Pulmonary rehabilitation in chronic respiratory insufficiency · 5

Home mechanical ventilation

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Definition and goals of home mechanical ventilation

Born during the polio epidemics of 50 years ago, home mechanical ventilation has evolved towards two distinct concepts—a life support system for patients without respiratory independence (high cervical spinal cord injury, end stage neuromuscular disease, or chronic obstructive pulmonary disease), and elective therapy for patients with progressive chronic respiratory insufficiency (mostly restrictive parietal or neuromuscular insufficiency) which prevents acute respiratory failure, preserves function, and increases survival.[1]

Patients on home mechanical ventilation use their respirator intermittently (at least three hours per day) or continuously, with either a tracheostomy, a mouthpiece, or a face or nasal mask, or an external device such as a cuirass or a poncho suit. Consequently, home mechanical ventilation is the longer term application of ventilatory support to patients who are no longer in acute respiratory failure and do not need the sophistication of an intensive care unit.[1]

According to the guidelines for long term mechanical ventilation published by a task force of the American College of Chest Physicians,[2] the goals of home mechanical ventilation must include the following: (1) extend life; (2) enhance the quality of life; (3) provide an environment which will enhance individual potential; (4) reduce morbidity; (5) improve physical and physiological function; and (6) be cost beneficial.

Several meetings and consensus conferences[1 3] have emphasised the importance of identifying the problems relative to equipment, the assessment of individual needs, appropriate levels of care, and adequate long term reimbursement which have been treated differently in most countries.

History

There have been four main periods of development of home mechanical ventilation in chronic respiratory insufficiency.

Long term home mechanical ventilation was introduced into clinical practice more than 50 years ago when the iron lung made survival possible for thousands of patients with poliomyelitis and other diseases associated with neuromuscular ventilatory failure.

After the acute phase of the illness the presence of severe motor damage, sometimes with total respiratory paralysis, required life long respiratory support compatible with management in the home.[4]

The 1950s marked a period of rapid progress in technology, with the development of endotracheal ventilation and tracheostomy, and improved survival after acute respiratory failure,[5] as reported by Robert et al[6] in an important retrospective study which included patients with chronic respiratory insufficiency from several causes. After the eradication of poliomyelitis, improvements of mechanical ventilation techniques were mainly concerned with managing acute respiratory failure using continuous positive pressure ventilation, although there was also interest in phrenic nerve stimulation for patients with cervical cord injuries unable to achieve an adequate level of respiration.[7] Interest was also turning towards patients with chronic respiratory insufficiency due to chronic obstructive pulmonary disease (COPD) and restrictive disorders including chronic post-tuberculosis scanning or chest wall deformities as well as those with respiratory insufficiency due to pure neurological problems. In the early 1970s home mechanical ventilation administered via a mouthpiece or a tracheostomy was compared with long term oxygen therapy (LTOT) in patients with COPD.[8]

By the end of the 1970s the multicentre study by the British Medical Research Council[9] confirmed the results of the Denver group[10] by showing a significant improvement of survival in patients with COPD receiving LTOT compared with a control group. Some months later the publication of the American NOTT study[11] also demonstrated benefit for those receiving LTOT compared with a control group who only received oxygen therapy at night. These results clearly indicated the benefit of LTOT, as well as introducing transtracheal oxygen therapy.[12] In parallel, the important study of Anthonisen et al[13] of intermittent positive pressure breathing (IPPB), admittedly in less severe patients, showed no advantage for IPPB over compressor nebulisers in outpatients with COPD.

During the last 10 years interest in home mechanical ventilation has again increased dramatically. Several factors explain this and include advances in general respiratory care

and rehabilitation, better home care services, and new generations of compact, portable ventilators. Thus, thousands of patients around the world (mainly with a restrictive ventilatory defect) are treated at home by mechanical ventilation. About 6800 patients are receiving home mechanical ventilation in the USA.[14] In France the National Association for Home Respiratory Care (ANTADIR) manages 26 000 patients with respiratory insufficiency in their own homes and estimated in 1992 that more than 5000 chronic ventilator assisted individuals were receiving home mechanical ventilation. At the end of the 1980s a desire for non-invasive mechanical ventilation using improved types of connection devices such as nasal masks increased.[15] More recently, the use of home mechanical ventilation was reconsidered in patients with COPD who were severely hypoxic and hypercapnic with unstable disease and inadequately controlled on LTOT.[16 17]

Physiological basis for chronic mechanical ventilation

PATIENTS WITH RESTRICTIVE LUNG DISEASE
Ventilatory insufficiency with hypercapnia is encountered in most of the diseases associated with a deficiency of the rib cage and chest wall musculature. Hypercapnia frequently develops insidiously without any clear precipitating factor. There is a relation between the extent of respiratory muscle weakness and the degree of hypercapnia, but the association is loose, implying that other factors such as alterations of the static mechanical properties of the respiratory system, respiratory muscle fatigue, and alterations of the central control of respiration all contribute.[18]

In chronic neuromuscular diseases there is reduced distensibility of the respiratory system secondary to the lowering of chest wall and lung compliance. It explains the important reduction of vital capacity, and causes the elastic work linked to breathing to increase. It could also explain the adoption of the typical respiratory pattern of a low tidal volume and a high respiratory rate. If there is no compensatory increase of minute ventilation, hypercapnia results.

Respiratory muscle fatigue may also cause a reduction in inspiratory time and tidal volume and, if it is severe, reduce minute ventilation.[19] In chronic respiratory insufficiency secondary to muscle disease or chest deformity the respiratory muscles are prone to acute or chronic fatigue as their energy requirements are increased above energy intake.[20]

It is also possible that hypercapnia in restrictive lung diseases is the result of an inadequate response of the respiratory centres. It is well known that nocturnal hypoventilation appears early during follow up of the causal disease, before diurnal hypoventilation. Alteration of nocturnal gas exchange could progressively reduce the sensitivity of the central and peripheral chemoreceptors and thus enhance the amplitude of diurnal hypercapnia.

In most of these patients, therefore, nocturnal respiratory assistance may improve the diurnal level of hypercapnia by several mechanisms including an improvement of pulmonary and thoracic compliance secondary to breathing with large tidal volumes during the night, resting of the respiratory muscles,[21] and resting respiratory drive to a normal level following correction of the nocturnal alterations of gas exchange.[22]

PATIENTS WITH COPD
Long term mechanical ventilation causes several changes to occur in respiratory control and performance. The correction of arterial blood gases is one of the principal objectives which determines the adjustment of the settings on the respirator. Home mechanical ventilation is preferably used at night to correct the episodes of arterial oxygen desaturation that occur during rapid eye movement (REM) sleep when the patient breathes ambient air.[23] The improvement in nocturnal PaO_2 can also increase the diurnal PaO_2,[24 25] an effect that can be related to the improvement of alveolar-arterial oxygen gradients and to an improved level of spontaneous ventilation following mechanical ventilation. This could reflect better compliance of the chest wall and lungs, improved respiratory muscle function, increased respiratory drive, a lowering of minute oxygen consumption due to a decrease in the work of breathing, or an increase in the efficiency of the respiratory muscles. If the cardiac output remains unaltered, this may provide a higher PaO_2.[26]

The reduction in $PaCO_2$ is a sign of improvement in alveolar ventilation with mechanical ventilation which can persist temporarily after discontinuing support, and could be associated with a change in the pattern of breathing to one more favourable to alveolar ventilation.[27] The respiratory centre may recover its CO_2 sensitivity which had become blunted.[28] The increase in lung volumes (FRC, FVC) and compliance[29] in emphysematous patients could limit the efficiency of home ventilation and could even aggravate underlying disease.[20] The most important effect seems to be the reduction of respiratory muscle fatigue in emphysematous patients whose respiratory muscles become disadvantaged by the very large lungs, and who are also subjected to an increase in respiratory work due to the increased resistance of the respiratory tract.[25] It is clear, however, that there is a balance between potential benefit for emphysematous patients and the increase in the pulmonary inflation caused by mechanical ventilation.[30] The long term haemodynamic effects of home mechanical ventilation are mainly secondary to the correction of the hypoxaemia and the reduction of consequent pulmonary hypertension. In addition, improved function of the right ventricle can occur,[31] as well as an increase in cardiac output. Secondary polycythaemia is also controlled by the improvement of PaO_2.

The red cell mass is correlated to the mean level of SaO_2 and the lowest nocturnal SaO_2.

In patients with COPD tracheostomy itself could be of value for several reasons[32]: (1) reduction of the anatomical dead space; (2) facilitation of endotracheal aspiration and drainage; (3) facilitation of endotracheal ventilation; (4) possible reduction of the airway resistance with subsequent reduction in the respiratory work; (5) modification of FRC (as tracheostomy makes "pursed lips" breathing no longer possible, a reduction in FRC can occur, which is also assisted by a reduction of airways resistance); (6) inhibition of obstructive apnoeas which are present in the "overlap syndrome"—that is, the association of COPD and the sleep apnoea syndrome.[33]

Selection of patients

The general condition of any potential patient for home mechanical ventilation must be good enough, with reasonably stable disease and sufficient support available at home, to make this procedure worthwhile.[34]

INDICATIONS FOR HOME MECHANICAL
VENTILATION (table 1)
Chronic respiratory insufficiency due to restrictive lung and chest wall disorders

The best long term results[6] have been obtained with home mechanical ventilation and tracheostomy in patients who are otherwise healthy, with disease confined to the respiratory system (often young people or those with slowly progressive conditions)—for example, polio victims (five year survival 95%; 10 years 87%). Other conditions include chest deformities such as kyphoscoliosis, spinal cord injury, some types of neuromuscular diseases[35]—for example, muscular dystrophy, Steinert disease, central hypoventilation syndrome. In patients with restrictive lung disease secondary to tuberculous scarring, with and without sequelae of collapse therapy, long term results are poorer.[6] Some other diseases are unsuitable for home mechanical ventilation such as amyotrophic lateral sclerosis and severe lung interstitial lung disease. Results are similar for patients

with neuromuscular and skeletal disorders with a five year survival rate of 70–80%, and associated with a reduction in admission to hospital for respiratory failure (tables 2 and 3).[6] The obesity-hypoventilation syndrome[36] should be a good indication but warrants further investigation.[37]

Patients with COPD acute on chronic respiratory failure: should they be ventilated?[16]

Respiratory intensive care has a major influence on the survival rates of patients with acute respiratory failure due to COPD.[38 39] In these patients acute respiratory failure is caused by disease progression or an acute event such as bronchial infection, bronchospasm, pulmonary emboli, or cardiac failure associated with chronic airway obstruction.[38] There is general agreement that mechanical ventilation should be avoided in patients with severe chronic respiratory insufficiency as it is associated with great difficulty in weaning.[40 41] The prognosis for those patients with COPD who survive an episode of acute respiratory failure also remains controversial.[42] Hospital mortality has varied from 6% to 38%,[43 44] the need for intubation from 4% to 54%,[43 45 46] and the two year survival from 25% to 68%.[46-50] These differences in outcome may arise from differences in patient selection and definitions of COPD[46] as well as from inconsistencies in defining the causes and severity of acute respiratory failure.[48] In most series COPD has only been defined by clinical criteria or by radiological signs of hyperinflation, and pulmonary function tests were not available for all patients.[45] Asthmatic patients have not always been excluded from these studies. The precipitating cause of the acute respiratory failure is also important, as the mortality rate secondary to infection (20%) is very different from that secondary to heart failure (40%).[47] There is also disagreement about defining the severity of acute respiratory failure, although it is generally accepted that this requires initial arterial blood gases breathing air of a $PaO_2 < 7$ kPa (<50 mm Hg) and $PaCO_2 > 7-9$ kPa (>50–70 mm Hg). Both criteria appear to be good indicators of the severity of acute respiratory failure.[43] The severity of acidosis on admission, expressed as arterial pH, correlates better with survival than does the absolute level of $PaCO_2$.[49] Mortality increases markedly if pH falls below 7·23. This is supported by other studies[41] where a good correlation was found between the severity of respiratory acidosis and the absolute PaO_2 and the need for ventilatory support, with the increased mortality risk. However, in a retrospective study of 135 patients with COPD complicated by acute respiratory failure, who were treated primarily with controlled oxygen therapy, Jeffrey et al[44] found that the death rate increased with the age of the patient, but did not correlate with the severity of hypoxaemia on admission when the patient was breathing air, but was significantly higher in those patients in whom the pH fell below 7·26 during controlled oxygen therapy. Age alone, however, does not

Table 1 Diagnosis suitable for consideration for long term mechanical ventilation (after reference 1)

Site or type of defect	Favourable diagnosis	Unfavourable diagnosis
Ventilatory drive	Central hypoventilation syndromes Ondine's curse Arnold-Chiari malformation	Cerebrovascular accident (stroke) Malignancy
Neural transmission to the ventilatory muscles	High cervical spinal cord injury Poliomyelitis Guillain-Barré syndrome Bilateral phrenic nerve paralysis	Amyotrophic lateral sclerosis Multiple sclerosis
Ventilatory muscles	Muscular dystrophy Congenital myopathies	
Thoracic cage	Kyphoscoliosis Post-thoracoplasty	
Lungs and airways	Bronchopulmonary dysplasia	Chronic obstructive pulmonary disease Bronchiectasis Cystic fibrosis Interstitial lung disease

Table 2 Results of long term mechanical ventilation with tracheostomy in neuromuscular diseases (after reference 35)

References	Treatment	No of patients	Results (% survival)
Splaingard, *et al*	INPV	40	5 years (76%)
Splaingard, *et al*	IPPV	47	3 years (67%)
Robert, *et al*	IPPV	41 (polio)	5 years (95%)
		13 (myopathy)	5 years (62%)
Goulon, *et al*	INPV or IPPV	110	5 years (80%)

INPV—intermittent negative pressure ventilation; IPPV—intermittent positive pressure ventilation.

seem to be a determinant of survival.[50] The influence on outcome of mechanical ventilation also remains unclear.[51] Hudson[52] divided several series of patients with COPD complicated by acute respiratory failure into two groups: those treated before and after 1975. The overall survival of patients treated before 1975 was 72%, but this increased to 91% after 1975. In a prospective study Martin *et al*[48] found a 94% survival in hospital and a 72% survival at two year follow up, but he included patients who had less severe respiratory failure and some with only acute bronchitis as the precipitating illness in whom there was no indication for mechanical ventilation. Controversy remains, however, if we consider the series of Vandenbergh *et al*[53] who, in 1968, described a survival rate of 78% with a low rate of mechanical ventilation (8%). In contrast, Petty[10] included only patients needing respiratory assistance and found a 76% survival rate before 1975 compared with 87% in those treated after 1975. Mechanical ventilation itself could introduce a factor of prognostic significance.[54] Thus, Sluiter *et al*[49] presented data supporting the concept that the characteristics of patients selected for mechanical ventilation were a more important factor in determining the prognosis than mechanical ventilation itself.

Thus, most patients with COPD survive an episode of acute respiratory failure, even if the subsequent prognosis for survival is poor and similar to that of other patients with COPD without an episode of acute respiratory failure, both being strongly related to the severity of the underlying process.

Chronic daytime respiratory failure

Mechanical ventilation is commonly considered in severe hypoxic and hypercapnic patients with COPD who have unstable respiratory drive and abnormal blood gas tensions leading to frequent episodes of decompensation with acute respiratory failure despite using long term oxygen therapy. Mechanical ventilation should be considered in the following situations.

1. During the episode of acute respiratory failure[25 54]:
(a) The patient had to be intubated:
   (i) It was possible to wean him from the respirator: nasal mask ventilation may be used to facilitate the period immediately following extubation[55] and may be continued on a long term basis if hypercapnia persists or worsens.
   (ii) It was impossible to wean and extubate the patient: a tracheostomy is often performed when difficulties arise in weaning from the respirator or if the period of mechanical ventilation is prolonged—for example, in patients with several previous episodes of acute respiratory failure whose status is declining and in whom a new episode of acute respiratory failure could be fatal (12% of cases in the ANTADIR study[56]) the tracheostomy is generally maintained after the episode of acute respiratory failure with long term mechanical ventilation, or if the status of the patient is less severe the tracheostomy may be closed and followed by LTOT or nasal mask ventilation according to the baseline $Paco_2$.
(b) The patient was treated only by nasal mask ventilation during the acute episode. Nasal ventilation may be maintained if the status of the patient is poor or declining. If nasal ventilation fails, a tracheostomy may be performed and followed by long term home mechanical ventilation via the tracheostomy. The invasive character of tracheostomy is a stimulus for non-invasive techniques such as nasal mask ventilation in those patients.[57]

2. Outsite an episode of respiratory failure nasal mask ventilation is nowadays proposed if the patient is deteriorating in spite of LTOT, with frequent readmissions into hospital, and worsening arterial blood gases. A European long term controlled study is being conducted to evaluate the real benefit of a long term nasal mask ventilation *v* LTOT in patients with severe COPD.[58]

Chronic severe nocturnal hypoxia

Severe nocturnal hypoxia can occur without significant daytime hypoventilation in either restrictive or obstructive pulmonary disease. The symptoms are early morning headaches, tiredness, personality change, daytime sleepiness, or frequent arousals at night, and are often unrecognised.[26] Polycythaemia and pulmonary hypertension may be present by the time of diagnosis, and they provide valuable clues to the presence of nocturnal hypoxia.[35]

In common practice, the overlap

Table 3 Clinical status of 222 patients on home positive pressure ventilation via tracheostomy (adapted from references 1 and 35)

	Polio	Myopathy	Kypho-scoliosis	Sequelae of tuberculosis	COPD	Bronchiectasis
No of patients	41	13	53	55	50	10
Hours/day on ventilator (mean)	15	17	11	12	12	20
Frequency of suctioning (times/day)	<1	<1	4	10	15	50
Days acutely ill at home/year	7	7	13	18	23	66
Days hospitalised/year	3	7	6	8	12	22
Able to leave home >3 times/week (%)	38	—	33	24	25	0
Five year survival (%)	95	62	77	70	18	0

syndrome[33] associates COPD and sleep apnoea. Relief of upper airway obstruction may be necessary, but if the hypoxia is due at least in part to hypopnoeas and apnoeas, home mechanical ventilation should be considered. In the ANTADIR study[56] there was a better prognosis for overweight patients undergoing home mechanical ventilation via a tracheostomy for the overlap syndrome. These results were confirmed in a recent multicentre study using nasal mask ventilation for patients with respiratory insufficiency for several causes.[59]

Methods

POSITIVE PRESSURE VENTILATION

Home mechanical ventilation employs mainly intermittent positive airway pressure ventilation, which can be applied intermittently by a nasal mask or mouthpiece or via a tracheostomy. Intermittent positive pressure breathing (IPPB) is used for short periods using pressure cycled ventilators[60–62] in order to deliver aerosolised bronchodilators. Intermittent positive pressure ventilation (IPPV) is used for longer periods of time with volume cycled ventilators.[26]

IPPV/IPPB

Mouth IPPV with volume cycled respirators was popular in Europe in the 1970s, but it rapidly became apparent that it did not provide a real benefit for patients.[8] It differs from IPPB whose main role is the administration of bronchodilator medication.[26]

The major disadvantage of mouth IPPV is that it can only be used for short periods of time, up to 2–3 hours per day, because of difficulties in achieving an adequate mouth seal, particularly during sleep. It can be useful, however, in severe neuromuscular diseases, especially spinal cord injuries.[63] The beneficial effects on arterial gases wear off after two hours.[60] The activity of the respiratory muscles persists during ventilation, even if inspiration is self triggered,[61] which explains the increase in oxygen consumption.[62] The long term effects of the mouth IPPV in patients with COPD have not been docu-

mented by any study comparable to that of Anthonisen *et al*[13] for IPPB, although there are many uncontrolled or retrospective studies.[8 64]

In the French home mechanical ventilation programmes, the number of patients treated with mouth IPPV has decreased greatly in recent years, initially with an increase in the use of tracheal ventilation[64–67] and, more recently, nasal mask ventilation.[15 68]

Home mechanical ventilation via tracheostomy (HMVT)

The major interest of HMVT is the technical opportunity for prolonged mechanical ventilation sessions, especially during the night. Apart from the psychological effects, the existence of a tracheostomy in itself reduces the resistance of the upper airways and respiratory dead space, facilitates aspiration, and alleviates some work of breathing.[26 32]

The disadvantages are that it is invasive, requiring more support and ideally an adherence to a general programme of rehabilitation,[14] and requires home care support and has a socioeconomic cost. The risks include tracheal stenosis, although according to the results of the multicentre study coordinated by the ANTADIR the incidence is low, with 14 cases reported in 256 patients with COPD.[56]

The long term results of HMVT have been evaluated by Robert *et al*[6] who compared the outcome of patients with different causes of chronic respiratory failure treated with HMVT. In 1973 studies with HMVT suggested that the prognosis in patients with COPD was worse (10 year survival: 35%) than in patients with a restrictive chest wall or neuromuscular condition. A more recent study by Robert *et al*[66] confirmed a very poor prognosis for severely disabled patients with COPD who were treated by HMVT compared with patients with restrictive disorders treated by the same methods. These results must, however, also be evaluated to include an attempt at improving the comfort of the patient in terms of reducing the frequency of hospital admissions.[64 67] In the recent series reported by Robert *et al*[66] the five year survival was 30% and the 10 year survival 8% for a population of 112 patients with COPD treated by HMVT (fig 1).

We have conducted a similar retrospective multicentre study in a large population of 259 patients with COPD treated with HMVT, including 58% "blue and bloated," 20% "pink and puffing," and 22% intermediate type.[56] Their survival (fig 2) at five years was 42% and at eight years was 22%, which was between that of Robert's study and the previous reports of long term oxygen therapy alone reported in the NOTT and British Medical Research Council (BMRC) trials[9 11] which comprised patients with less severe COPD. The survival of the patients in our series was better than for the patients in the BMRC trial until the fourth year when the survival curves converged. As the severity of the condition seems identical in the two
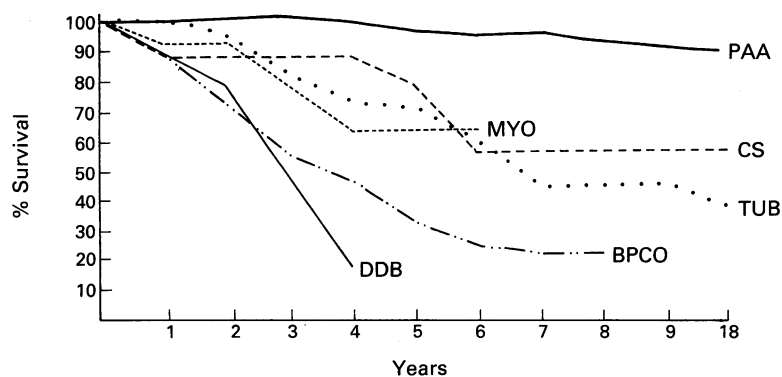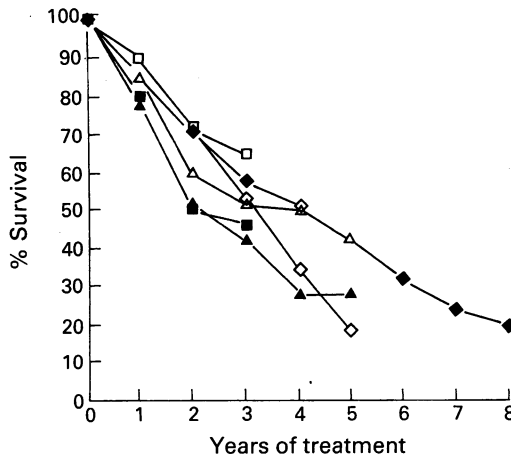

Figure 1   Actuarial survival of patients under home mechanical ventilation with tracheostomy. PAA—41 post-polio patients; MYO—13 patients with myopathies; CS—53 patients with kyphoscoliosis; TUB—55 patients with sequelae of tuberculosis; BPCO—50 patients with chronic obstructive disease; DDB—10 patients with bronchiectasis. Reproduced from reference 6 with permission.

Figure 2 Actuarial survival of patients with COPD receiving home mechanical ventilation with tracheostomy (HMVT): Comparison between study by Robert et al[6] (◇) and the retrospective ANTADIR series[56] (◆). The results are also shown for patients in the BMRC study[9] (△ oxygen, ▲ controls) and the NOTT study[11] (■ 12 hours/day oxygen, □ 24 hours/day oxygen).

patients with skeletal chest wall disease or with neuromuscular disease NIPPV improves the arterial blood gases, prolonging survival and improving their quality of life.

Survival rates on home IPPV are much lower for patients with chronic airflow obstruction than for those with restrictive chest wall or neuromuscular disease. The 10 year survival rate is approximately 10%[64-66] with further admissions to hospital and some improvement in right heart failure and arterial blood gases.

This type of ventilation could be proposed as a preventive treatment in patients with severe COPD with fluctuating hypercapnia and episodes of acute respiratory failure, avoiding the need for a tracheostomy or to facilitate weaning.[55] NIPPV has also been successfully used during episodes of acute respiratory failure in patients with COPD,[72 76] as well as in patients with restrictive problems.[70-72] In patients with COPD, however, there are few data assessing the value of NIPPV on a long term basis.[57] In an open study, 12 patients treated with NIPPV for 12 months achieved an improvement in PaO_2 and diurnal $PaCO_2$. An increment in the total sleep time was also observed and was associated with improved sleep efficiency.[17]

NIPPV relieves upper airway obstruction and has been proposed for patients with sleep apnoea who do not tolerate nasal CPAP.[34] In severe diffuse bronchiectasis where HMVT appears to be ineffective, NIPPV should be tested in spite of the high airway resistance of these patients and their considerable bronchial hypersecretion. Good results have been obtained in patients with cystic fibrosis.[77] However, NIPPV fails to prevent worsening of chronic respiratory insufficiency in patients with Duchenne muscular dystrophy.[75]

An algorithm summarising the indications for HMVT, LTOT, and NIPPV, respectively, is presented in fig 3.

studies, it appears that a more invasive approach to these patients is worthwhile, especially if LTOT is not successful. HMVT should therefore be reconsidered in the most severely affected patients with COPD as it may prolong survival, particularly if target levels of SaO_2 are not achieved with LTOT.

Nasal intermittent positive pressure ventilation (NIPPV)

Initial experience with NIPPV during sleep has been promising and improvement in ventilatory failure has been recorded particularly in patients with restrictive and neuromuscular diseases.[68] NIPPV has been intensively studied during the past five years[57] with good results in acute respiratory failure[69-72] as well as in chronic respiratory failure, especially in patients with chronic restrictive respiratory insufficiency mainly of neuromuscular origin.[73-75] The nasal mask which is commonly used is similar to the device used to treat the sleep apnoea syndrome. In a recent study NIPPV at night also provided good results in patients with kyphoscoliosis and lung damage resulting from tuberculosis.[15] Clearly, in

Figure 3 Route of decision making as to best treatment for patients with chronic respiratory insufficiency (CRI). LTOT—long term oxygen therapy; NIPPV—nasal intermittent positive pressure ventilation; HMVT—long term home mechanical ventilation with tracheostomy; TTO—transtracheal oxygen therapy; ⊕—effective treatment; ⊖—ineffective treatment. After reference 88.
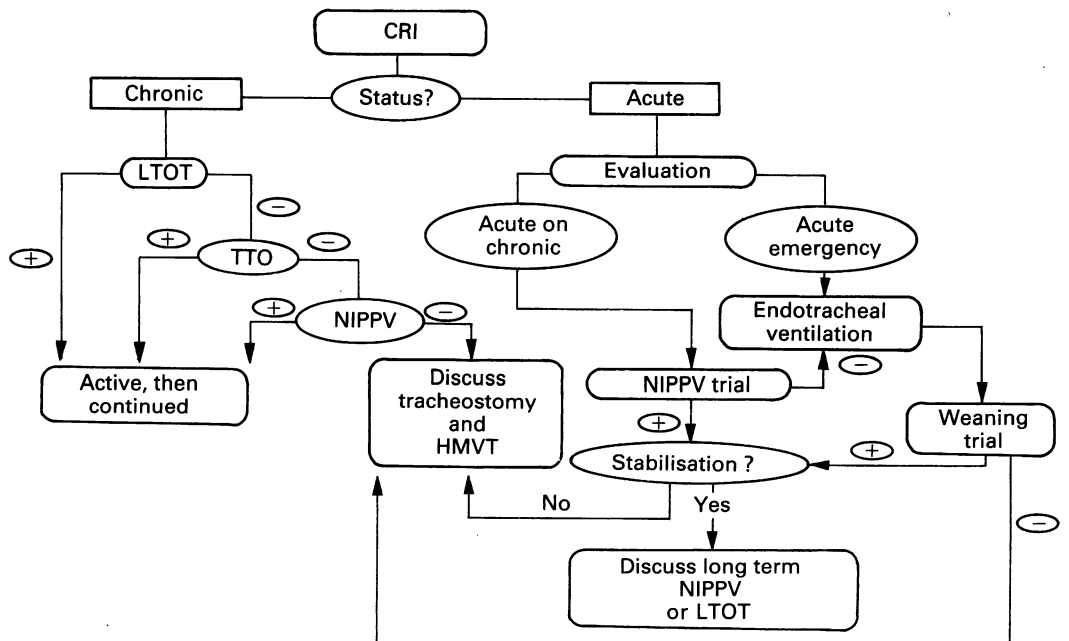
NEGATIVE INTERMITTENT PRESSURE
VENTILATION (NIPV)
NIPV was introduced by Dalziel in 1832, but
its real use began with Drinker who built the
first reliable iron lung in 1928. Emerson
further improved it and its real use was
during the polio epidemics of 50 years ago,[78]
reducing mortality by 50%.[26 74] NIPV has
been used in other neuromuscular diseases,
and appeared useful in cases of muscular
dystrophy, kyphoscoliosis, and also in
patients with post-tuberculosus fibrosis.[79]

Use of NIPV has recently been reconsid-
ered for patients with COPD through new
devices using light shells and jackets (poncho,
wrap, or cuirass) applied around the thorax
and upper abdomen.[80]

Several trials are being conducted to ascer-
tain whether the respiratory muscles can be
rested with benefit. Preliminary results show
that there is a real effect on the level of
dyspnoea, on the control of diaphragmatic
activity, and on improving the respiratory
muscle strength.[79–81] NIPV seems better toler-
ated by patients with type B COPD than by
those with emphysema in terms of levels of
dyspnoea. However, other studies[82] failed to
confirm these early results.

Apparatus

VENTILATORS
Ventilators for IPPV are generally used for
long periods of time, often overnight. Most of
them are volume cycled and they should be
simple, reliable, and easy for the patient to
adjust. For patients with COPD who are
generally only partially dependent on their
machine, the need for a battery operated
ventilator is mandatory, which should also be
light and portable. Both high and low pres-
sure alarms are needed to indicate airflow
obstruction, disconnection, or failure of the
ventilator. These alarms must be independent
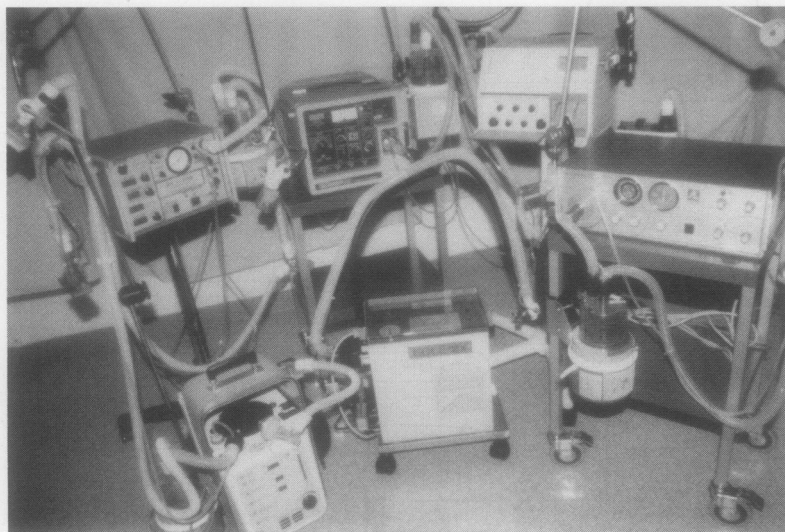of any external power source.

Figure 4   Volume cycled ventilators available in Europe. From left to right: (front) Airox Home (BioMMS), Eole 2A (SAIME), Monnal D (CFPO); (back) PLV 100 (Lifecare), Companion 2801 (Puritan Bennett), EV 800 (Draeger).

In Europe several types of ventilator are
commonly used, the Monnal D (CFPO)
respirator being the most popular. Many
other IPPV ventilators are available (fig 4).
Negative pressure generators are also avail-
able including the Myoclet in France, the
Newmarket pump in the UK, the Emerson
iron lung, the Portalung, and the Lifecare
170C which has recently been improved in
the USA.

Simple patterns of ventilation are used with
controlled rather than assisted or controlled-
assisted mode (VT 10–15 ml/kg with a cuffed
tracheostomy tube, 15–20 ml/kg using nasal
mask; inspiratory/expiratory ratio (I/E) 1/2; a
respiratory rate of 12–18/min; FIO_2 < 35%).
Supplemental oxygen can be provided by
cylinders, liquid oxygen, or a concentrator.

In addition to nasal CPAP generators,
bilevel pressure preset ventilators (BIPAP®)
are now available which allow the ventilator
to cycle between a preset inspiratory and
expiratory airway pressure.[83] They are more
comfortable to use than conventional respira-
tors which provide NIPPV, and they can also
spontaneously adjust for leaks. Bilevel
pressure preset ventilator devices are under
evaluation for long term use[84] and could be as
effective as NIPPV for several types of
cardiorespiratory failure.[85]

CONNECTION TO THE RESPIRATOR
Tracheostomy (fig 5)
The use of a tracheostomy enables IPPV to
be used for longer periods than with a mouth-
piece, so nocturnal or continuous assisted
ventilation is possible.

Uncuffed or deflated tubes permit speech,
provided there is a speaking valve attach-
ment,[74] and allows spontaneous breathing in
the event of incoordination between patient
and ventilator or ventilator failure, although
flow of air through the pharynx may be
uncomfortable. It is possible to achieve a
sufficient improvement of arterial blood gases
with an uncuffed cannula, provided the
respirator is volume cycled and can deliver
about three times the volume of air that
would be required using a cuffed tube.[74]

A cuffed tube is necessary if aspiration of
pharyngeal secretions is a problem, or if the
respiratory status and the level of compliance
and airway resistance needs airtight ventila-
tion. At home the presence of a cuffed tube is
more difficult to manage, and must be
deflated to permit speech. The patient must
be educated to deflate and inflate as well as to
change his tracheostomy tube, to clean it, and
to suck out his own secretions as carefully as
possible to avoid tracheal irritation. He must
be told to ask immediately for assistance if he
cannot replace the cannula as the stoma can
close within hours. Conversely, difficulty in
replacing a tracheostomy tube may be the
first symptom of a tracheal stenosis caudal to
the stoma.

A silver tracheostomy tube is no longer
recommended as plastic tubes are becoming
more available. When the patient returns
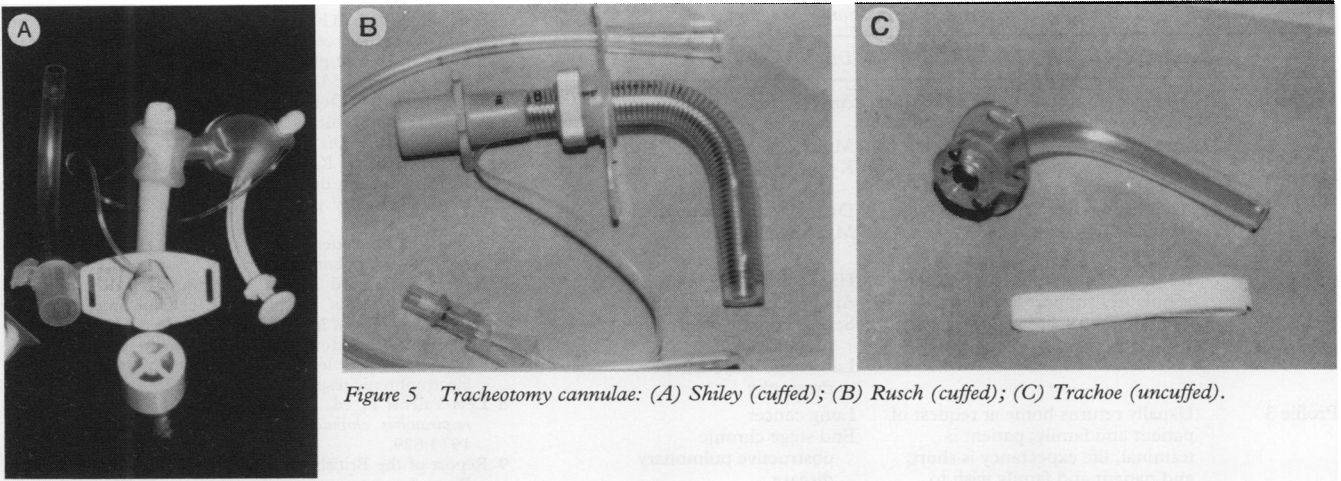from the intensive care unit he is generally

Figure 5   Tracheotomy cannulae: (A) Shiley (cuffed); (B) Rusch (cuffed); (C) Trachoe (uncuffed).

equipped with a cuffed tracheostomy tube, which will be deflated as soon as possible to minimise the chances of later tracheal stenosis. If the respiratory status is compatible with it, the patient will then be ventilated through a speaking tracheostomy cannula.

Humidification of the inspired air is necessary during IPPV and after tracheostomy. The presence of a tracheostomy requires suction equipment at home.

Management of a tracheostomy at home presents considerable psychological and nursing problems both for the patient and attendant. It requires an educational and rehabilitation programme to facilitate the return to home and rehabilitation of the patient.[35 86 87] One of the goals is for the patient to manage his own tracheostomy. Modern tracheostomy tubes have an inner cannula which should be changed and cleaned twice daily. The outer cannula need only be changed once or twice a week, except if the patient produces a lot of secretions. The technique of changing the tube should be understood by the patient and his relatives, but until the tracheostomy tract is fully developed the outer cannula should be removed only by experienced medical or nursing staff.

Nasal facial mask ventilation[15]
The development of comfortable and airtight standard nasal and facial masks has enabled IPPV to be given via the nose. The same masks can be used as with nasal CPAP (fig 6), but it is often necessary to make customised nasal masks which are better tolerated and which minimise air leaks.[72] No humidification is required on the breathing circuit.

From hospital to home
A successful return home by the patient treated with home mechanical ventilation requires several steps.

IDENTIFYING THE PRIME CANDIDATE
The patient must be relatively stable on a day-to-day basis without any gross fluctuation that requires inpatient care.[35] Depending on the cause of the respiratory insufficiency, two profiles can be encountered[86]: (1) the patient is unable to maintain adequate spontaneous ventilatory function over prolonged time periods; and (2) the patient has stable ventilator failure but is completely dependent on continuous ventilatory support.

In clinical practice two distinct situations are present: (1) the patient who has stayed in the intensive care unit, has a tracheostomy, but cannot be weaned from the respiratory support; and (2) the patient with chronic progressive hypoxia and hypercapnia in whom non-invasive assisted ventilation might be more useful than oxygen therapy alone. The majority of the patients are of this type, with a slowly progressive worsening of their hypercapnia and clinical status.

HOME CARE TEAM MEMBERS
Once the physician, patient, and family have decided that a return to home is feasible, an overall assessment of the patient and home is

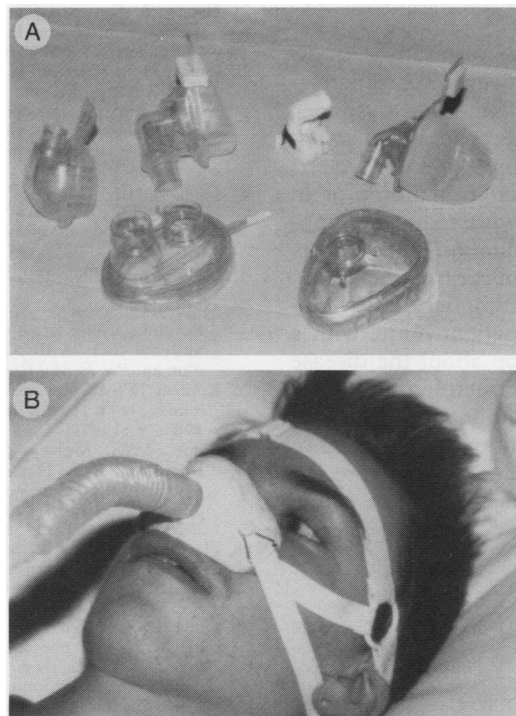Figure 6   Masks for nasal ventilation: (A) industrial masks; (B) moulded mask.

Table 4 Patient profile types requiring assisted ventilation in the home.

	Group description	Diseases involved
Profile 1	Mainly composed of neuromuscular and thoracic wall disorders; particular stage of disease process allows patient certain periods of spontaneous breathing time during day; generally require only nocturnal mechanical support.	Amyotrophic lateral sclerosis Multiple sclerosis Kyphoscoliosis and related chest wall deformities Diaphragmatic paralysis Myasthenia gravis
Profile 2	Requires continuous mechanical ventilatory support associated with long term survival rates.	High spinal cord injuries Apnoeic encephalopathies Severe chronic obstructive lung disease Late-stage muscular dystrophy
Profile 3	Usually returns home at request of patient and family; patient is terminal, life expectancy is short, and patient and family wish to spend remaining time at home; patients usually pose significant management problems in the home due to rapidly deteriorating condition.	Lung cancer End stage chronic obstructive pulmonary disease

initiated, the best primary care givers being the close members of the family (table 4).[86]

The professional staff involved in the continued care should include a complete team (primary physician, clinician psychologist, respiratory therapist, nurse, social service or home care planner, home care and equipment supplier) which acts in conjunction with the hospital based team. In France the home care service is divided into public and private organisations, the most important being ANTADIR, which treats 25 000 patients in their homes, and 33 regional associations which have the responsibility of LTOT (partly with the private home care associations) for nearly all patients on home mechanical ventilation.

Home mechanical ventilation and rehabilitation

Despite medication and respiratory assistance, many patients with severe chronic respiratory insufficiency suffer from dyspnoea which limits their physical capacity and activities of daily living. Methods to improve the ability of patients to function at home or in their work environment with reduced symptoms (the goals of rehabilitation) have therefore become accepted forms of treatment.[26]

The key elements of pulmonary rehabilitation[87] which will get the patient home with a respirator are (1) education about the disease and management of its treatment—that is, home mechanical ventilation; (2) physical therapy; (3) exercise conditioning (adapted to those severely disabled patients) and breathing retraining; (4) psychosocial counselling; and (5) vocational training.[88]

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