v.andersen@auckland.ac.nz.

Yours sincerely

Victoria Andersen  
PhD student

Bruce Arroll  
Professor & HoD,  
General Practice & Primary Health Care

Enclosed:
General Practitioner Survey  
General Practitioner Questionnaire  
Summary of CME & HF Management Study  
Declaration by Senior Partner or Practice Manager  
3 Freepost Envelopes

This study has received ethical approval from the Northern X Ethics Committee.
20 August 2004

Dr «firstname» «lastname»
«practice»
«address_1»
«address_2»
«address_3»

Dear Dr «lastname»

Re: Heart Failure Management and CME

Approximately 20 months ago you participated in a small-group session run by the Department of General Practice & Primary Health Care. This was part of a 30 MoPS points Practice Review Activity. We would now like to complete the second half of the PRA for you.

This will involve one of the research team contacting you in order to arrange a time to visit the practice to help identify your heart failure patients who would then be sent information sheets and consent forms for their medical records to be accessed. Once the consent forms have been received, data will be collected from their medical records.

Enclosed with this letter are some items that need to be filled out.

1. Consent form (blue paper). This is a requirement by the Auckland Ethics Committee which has given this project Ethics approval (#2001/280). **Only one needs to be filled out per practice**, and the signatory needs to either be the senior partner or practice manager. This is to be returned to the University of Auckland. A freepost envelope is attached.

2. Survey (pink paper). This is a repeat of the survey you completed at the start of the project.

3. Questionnaire (lilac paper).
Please complete the survey and questionnaire and return them in the enclosed freepost envelope.

If you have any questions, please contact Victoria on 3737 599 ext 84450 or v.andersen@auckland.ac.nz.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Associate Professor, General Practice

This study has received ethical approval from the Auckland Ethics Committee.
12 August 2005

Dr «firstname» «lastname»
«practice»
«address1»
«address2»
«address3»

Dear Dr «lastname»

Re: Heart Failure Management and CME

Approximately 2 years ago you were invited to participate in an Internet-based CME session run by the Department of General Practice & Primary Health Care and the Goodfellow Unit. The content included a series of case studies about the management of heart failure. This was part 1 of a 30 MoPS points Practice Review Activity. We would now like to complete the second half of the PRA for you. Or if you did not complete a PRA, this will become the first round of the PRA. (It does not matter if you did not visit the Internet site.)

This research will involve one of the research team contacting you in order to arrange times to visit the practice to help identify your heart failure patients. The identification of patients will require several visits. Eligible patients will then be sent information sheets and consent forms for their medical records to be accessed. Once the consent forms have been received, data will be collected from their medical records.

Enclosed with this letter are some items that need to be filled out.

1. Consent form (blue paper). This is a requirement by the Auckland Ethics Committee which has given this project Ethics approval (#2001/280). Only one needs to be filled out per practice, and the signatory needs to either be the senior partner or practice manager. This is to be returned to the University of Auckland. A freepost envelope is attached.
2. Survey (pink paper). This is a repeat of the survey you completed at the start of the project.

3. Questionnaire (lilac paper).

Please complete the survey and questionnaire and return them in the enclosed freepost envelope.

If you have any questions, please contact Victoria on 3737 599 ext 84450 or v.andersen@auckland.ac.nz.

Yours sincerely

Victoria Andersen
PhD student

Bruce Arroll
Professor & HoD,
General Practice & Primary Health Care

Enclosed:
General Practitioner Survey
General Practitioner Questionnaire
Summary of CME & HF Management Study
Declaration by Senior Partner or Practice Manager
2 Freepost Envelopes

This study has received ethical approval from the Auckland Ethics Committee.
Survey: Heart Failure Management.

1. What changes in heart failure management have you heard of in the past 3 years?
   (e.g. Investigations Medications Other management)

If you HAVE answered QUESTION 1 go to QUESTION 3.
If you have NOT answered QUESTION 1 go to QUESTION 2 & 2a.

2. In the past 3 years, have you made any changes in the way you manage your heart failure patients? (Please circle) YES NO
If YES, what sort of changes?

2a. Has the hospital made any changes in the management of your heart failure patients? (Please circle) YES NO
If YES, what sort of changes?

IF you have answered Question 2 & 2a, finish the questionnaire here and return it.
3. Where did you hear about these changes?
(Note: Consider the points described in Question 1.) (Consider the following sources e.g. colleagues, IPA meetings/cell groups, journal articles, e-journals, Internet sites, drug companies.)

4. Which of these changes have you implemented?
(Note: Consider the points described in Questions 1 & 3.)

5. What proportion of your heart failure patients have you changed the management of?

6. Have you had any problems or concerns with these patients during these changes in management?  
   YES  
   NO
If YES, what sort of problems have you encountered?
7. Has the hospital made any changes in the management of your heart failure patients? (Please circle) YES NO

If YES, what sort of changes?

Additional Information. (Please number question.)

Thank you.

Please return the survey in the free post envelope provided, or if it is missing to:

FREEPOST 122259, Victoria Andersen, Department of General Practice, Private Bag 92019, AUCKLAND.
General Practitioner Questionnaire:
Experiences of CME in the Management of Heart Failure.

The purpose of this questionnaire is to discover what you thought of the CME modality you participated in and what the outcomes of the CME were in your practice. This questionnaire is part of a study evaluating different CME modalities and how effective these are in changing general practitioner behaviour.

Neither your name nor the name of your surgery is written on this questionnaire. Only the principal investigator has access to the list of general practitioners who have participated in the questionnaire. Any identifying features will be removed before information is published. You are under no obligation to answer all of the questions.

Answers from the questions that ask for your opinion or your reasons for doing certain things help the researchers gain a picture of how different types of CME are viewed and how changes in disease management are implemented at practice level. There is room at the end of the questionnaire for longer answers or for any further comments you would like to make.

If you have any queries, please contact Victoria Andersen at the Department of General Practice and Primary Health Care, Faculty of Medical and Health Sciences, ph: 3737 599 ext. 84450, fax 3737 624 or e-mail v.andersen@auckland.ac.nz

Thank you for your time.

Victoria Andersen
PhD Student
Principal Investigator, CME in heart failure management in general practice.
1.) Which CME modality did you use? ________________________________

1a.) How long did you spend on this? ________________________________

2.) What did you like about this modality? ____________________________

3.) What did you dislike about this modality? ________________________
4.) How could it have been improved? __________________________________________

___________________________________________________________________________

___________________________________________________________________________

___________________________________________________________________________

___________________________________________________________________________

___________________________________________________________________________

___________________________________________________________________________

___________________________________________________________________________

5.) Did you have any questions that were not answered by the information you received?

   YES                              NO

   If YES, what were/are these questions? _______________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

6.) Has this education changed your heart failure management?  YES      NO

   If YES, in what ways has your management changed?

   If NO, what would make you change your management?

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________

   ___________________________________________________________________________
7.) How did you organise change?

8.) Did you add decision / prompt tools to your computerised records?  **YES**  **NO**
If **YES**, give examples.  
If **NO**, why not?

9.) Did you collaborate with colleagues (other general practitioners) at your practice?  **YES**  **NO**
If **YES**, give examples.
10.) Were nurses involved in management?     YES     NO
If YES, give examples.

11.) Have you done any additional reading?     YES     NO
If YES, give examples.

12.) Have you had any further discussions with colleagues about the management of heart failure?     YES     NO
If YES, were the colleagues from your practice another practice both?
What issues did you discuss?
12a.) Were these discussions helpful?  
YES  NO

If YES, in what ways were the discussions helpful?

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

13.) Have you had any additional CME on heart failure diagnosis and management?  
YES  NO

Demographics.
14.) Are you:  
MALE  FEMALE

15.) What sort of practice do you work in  
SINGLE  GROUP

15a.) If in a GROUP, how many general practitioners (full and part-time) ___________

16.) Which year did you qualify as a general practitioner? ________________

Additional information from questions (please number).
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
4J. Patient mail-out pack

- Letter from GP
- Participant information sheet (double sided)
- Participant consent form.

The participant information sheet and participant consent form were colour coded by study arm for easy identification on their return.

Pale blue = guideline study arm
Pale pink = small group study arm
Light yellow = Internet study arm.

All participant information sheets and consent forms contained the same information. Each batch of consent forms for a particular practice had the GPs study ID numbers on the lower right hand corner.
16 January 2006

Dear

Recently I participated in a study that is being carried out by members of the Auckland Medical School who are doing research on different ways of delivering medical education to GPs. They now need to collect data to find out which type of education has been most effective. I support this study and would like to invite you to participate in it.

The researchers would like to have access to your medical records in order to take notes about certain drugs you may have been prescribed and tests you may have had done. They are strictly bound by The Privacy Act and cannot look at your medical records without your consent. Any information they receive is confidential and your name or any other identifying details will not be in any reports published by the research team.

You are not obliged to take part in this study. Your decision will not in any way effect your current or future healthcare. This study has received ethical approval from the Auckland Ethics Committee.

Please read the accompanying information sheet. If you wish to take part in this study, fill out the reply form and post in the freepost envelope as soon as possible.

Yours sincerely
Evaluation of Educational Modalities in General Practice

PARTICIPANT INFORMATION SHEET

Principal Investigator.
Victoria Andersen, PhD student, Department of General Practice and Primary Health Care, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland. Ph: (09) 3737 599 ext 84450. Fax: (09) 3737 624. E-mail: v.andersen@auckland.ac.nz

Supervisor.
Dr Bruce Arroll, Professor & HoD, Department of General Practice and Primary Health Care, School of Population Health, University of Auckland. Ph: (09) 3737 599 ext 86978. Fax: (09) 3737 624. E-mail: b.arroll@auckland.ac.nz

Introduction.
You are invited to participate in this study which will investigate how general practitioners respond to different types of education. You are under no obligation to be involved in this research. However if you do decide you would like to take part, please fill out the accompanying consent form and return it within 14 days using the stamped addressed envelope. If after that period of time the signed consent form has not been received the practice nurse at your doctor’s surgery will contact you to check whether you wish to be involved or not. If you choose not to take part, this will not affect your future health care. If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason and this will in no way affect your continuing or future health care.

Study Details.
The study aims to evaluate how different types of education for general practitioners affect their management of certain conditions. Your doctor is participating in this study and has been asked to select specific patients that he/she treats.

Continued over the page . . .
We are hoping to recruit close to 2000 patients in the Waitemata Health Area and would need to look at your medical records soon and make notes of medications you may have been prescribed and tests that you might have had done. Information from your records will be collected by one of the study team.

The data from your records will be used to assess how effective the different types of medical education have been in changing your doctor's management of heart conditions.

You do not need to make any special visits to your doctor or to the study team. You do not have to attend any lectures or seminars. There are no experimental medications that we are asking you to test.

Details from your medical records are confidential. No material which could personally identify you will be used in any reports on this study. Any data collected will be identified only by a code number (e.g. your file number), making it impossible to identify you without contacting your doctor's surgery. Only the person conducting the study in Auckland will have access to this data once it has been collected. Data will be stored for 10 years as required by the Ethics Committee guidelines.

Please feel free to ask any questions about the research project. Contact your doctor or Victoria Andersen ph: (09) 3737 599 ext. 84450, e-mail v.andersen@auckland.ac.nz

If you have any queries about your rights as a participant in this study, you may wish to contact a Health Advocate, Auckland ph: 623 5799 or Northland to Franklin ph: 0800 555 050.

This study has received ethical approval from the Northern X Ethics Committee.

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.

If you have any questions about ACC, contact your nearest ACC office or the investigator.
Participant Consent Form for the Evaluation of Educational Modalities in General Practice.

This consent form will be held for a period of 10 (ten) years.

Researcher: Victoria Andersen
Department of General Practice and Primary Health Care
School of Population Health
Faculty of Medical and Health Science
The University of Auckland

I give consent for information regarding my heart condition and related medication to be gathered from my medical records.

YES ☐ NO ☐ Please print your name below.

I have been given and have understood an explanation of this research project. I have had opportunity to ask questions and have them answered. I understand that taking part in this project is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care. I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study. I understand that my general practitioner may be aware that I am taking part in this study.

I consent to take part in this research.

Signed: ________________________________

Name: ________________________________
(Please print) First name Last name Title (e.g. Mr, Mrs, etc)

Date: ________________________________

Contact phone number: ________________________________

Address: ________________________________

Ethnicity: ________________________________

This study has received ethical approval from the Northern X Ethics Committee
4K. Boston Score sheet and completion instructions for research assistants
# BOSTON SCORING SYSTEM - guide sheet.

<table>
<thead>
<tr>
<th></th>
<th>History</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>History</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Rest dyspnoea</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>Orthopnoea</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>Paroxysmal nocturnal dyspnoea</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>Dyspnoea on level</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>Dyspnoea on climbing</td>
<td>1</td>
</tr>
</tbody>
</table>

**Sub Score**

<table>
<thead>
<tr>
<th></th>
<th>Physical Examination</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>Physical Examination</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Heart rate 91 – 110/min (1) / &gt; 110/min (2)</td>
<td>1 / 2</td>
</tr>
<tr>
<td>10</td>
<td>Elevated JVP &gt; 6cm H₂O (2) / &gt; 6cm + hepatomegaly or oedema (3)</td>
<td>2 / 3</td>
</tr>
<tr>
<td>11</td>
<td>Rales Basilar (1) / &gt; Basilar (2)</td>
<td>1 / 2</td>
</tr>
<tr>
<td>12</td>
<td>Wheezing</td>
<td>3</td>
</tr>
<tr>
<td>13</td>
<td>S₁, gallop</td>
<td>3</td>
</tr>
</tbody>
</table>

**Sub Score**

<table>
<thead>
<tr>
<th></th>
<th>Chest X-ray</th>
<th>Score</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>Chest X-ray</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Alveolar pulmonary oedema</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Interstitial oedema</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Bilateral pleural effusion</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Cardiotoracic ratio ≥ 0.5</td>
<td>3</td>
<td>CTR =</td>
</tr>
<tr>
<td>20</td>
<td>Upper–zone flow redistribution</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

**Sub Score**

<table>
<thead>
<tr>
<th></th>
<th>TOTAL</th>
<th>GRADE</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>TOTAL</td>
<td>GRADE</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th></th>
<th>ADDITIONAL MEASUREMENTS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>23</td>
<td>ADDITIONAL MEASUREMENTS</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>Response to diuretics</td>
<td>YES</td>
</tr>
<tr>
<td>25</td>
<td>Echocardiography</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>BNP / NT pro—BNP</td>
<td></td>
</tr>
</tbody>
</table>
Additional Measurements.

15 Chest X-ray | Score | Date:
16 Alveolar pulmonary oedema | 4 |
17 Interstitial oedema | 3 |
18 Bilateral pleural effusion | 3 |
19 Cardiotoracic ratio ≥ 0.5 | 3 | CTR =
20 Upper-zone flow redistribution | 2 |

Sub Score

15 Chest X-ray | Score | Date:
16 Alveolar pulmonary oedema | 4 |
17 Interstitial oedema | 3 |
18 Bilateral pleural effusion | 3 |
19 Cardiotoracic ratio ≥ 0.5 | 3 | CTR =
20 Upper-zone flow redistribution | 2 |

Sub Score

15 Chest X-ray | Score | Date:
16 Alveolar pulmonary oedema | 4 |
17 Interstitial oedema | 3 |
18 Bilateral pleural effusion | 3 |
19 Cardiotoracic ratio ≥ 0.5 | 3 | CTR =
20 Upper-zone flow redistribution | 2 |

Sub Score

15 Chest X-ray | Score | Date:
16 Alveolar pulmonary oedema | 4 |
17 Interstitial oedema | 3 |
18 Bilateral pleural effusion | 3 |
19 Cardiotoracic ratio ≥ 0.5 | 3 | CTR =
20 Upper-zone flow redistribution | 2 |

Sub Score

EF (%) | FS (%) | LVEDD/LVESD | Date

25 Echocardiography

EF (%) | FS (%) | LVEDD/LVESD | Date

25 Echocardiography

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date

26 BNP / NT pro—BNP

Value | Range | Date
**BOSTON SCORE for HEART FAILURE.**

<table>
<thead>
<tr>
<th>HEART FAILURE GRADING</th>
<th>TOTAL</th>
<th>0—4</th>
<th>No</th>
<th>N</th>
<th>5—7</th>
<th>Possible</th>
<th>P</th>
<th>8—12</th>
<th>Definite</th>
<th>D</th>
</tr>
</thead>
</table>

The Boston Score was developed in the 1970s, Echocardiography and BNP were not available. These have been added in as has response to diuretics to augment the diagnostic capability of the Boston Score.

It is possible that the signs and symptoms occur in the pre-intervention period and tests are only carried out after the intervention date (see also the Scenarios detailed at the end of this sheet). Work forwards. The symptoms (History and Physical Examination) would be expected to occur within a short time-frame e.g. over a few days, weeks or several months. However it is possible that a patient may not show any symptoms but be admitted to hospital for MI or in an acutely decompensated state, and relevant tests be carried out in hospital.

**Getting Started.**

Identify the educational intervention date which is available from the Principal Investigator. Review notes from approximately 5 years prior to this date for symptoms and any diagnostic tests that have been done.

1. **History.** These symptoms relate to breathing / shortness of breath / fluid overload and will be patient reported. Circle the relevant score. The symptoms may not be as directly described as below – it may be necessary to interpret, and be aware of shorthand. The symptoms could be recorded in the patient’s notes, letters to the GP or in discharge summaries.
2. Rest dyspnoea. Shortness of breath at rest i.e. when seated.
3. Orthopnoea. Shortness of breath on lying down. Notes may refer to requiring extra pillows at night.
4. Paroxysmal nocturnal dyspnoea. Waking up due to being short of breath.
5. Dyspnoea on level. Shortness of breath walking on the flat.
6. Dyspnoea on climb. Shortness of breath at some exertion e.g. up a slope.
7. Sub Score. The maximum score allowable is 4. Score all History symptoms as they appear, but one or combination higher than 4 is reduced to 4.

8. **Physical Examination.** Observations made by the GP / PN or external health professionals. The symptoms could be recorded in the patient’s notes, letters to the GP or in discharge summaries. Circle the relevant score.
9. Heart Rate. The score depends on the rate. 1 mark for 91 to 110 / minute (bpm); 2 marks >110 / minute.
10. Elevated Jugular Venous Pressure. > 6 cm H₂O (e.g. “+ 7” or “+ 7 cm” or “JVP +7”) scores 2 marks; including hepatomegaly or oedema, 3 marks. There is no score for oedema alone.
11. Rales. These can also be called crepitations, creps. Heard at the base of the lung, these score 1 mark. Any higher, scores 2 marks. Take care that these are not due to a respiratory infection.
12. Wheezing. Take care that this is not due to asthma, CORD etc.
13. S₃ gallop. The third heart sound may be listed as S₃, or HS 1 + 2 + 3. S₁ and S₂ are normal. There is no score for S₄.
14. Sub Score. The maximum score allowable is 4. Score all Physical Examination symptoms as they appear, but one or a combination higher than 4 is reduced to 4.
DIAGNOSTIC TESTS

GENERAL INFORMATION: There may be more than one of each test performed in the evaluation period. Record all including the dates – see additional sheet. Date format is dd/mm/yyyy.

15. Chest X-ray. Include the date or dates this was done. If there is mention of a CXR but no report or discussion, ask practice staff if it is possible to contact the hospital or cardiology clinic. However, make a thorough check of the hand-written, typed and electronic discharge summaries and radiology reports first. There may be more than one CXR – include details on the separate sheet.


17. Interstitial oedema. Hilar haziness / peribronchial cuffing / Kerley B lines.

18. Bilateral pleural effusion.

19. The cardiothoracic ratio (CTR) is usually represented by a fraction. Record the fraction or the percentage given in the report. However sometimes this is simply noted as ‘increased’ or ‘cardiomegaly’. Record these as ↑, or as cardiomegaly.


21. Sub Score. The maximum score allowable is 4. Score all CXR symptoms as they appear, but one or a combination higher than 4 is reduced to 4.

22. Total and Grade for Boston Score

Add the subscores, and write the grade.

<table>
<thead>
<tr>
<th>HEART FAILURE GRADING</th>
<th>TOTAL</th>
<th>0—4</th>
<th>No</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5—7</td>
<td>Possible</td>
<td>P</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8—12</td>
<td>Definite</td>
<td>D</td>
</tr>
</tbody>
</table>
Be aware that some diagnostic tests may have been carried out after the educational intervention. A low Boston Score may be obtained but then heart failure confirmed by Echo or BNP.

23. **Additional Measurements.**
As mentioned earlier, it is possible for a patient to have a low Boston Score prior to educational intervention but to then have heart failure confirmed by additional diagnostic testing later on. However a patient who has no Boston Score prior to the educational intervention but has a positive Echo / BNP / response to diuretic, after the intervention cannot be included in the study – there must be some indication of symptoms prior to the intervention. Record dates as dd/mm/yyyy.

24. **Response to diuretics.** This requires 2 visits / medical notes close together e.g. a few days apart, no more than a week, which demonstrate a weight loss of \( \geq 2 \text{kg} \) or a comment regarding response to diuretic.

25. **Echocardiograph.** This is the gold standard in diagnostic testing for heart failure. Record information such as;

- ejection fraction (EF), or
- fractional shortening (FS), or
- left ventricular end systolic diameter (LVESD) and left ventricular end diastolic diameter (LVEDD), or

**Ejection Fraction** is the most commonly reported (write as EF = xx%), and "the" variable of interest. (Heart failure is indicated by an EF of \( \leq 50\% \). Record whatever echo result is written as there is no need to try and make a clinical judgement.)

If EF is not reported, then **Fractional Shortening** (FS = xx%) can be used as a substitute, reported on its own. An FS of \( \leq 28\% \) is generally indicative of heart failure.

The next bullet point has 2 variables and both need to be recorded. **LVEDD** and **LVESD** are reported in mm or cm. These are the variables that are used to calculate the FS.
If there is mention of an Echo but no report or discussion, ask practice staff if it is possible to contact the hospital or cardiology clinic. Again, make a thorough check of the hand-written, typed and electronic discharge summaries first. There may be more than one Echo – record details on the separate sheet.

It is possible that EF is recorded when the patient undergoes Angiography. If this is the case, note ‘angiogram’ by the value.

26. BNP. Indicate (circle) which of these tests was used and the value, including upper and lower or indicative limits given by the lab. In computerised records this may be listed as “AH Bio Sendaway” or similar. Or it may be recorded with the laboratory test on hospital discharge letters. There may be more than one BNP test – record details on the separate sheet.

Administration.

27. Patient’s chart number. The clinic ID number for the patient.

    GP ID: note the GP that the patient attends most frequently – seen by clinical notes. NB: take care not to count scripts-only as consultations.

    Today’s date. Record as dd/mm/yyyy.

28. If a patient has a diagnosis of heart or left ventricular failure in the problem list, record “Diagnosis dd/mm/yyyy” (or whatever date is listed) here. The diagnosis could be in the Problem list, a note in the clinical records or an endorsement on a prescription or a letter written by the GP, a hospital discharge letter or a letter from a private specialist

29. Indicate where the ‘diagnosis’ was found. See above for explanation. If more than one source is found, the earliest one should be listed.

30. Write your initials here.

31. When did the symptoms occur e.g. give month or month range and year. Just a reminder that symptoms need to occur within a few months.
Additional notes:
If the clinical records note that the patient has diastolic failure, record this also on the top of the sheet with date if possible. Some dates may not be clear or precise – record as mm/yyyy or just yyyy. Always be aware of the possible side effects of drugs that could mimic symptoms, and of concomitant comorbidities.

Scenarios to consider:

- Patient who scores a ‘Possible’ or ‘Definite’ on the Boston Score. Continue collecting data on Echo, BNP, even if this is after the Intervention date.
- Patient who scores a ‘No’ (1 to 4 points) on the Boston Score but who also has a response to diuretic. Include this patient.
- Patient who scores a ‘No’ (1 to 4 points) on the Boston Score and has none (either they were not done or are negative / normal) of the Additional Measurements. This patient is excluded.
- Patient who has ‘Heart Failure’ listed in the Problem List but no objective evidence of heart failure. Head up a Boston Score Sheet, write ‘no evidence’ across the sheet, and include.
- Also check the notes to see if Heart Failure is written in (may not be in the Problem List).
- Also check any correspondence. This could be a hospital discharge letter, or a letter from a private specialist or a letter from the GP to an external recipient.
4L. Patient Demographics data collection sheet and completion instructions for research assistants
CME & HF Mgmt: Patient demographics.

**PRACTITIONER**
2. GP attributed to patient (study number)
3. Actual GP (study number)
4. Date of educational intervention

**PATIENT**
5. Patient chart number and / or computer file number
6. NHI
7. Date of birth
8. Gender
9. Recorded Ethnicity
10. Deceased? Yes | No | If Yes; DATE:

**DIAGNOSES**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>11.</td>
<td>HTN</td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>DM</td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>AF</td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>RHEUMATIC FEVER</td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>VALVULAR DISEASE</td>
<td></td>
</tr>
<tr>
<td>16.</td>
<td>IHD</td>
<td></td>
</tr>
<tr>
<td>17.</td>
<td>HF</td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>MI</td>
<td></td>
</tr>
</tbody>
</table>

19. **OTHER** (Refer to notes for Patient Demographics.)

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
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<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Patient Demographics Definitions.

DEMOGRAPHIC INFORMATION.
1. Patient identifier. This is the study number assigned to the patient.
   Date of data collection. Today’s date.

PRACTITIONER.
2. GP attributed to patient. Patient’s GP as given in practice records – list as study number.
3. Actual GP (study number). In practices of 2 or more GPs, the most frequently attended GP (>50% of visits) becomes the GP for that patient regardless of the attributed GP in the practice records. The Actual GP (study number) is the first 3 digits of the patient identifier.
4. Date of educational intervention. Held by the P.I. (Victoria), also refer to practice information. Data collection starts 18 months prior to this date and ends 18 months after this date.

PATIENT.
5. Patient chart number and/or computer file number. If the practice has both patient identifiers, record them by the appropriate headings.
6. NHI. This may be required if it is necessary to contact the hospital for records, diagnostic tests or checking duplicate records. State if not recorded.
7. Date of birth.
8. Gender.
9. Recorded ethnicity. State what is given in medical records. If not available, state ‘not recorded’.
10. Deceased. This may be indicated by a change in the patient’s clinical record number. This date should be after the date of 4. If not, please indicate.
DIAGNOSES.
As recorded in the medical records. This could be under “Problems” and / or “Resolved Problems”. Include date and any other relevant comments.

11. **HTN.** Has the patient ever been diagnosed with hypertension?

12. **Diabetes Mellitus.** Has the patient ever been diagnosed with either Type I or Type II?

13. **Atrial Fibrillation.** Has the patient ever been diagnosed with AF? Has cardioversion been attempted?

14. **Rheumatic Fever.** Has the patient ever suffered from Rheumatic Fever?

15. **Valvular Disease.** Has the patient ever suffered from valvular disease, and if so, which valve.

16. **Ischaemic Heart Disease.** Has this ever been diagnosed?

17. **Heart Failure.** Has the patient had this recorded in the Problems list?

18. **Myocardial infarction.** Date any occurrence and site.

19. **OTHER.** Include other conditions that may impact on heart failure e.g.
   - anaemia,
   - COPD or asthma (or other respiratory conditions),
   - obesity,
   - hypo-/hyperthyroidism,
   - cardiomyopathy,
   - smoking status,
   - alcohol dependency.
4M. Pre- and Post-Intervention data collection sheet and completion instructions for research assistants
Pre-intervention data.

Patient demographics

3. BP
   | Start | mmHg |
   | End   | mmHg |

4. HR
   | Start | bpm |
   | End   | bpm |

5. Height | m |

6. Weight
   | Start | kg |
   | End   | kg |

7. BMI
   | Start | kg/m$^2$ |
   | End   | kg/m$^2$ |

8. Electrolytes
   General
   | Cr   | mmol/l | Date |
   | Na$^+$ | mmol/l |     |
   | K$^+$  | mmol/l |     |

9. Spironolactone
   Date of initiation: 
   | Prior to initiation | Yes | Don't know | Cr | mmol/l | K+ | mmol/l | Date |
   | Post initiation    | Yes | Don't know | Cr | mmol/l | K+ | mmol/l |     |
Pre-Intervention: 10. Medications

<table>
<thead>
<tr>
<th>ACE Inhibitor</th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continued</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes: GP H C</td>
</tr>
<tr>
<td>Stopped</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diuretic</th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continued</td>
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<td>No</td>
</tr>
<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes: GP H C</td>
</tr>
<tr>
<td>Stopped</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>β–Blocker</th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continued</td>
<td>Yes</td>
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<td>Yes</td>
<td>If Yes: GP H C</td>
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<tr>
<td>Stopped</td>
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<td>No</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Spironolactone</th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
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</thead>
<tbody>
<tr>
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<td>No</td>
</tr>
<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes: GP H C</td>
</tr>
<tr>
<td>Stopped</td>
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<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
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<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
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<tbody>
<tr>
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<td>No</td>
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<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes: GP H C</td>
</tr>
<tr>
<td>Stopped</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

11. Other Medications

<table>
<thead>
<tr>
<th>Anti-arrhythmic</th>
<th>β– blocker</th>
<th>Nitrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digoxin</td>
<td>Ca²⁺ channel</td>
<td>Lipid lowering</td>
</tr>
<tr>
<td>Anti-platelet</td>
<td>DHP</td>
<td>NSAID</td>
</tr>
<tr>
<td>Anti-coagulant</td>
<td>Other</td>
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</table>
### Post-intervention data.

#### Patient demographics

3. **BP**

<table>
<thead>
<tr>
<th></th>
<th>Start</th>
<th>mmHg</th>
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</thead>
<tbody>
<tr>
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<td>End</td>
<td>mmHg</td>
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4. **HR**

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<thead>
<tr>
<th></th>
<th>Start</th>
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<tr>
<td></td>
<td>End</td>
<td>bpm</td>
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</table>

5. **Height**

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<tr>
<th></th>
<th>m</th>
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6. **Weight**

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<tr>
<th></th>
<th>Start</th>
<th>kg</th>
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<tr>
<td></td>
<td>End</td>
<td>kg</td>
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7. **BMI**

<table>
<thead>
<tr>
<th></th>
<th>Start</th>
<th>kg/m²</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>End</td>
<td>kg/m²</td>
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8. **Electrolytes**

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<thead>
<tr>
<th></th>
<th>Cr</th>
<th>mmol/l</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Na⁺</strong></td>
<td>mmol/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>K⁺</strong></td>
<td>mmol/l</td>
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<td></td>
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</table>

9. **Spironolactone**

#### Date of initiation:

<table>
<thead>
<tr>
<th>Prior to initiation</th>
<th>Yes</th>
<th>Don’t know</th>
<th>Cr</th>
<th>mmol/l</th>
<th>Date</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>K⁺</td>
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<table>
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<tr>
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<th>Cr</th>
<th>mmol/l</th>
<th>Date</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>K⁺</td>
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Post–Intervention: 10. Medications

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<th><strong>ACE Inhibitor</strong></th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
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</thead>
<tbody>
<tr>
<td>Continued</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>H</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>Stopped</td>
<td>Yes</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th><strong>Diuretic</strong></th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
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</thead>
<tbody>
<tr>
<td>Continued</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
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<td>Yes</td>
<td>If Yes:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GP</td>
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<tr>
<td></td>
<td></td>
<td>H</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>Stopped</td>
<td>Yes</td>
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</table>

<table>
<thead>
<tr>
<th><strong>β–Blocker</strong></th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
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</thead>
<tbody>
<tr>
<td>Continued</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>H</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C</td>
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<tr>
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<table>
<thead>
<tr>
<th><strong>Spironolactone</strong></th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
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</thead>
<tbody>
<tr>
<td>Continued</td>
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<td>No</td>
</tr>
<tr>
<td>Initiated</td>
<td>Yes</td>
<td>If Yes:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GP</td>
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<td></td>
<td>C</td>
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<tr>
<td>Stopped</td>
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<table>
<thead>
<tr>
<th><strong>AT–II blocker</strong></th>
<th>Chemical Name:</th>
<th>Max. Dose (mg):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continued</td>
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<td>No</td>
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<td></td>
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<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>

11. Other Medications

<table>
<thead>
<tr>
<th><strong>Anti–arrhythmic</strong></th>
<th><strong>β– blocker</strong></th>
<th><strong>Nitrate</strong></th>
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<tr>
<td>Anti–platelet</td>
<td>DHP</td>
<td>NSAID</td>
</tr>
<tr>
<td>Anti–coagulant</td>
<td>Other</td>
<td></td>
</tr>
</tbody>
</table>
Pre- & Post-Intervention Data Collection Definitions:

1. **Patient identifier.** GP identifier + number assigned to patient by the study.
   - **Date of data collection.** Today’s date.

2. **Date of intervention.** This is the index date. Everything on or before this is ‘pre-intervention’ data, everything after this date is ‘post-intervention’ data. The time frame is 18 months on either side of the index date.

**PATIENT DEMOGRAPHICS.**

3. **Blood pressure recorded.** Value at start of the 18 months and at the end of the 18 months.

4. **Heart rate recorded.** Value at start of the 18 months and at the end of the 18 months.

5. **Height recorded.**

6. **Weight recorded.** Value at start of the 18 months and at the end of the 18 months.

7. **BMI recorded.** Value at start of the 18 months and at the end of the 18 months. (This is not necessary if the weight and height have already been recorded.)

8. **ELECTROLYTES.**
Collect these values as close to the end of the 18 months as possible.

9. **SPIRONOLACTONE.**
This has separate data requirements. Note the data of the first prescription. Double-check the date with any hospital records in case it was initiated in hospital. Identify the closest lab tests of electrolytes prior to the initiation (these may be the date of the prescription), and the closest lab tests after initiation. These may be on hospital records if the drug was initiated in hospital.
10. MEDICATIONS.
Information is needed on whether the drug is being prescribed. List the chemical name and the maximum dose achieved during the data collection period.

Note whether the drug was continued from previously (circle Yes or No).

Note whether the drug was initiated during that period (circle Yes or No). If Yes, circle whether the GP (GP), Hospital (H) or Cardiologist (C) made the initial prescription.

Note whether the drug was stopped during that period (circle Yes or No).

ACE Inhibitor. There are many on the market and some of those available have had supplier and therefore name changes over the years. The more common drugs are:

<table>
<thead>
<tr>
<th>Chemical Name</th>
<th>Brand Name/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>Capoten, Captohexal, Capril, Apo–Capoten</td>
</tr>
<tr>
<td>Cilazapril</td>
<td>Inhibace</td>
</tr>
<tr>
<td>Enalapril</td>
<td>Renitec, Enahexal, m–Enalapril, Apo–Enalapril</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>Prinivil</td>
</tr>
<tr>
<td>Perindopril</td>
<td>Coversyl</td>
</tr>
<tr>
<td>Quinapril</td>
<td>Accupril</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>Gopten, Odrik</td>
</tr>
<tr>
<td>Captopril + hydrochlorothiazide</td>
<td>Capozide</td>
</tr>
<tr>
<td>Cilazapril + hydrochlorothiazide</td>
<td>Inhibace Plus</td>
</tr>
<tr>
<td>Enalapril + hydrochlorothiazide</td>
<td>Co–Renitec</td>
</tr>
<tr>
<td>Lisinopril + hydrochlorothiazide</td>
<td>Prinzide</td>
</tr>
<tr>
<td>Quinapril + hydrochlorothiazide</td>
<td>Accuretic</td>
</tr>
</tbody>
</table>

Diuretic. Please note that Spironolactone is not to be counted as ‘Diuretic’ as it has a separate category. See Pharmaceutical Schedule for all diuretics.
β-blockers. Only those licensed for heart failure. These are:

<table>
<thead>
<tr>
<th>Chemical Name</th>
<th>Brand Name/s</th>
<th>Maintenance Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoprolol succinate</td>
<td>Betaloc CR</td>
<td>190 mg</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>Dilatrend</td>
<td>25mg BD (severe, mild-moderate &lt;85kg) 50mg BD (mild-moderate &gt;85kg)</td>
</tr>
</tbody>
</table>

Spironolactone. This is separate from the Diuretics

<table>
<thead>
<tr>
<th>Chemical Name</th>
<th>Brand Name/s</th>
<th>Maintenance Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spironolactone</td>
<td>Spirotone or Aldactone</td>
<td>25mg</td>
</tr>
</tbody>
</table>

Angiotensin II Receptor Blockers. Record regardless of whether the drug has been licensed for heart failure or not.

<table>
<thead>
<tr>
<th>Chemical Name</th>
<th>Brand Name/s</th>
<th>Maintenance Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candesartan</td>
<td>Atacand</td>
<td>32 mg</td>
</tr>
<tr>
<td>Losartan</td>
<td>Cozaar</td>
<td>50 mg</td>
</tr>
<tr>
<td>Losartan + hydrochlorothiazide</td>
<td>Hyzaar</td>
<td>50/12.5 mg (100/25 mg)</td>
</tr>
</tbody>
</table>

11. OTHER MEDICATIONS.
These are listed in the data collection sheets. Please tick what is prescribed. Refer to the accompanying list for drugs sorted by class, trade name and chemical name.

12. INITIALS
The initials of who is collecting the data.
4N. Current Management data collection sheet
Current Management Data.

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Chemical Name</th>
<th>Dose (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE Inhibitor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β –Blocker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spironolactone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AT–II blocker</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Other Medications

<table>
<thead>
<tr>
<th>Anti–arrhythmic</th>
<th>β– blocker</th>
<th>Nitrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digoxin</td>
<td>Ca²⁺ channel</td>
<td>Lipid lowering</td>
</tr>
<tr>
<td>Anti–platelet</td>
<td>DHP</td>
<td>NSAID</td>
</tr>
<tr>
<td>Anti–coagulant</td>
<td>Other</td>
<td></td>
</tr>
</tbody>
</table>

‡ To find the current medications it may be necessary to go backwards a few months from this date to find the prescription details. Note that not all drugs may be prescribed at the same consultation.
40. **Grouping of listed diagnoses and free text comorbidities**

The listed Diagnoses (numbered 11 to 18) in Patient Demographics (appendix 4L) and ‘Other’ (free-text co-morbidities) were reorganised into related groups after data entry.

**Diagnoses**

**12. DM**

Impaired glucose tolerance was also included.

**15. Valvular disease**

This included references to valvular disease or specific valves and also the following:

- Aortic stenosis
- Murmur
- Valve . . .
- Regurgitation
- Valve repair
- Endocarditis

**16. IHD**

Any references to Ischaemic Heart Disease were included as well as:

- Coronary Heart Disease
- Coronary Artery Disease
- CABG
- Angina
- Angioplasty / PTCA / stent

‘Other’ comorbidities (in bold includes the bullet points listed underneath):

**Rhythm Disturbances**

- Pacemaker
- Defibrillator
- Atrial ectopy
- Paroxysmal supraventricular tachycardia
- Bradycardia
- Heart Block / LBBB / RBBB

**Lipid Disorder**

- Hyperlipidaemia
- Dyslipidaemia
- Hypercholesterolaemia
- High cholesterol

**Impaired Renal Function**
- Acute renal failure
- Chronic renal failure / impairment / abnormality
- Reduced creatinine clearance

**Impaired Respiratory Function**
1. **COAD** / **COPD** / **CORD**, plus
   - Bronchiecstatis
   - Emphysema
   - Type II respiratory failure
   - Asbestos exposure
   - Right HF
   - Cor pulmonale

2. **Asthma** / **Bronchial asthma**

**Blood / Endocrine Disorders**
- Hypothyroid
- Hyperthyroid
- Anaemia

**Cardiomyopathy including**
- Alcoholic
- Dilated
- Congestive

**Alcohol mis-use**
- ‘Alcohol’ / Et-oh
- Dependency
- Alcoholism
- Ex – alcohol

**Smoker**: current or previous.

**Obesity**: where this was listed.
4P. Coding schedule for medications and doses

Data Coding Instructions.

1. Drug given?
   YES = 1
   NO = 0
   e.g. Any diuretic? Yes = 1    No = 0

2. Type of drug within that class.

ACE-Inhibitor

<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Trade name</th>
<th>CODE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benazapril</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Captopril</td>
<td>Capoten, Apo-Captopril</td>
<td>2</td>
</tr>
<tr>
<td>Cilazapril</td>
<td>Inhibace</td>
<td>3</td>
</tr>
<tr>
<td>Enalapril</td>
<td>Renitec, m-Enalapril</td>
<td>4</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>Prinivil</td>
<td>5</td>
</tr>
<tr>
<td>Perindopril</td>
<td>Coversyl</td>
<td>6</td>
</tr>
<tr>
<td>Quinapril</td>
<td>Accupril</td>
<td>7</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>Gopten, Odrik</td>
<td>8</td>
</tr>
</tbody>
</table>

ACE-Inhibitor + diuretic

<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Trade name</th>
<th>CODE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cilazapril with hydrochlorothiazide</td>
<td>Inhibace Plus</td>
<td>3A</td>
</tr>
<tr>
<td>Enalapril with hydrochlorothiazide</td>
<td>Co-Renitec</td>
<td>4A</td>
</tr>
<tr>
<td>Quinapril with hydrochlorothiazide</td>
<td>Accuretic</td>
<td>7A</td>
</tr>
</tbody>
</table>
3. Dose scale.

**ACE Inhibitor – most often used**

<table>
<thead>
<tr>
<th>Dose code</th>
<th>Captopril</th>
<th>Cilazapril</th>
<th>Enalapril</th>
<th>Quinapril</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>150 mg</td>
<td>5 mg</td>
<td>20 - 15 mg</td>
<td>20 - 15 mg</td>
</tr>
<tr>
<td>2</td>
<td>100 – 75</td>
<td>2.5 – 2.0</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>25 – 50</td>
<td>1.25 – 1.5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>12.5</td>
<td>0.5 – 1.0</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>5</td>
<td>6.25</td>
<td>0.25</td>
<td>1.25</td>
<td>1.25</td>
</tr>
<tr>
<td>9</td>
<td>Any dose over those in the first row.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**ACE Inhibitor – less frequently used**

<table>
<thead>
<tr>
<th>Dose code</th>
<th>Benazepril</th>
<th>Lisinopril</th>
<th>Perindopril</th>
<th>Trandolapril</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20 mg</td>
<td>4 mg</td>
<td>2 mg</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>1</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Any dose over those in the first row.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**ACE Inhibitor + Diuretic**

<table>
<thead>
<tr>
<th>Dose code</th>
<th>Cilazapril</th>
<th>Enalapril</th>
<th>Quinapril</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5 + 12.5 mg</td>
<td>20 + 12.5 mg</td>
<td>20 + 12.5 mg</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td>10 + 12.5</td>
</tr>
<tr>
<td>9</td>
<td>Any dose over those in the first row.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Code ACE inhibitor dose for ACE inhibitor + diuretic (3A, 4A, & 7A).  

---

4 Recommended dose from NZ Guidelines. Stated as TOTAL daily dose.
### Diuretics

<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frusemide</td>
<td>1</td>
</tr>
<tr>
<td>BFZ</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Frusemide</th>
<th>Dose Code</th>
<th>BFZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>240 – 200 mg</td>
<td>1</td>
<td>10 mg</td>
</tr>
<tr>
<td>160 – 120</td>
<td>2</td>
<td>7.5</td>
</tr>
<tr>
<td>100 – 80</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>60 – 40</td>
<td>4</td>
<td>2.5</td>
</tr>
<tr>
<td>&lt; 40</td>
<td>5</td>
<td>&lt; 2.5</td>
</tr>
<tr>
<td>Any dose over ‘1’</td>
<td>9</td>
<td>Any dose over ‘1’</td>
</tr>
</tbody>
</table>

All other diuretics are ‘1’ in the ‘Other diuretic’ column.

### β-Blocker

<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Trade name</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carvedilol</td>
<td>Dilatrend</td>
<td>1</td>
</tr>
<tr>
<td>Metoprolol Succinate</td>
<td>Betaloc CR</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Carvedilol</th>
<th>Dose code</th>
<th>Metoprolol Succinate</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 mg</td>
<td>1</td>
<td>190 mg</td>
</tr>
<tr>
<td>25 – 37.5</td>
<td>2</td>
<td>71.5 – 95</td>
</tr>
<tr>
<td>12.5</td>
<td>3</td>
<td>37.5 – 47.5</td>
</tr>
<tr>
<td>≤ 6.25</td>
<td>4</td>
<td>≤ 23.75</td>
</tr>
</tbody>
</table>
**Spironolactone**

Only one medication, no code

<table>
<thead>
<tr>
<th>Dose code</th>
<th>Spironolactone</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>75 – 100 mg</td>
</tr>
<tr>
<td>2</td>
<td>47.5 – 50</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
</tr>
<tr>
<td>4</td>
<td>12.5</td>
</tr>
</tbody>
</table>

**AT II Blocker or ARB**

<table>
<thead>
<tr>
<th>Chemical Name</th>
<th>Trade name</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candesartan</td>
<td>Atacand</td>
<td>1</td>
</tr>
<tr>
<td>Losartan</td>
<td>Cozaar</td>
<td>2</td>
</tr>
<tr>
<td>Losartan + hydrochlorothiazide</td>
<td>Hyzaar</td>
<td>2A</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Candesartan</th>
<th>Dose code</th>
<th>Losartan</th>
</tr>
</thead>
<tbody>
<tr>
<td>32 mg</td>
<td>1</td>
<td>50 mg</td>
</tr>
<tr>
<td>16</td>
<td>2</td>
<td>25 – 38</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>12.5 – 13</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>6.25</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Any dose over ‘1’</td>
<td>9</td>
<td>Any dose over ‘1’</td>
</tr>
</tbody>
</table>
4Q. **HF scoring systems**

Diagnosis of HF in general practice could be assisted with the use of practical and relevant system to record and classify significant signs, symptoms and the outcomes of diagnostic tests. HF scoring systems were reviewed for this thesis in order to employ a standard classification system across all practices participating in the study and also for the PhD student and research assistants who were identifying patients. In the first instance the criteria that are used also need to be sufficiently broad to encompass the different signs and symptoms that patients present with and that GPs are able to detect.

Even as early as 1992 researchers were calling for specific criteria for HF (in estimating severity) and the development of a scoring system that included information from:

- Clinical observations
- CXR
- Exercise capacity
- Echocardiography. (481)

All of these aspects need to be considered together as there are the following problems if they are considered as individual elements;

- History and physical examinations do not necessarily equate to the actual severity of the failing heart.
- CXR. This may not give an accurate picture of congestion in patients with severe long standing HF i.e. it may not exist.
- Exercise capacity. Not necessarily able to be predicted in all patients. Not necessarily a predictor of LV dysfunction and severity.
- LV Ejection Fraction. Clinical findings may not represent level of dysfunction. Ejection fraction does not necessarily indicate HF symptoms or exercise capacity. (481)

This appendix considers some of the major scoring systems (for use in primary care patients) that were published at the time the data collection was due to commence. All of the following were included in a 1997 review by Mosterd et al. of six HF scores. (482) (The WHO Criteria, while not tested in the review, is included here as it was used in the CASE study (260) and has been referenced by other papers used in this thesis).

The scoring systems were tested in patients who participated in the Rotterdam Study who had undergone echocardiography and answered the questions in the ‘Men born in 1913’ study, and had randomly selected patients in the different levels to participate in
the review. (The authors do indicate that the ‘men born in 1913’ was developed and tested only in men but that there is no evidence to indicate that the score would perform differently in women. Since there is a tendency for women to present with different symptoms, this theory does need to be tested.) One researcher (cardiologist) then classified all patients according to the six HF scoring systems using information from medical history, clinical examination, ECG, CXR and echocardiograms. (482) The cardiologist’s diagnosis was taken as the gold standard that the scoring systems were reviewed against. (482) The presence of definite and possible (definite + possible) HF was determined and the sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) are given for each scoring system. The values of PPV and NPV value are of more use in clinical practice as they give the probability that the test results provide the correct diagnosis. (482)

Another study prefaced the comparison of three scoring systems by the statement;

“Epidemiological studies in congestive heart failure have been hampered by the lack of uniform diagnostic criteria, relying instead on physician diagnosis of the disease.” (483)

The object of this study was to evaluate the relationship between the clinical diagnosis of HF against an objective measurement of systolic function and the results (HF present, possible or absent) of the three HF scoring systems were compared against LVEF. The authors do state that it was not a reference standard as such but had clinical significance as the most important predictor of prognosis in patients with CHD. EF values were graded as ‘low’ \( \leq 40\% \), ‘borderline’ 41 to 49\% and normal LVEF as \( \geq 50\% \). The majority of these patients were in-patients at the time of the study, having been referred for testing by radionuclide ventriculogram (1982 to 1983). The sensitivity and specificity are given for Framingham and Boston scores\(^b\). (483)

Criticisms of the above study is that it took place in a hospital setting and that only patients with a certain EF were classified as having HF, which excludes patients who have clinical evidence of HF.

In the following sections each of the diagnostic criteria are given and critiqued for their inclusions and exclusions and for their usefulness in primary care.

\(^b\) The Duke criteria (the third scoring system in this paper) are not reported on. These consist of \( S_3 \) gallop and cardiomegaly on CXR (CTR > 0.48). (484)
1. **WHO criteria**
The WHO criteria clearly set out the signs and symptoms and is far sighted enough to include causative factors. These are listed in table 4Q.i. (260)

Table 4Q.i. WHO criteria for HF diagnosis

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Dyspnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chronic fatigue</td>
</tr>
<tr>
<td></td>
<td>Oedema</td>
</tr>
<tr>
<td></td>
<td>Exercise intolerance</td>
</tr>
<tr>
<td>Signs</td>
<td>3rd or 4th heart sounds</td>
</tr>
<tr>
<td></td>
<td>Heart murmur</td>
</tr>
<tr>
<td></td>
<td>Cardiomegaly</td>
</tr>
<tr>
<td></td>
<td>Pulmonary crackles</td>
</tr>
<tr>
<td></td>
<td>Raised jugular venous pressure</td>
</tr>
<tr>
<td></td>
<td>Dependent oedema</td>
</tr>
<tr>
<td>Causative factors</td>
<td>Angina</td>
</tr>
<tr>
<td></td>
<td>Previous MI</td>
</tr>
<tr>
<td></td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Valvular heart disease / rheumatic fever</td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathy</td>
</tr>
</tbody>
</table>

The main omission from the criteria is any objective evidence of function, not even CXR, although the inclusion of cardiomegaly does suggest further testing. The third and fourth hear sounds may be difficult to detect by GPs.

The criteria become much more complex by the definition of HF from the combination of factors from above. HF was considered possible if the patient had:

- > 2 symptoms,
- > 2 signs,
- > 1 symptom + > 1 sign, or
- > 1 symptom + > 1 causative factor. (260)

2. **Framingham criteria**
The Framingham Study criteria have been called ‘sensible’ i.e. they have face validity (256) and they have developed a method of ‘practical, reliable and reproducible diagnosis’ (250) However few patients were very old at the time of observation (follow up 16 years) as the enrolled population was aged 30 to 62 years at entry. (267, 485)
The criteria are split into major and minor with one criterion described as either major or minor. The criteria in table 4Q.ii from table 1 of the 1971 Framingham article. (485)

Table 4Q.ii. Framingham criteria for the diagnosis of HF

<table>
<thead>
<tr>
<th>MAJOR CRITERIA</th>
<th>MINOR CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal nocturnal dyspnoea or othopnoea</td>
<td>Ankle oedema</td>
</tr>
<tr>
<td>Neck vein distension</td>
<td>Night cough</td>
</tr>
<tr>
<td>Râles</td>
<td>Dyspnoea on exertion</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>Hepatomegaly</td>
</tr>
<tr>
<td>Acute pulmonary oedema</td>
<td>Pleural effusion</td>
</tr>
<tr>
<td>S₃ gallop</td>
<td>Vital capacity ↓ ⅓ from maximum</td>
</tr>
<tr>
<td>Increased venous pressure &gt;16cm H₂O</td>
<td>Tachycardia (rate of ≥120/min)</td>
</tr>
<tr>
<td>Circulation time ≥25 seconds</td>
<td></td>
</tr>
<tr>
<td>Hepatojugular reflux</td>
<td></td>
</tr>
</tbody>
</table>

| Weight loss ≥4.5 kg in 5 days.⁺                      |

⁺ If weight loss could have been due to factors other than treatment for heart failure, it is considered a minor criterion.

It is questionable whether some of these criteria are seen or measured in general practice for the diagnosis of HF (e.g. increased venous pressure > 16cm H₂O, circulation time, and vital capacity. Even if these criteria were measured in order to have any meaning there would need to be a prior measurement for comparison.). The criteria are a mix of signs, symptoms and CXR findings.

The diagnosis itself becomes complicated as definite HF diagnosis requires 2 major or 1 major and 2 minor criteria present concurrently, but the minor criteria must not be attributable to any other condition. (485) This however is one of the problems with diagnosis HF: signs and symptoms are not necessarily specific to HF which makes diagnosis on clinical ground difficult and inaccurate, so attribution may be problematic.

Then the weight loss criterion needs to be considered. The criterion does not specify or suggest any potential ‘other factors’ that may be responsible for this level of weight loss and if these occurred concurrently with treatment it may be difficult to determine the relative contribution to weight loss. The pre-treatment weight loss would need to have been measured and recorded for comparison.

The definition of HF is then split further into probable and questionable HF. Probable HF was defined as a doctor’s listed diagnosis of CHF and if there were no reasons to
doubt the presence of HF even if there were incompletely recorded criteria. Questionable HF was present when the criteria for diagnosis were present but their relation to HF was in doubt as they did not occur concomitantly or could be attributed to another condition. (485)

It has been commented that the Framingham criteria have not been validated against a physiological or reference standard. (250, 256, 483) An early attempt to validate the Framingham criteria found that they were shown to be 63% sensitive and 63% specific for reduced LVEF (274) although HF and impaired LVSF are not synonymous.

The figures from the Rotterdam Study (from tables 4a and 4b) were calculated as follows; (482)

Table 4Q.iii. Calculations for Framingham criteria from the Rotterdam Study

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible HF</td>
<td>0.71</td>
<td>0.89</td>
<td>0.75</td>
<td>0.87</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.49 – 0.93</td>
<td>0.79 – 0.99</td>
<td>0.54 – 0.96</td>
<td>0.76 – 0.98</td>
</tr>
<tr>
<td>Definite HF</td>
<td>1</td>
<td>0.78</td>
<td>0.31</td>
<td>1</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.66 – 0.90</td>
<td>0.08 – 0.54</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Framingham Study criteria were better at identifying non-HF patients than it was in predicting which patients had possible HF. However the predictive value for definite HF was very low.

The EFs compared for patients who scored HF present or HF absent are presented in table 4Q.iv. (483)

Table 4Q.iv. HF present or absent by Framingham score and comparative EF

<table>
<thead>
<tr>
<th>Score result</th>
<th>HF present</th>
<th>HF absent</th>
<th>P &lt;0.001</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection Fraction</td>
<td>0.45 ± 0.018</td>
<td>0.53 ± 0.014</td>
<td></td>
</tr>
</tbody>
</table>

There was a significant difference in the EF between patients who had HF by the Framingham Study criteria when compared with patients who did not have HF.

The Framingham Study HF criteria includes signs that probably are not used by GPs, and while it seemed to detect low EF values, the PPV for definite HF was quite low but much higher when determining possible HF patients.
3. **NHANES**

The National Health And Nutrition Examination Survey (NHANES) system was designed from data taken from medical record reviews of patients who self-reported HF or similar on a medical history questionnaire. (Prevalence was assessed by questionnaire and clinical score but there were substantial differences between these two methods.) Patients were non-institutionalised and aged from 25 to 74 years. (486)

The resulting criteria are very similar to the Boston Score (see section 3.3.7). The sections are split into three; dyspnoea / difficulty breathing, physical examination, and CXR. The maximum value for any variable is 2 points, and congestive HF was defined as a total score of ≥3 points. There is no limit as to the maximum amount that can be ‘scored’ in a section unlike the Boston score. (486)

The four questions on breathing are highly detailed and relate only to walking. The precision in these questions is unlikely to be asked in general practice. This is however one of the few scoring systems that includes any objective evidence in the questions.

The table below displays the calculations for sensitivity, specificity, PPV and NPV from the Rotterdam study comparing HF scoring systems (from tables 4a and 4b). (482)

Table 4Q.v. Calculations for NHANES criteria from the Rotterdam Study

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible HF</td>
<td>0.53</td>
<td>0.86</td>
<td>0.64</td>
<td>0.80</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.29 – 0.77</td>
<td>0.75 – 0.97</td>
<td>0.69 – 0.89</td>
<td>0.68 – 0.92</td>
</tr>
<tr>
<td>Definite HF</td>
<td>1</td>
<td>0.82</td>
<td>0.36</td>
<td>1</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.71 – 0.93</td>
<td>0.11 – 0.61</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Few points are required to diagnose HF using the NHANES scoring system. The sensitivity and PPV are low for possible HF, and the PPV is very low for definite HF.

4. **Men born in 1913**

This study evaluated factors in a random population sample of men born in 1913 who were assessed between 1963 and 1980. The scoring system splits findings into cardiac and pulmonary. Absent factors score ‘0’; any factor that had occurred in the past scored ‘1’; and any factor that had occurred within the previous year scored ‘2’ (see below, from table 3 of Men born in 1913 article). (487) A twelve month period for symptoms to occur seems a long time and there is no mention of concurrence of symptoms. Compare this with the symptom requirements for the Walma score (see section 5). (328)
Table 4Q.vi. Scoring system for HF from Men born in 1913

<table>
<thead>
<tr>
<th>Factors</th>
<th>Score</th>
<th>Absent</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac Score 0 – 6 points</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of heart disease</td>
<td>0</td>
<td>1 – 2</td>
<td></td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>0</td>
<td>1 – 2</td>
<td></td>
</tr>
<tr>
<td>Swollen legs at the end of the day</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Dyspnoea at night</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Pulmonary râles</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Pulmonary Score 0 – 5 points</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of bronchitis / asthma</td>
<td>0</td>
<td>1 – 2</td>
<td></td>
</tr>
<tr>
<td>Cough, phlegm, or wheezing</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Rhonchi at examination</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

The score is then used to give the ‘stage’ of HF the patient is currently in. (488) Stage ‘0’ represents clearly no sign of HF; stage 1 is latent HF as there is an indication of cardiac disease; stage 2 indicates CVD but the patient also has dyspnoea or is being treated, by stage 3 the patient is being treated but still dyspnoeic, and at stage 4 the patient is severely unwell and survival is considered to be limited. (488)

At some point during the diagnosis procedures, the WHO dyspnoea grade questionnaire is applied. It is similar to the dyspnoea questions included in the NHANES scoring system except these questions are graded from ‘0’ meaning no dyspnoea through to grade 4 meaning shortness of breath when walking or dressing. (487)

The diagnostic accuracy has also been calculated for this scoring system. (482)

Table 4Q.vii. Calculations for Men born in 1913 criteria from the Rotterdam Study

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Possible HF</strong></td>
<td>0.76</td>
<td>0.76</td>
<td>0.59</td>
<td>0.88</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.56 – 0.96</td>
<td>0.62 – 0.90</td>
<td>0.38 – 0.80</td>
<td>0.77 – 0.99</td>
</tr>
<tr>
<td><strong>Definite HF</strong></td>
<td>0.80</td>
<td>0.63</td>
<td>0.18</td>
<td>0.96</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.45 – 1.00</td>
<td>0.49 – 0.77</td>
<td>0.02 – 0.34</td>
<td>0.89 – 1.00</td>
</tr>
</tbody>
</table>

The age of patients was young for HF patients – 50 at age of first evaluation and aged 67 at the final evaluation. The system itself if quite complex, needing to use a cardiac score, pulmonary score, and dyspnoea score before determining what level of HF the
patient has. Even then there are different states of HF stage 2, which also take into account treatment and response to treatment. It is quite a long scoring system and it does not include objective evidence of ventricular function. The NPV of men born in 1913 scoring system is high for both possible and definite HF, however the PPV of this scoring system is very low for definite HF and it does not perform well when compared against other scoring systems in PPV for possible HF.

5. **Walma**

The study was based on withdrawal of long-term diuretics from elderly patients in primary care which is possibly a more appropriate study group. (489) The Walma score used weighted symptoms (see table 4Q.viii). (328, 489)

Table 4Q.viii. Walma HF scoring system

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal nocturnal dyspnoea (within past week)</td>
<td>3</td>
</tr>
<tr>
<td>Dyspnoea on exertion (past week)</td>
<td>2</td>
</tr>
<tr>
<td>Raised JVP</td>
<td>2</td>
</tr>
<tr>
<td>HR &gt; 100 bpm</td>
<td>1</td>
</tr>
<tr>
<td>Hepatoujugular reflex</td>
<td>1</td>
</tr>
<tr>
<td>Lower pulmonary crepitations</td>
<td>1</td>
</tr>
<tr>
<td>S₃ gallop rhythm</td>
<td>1</td>
</tr>
<tr>
<td>Bilateral pitting ankle oedema</td>
<td>1</td>
</tr>
</tbody>
</table>

HF is considered present if the score is ≥3. This score does not include any measures of objective evidence (ECG or CXR) as they were routinely unavailable in a general practice setting. (489) This is not the situation in current primary care.

The accuracy values for the Walma criteria are below. (482)

Table 4Q.ix. Calculations of diagnostic accuracy for Walma criteria

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Possible HF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>95% CI</td>
<td>0.64 – 1.00</td>
<td>0.75 – 0.97</td>
<td>0.54 – 0.94</td>
<td>0.82 – 1.00</td>
</tr>
<tr>
<td><strong>Definite HF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>95% CI</td>
<td>0.58 – 0.84</td>
<td>0.06 – 0.46</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The negative variables calculated in the Rotterdam study were quite high, however the Walma score was poor at predicting definite HF although it was better at possible HF.
6. **Gheorghiade**

The Gheorghiade scoring system (490) was used in a trial comparing patient variables on and off digoxin. The variables included are similar to those used in the Boston Score but each variable is worth one point. (490) The following variables were used:

- Dyspnoea on exertion,
- Paroxysmal nocturnal dyspnoea,
- Sinus tachycardia,
- Jugular venous distention,
- Pulmonary end-inspiratory râles,
- S3 gallop,
- CTR > 0.5,
- Radiographic interstitial oedema,
- Alveolar changes,
- Pulmonary venous hypertension,
- Pleural effusion,
- Peripheral oedema. (490)

It is not made clear how many points are indicative of HF as the grading system is used to compare assessments of patients on and off digoxin. This scoring system utilises signs and symptoms (although not particularly many) as well as radiographic evidence.

The Rotterdam study (482) calculated the variables for diagnosing HF as follows;

Table 4Q.x. Calculations for Gheorghiade criteria

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Possible HF</strong></td>
<td>0.47</td>
<td>0.95</td>
<td>0.80</td>
<td>0.80</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.23–0.71</td>
<td>0.88–1.00</td>
<td>0.55–1.00</td>
<td>0.68–0.92</td>
</tr>
<tr>
<td><strong>Definite HF</strong></td>
<td>1</td>
<td>0.90</td>
<td>0.50</td>
<td>1</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.82–0.98</td>
<td>0.19–0.81</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The specificity values for both possible and definite HF are very high, and the PPVs for possible and definite HF are higher than any of the studies reported so far, however it is uncertain how many points are needed to determine what is HF.
7. **Boston score**

The Boston score was first published in 1980 in a text book, revised to measure clinical CHF in an RCT of digoxin (491) and revised again for a study of community use of digoxin. (491)

The Boston score is split into three categories: history, physical examination, and CXR. Each criterion is weighted and no more than four points can be scored in each section. The subscores for each section are then totalled to indicate whether the patient has definite HF (8 to 12 points), possible HF (5 to 7 points) or unlikely HF (0 to 4 points). (38) The table 4Q.xi below is taken from table 1 of the Boston score article. (38)

Table 4Q.xi. Criteria for scoring the certainty of HF diagnosis, the Boston score

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Point value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category I: History</strong></td>
<td></td>
</tr>
<tr>
<td>Rest dyspnoea</td>
<td>4</td>
</tr>
<tr>
<td>Orthopnoea</td>
<td>4</td>
</tr>
<tr>
<td>Paroxysmal Nocturnal Dyspnoea</td>
<td>3</td>
</tr>
<tr>
<td>Dyspnoea on walking on level</td>
<td>2</td>
</tr>
<tr>
<td>Dyspnoea on climbing</td>
<td>1</td>
</tr>
<tr>
<td><strong>Category II: Physical Examination</strong></td>
<td></td>
</tr>
<tr>
<td>Heart-rate abnormality (if 91 – 110 beats/min, 1 point; if &gt;110 beats/min, 2 points)</td>
<td>1 – 2</td>
</tr>
<tr>
<td>Jugular-venous pressure elevation (2 points if &gt;6 cm H$_2$O; 3 points if &gt;6 cm H$_2$O, plus hepatomegaly or oedema)</td>
<td>2 – 3</td>
</tr>
<tr>
<td>Lung crackles (1 point if basilar; 2 points if more than basilar)</td>
<td>1 – 2</td>
</tr>
<tr>
<td>Wheezing</td>
<td>3</td>
</tr>
<tr>
<td>Third heart sound</td>
<td>3</td>
</tr>
<tr>
<td><strong>Category III: Chest Radiography</strong></td>
<td></td>
</tr>
<tr>
<td>Alveolar pulmonary oedema</td>
<td>4</td>
</tr>
<tr>
<td>Interstitial pulmonary oedema</td>
<td>3</td>
</tr>
<tr>
<td>Bilateral pulmonary effusions</td>
<td>3</td>
</tr>
<tr>
<td>Cardiotoracic ratio $\geq$0.50</td>
<td>3</td>
</tr>
<tr>
<td>Upper-zone flow redistribution</td>
<td>2</td>
</tr>
</tbody>
</table>

The score has been validated against resting PCWP $>$12 mmHg (measure of LV failure severity) and it was found that definite or possible HF had a 90% sensitivity and 85%
specificity for an elevated PCWP. (38, 250) The use of the classification by category also has excellent inter-rater and intra-rater agreement, indicating a high reliability. (38)

A scoring system may be better at identifying patients than a non-standardised recording of signs and symptoms. The Boston score was used to review the signs and symptoms that patients were referred with from primary care to study cardiologists and found that only 32 of 88 patients (36%) scored as ‘definite HF’ and 24 of 88 (27%) were ‘possible HF’. This was followed up by further testing and most of the definite HF patients (28 of 32 patients) retained this classification. Out of the 24 possible HF patients determined by the Boston score, 9 were confirmed as possible, 7 revised as definite and 8 revised as unlikely. Overall the Boston score was better at predicting HF than usual GP practice. Definite HF was likely to be confirmed but there was some doubt over patients with possible HF. (244)

When EFs were compared for HF that was diagnosed by the grading criteria, there was a significant difference seen between groups. (483) The table below (4Q.xii) is taken from table 3 of the above reference.

Table 4Q.xii. Relationship between clinical classification and LVEF

<table>
<thead>
<tr>
<th></th>
<th>Definite HF</th>
<th>Possible HF</th>
<th>Unlikely HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ejection Fraction</strong></td>
<td>0.41 ± 0.021</td>
<td>0.51 ± 0.020</td>
<td>0.55 ± 0.016</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

The PPVs calculated for the Boston score were the highest for both definite and possible HF out of the six scoring systems evaluated in the Rotterdam study. The table below is from tables 4a and 4b of the Rotterdam study. (482)

Table 4Q.xiii. Calculations for Boston score from the Rotterdam Study

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Possible HF</strong></td>
<td>0.41</td>
<td>0.97</td>
<td>0.88</td>
<td>0.78</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.17 – 0.65</td>
<td>0.89 – 1.00</td>
<td>0.64 – 1.00</td>
<td>0.66 – 0.90</td>
</tr>
<tr>
<td><strong>Definite HF</strong></td>
<td>1</td>
<td>0.94</td>
<td>0.63</td>
<td>1</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.87 – 1.00</td>
<td>0.30 – 0.96</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

While the Boston score includes symptoms, signs and CXR evidence, and the organisation of the scoring system means that no one category can determine the diagnosis, it is a long list of criteria. Moreover the weighting given to different criteria would make it difficult to memorise and so would need to be available in paper or electronic format if it were to be used in primary care. It does have an extensive and
inclusive list of criteria and has been demonstrated to perform well when identifying possible and definite HF patients. However echocardiography is always the next step after making an initial diagnosis in order to determine function of the heart.

**SUMMARY:** The application of a HF scoring system to research or to patients in primary care is not to say that echocardiography is redundant. Rather, it is to standardise the diagnostic process and to determine which patients are most likely to have HF and as such to be referred for echocardiography to ascertain the heart’s function and the underlying reasons for cardiovascular dysfunction. The scoring systems are unable to do this as they solely report on external manifestations of the dysfunction, with only a few including objective evidence in the form of CXR. Thought needs also to be given to the different symptoms and signs that patients present with and that GPs are able to distinguish. The availability of a standardised scoring system in primary care would simplify the diagnostic process and the use of a scoring system in research would simplify translation of research findings to patients.
Appendices for Chapter 5. Results
5A. Patient numbers by GP, practice size and IPA / PHO

These tables utilise information from the set of tables in Chapter 5 section 1.3 and break it down further to give details of how many eligible patients participated per GP. The GPs are individually identified by their study number.

Table 5A.i. Patients per GP for CHS/Comprehensive guideline arm.

<table>
<thead>
<tr>
<th>Practice size</th>
<th>GPs</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>052</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>093</td>
<td>2</td>
</tr>
<tr>
<td>Medium</td>
<td>030</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>031</td>
<td>7</td>
</tr>
<tr>
<td>Large</td>
<td>025</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>026</td>
<td>7</td>
</tr>
<tr>
<td>TOTALS</td>
<td>6</td>
<td>38</td>
</tr>
</tbody>
</table>

Table 5A.ii. Patients per GP for IPCS/HealthWest guideline arm.

<table>
<thead>
<tr>
<th>Practice size</th>
<th>GPs</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>107</td>
<td>22</td>
</tr>
<tr>
<td>Medium</td>
<td>178</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>179</td>
<td>2</td>
</tr>
<tr>
<td>Large</td>
<td>142</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>143</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>145</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>159</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>160</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>161</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>162</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>163</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>165</td>
<td>5</td>
</tr>
<tr>
<td>TOTALS</td>
<td>12</td>
<td>99</td>
</tr>
</tbody>
</table>
Table 5A.iii. Patients per GP for CHS/Comprehensive small group arm

<table>
<thead>
<tr>
<th>Practice size</th>
<th>GPs</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Medium</td>
<td>006</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>007</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>037</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>058</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>059</td>
<td>1</td>
</tr>
<tr>
<td>Large</td>
<td>001</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>002</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>004</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>090</td>
<td>7</td>
</tr>
<tr>
<td>TOTALS</td>
<td>9</td>
<td>38</td>
</tr>
</tbody>
</table>

Table 5A.iv. Patients per GP for IPCS/HealthWest GPs in the small group arm.

<table>
<thead>
<tr>
<th>Practice size</th>
<th>GPs</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>147</td>
<td>12</td>
</tr>
<tr>
<td>Medium</td>
<td>111</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>112</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>113</td>
<td>1</td>
</tr>
<tr>
<td>Large</td>
<td>166</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>167</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>168</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>169</td>
<td>10</td>
</tr>
<tr>
<td>TOTALS</td>
<td>8</td>
<td>60</td>
</tr>
</tbody>
</table>
There was considerable variation in the numbers of eligible HF patients per GP. The number of patients per GP ranged from 1 to 22. This gives a mean number of approximately 7 HF patients per GP.
5B. Expanded grouping of ethnicities

5B.i. Reported ethnicities for all patients who participated in the study.

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>N (%) of patients</th>
<th>All patients</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. European</td>
<td>315 (88%)</td>
<td>111 (81%)</td>
<td>90 (92%)</td>
<td>114 (92%)</td>
<td></td>
</tr>
<tr>
<td>2. Maori</td>
<td>15 (4%)</td>
<td>7 (5%)</td>
<td>4 (4%)</td>
<td>4 (3%)</td>
<td></td>
</tr>
<tr>
<td>3. Pacific</td>
<td>13 (4%)</td>
<td>9 (7%)</td>
<td>1 (1%)</td>
<td>3 (2%)</td>
<td></td>
</tr>
<tr>
<td>4. Asian</td>
<td>5 (1%)</td>
<td>4 (3%)</td>
<td>0</td>
<td>1 (1%)</td>
<td></td>
</tr>
<tr>
<td>5. Indian / Sri Lankan</td>
<td>6 (2%)</td>
<td>4 (3%)</td>
<td>0</td>
<td>2 (2%)</td>
<td></td>
</tr>
<tr>
<td>9. Not determined</td>
<td>5 (1%)</td>
<td>2 (2%)</td>
<td>3 (3%)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>359</td>
<td>137</td>
<td>98</td>
<td>124</td>
<td></td>
</tr>
</tbody>
</table>

Rounding of proportions may result in total percentages being greater than 100%.
5C. Angiotensin inhibition prescription by HF group over time

Table 5C.i. Timing of diagnosis and Angiotensin inhibitor prescription, pre-intervention

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>ACEi / ARB</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>145 (78%)</td>
<td>40 (22%)</td>
<td>185</td>
</tr>
<tr>
<td>Mid</td>
<td>20 (71%)</td>
<td>8 (29%)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>78 (63%)</td>
<td>45 (37%)</td>
<td>123</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>243</td>
<td>93</td>
<td>336</td>
</tr>
</tbody>
</table>

Table 5C.ii. Timing of diagnosis and Angiotensin inhibitor prescription, post-intervention

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>ACEi / ARB</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>134 (72%)</td>
<td>51 (28%)</td>
<td>185</td>
</tr>
<tr>
<td>Mid</td>
<td>23 (82%)</td>
<td>5 (18%)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>77 (63%)</td>
<td>46 (37%)</td>
<td>123</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>234</td>
<td>102</td>
<td>336</td>
</tr>
</tbody>
</table>

Table 5C.iii. Timing of diagnosis and Angiotensin inhibitor prescription, current management

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>ACEi / ARB</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>120 (65%)</td>
<td>64 (35%)</td>
<td>184</td>
</tr>
<tr>
<td>Mid</td>
<td>19 (68%)</td>
<td>9 (32%)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>68 (56%)</td>
<td>54 (44%)</td>
<td>122</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>207</td>
<td>127</td>
<td>334</td>
</tr>
</tbody>
</table>

*Two patients had no data for the current management period, 3 years after the educational intervention.*
5D. β-blocker prescription by HF group over time

Table 5D.i. Timing of diagnosis and β-blocker prescription, pre-intervention

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blocker</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>46 (25 %)</td>
<td>139 (75 %)</td>
<td>185</td>
</tr>
<tr>
<td>Mid</td>
<td>9 (32 %)</td>
<td>19 (68 %)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>16 (13 %)</td>
<td>107 (87 %)</td>
<td>123</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>71 (21 %)</td>
<td>265 (79 %)</td>
<td>336</td>
</tr>
</tbody>
</table>

Table 5D.ii. Timing of diagnosis and β-blocker prescription, post-intervention

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blocker</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>54 (29 %)</td>
<td>131 (71 %)</td>
<td>185</td>
</tr>
<tr>
<td>Mid</td>
<td>10 (36 %)</td>
<td>18 (64 %)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>21 (17 %)</td>
<td>102 (83 %)</td>
<td>123</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>85 (25 %)</td>
<td>251 (75 %)</td>
<td>336</td>
</tr>
</tbody>
</table>

Table 5D.iii. Timing of diagnosis and β-blocker prescription, current management

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blocker</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>64 (35 %)</td>
<td>120 (65 %)</td>
<td>184</td>
</tr>
<tr>
<td>Mid</td>
<td>10 (36 %)</td>
<td>18 (64 %)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>36 (29.5 %)</td>
<td>86 (70.5 %)</td>
<td>122</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>110 (33 %)</td>
<td>224 (67 %)</td>
<td>334</td>
</tr>
</tbody>
</table>
5E. Spironolactone prescription by HF group over time

Table 5E.i. Timing of diagnosis and spironolactone prescription, pre-intervention

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blocker</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>50 (27%)</td>
<td>135 (73%)</td>
<td>185</td>
</tr>
<tr>
<td>Mid</td>
<td>2 (7%)</td>
<td>26 (93%)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>14 (11%)</td>
<td>109 (89%)</td>
<td>123</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>66 (20%)</td>
<td>270 (80%)</td>
<td>336</td>
</tr>
</tbody>
</table>

Table 5E.ii. Timing of diagnosis and spironolactone prescription, post-intervention

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blocker</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>52 (28%)</td>
<td>133 (72%)</td>
<td>185</td>
</tr>
<tr>
<td>Mid</td>
<td>4 (14%)</td>
<td>24 (86%)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>16 (13%)</td>
<td>107 (87%)</td>
<td>123</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>72 (21%)</td>
<td>264 (79%)</td>
<td>336</td>
</tr>
</tbody>
</table>

Table 5E.iii. Timing of diagnosis and spironolactone prescription, current management

<table>
<thead>
<tr>
<th>Timing of diagnosis</th>
<th>β-blocker</th>
<th>None</th>
<th>Totals (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>45 (24.5%)</td>
<td>139 (75.5%)</td>
<td>184</td>
</tr>
<tr>
<td>Mid</td>
<td>5 (18%)</td>
<td>23 (82%)</td>
<td>28</td>
</tr>
<tr>
<td>Late or no diagnosis</td>
<td>18 (15%)</td>
<td>104 (85%)</td>
<td>122</td>
</tr>
<tr>
<td>Totals (n)</td>
<td>68 (20%)</td>
<td>266 (80%)</td>
<td>334</td>
</tr>
</tbody>
</table>
5F. Prescribed doses of ACE inhibitors

5F.i. ACEi doses prescribed over time, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention n (%)</th>
<th>Post-intervention n (%)</th>
<th>Current management n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>15 (6 %)</td>
<td>18 (7 %)</td>
<td>17 (8 %)</td>
</tr>
<tr>
<td>1</td>
<td>100 (39 %)</td>
<td>96 (38 %)</td>
<td>83 (37 %)</td>
</tr>
<tr>
<td>2</td>
<td>72 (28 %)</td>
<td>68 (27 %)</td>
<td>57 (26 %)</td>
</tr>
<tr>
<td>3</td>
<td>51 (20 %)</td>
<td>45 (18 %)</td>
<td>43 (19 %)</td>
</tr>
<tr>
<td>4</td>
<td>20 (8 %)</td>
<td>20 (8 %)</td>
<td>23 (10 %)</td>
</tr>
<tr>
<td>5</td>
<td>1 (&lt;1 %)</td>
<td>3 (1 %)</td>
<td>0</td>
</tr>
<tr>
<td>N&lt;sup&gt;b&lt;/sup&gt;</td>
<td>259</td>
<td>250</td>
<td>223</td>
</tr>
</tbody>
</table>

5F.ii. ACEi doses prescribed over time by each study arm, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline n (%)</th>
<th>Small group n (%)</th>
<th>Internet n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>9</td>
<td>8 (8 %)</td>
<td>9 (10 %)</td>
<td>12 (14 %)</td>
</tr>
<tr>
<td>1</td>
<td>35 (34 %)</td>
<td>32 (34 %)</td>
<td>25 (29 %)</td>
</tr>
<tr>
<td>2</td>
<td>26 (27 %)</td>
<td>24 (26 %)</td>
<td>21 (24 %)</td>
</tr>
<tr>
<td>3</td>
<td>17 (18 %)</td>
<td>19 (20 %)</td>
<td>19 (22 %)</td>
</tr>
<tr>
<td>4</td>
<td>12 (12 %)</td>
<td>9 (10 %)</td>
<td>10 (11 %)</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>1 (1 %)</td>
<td>0</td>
</tr>
<tr>
<td>N</td>
<td>97</td>
<td>94</td>
<td>87</td>
</tr>
</tbody>
</table>

<sup>a</sup> Values for percentages have been rounded and may not always equal 100% when added together.

<sup>b</sup> Total number of patients in that group or sub-group being prescribed ACE inhibitors for whom actual dose information was available.
5G. Prescribed doses of Angiotensin Receptor Blockers

5G.i. ARB doses prescribed over time, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention %</th>
<th>Post-intervention %</th>
<th>Current management %</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>0</td>
<td>2 (7%)</td>
<td>2 (7%)</td>
</tr>
<tr>
<td>1</td>
<td>5 (20%)</td>
<td>6 (20%)</td>
<td>3 (11%)</td>
</tr>
<tr>
<td>2</td>
<td>9 (36%)</td>
<td>10 (33%)</td>
<td>12 (43%)</td>
</tr>
<tr>
<td>3</td>
<td>9 (36%)</td>
<td>10 (33%)</td>
<td>7 (25%)</td>
</tr>
<tr>
<td>4</td>
<td>1 (4%)</td>
<td>2 (7%)</td>
<td>4 (14%)</td>
</tr>
<tr>
<td>5</td>
<td>1 (4%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>N</td>
<td>25</td>
<td>30</td>
<td>28</td>
</tr>
</tbody>
</table>

5G.i. ARB doses prescribed over time by each study arm, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>9</td>
<td>0</td>
<td>1 (9%)</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>1</td>
<td>1 (11%)</td>
<td>1 (9%)</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>2</td>
<td>3 (33%)</td>
<td>4 (36%)</td>
<td>5 (42%)</td>
</tr>
<tr>
<td>3</td>
<td>4 (44%)</td>
<td>5 (45%)</td>
<td>3 (25%)</td>
</tr>
<tr>
<td>4</td>
<td>1 (11%)</td>
<td>0</td>
<td>2 (17%)</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>N</td>
<td>9</td>
<td>11</td>
<td>12</td>
</tr>
</tbody>
</table>
5H. Combined prescribed doses of ACEI and ARBs

5H.i. ACEi and ARB doses prescribed over time, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention %</th>
<th>Post-intervention %</th>
<th>Current management %</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>15 (5%)</td>
<td>20 (7%)</td>
<td>19 (8%)</td>
</tr>
<tr>
<td>1</td>
<td>105 (37%)</td>
<td>102 (36%)</td>
<td>86 (34%)</td>
</tr>
<tr>
<td>2</td>
<td>81 (29%)</td>
<td>78 (28%)</td>
<td>69 (27%)</td>
</tr>
<tr>
<td>3</td>
<td>60 (21%)</td>
<td>55 (20%)</td>
<td>50 (20%)</td>
</tr>
<tr>
<td>4</td>
<td>21 (7%)</td>
<td>22 (8%)</td>
<td>27 (11%)</td>
</tr>
<tr>
<td>5</td>
<td>2 (1%)</td>
<td>3 (1%)</td>
<td>0</td>
</tr>
<tr>
<td>N</td>
<td>284</td>
<td>280</td>
<td>251</td>
</tr>
</tbody>
</table>

5G.i. ACEi and ARB doses prescribed over time by each study arm, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline</th>
<th>Small group</th>
<th>Internet</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
<td>Pre</td>
</tr>
<tr>
<td>9</td>
<td>8 (8%)</td>
<td>10 (10%)</td>
<td>13 (13%)</td>
<td>3 (3%)</td>
<td>4 (5%)</td>
<td>1 (1%)</td>
<td>5 (6%)</td>
</tr>
<tr>
<td>1</td>
<td>36 (34%)</td>
<td>33 (31%)</td>
<td>26 (26%)</td>
<td>31 (35%)</td>
<td>30 (35%)</td>
<td>24 (34%)</td>
<td>39 (44%)</td>
</tr>
<tr>
<td>2</td>
<td>29 (27%)</td>
<td>28 (27%)</td>
<td>26 (36%)</td>
<td>33 (37%)</td>
<td>28 (33%)</td>
<td>24 (34%)</td>
<td>19 (21%)</td>
</tr>
<tr>
<td>3</td>
<td>21 (20%)</td>
<td>24 (23%)</td>
<td>22 (22%)</td>
<td>18 (20%)</td>
<td>15 (18%)</td>
<td>16 (23%)</td>
<td>21 (24%)</td>
</tr>
<tr>
<td>4</td>
<td>13 (12%)</td>
<td>9 (9%)</td>
<td>12 (12%)</td>
<td>4 (4%)</td>
<td>6 (7%)</td>
<td>6 (8%)</td>
<td>4 (4%)</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>1 (1%)</td>
<td>0</td>
<td>1 (1%)</td>
<td>2 (2%)</td>
<td>0</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>N</td>
<td>106</td>
<td>105</td>
<td>99</td>
<td>89</td>
<td>85</td>
<td>71</td>
<td>89</td>
</tr>
</tbody>
</table>
**5I. Adjusted means of ACEI for recommended dose and greater**

5I.i. Adjusted means and 95%CI for ACEi guideline-recommended dose and higher by study arm and time period

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline % (95% CI)</th>
<th>Small group % (95% CI)</th>
<th>Internet % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>41% (27–55)</td>
<td>37% (24–53)</td>
<td>53% (38–67)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>40% (27–55)</td>
<td>39% (25–55)</td>
<td>57% (41–70)</td>
</tr>
<tr>
<td>Current Management</td>
<td>38% (25–54)</td>
<td>34% (21–50)</td>
<td>54% (39–69)</td>
</tr>
</tbody>
</table>

**5J. Adjusted means of ACEI and ARBs for recommended dose and greater**

5J.i. Adjusted means and 95%CI for ACEI and ARBs at guideline-recommended dose and higher by study arm and time period

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline % (95% CI)</th>
<th>Small group % (95% CI)</th>
<th>Internet % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>40% (27–54)</td>
<td>42% (28–57)</td>
<td>52% (37–66)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>40% (27–54)</td>
<td>45% (31–60)</td>
<td>52% (37–66)</td>
</tr>
<tr>
<td>Current Management</td>
<td>37% (25–51)</td>
<td>37% (24–52)</td>
<td>52% (37–66)</td>
</tr>
</tbody>
</table>
5K. Prescribed doses of β-blockers

5K.i. β-blocker doses prescribed over time, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention %</th>
<th>Post-intervention %</th>
<th>Current management %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6 (8%)</td>
<td>11 (12%)</td>
<td>13 (11%)</td>
</tr>
<tr>
<td>2</td>
<td>20 (26%)</td>
<td>19 (21%)</td>
<td>27 (23%)</td>
</tr>
<tr>
<td>3</td>
<td>30 (39%)</td>
<td>34 (37%)</td>
<td>43 (37%)</td>
</tr>
<tr>
<td>4</td>
<td>19 (25%)</td>
<td>26 (29%)</td>
<td>34 (29%)</td>
</tr>
<tr>
<td>5</td>
<td>1 (1%)</td>
<td>1 (1%)</td>
<td>0</td>
</tr>
<tr>
<td>N</td>
<td>76</td>
<td>91</td>
<td>117</td>
</tr>
</tbody>
</table>

5K.ii. β-blocker doses prescribed over time by each study arm, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline %</th>
<th>Small group %</th>
<th>Internet %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>1</td>
<td>3 (12%)</td>
<td>3 (10%)</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>2</td>
<td>4 (16%)</td>
<td>4 (14%)</td>
<td>6 (15%)</td>
</tr>
<tr>
<td>3</td>
<td>12 (48%)</td>
<td>13 (45%)</td>
<td>19 (46%)</td>
</tr>
<tr>
<td>4</td>
<td>6 (24%)</td>
<td>9 (31%)</td>
<td>13 (32%)</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>N</td>
<td>25</td>
<td>29</td>
<td>41</td>
</tr>
</tbody>
</table>
### 5L. Adjusted means of β-blockers for half to recommended dose

5L.i. Adjusted means and 95%CI for β-blockers at half to guideline-recommended dose by study arm and time period

<table>
<thead>
<tr>
<th>Time period</th>
<th>Guideline % (95 % CI)</th>
<th>Small group % (95 % CI)</th>
<th>Internet % (95 % CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>19 % (6 – 47)</td>
<td>25 % (7 – 58)</td>
<td>32 % (14 – 59)</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>17 % (5 – 43)</td>
<td>42 % (16 – 72)</td>
<td>23 % (9 – 48)</td>
</tr>
<tr>
<td>Current Management</td>
<td>19 % (7 – 45)</td>
<td>33 % (12 – 64)</td>
<td>37 % (17 – 63)</td>
</tr>
</tbody>
</table>
5M. Prescribed doses of Spironolactone

5M.i. Spironolactone doses prescribed over time, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Pre-intervention %</th>
<th>Post-intervention %</th>
<th>Current management %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3 (4%)</td>
<td>2 (3%)</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>5 (7%)</td>
<td>10 (14%)</td>
<td>7 (10%)</td>
</tr>
<tr>
<td>3</td>
<td>59 (87%)</td>
<td>59 (81%)</td>
<td>60 (86%)</td>
</tr>
<tr>
<td>4</td>
<td>1 (1%)</td>
<td>2 (3%)</td>
<td>3 (4%)</td>
</tr>
<tr>
<td>N</td>
<td>68</td>
<td>73</td>
<td>70</td>
</tr>
</tbody>
</table>

5M.ii. Spironolactone doses prescribed over time by each study arm, all dose scales

<table>
<thead>
<tr>
<th>Dose scale</th>
<th>Guideline %</th>
<th>Small group %</th>
<th>Internet %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Current</td>
</tr>
<tr>
<td>1</td>
<td>2 (8%)</td>
<td>1 (4%)</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>4 (17%)</td>
<td>7 (29%)</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>3</td>
<td>18 (75%)</td>
<td>15 (63%)</td>
<td>17 (81%)</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>1 (4%)</td>
<td>2 (10%)</td>
</tr>
<tr>
<td>N</td>
<td>24</td>
<td>24</td>
<td>21</td>
</tr>
</tbody>
</table>