Breathlessness is a sensation most of us have experienced, yet which can prove difficult to evaluate and manage in our patients. This paper aims to provide an approach to the problem of chronic breathlessness in the elderly.

Breathlessness cannot be explained by any one physiological mechanism and may be made up of several sensations. It usually arises in situations where there is increased respiratory effort. Respiratory effort may result from increased respiratory drive, increased respiratory load or through both these mechanisms. The perception of effort arises through activation of the sensory cortex (from chest wall and airway afferents) at the same time that the respiratory muscles are signalled to contract. Yet, in paralysed normal volunteers, an increase in the amount of inspired CO₂ alone has been shown to cause respiratory discomfort. Cortical influences such as previous experience and learning, and psychological status are thought to be important in the modulation of breathlessness. It may be helpful to consider breathlessness in an analogy with pain: both are a subjective response to a physiological stimulus, which may be modified by presence of other diseases and by experience.

Although breathlessness is common in the elderly it should not be considered an inevitable consequence of aging. Older people are less sensitive to added respiratory loads, and their ventilatory response to hypercapnia and hypoxia are reduced by about 50% compared with younger people. Elderly asthmatics, despite a greater reduction in FEV₁ with methacholine, have less awareness of the symptoms of bronchoconstriction than younger subjects do. These findings suggest that the elderly may become breathless later in the course of disease than younger people do or may present later during an exacerbation of disease. However, these differences may be outweighed to some extent by age-related loss of physiological reserve, particularly in the cardiovascular, respiratory, and musculoskeletal systems.

**Causes of breathlessness in older patients**

These can be divided into five categories, namely respiratory, cardiovascular, deconditioning, respiratory muscle weakness/mechanical dysfunction and miscellaneous (including anaemia and anxiety). Causes of breathlessness are listed in Table 1. Breathlessness is a problem in about 50% of terminal cancer patients and may be due to the tumour itself, its treatment or to comorbidities. There may be more than one mechanism for an older person's breathlessness. An example of this is the cycle that occurs when breathlessness from cardiac or respiratory disease leads to exercise limitation. The resultant deconditioning means that the intensity of breathlessness on exertion will be greater and lead to a further reduction in exercise. Another example is the relationship between chronic disease and psychiatric morbidity. Severe chronic obstructive pulmonary disease (COPD) is commonly associated with hyperventilation and psychiatric disorders. In an Australian study of 50 consecutive patients hospitalised with COPD, 72% had experienced hyperventilation at some time in their illness; 34% had an anxiety disorder (the majority of these had panic disorder) and 16% were depressed. As well as psychiatric disorders, comorbidities such as ischaemic heart disease and lung cancer, are more common in patients with COPD and may contribute to breathlessness.

**Assessment of the breathless older patient**

Because of the wide range of contributing factors, the approach to the breathless elderly patient must be comprehensive and thorough.

<table>
<thead>
<tr>
<th>Respiratory</th>
<th>Airway disease</th>
<th>Upper-airway obstruction</th>
<th>Asthma</th>
<th>Chronic bronchitis</th>
<th>Emphysema</th>
</tr>
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<tbody>
<tr>
<td>Parenchymal Lung Disease</td>
<td>Interstitial lung disease</td>
<td>Malignancy-primary or metastatic</td>
<td>Pneumonia</td>
<td></td>
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<tr>
<td>Pulmonary Vascular Disease</td>
<td>Arteriovenous malformation</td>
<td>Intravascular obstruction</td>
<td>Vasculitis</td>
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<tr>
<td>Pleural disease</td>
<td>Effusion</td>
<td>Fibrosis</td>
<td>Malignancy</td>
<td></td>
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<tr>
<td>Cardiovascular</td>
<td>Decreased cardiac output</td>
<td>Elevated pulmonary venous pressure</td>
<td>Right to left shunt</td>
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<tr>
<td>Deconditioning</td>
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</tr>
<tr>
<td>Respiratory muscle weakness/mechanical dysfunction</td>
<td>Neuromuscular disorders (e.g. myasthenia gravis)</td>
<td>Phrenic nerve dysfunction or injury</td>
<td>Weakness (e.g. malnutrition, thyroid disease, myositis)</td>
<td>Deformities (e.g. kyphoscoliosis)</td>
<td>Abdominal “loading” (e.g. ascites, obesity)</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Anaemia</td>
<td>Anxiety / psychological</td>
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History. This should include a detailed description of the symptoms, including time course, intensity, aggravating and relieving factors, and associated symptoms. The type of breathlessness may be helpful. In a study of 53 patients with chronic breathlessness, “rap” (D1 CO2) breathing was more often described by those with pulmonary vascular disease or heart failure, increased “work” of breathing by patients with obstructive or interstitial lung disease, or neuromuscular disease, and “tightness” by those with asthma.1 Breathlessness on lying down (orthopnoea) suggests significant cardiorespiratory disease,10 whereas paroxysmal nocturnal dyspnoea is more specific for left heart failure. Episodic breathlessness suggests asthma, myocardial ischaemia or recurrent pulmonary emboli. Details of comorbidities (particularly ischaemic heart disease and hypertension), past history, medications, smoking, alcohol and occupational exposure should be recorded. A full systems review should also be undertaken.

Examination. Particular attention should be paid to general status, cardiorespiratory and neurological aspects of the examination. Signs of right or left heart failure should be especially looked for. Clinical signs suggesting increased respiratory effort include tachypnoea and accessory muscle use.

Investigations. Use of these should be guided by the provisional diagnosis at the completion of the history and examination. A full blood count excludes anaemia and a resting electrocardiogram yields information on heart rate, rhythm, ischaemia and heart size. Chest radiograph may indicate the presence of cardiac, mediastinal, parenchymal, pulmonary-vascular or pleural disease. Spirometry is indicated where there is any possibility of airways or lung disease. An ‘restrictive’ pattern (FEV1/FVC < 70% and FEV1 < 70% predicted for age, sex and height) suggests COPD or asthma and in these patients the most important measure is the post-bronchodilator FEV1, which gives an indication (along with symptoms) of severity and has independent prognostic value.11,12 More detailed lung function testing such as bronchial provocation tests, flow-volume loops, lung volume measurements, diffusing capacity for carbon monoxide (DL CO) and mouth pressures may be required in difficult cases. Patients with suspected interstitial lung disease have a ‘restrictive’ pattern on spirometry and high resolution chest CT may help determine the likely underlying pathology. The presence of pulmonary emboli can be confirmed by spiral chest CT or pulmonary angiography. If cardiac disease is suspected, an echocardiogram can give information on ventricular systolic and diastolic function, as well as heart valves and pericardium.

Pulse oximetry is useful to rule out significant hypoxia, however, an arterial blood gas is required to determine whether or not there is CO2 retention or an increase in the alveolar-arterial oxygen gradient.

Management of breathlessness

Once a diagnosis is made, the reversible factors contributing to the breathlessness should be corrected as far as practicable. If breathlessness continues to be a problem there are some other approaches that may be used. Most of the research into the symptomatic treatment of chronic breathlessness relates to patients with COPD or normal volunteers and may not be generalisable to patients with other conditions.

Measurement of breathlessness

There is a relatively poor correlation between physiological impairment and the severity of breathlessness. For example, breathlessness in panic disorder occurs with a physiologically normal cardiorespiratory system, whereas patients with COPD and a low FEV1 are not necessarily the most breathless.20,21 Assessment of treatment efficacy should, therefore, incorporate a symptom measure at baseline and after treatment, and not rely solely on improvement in physiological function (e.g. FEV1). An objective measurement of breathlessness at a given time can be made using a visual analogue scale or modified Borg scale.22,23 A measure of breathlessness over the previous few weeks can be made with the dyspnoea component of the Chronic Respiratory Questionnaire,24 or the Baseline and Transition Dyspnea Indices.25 Exercise tolerance can be assessed reliably using a timed-walk test17 or shuttle-walking test.26,27 The six-minute walk has been validated in patients with COPD,17 heart failure28 and in older patients with COPD.21 It can be combined with a breathlessness score.29

Medical therapy for breathlessness

There is no convincing evidence that any medicine specifically reduces breathlessness. The efficacy of any agent can usually be explained in terms of decreased respiratory drive or improvement in respiratory mechanics.4 The treatment of all the underlying causes of breathlessness is outside the...
scope of this article, however, there are some specific considerations in elderly patients. Elderly are at particular risk of adverse effects from medication because of loss of renal clearance, reduced physiological reserve to cope with actions of medicines, increased comorbidity and more drug interactions. The effect of the intervention should, therefore, be carefully assessed against predetermined endpoints. If therapy is not efficacious it should be stopped.

**Bronchodilator medications.** In the elderly, there are theoretical reasons why β-agonists may not be as effective as anticholinergic agents, although they remain effective bronchodilators in this group. Overall, the size of the bronchodilator effect seen with β-agonists is similar to that seen with anticholinergic agents. In 20 elderly patients with stable COPD, FEV1 improved on average by 20% after 5 mg nebulised salbutamol. In a study in 12 elderly patients with COPD, oxitropium caused an 18% increase in FEV1, and this effect was of similar magnitude to that seen in younger patients in the same study.

With respect to their effect on breathlessness, β-agonists, anticholinergics and theophylline have each been shown in randomised controlled trials in COPD patients to improve symptoms and this is not dependent on their effect on FEV1. Lack of a bronchodilator response should therefore not limit the use of these agents if the patient gets symptomatic relief. Because similar numbers of patients prefer β-agonists and ipratropium, a trial of each may be appropriate, so that the patient can decide which they prefer.

Bronchodilators can be delivered by a wide range of devices and the choice is common; checks should be undertaken regularly to make sure the patients can actually use the delivery system prescribed. Because of cost and inconvenience, nebuliser therapy should be reserved for patients in whom there is a clear symptomatic response. Nebulised saline affords nearly as much relief of breathlessness as a β-agonist and could be used where patients are experiencing side-effects from excessive doses of bronchodilators. Why saline is effective is not clear but may be due to mechanical factors such as cooling or facial stimulation.

**Opiates.** Opiates reduce breathlessness by decreasing ventilatory drive in response to carbon dioxide, hypoxia and exercise. The acute effect of opiates has been confirmed in COPD patients, heart failure and in terminal malignancy. However, longer term use has no significant effect on breathlessness or exercise tolerance in COPD. Adverse effects such as confusion, constipation and nausea and anticholinergic agents, particularly in the elderly, are troublesome particularly in older patients. Morphine has it advocates for the management of breathlessness in terminal disease. However, for reasons outlined previously it is possible that the nebulising process itself contributes to the relief of breathlessness.

**Benzodiazepines.** Benzodiazepines have no specific effect on breathlessness but may be useful where some of the breathlessness is due to anxiety or panic. Those in whom a significant component of anxiety or depression is suspected should be assessed by a psychiatrist with an interest in breathlessness and/or elderly patients. Care needs to be taken in the elderly with the choice of benzodiazepines and benzodiazepines with a shorter half-life that are cleared by renal clearance, reduced physiological reserve to cope with actions of medicines, increased comorbidity and more drug interactions. The effect of the intervention should, therefore, be carefully assessed against predetermined endpoints. If therapy is not efficacious it should be stopped.

**Oxygen.** The provision of long-term oxygen therapy in New Zealand is reserved for those with chronic lung disease who are significantly hypoxic or for breathlessness due to hypoxia in terminally ill patients. Oxygen has, however, been shown to relieve breathlessness in patients with COPD who are only mildly hypoxic and who are breathless on exertion. Portable oxygen is often used in hospital for early mobilisation of patients with an exacerbation of their respiratory disease or to allow greater exercise intensity in training programmes.

**Nonpharmacological approaches to breathlessness.** 

**Breathing pattern.** Diagnosis of hyperventilation (whether primary or associated with underlying disease) can be difficult, but may be suggested by the clinical presentation and laboratory data. Score on the Nijmegen questionnaire may support the diagnosis and provide an objective measurement of treatment efficacy. In other clinical situations, changes in the breathing pattern can reduce breathlessness. In nine patients (mean age 52 years) with moderately severe but stable heart failure, one month of training to control the respiratory rate to approximately six breaths per minute was associated with a significant reduction in breathlessness, improved exercise tolerance and improved diastolic oxygen saturation. In severe COPD, the adoption of pursed lips breathing relieves breathlessness by reduction in respiratory rate, increase in end-expiratory pressure and tidal volume, and improvement in oxygen saturation. In breathless patients, an assessment by a physiotherapist with expertise in correction of breathing patterns can be invaluable.

**Pulmonary rehabilitation and exercise programmes.** Comprehensive pulmonary rehabilitation combines elements of education, support and exercise training, and this approach has been shown to reduce breathlessness and improve exercise tolerance in COPD. In a controlled trial in 89 patients with severe COPD, subjects received either respiratory rehabilitation or usual care. The rehabilitation was intensive and expensive, consisting of inpatient rehabilitation for eight weeks followed by outpatient treatment for 16 weeks. There were significant improvements in breathlessness scores and exercise tolerance, and the benefits were maintained for at least six months. Other studies suggest that the benefits of pulmonary rehabilitation are maintained for longer if the therapy can be incorporated into the home environment. A recent meta-analysis of 14 randomised trials of respiratory rehabilitation in COPD showed significant and clinically useful benefits in dyspnoea and exercise tolerance, with the distance walked in six minutes improving by 56 m on average. The improvement in breathlessness scores seen was approximately that seen from a short course of oral prednisolone. In populations where rehabilitation is not available, it is possible that the benefits of rehabilitation are at least as great as the improvement seen with bronchodilators or oral theophylline.

In order to improve breathlessness, rehabilitation programmes need to include an element of exercise training. Exercise training appears to reduce breathlessness through decreased ventilatory requirements, possibly through desensitisation, rather than by improving muscle function. Elderly patient can tolerate intensive pulmonary rehabilitation and should not be excluded on the basis of age alone.

Exercise programmes in severe heart failure can improve breathlessness and exercise tolerance. In one study of 18 patients (mean age 52, ejection fraction 21%), three weeks of interval training was compared in a crossover study with three weeks of activity restriction. The treatments were given in random order. At the end of the training period, work rate increased by 57% and at peak work performance, breathlessness and leg fatigue were significantly reduced. The extent to which inspiratory muscle weakness contributes to breathlessness remains controversial and targeted respiratory muscle training does not clearly have a greater effect than exercise training programmes. Nutritional supplementation. Low body weight is common in end-stage cardiorespiratory disease and is an independent predictor of mortality in COPD. Randomised controlled trials of weight gain show that the benefits of nutritional replacement however, are disappointing in that they have shown improvements in parameters such as respiratory muscle strength but the effects on breathlessness and exercise tolerance are small and not maintained after stopping the programme. Other studies suggest that the benefits of nutritional replacement contribute to breathlessness remains controversial and targeted respiratory muscle training does not clearly have a greater effect than exercise training programmes.
Summary

Breathlessness in the elderly is a common clinical problem but should not be considered an inevitable component of the aging process. Because of the diverse causes and the possibility of more than one underlying mechanism, the approach to the breathless patient needs to be comprehensive with investigations guided by specific clinical questions. Once the underlying reversible factors have been identified and treated as far as practicable, management of the chronically breathless patient is based on symptom relief, exercise conditioning, and identification of breathing patterns and patient education. Interventions should be objectively evaluated using symptom scores or a measure of exercise tolerance rather than a physiological measure alone. As there is the potential for harm (including cost), treatments offering no benefit should be promptly withdrawn.

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