Home ventilation: the Green Lane Hospital experience

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Abstract

Aims. To describe the characteristics and outcomes of patients treated with domiciliary nocturnal support ventilation (NSV).

Methods. Case-note review of all patients treated with home NSV by Green Lane Hospital.

Results. 111 patients received home NSV between 1990 and 1999. 59 had respiratory failure due to obesity-hypoventilation syndrome (OHS), most of whom were Maori or Pacific Island people. Their mean BMI was 53 kg/m². They frequently presented acutely, and often *in extremis*. After a median duration of 22 months treatment, 37 patients continued treatment. Four have died, but none from respiratory failure. Other causes of repsiratory failure

included: neuromuscular disease (26), kyphoscoliosis (19) and obstructive sleep apnoea (8). Patients who did not have OHS were mostly of New Zealand European ethnicity, required lower ventilation pressures than patients with OHS, and had better arterial blood gases on treatment. After a median follow-up of 35 months, however, fourteen have died. 33 continued on treatment. Both OHS and non-OHS patients had high deprivation scores according to NZdep96. This was most apparent for patients with OHS.

Conclusions. OHS is an important cause of respiratory failure in New Zealand, particularly affecting Maori and Pacific people. The prognosis of OHS treated with NSV appears to be good despite significant co-morbidity.

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Non-invasive ventilation is an established treatment for chronic respiratory failure.[1] Nocturnal positive pressure ventilatory assistance, using nasal masks, can be used at home and is effective in improving daytime respiratory function, quality of life and prolongs survival in patients with restrictive thoracic wall conditions.[2-4] In primary lung conditions, particularly chronic obstructive pulmonary disease (COPD), the evidence is less convincing, although some patients probably benefit.[4]

Green Lane Hospital has provided nocturnal support ventilation (NSV) to occasional patients since 1990, using bi-level positive airway pressure. This supplies positive airway pressure at two levels, inspiratory (IPAP) and expiratory (EPAP), to assist the patient's own respiratory effort. Despite evidence of benefit, funding of home non-invasive ventilation in New Zealand is haphazard.[1] At Green Lane Hospital, this therapy has largely been provided from other budgets.

We have noted numerous patients presenting with respiratory failure due to obesity (the obesity-hypoventilation syndrome, OHS). This is an unproven indication for non-invasive ventilation. We decided to review the provision and outcomes of home NSV therapy at Green Lane Hospital.

Methods

All patients treated with home NSV before November 1999 (the date of the review) were included. Cases were identified from the ventilatory support service records, and more recently from a computer database. Patients were included if they had been provided with a NSV machine for home use, or if support had been provided for use of a NSV machine at home. Patients using CPAP for obstructive sleep apnoea were not included.

The ventilatory support service records and Green Lane Hospital records were reviewed. Where available, other hospital records were also reviewed. Information was collected on patient demographics, primary and secondary diagnosis, comorbidities, ventilator settings, arterial blood gases on treatment, reason for stopping treatment and outcome. Ethnicity was recorded as stated in the case-notes. Patient addresses (the address at the time of starting NSV) were geocoded and matched with an NZDep96 score if possible.

Results

111 patients were commenced on home NSV between October 1990 and November 1999 (Table 1). In 59

patients, the principal cause of respiratory failure was OHS. Four OHS patients also had another significant cause of respiratory failure. Obesity-related hypoventilation was thought to be a significant contributing factor in a further three patients with other major disorders (pneumonectomy, myotonic dystrophy, quadriplegia). Other causes of respiratory failure included neuromuscular disease (26 patients), kyphoscoliosis (19) and OSA without evidence of OHS (8 - only two had respiratory failure due to OSA alone).

Table 1. Patients treated with nocturnal support ventilation.

Total	111
Obesity-hypoventilation syndrome	**59**
Obesity-hypoventilation syndrome + OSA	39
Obstructive Sleep Apnoea (not OHS)*	**8**
Neuromuscular	**26**
Duchenne muscular dystrophy	8
Myopathy	6
Spinal injury/disease	6
Motor Neuron Disease	2
Unknown/Miscellaneous	4
Kyphoscoliosis	**19**
Thoracoplasty	5
Potts disease	4
Polio	3
Idiopathic	2
Associated neurological disorder†	5
Central apnoeas (all have other problems)	**6**
Lung disease‡	**7**
COPD	4
Pneumonectomy	2
Asbestosis	1
Bridge to transplant	**3**
Cystic firbosis	2
Broncheictasis	1

Principal causes of respiratory failure in 111 patients are shown. Some patients had more than one cause. *Most patients with obstructive sleep apnoea (OSA) also had other causes of respiratory failure. Only two had pure OSA. †Several patients had kyphoscoliosis due to a neurological disorder and both the skeletal and neurological problems contributed to the respiratory failure. ‡Many more patients had a component of asthma or chronic obstructive pulmonary disease. Only included here if this was part of the principal cause of respiratory failure.

Obesity-hypoventilation syndrome. The majority of patients with OHS were either Maori (26) or Pacific Island people (27, Table 2). Only seven were of New Zealand/European ethnicity (including one also recorded as Maori elsewhere in the notes). 63% were male. All were morbidly obese (BMI>35 kg/m²). 39 patients (66%) had co-existing obstructive sleep apnoea (OSA), 33 (56%) were transferred to Green Lane Hospital following an acute-hospitalisation elsewhere with respiratory failure. Sixteen of these had been admitted to an intensive care unit (ICU). At least five others had had previous ICU admissions.

There was a high prevalence of co-morbidity (Figure 1). 43 had evidence of right heart failure and 30 of these had evidence of biventricular failure. Polycythaemia, hypertension, gout and non-insulin dependent diabetes were common. Cellulitis was also common (13) and often precipitated the inital admission.

The median duration of NSV was 22 months (range 1-79) at the time of review. 37 patients continue on treatment. Some patients proved difficult to follow-up; two were lost completely, eight did not attend their most recent appointment and two were followed up at other hospitals. Seven patients were transferred to CPAP treatment and five decided not to continue NSV. One patient successfully stopped NSV after aortic valve surgery. The mean (SD) weight loss since presentation was 7.7 kg (19). Only two lost sufficient weight to discontinue treatment, one of whom has since re-presented with respiratory failure.

Four patients have died, one of acute on chronic renal failure, one from obesity-related cardiomyopathy and two probably from respiratory failure (one had refused to continue NSV eight months previously, the other still had a NSV machine but was thought to be non-compliant).

Non-obesity-hypoventilation syndrome patients. Patients without obesity-hypoventilation syndrome were mostly of New Zealand/European ethnicity (Table 1) and most had chest wall problems - either kyphoscoliosis or neuromuscular conditions (some patients had both). Three (one bronchiectasis, two cystic fibrosis) received NSV as a 'bridge' to lung transplantation. Eight patients had obstructive sleep apnoea, however, six of these had other important conditions causing respiratory failure (three neurological, two COPD, pneumonectomy). Ten patients had a diagnosis of either asthma or COPD, but these were associated with other disorders and not thought to be the major cause of respiratory failure. Six patients had a central component to their respiratory failure, but all also had other conditions (two neuromuscular, asbestosis, COPD, stroke, cardiac disease).
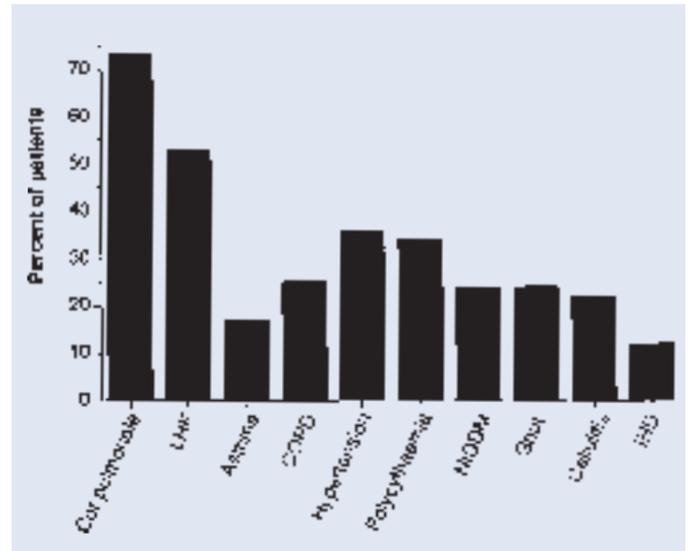

Figure 1. Co-morbidity in patients with obesity-hypoventilation syndrome LHF=left heart failure, NIDDM=non-insulin dependent diabetes mellitus, IHD=ischaemic heart disease.

The median duration of NSV in non-OHS patients was 35 months (range 2 - 108). 33 continue on treatment. Three patients were intolerant or chose to stop. Two have had lung transplants and in two patients NSV was no longer needed. Fourteen non-OHS patients have died, five of whom had progressive neuromuscular disease, five kyphoscoliosis and two quadriplegia. It was difficult to be certain how many of these died from progression of their original disease, respiratory failure or from other disorders. Some died of respiratory infection, which was probably due to problems with bronchial toilet. One died after a major stroke.

Table 2. Patient demographics, nocturnal support settings and arterial gases.

	OHS			nonOHS*			
	n	mean (sd)	range	n	mean (sd)	range	p§
Age (years)	59	45.5 (11.2)	22-72	49	43.4 (17.7)	10-71	ns
Weight (kg)	59	152.3 (30.5)	100-240	38	67.8 (23.1)	27-143.5	
BMI (kg/m²)	58	53 (9.9)	37.3-80.1	35	26.7 (8.6)	13.2-57.5	
Acute presentation	33 (56%)			12 (24%)			<0.002
ICU	16 (27%)			7 (14%)			ns
Ethnicity							
NZ/European	7 (12%)			38 (78%)			
Maori	26 (44%)			8 (16%)			<0.0001
Pacific	27 (46%)			3 (6%)			
NSV settings†							
IPAP (cmH₂O)	59	21.2 (4.1)	10-28	49	17.6 (3.1)	10-22	<0.001
EPAP (cmH₂O)	59	5.9 (1.72)	2-10	49	4.2 (1.6)	2-8	<0.001
Rate (breaths/minute)	56	18.5 (3.5)	10-26	49	17.8 (3.3)	10-24	ns
Supplementation oxygen	31 (53%)			11 (22%)			<0.003
Morning arterial blood gases on treatment‡							
pH	48	7.37 (0.048)	7.29-7.51	29	7.41 (0.047)	7.33-7.50	<0.001
pCO₂ (kPa)	48	7.1 (1.0)	4.7-8.9	29	6.3 (1.0)	4.9-8.8	<0.002
pO₂ (kPa)	48	9.1 (1.3)	5.6-12.1	29	11.1 (3.3)	5.6-18.3	<0.001
HCO₃ (mmol)	48	30.0 (2.9)	22-38	29	29.6 (4.5)	22-43	ns

*Excludes 3 patients in whom OHS was thought to contribute to respiratory failure from other causes. †Final/latest NSV settings. ‡Latest arterial blood gas taken on waking after overnight NSV. §pvalue comparing OHS to non-OHS. Significance tested using one-way ANOVA for continuous variables and Chi-squared for categorical variables. ns=not significant.

NSV settings and blood gases. The NSV pressures needed in patients with OHS tended to be higher than in patients with other conditions. Despite this, overnight ventilation remained sub-optimal and morning PCO_2 values were higher (Table 2). The choice of inspiratory pressure (IPAP) was generally a compromise between achieving adequate ventilation and patient tolerance. The expiratory pressures (EPAP) were more arbitrary. We tended to use low pressures so that there was a large difference between IPAP and EPAP to reduce the work of breathing.

Geocoding to NZDep96 scores. Ten patients had addresses which could not be geocoded because they were rural delivery addresses or PO Box numbers. The remainder were successfully matched with an NZDep96 score (Figure 2).
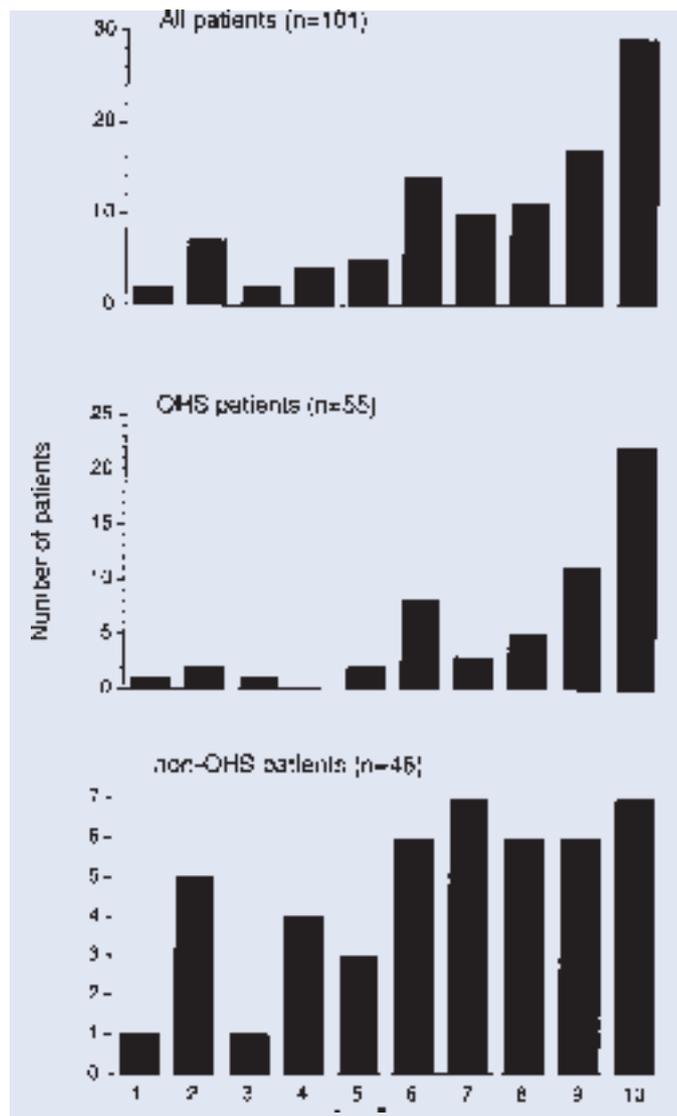

Figure 2. NZDep96 scores. Patients adresses have been matched to meshblocks from the 1996 census. Each meshblock is ranked according to indices of deprivation from responses to the census questions. Score 1 represents the least deprived areas and score 10 the most deprived. NSV=nocturnal support ventilation; OHS=obesity-hypoventilation syndrome.

Discussion

Green Lane Hospital has provided NSV services to the upper half of the North Island and this review probably includes the majority of patients within this region. More recently services have been provided also by other centres within the region. Nevertheless, demand for the Green Lane service has been sustained (Figure 3).
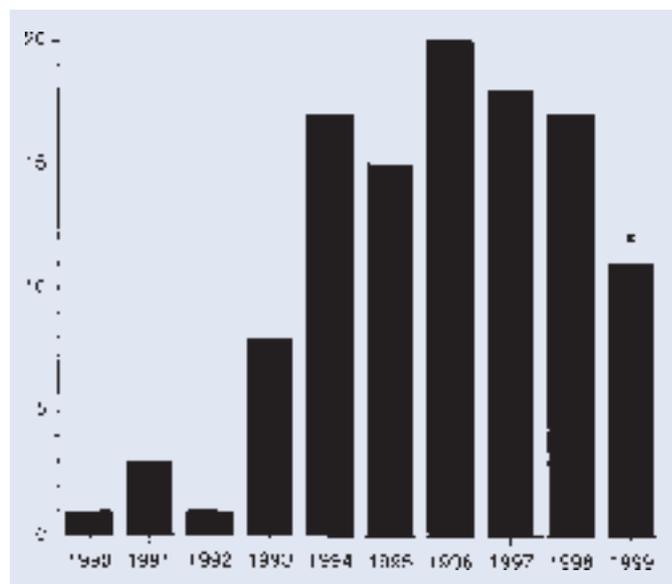

Figure 3. Number of patients commenced on NSV during each year 1990-1999. *Incomplete year (January-October inclusive). NSV=nocturnal support ventilation.

A striking feature is the number of patients whose principal cause of respiratory failure was OHS. The majority were Maori or Pacific Island people and, although the population served by Green Lane Hospital is difficult to define, this appears greater than would be expected from the ethnic composition of our population (approximately 15% and 5% respectively). The prevalence of obesity (BMI >32 kg/m[2]) is high in Maori (males 27%, females 28%) and Pacific people (males 26%, females 47%), compared with New Zealand/Europeans (13% in males and 17% in females).[5] The extent to which this contributes to the over-representation of Maori and Pacific people is unknown.

Apart from its predilection for Maori and Pacific Islanders, OHS is also associated with deprivation. The NZDep96 scores are based on data collected at the 1996 census. Each census meshblock (a geographical area with a median population of 90) is ranked according to nine indices of deprivation.[6] The areas are divided into deciles from the least deprived (1) to the most deprived (10). Over half of our OHS patients lived in areas ranked in the two most deprived deciles (Figure 2). There was a more even distribution of scores in the non-obesity-hypoventilation syndrome patients, although these also tended to cluster at the higher scores. The NZDep96 profile for Maori and Pacific Island people in general is skewed to the right (P Crampton - personal communication), but the distribution seen here is more extreme than expected. Whereas the association of deprivation with obesity-hypoventilation syndrome does not prove causality, there are many possible reasons for the association. Poverty leads to poor eating habits.[7,8] Maori and Pacific Island people, and those living in the most deprived areas are most likely to report being unable to afford to eat properly.[5] Poverty may also contribute to the patients' difficulty in losing weight and explain some of the problems they have with regular follow-up.

Patients with OHS tended to present late, and frequently *in extremis*. Often the presentation was precipitated by an infection, such as cellulitis. Respiratory infection was also commonly reported, but it was difficult to determine the accuracy of this diagnosis. These patients had a high incidence of significant co-morbidity, the extent of which might have been underestimated since we did not have access to all case notes from referring hospitals.

Our choice of inspiratory pressure (IPAP) was generally a compromise between achieving adequate ventilation and patient tolerance. We tended to use low expiratory pressures (EPAP) so that there is a large difference between IPAP and EPAP to reduce the work of breathing. On the other hand, OHS is closely associated with OSA and the EPAP pressures we used may have been too low to prevent upper airways collapse. Our experience is there is little benefit in using higher EPAP pressures, but we are unaware of studies on this issue.

Despite the severe clinical status of these patients and the difficulty in maintaining adequate ventilation, the mortality was surprisingly low. Only four patients are known to have died, and only one may have died of respiratory failure while on NSV. In fact, this patient was probably non-compliant with treatment. This suggests that NSV may improve survival in severe obesity-hypoventilation syndrome.

NSV is not currently funded within the Auckland region. Hence treatment has been offered only to severely ill patients in whom cheaper alternatives, such as nasal CPAP, have failed or were unlikely to work. The cost of the machine, maintenance and follow-up is estimated at $2000-$2500 per patient per year.

The involvement of culturally appropriate support workers may help to improve follow-up and weight reduction. We note with concern that the prevalence of obesity is increasing,[5] and this is likely to result in an increased prevalence of OHS.

In conclusion, NSV services in the upper half of the North Island are mostly unfunded. They have been provided to patients when there is no alternative. OHS is the most frequent cause of respiratory failure requiring NSV. This is primarily a condition of Maori and Pacific Island people and is associated with deprivation and significant co-morbidity. Although this is not one of the accepted indications for non-invasive ventilation, and whilst there is no unequivocal evidence of benefit, patients with OHS appear to have done surprisingly well on NSV. Attempts at weight reduction, however, have generally been unsuccessful. These patients would likely benefit from a comprehensive, fully funded ventilatory support service.

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