Pulmonary rehabilitation in chronic obstructive pulmonary disease (COPD) with recommendations for its use

Report of the European Respiratory Society Rehabilitation and Chronic Care Scientific Group (S.E.P.C.R. Rehabilitation Working Group)

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Although smoking, the major risk factor for COPD, seems to be declining in the Western World, its consequences will last for decades.

The last 30 years have seen widespread efforts to define the components and role of pulmonary rehabilitation in COPD patients; the task for the 1990's will be an extensive multidisciplinary application of scientific principles to pulmonary rehabilitation. In this perspective the empiric approach will be replaced by therapeutic principles aimed to achieve specific benefits for the individual patient, and to define the role of potential outcomes of pulmonary rehabilitation. Decreased mortality and morbidity may seem to be the most important outcome of a rehabilitation programme, but other issues such as physical and mental health, social and role functioning and perception of well-being can play a pivotal role to the individual response of a patient enrolled in a long-term rehabilitative programme. The effects of pulmonary rehabilitation on mortality will probably never be elucidated conclusively because the answer would require studies on large populations for many years in comparison to a control group.

Pulmonary rehabilitation aims to restore patients to an independent, productive and satisfying life and prevent further clinical deterioration to the maximum extent compatible with the stage of the disease. This goal may be accomplished, without materially improving lung function, by helping the patients to become more aware of their disease, more actively involved in their own health care and more independent in performing daily care activities, attempting to reverse the disability from disease.

Optimum drug therapy and smoking cessation are essential pre-requisites. An understanding of the factors limiting exercise, the effects and evaluation of retraining programmes will determine the selection of patients most likely to benefit from rehabilitation. Attention to psychosocial management, nutritional defects and physiotherapy form part of the treatment schedule. In respiratory failure, oxygen supplementation would appear essential and in advanced stages ventilatory support increases the therapeutic options. This statement crystallizes present views from a Working Party of the European Respiratory Society Rehabilitation and Chronic Care Scientific Group (formerly SEPCR Rehabilitation Working Group).

The rationale of pulmonary rehabilitation

The aims of pulmonary rehabilitation are: 1) a decrease of physical and psychological impairment due to the disease, 2) an increase in physical and mental fitness and performance and 3) maximal social re-integration of the patient to lower the handicap.

The methods to achieve these aims are incorporated into a programme of: a) an accurate diagnosis of the disease and the functional limitations, b) education about the disease to include its pathophysiology and the use of medication, c) physical training to improve physical fitness and d) psychosocial support. Rehabilitation treatment pre-supposes maximal medical treatment with cessation of smoking before the start of any programme to be conducted by a specialist multi-disciplinary team. Exercise testing is the most important single test used to plan the rehabilitation programme.

In light to moderate obstructive disease (FEV1 usually ≥60% of predicted normal values), exercise is limited by the cardiovascular system and/or peripheral muscle function. The rehabilitation programme is based on endurance training at high heart rate levels.

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Patients with ventilatory limitation (FEV1, 40–60% predicted normal values or less) often show failure of the respiratory pump. Rehabilitation treatment will contain exercise training ergonomics, calisthenics and breathing exercises. Many patients need nutritional advice. Respiratory failure requires special consideration. Psychological support and restoration of self confidence is best conducted around the home environment with involvement of family members and care givers. The benefits of pulmonary rehabilitation tend to decline after the initial treatment period. Follow-up programmes are essential.

Selection of patients

Any patient with symptomatic COPD should be considered. The Consultant Physician or Medical Director of rehabilitation should assess patient enthusiasm and commitment, family circumstances, clinical severity and stability of disease and appraise financial resources. Exclusion criteria will include distracting disease such as severe cardiac failure, cancer and neurological disability, poor patient commitment, adverse family circumstances and limited financial resource.

Respiratory rehabilitation is conducted through in-patient or out-patient programmes and for the severely disabled, at home. In-patient programmes are required for individuals with very limited walking capability, when clinical diagnosis and assessment are uncertain, for unstable disease and for the instruction of patient and family in the methods. Out-patient programmes benefit the symptomatic but less severely disabled; they must be fitted around a job and are ideally suited for periodic monitoring of progress. Home based programmes will follow hospital in-patient or out-patient training. Special facilities will be required for the very severely disabled who infrequently leave the home such as for long term oxygen therapy (LTOT), ventilatory support, nursing and psychosocial management. End-stage terminal patients need specialist care which should be capable of application to the home.

Evaluation of rehabilitation therapy

Limitation of exercise capacity is not the same as disability which is defined as the "total effect of impairment on the patient's life". Tests of rehabilitation effect must recognise the multiplicative nature of affective factors and must be adjusted to suit individual patient capabilities. While some degree of stratification in terms of disease severity, age and sex is required, of most importance is the establishment of stable base lines, good control data and a realistic time scale. Very short periods of evaluation of days or a few weeks will afford limited responses. Quarterly or half yearly intervals are better. Assessment will be on an individual or group basis using several parameters. For individuals, pre-treatment intervals provide the control data, for groups pre-treatment intervals or specifically selected control groups can be used. Tests must be applied in random order to avoid bias. Multivariate analysis rather than univariate analysis is more appropriate. It is also important to evaluate failure to attain physiological goals.

The evaluation of exercise programmes aims to answer the following two questions:
1. Does treatment improve exercise tolerance?
2. How is the improvement achieved?

Three types of measurement are used:
1. Progressive incremental exercise tests
2. Timed walk tests
3. Endurance at submaximal exercise

Progressive incremental exercise (PIE) is performed on a treadmill or bicycle ergometer with increments adjusted to patient capability. End points are maximal oxygen uptake (VO2max) or maximal power output of the equipment. Anaerobic threshold is measured where possible by analysis of the slope of the VO2/VO2max relationship. Breathlessness should be assessed using visual analogue scales or the categorical (modified Borg) scale. Wide intersubject variability will be a feature of the statistical analysis.

Sub-maximal PIE tests are less useful and should recognise the very wide subject and inter-subject variability, particularly in control data. Endurance at submaximal exercise is a better index but the conduct of the tests and selection of equipment will once again bestow peculiarities to the data.

Timed walk tests are indirect measurements of exercise capability but have the advantage of simplicity and minimal requirements of a corridor and trained supervisor. Six or 12 minute intervals are selected according to disease severity: distance and breathlessness are measured. Their main disadvantages are susceptibility to motivation of patient and supervisor, and inability to compare data between centres owing to the plethora of circumstances under which the tests are performed. Within individual centres stringent test/retest criteria are possible once learning effects on initial walks are taken into account. An analysis of failure of rehabilitation treatment will help dissect the components of individual disability. Factors such as programme adherence, motivation, psycho-neuroligical disease, socio-economic factors, severity of disease and frequency of intercurrent exacerbations will be revealed. An indication of the likely success of future rehabilitation attempts can be measured.

Mechanisms of exercise limitation

Decreased exercise capacity in COPD patients is measured by a reproducible decrease in maximum oxygen consumption (VO2max). The mechanism is multifactorial: 1) alteration in pulmonary mechanics which increases the work of breathing and leads to decreased ventilatory capacity and respiratory muscle fatigue, 2) impaired efficiency of pulmonary gas
exchange resulting in increased ventilatory requirement and 3) pulmonary vascular disease which inhibits cardiovascular accommodations to exercise. Maximum exercise ventilation (Vmax) is dictated by disturbed lung mechanics. In contrast to healthy persons, COPD patients increase ventilation during exercise, mainly by increasing respiratory rate. Expiratory flow during heavy exercise often impinges on the flow-volume loop recorded during a resting forced vital capacity manoeuvre. Expiratory flow limitation results from increased airway resistance and reduced elastic recoil of the lung. Functional residual capacity rises, forcing the respiratory muscle to operate on a less favourable length tension relationship. Premature fatigue sets in. The latter is enhanced by muscle wasting. Exercise hypoxaemia may further impair ventilatory muscle performance.

Mismatching of ventilation to perfusion leads to hypoxaemia and high physiological dead space (V̇Ed/V̇t) which increase the ventilatory requirement (V̇E for a given level of exercise).

Pulmonary artery pressure rises sharply on exercise and cardiac output may be prevented from an adequate response. Such effects have variable consequence on right heart muscle performance. Insufficient information is at present available to determine the effect of pulmonary vascular pathology on exercise limitation. Physiological training references are obtained at levels of exercise above the anaerobic threshold. The latter is therefore an important determinant of exercise training capability but cannot be reached by many patients with severe disability. Although their therapeutic responses will be less, benefits are nevertheless realised even though they are as yet not fully explained. Rehabilitation programmes intended to increase the exercise tolerance of COPD patients are designed after determination of the functional impairment of the pulmonary and cardiovascular systems.

**Exercise prescription**

Exercise tolerance improves in COPD patients through programmes of supervised exercise. It has yet to be established, however, whether these improvements are based on physiologic changes in the ability to perform work or whether psychological factors play the major role.

In normal subjects, training increases the oxygen uptake at which lactic acid begins to accumulate in the blood (the anaerobic threshold) by 25-40% and the maximal oxygen uptake (V̇Ȯ2max) by 5-20%.

Exercise training does nothing to reverse the basic disease process in COPD patients: resting lung mechanics and gas exchange efficiency do not improve. Conflicting results in terms of improved exercise endurance have been obtained after training respiratory muscles. The ventilatory requirement (the ventilation for a given level of exercise) can be lowered by training (through less CO₂ production from bicarbonate buffering of lactic acid) permitting the same work load with less dyspnoea. Despite such benefits, no clear change in pulmonary hypertension or arterio-venous PO₂ difference have been observed.

If no major contraindications (hypercapnia, arrhythmias or systemic hypertension) emerge after a preliminary exercise test the training programme should be initiated under the supervision of an expert therapist. It must be realised however that the consequences of such prescription in COPD patients, despite the goal of achieving a physiologic training effect, are not yet fully understood. Training at a work rate associated with lactic acidosis is more effective in inducing a training effect than at a work rate not associated with lactic acidosis. Thus, a programme involving exercise of 30-45 minutes per day, 3-5 days per week for 5-8 weeks at a work rate inducing substantial lactic acidosis seems a reasonable strategy. A maintenance programme of exercise follows which must be part of the rehabilitation process.

In patients with severe COPD, supplemental oxygen during training should be considered; oxygen is necessary when substantial desaturation occurs during exercise but it is still unclear whether the training effect is influenced by oxygen supplementation.

**Inspiratory muscle training and rest**

Respiratory muscle training aims to improve inspiratory muscle strength and/or endurance in the belief that it may lead to less breathlessness, less fatigue, improved capability for physical exercise and better clearance of airway secretions. Respiratory muscle training is achieved to a limited extent through general body training or schedules of hyperpnoea. Targeted inspiratory training is the best available technique. Reasonable guidelines involve training sessions twice per day for five to seven days per week. Suggested treatment time is 15 minutes and the course of treatment should be at least 8 weeks. Breathing is performed through an inspiratory resistance at a level between 30% and 40% of maximal inspiratory pressure. Failure to maintain and visualise inspiratory pressure is responsible for the variable outcome of many studies.

Paradoxically some patients with severe COPD suffer enhanced respiratory muscle fatigue after training. Respiratory muscle rest in these instances may be more appropriate than exercise training.

A conflict remains as to the recognition of patients who will respectively benefit from training or rest largely due to the difficulty of agreeing methods of measuring respiratory muscle fatigue. Muscle rest is variously achieved with negative and positive pressure non-invasive mechanical ventilation for defined periods. Tolerance of the equipment in COPD is inconsistent. Techniques of inspiratory muscle training and rest in COPD patients should remain topics for research at the present time and are not yet suitable for general clinical application.
Breathing retraining

Breathing retraining (BR) for stable chronic obstructive lung disease takes several forms, low frequency breathing (LFB), pursed lips breathing (PLB), abdomino-diaphragmatic breathing (ADB) and "directed" breathing (ventilation dirigée). All these imposed types of breathing have a common aim to modulate and create a new type of breathing pattern which enhances tidal volume and lowers respiratory frequency without effect on the duty cycle Ti/Ttot. In addition, pursed lips breathing may modulate expiratory airflow avoiding dynamic airway collapse. All types of breathing slightly improve gas exchange but more by a "frequency effect" than by changes in the distribution of ventilation and perfusion.

The work of breathing increases during breathing retraining. Despite this a reduction in breathlessness and small improvements in lung function tests such as the maximum breathing capacity and respiratory muscle endurance have been claimed. Caution is required as increasing Vr and/or Ti/Ttot induces in some COPD patients electromyographic signs of respiratory muscle fatigue. Breathing retraining techniques are not established. There is need for long term studies using slow breathing techniques which measure the effect on ventilatory control and lung mechanics to establish whether permanent adaptation of breathing pattern is really possible. Such studies must correlate changes of lung mechanics and the sensation of dyspnoea. Until more definitive evidence is available, breathing retraining cannot be recommended in COPD.

Chest physiotherapy and lung secretions

Chest physical therapy includes postural drainage, chest percussion and vibration administered by hand or mechanical means, shaking, cough and forced expiratory techniques. It is applied to many types of chest disorder which include COPD with increased production of sputum (>30 ml·day⁻¹), bronchiectasis, cystic fibrosis and atelectasis when the problem is retention of secretions in the proximal airways. In patients suffering acute attacks of asthma, acute pneumonia, respiratory failure, or in need of ventilatory support and in those COPD patients with scant sputum expectoration, chest physiotherapy is of little or no value or even harmful.

For hospitalised critically ill patients, four sessions per day would seem to offer the upper limit of effectiveness and tolerability. With more stable conditions twice daily sessions are more practical. At home physiotherapy to clear secretions is only required during acute exacerbations of infection, except in cystic fibrosis and bronchiectasis. The patient and family members should then receive an educational and technical programme of several sessions from a trained physiotherapist. Prior use of bronchodilator drugs (through a metered dose inhaler or nebuliser) and/or mucolytic agents enhance mucociliary clearance and avoid the induction or worsening of bronchospasm. The order of effectiveness of chest physiotherapy techniques in removing airway mucus is as follows:

1. Forced expiratory therapy (FET) + postural drainage
2. FET alone
3. Directed coughing
4. Percussion/vibration + Postural drainage
5. Postural drainage and clapping/vibration alone (very low efficiency)

Smoking cessation

Cigarette smoking is the single most important preventable cause of respiratory disease. It acts synergistically with a host of occupational and environmental factors to produce lung damage. Not all individuals who smoke are affected and detection of the more susceptible in advance of disease is still impossible. About 10% to 15% of smokers can be identified by accelerated decline of FEV₁. Motivation and smoking behaviour are important. Addiction to nicotine probably occurs to some degree in about two thirds of smokers. Smoking cessation must therefore have prime place in rehabilitation programmes and should precede definitive interventions such as exercise.

There are two broad therapeutic strategies: drug treatment principally involving nicotine replacement and psychological intervention. The most widely used nicotine substitute is nicotine polacrilex gum but other vehicles such as transdermal patches are under study. There is some evidence that clonidine in daily dose will augment nicotine substitution in alleviating withdrawal symptoms. The components of behavioural intervention include 1) self monitoring 2) goal setting or contracting, 3) stimulus control or alternative behaviours and 4) aversion. There is a strong community component to item (3) whereby environment associations of smoking can be progressively narrowed to reduce situations where it is possible to smoke. Maintenance programmes are required for some time after quitting tobacco. Nicotine or its stable breakdown products cotinine and carbon monoxide (e.g. HbCO) are the most frequently used biologic markers. Self help and group programmes should be tailored for all ages but targeted at high risk groups such as the young and all those with COPD. Current studies suggest a 30% success rate of all programmes. There is little evidence to support hypnosis or acupuncture as effective.

Nutrition

Both obesity and wasting are features of COPD. Obesity and hypoxia are components of the "blue bloater" type of COPD. Obesity needs to be controlled by dietary regulation. Less attention has been given to the weight loss of the pink and breathless patients. Quantitative studies of fat and muscle often reveal protein caloric malnutrition of marasmic proportions. The cause is unclear but loss of appetite, food aversion and hypermetabolism through increased energy demand...
of COPD all seem involved. Correction of malnutrition by appropriate feeding seems feasible but has been difficult to achieve in the few patients studied so far. Nutritional status should be measured in all COPD patients who lose weight without clear cause. Nutritional intervention has not been fully evaluated and particularly as to whether it might improve body weight or affect long term prognosis.

Patient education and psychosocial support

Disability in COPD includes an important component of psychosocial damage which in many individuals can be just as important as physical limitation. Psychosocial support aims to regain the patient's damaged self esteem and impaired psychosocial function by restoring coping skills and learning stress management. It is essential that the patient understands his own essential role and responsibility and that his beliefs are tuned to positive outcomes. Mutual understanding and support of family, friends and personal physicians must be created. Psychosocial therapy is an essential part of a comprehensive rehabilitation programme in COPD. After assessment, intervention will involve education, support to achieve mental and physical awareness and will undertake activities commensurate with the severity of disease. It is helpful to measure the improvements of the quality of life or alternatively failure of therapy by validated questionnaire. As pulmonary rehabilitation is a life-long activity, so will patient education be a never ending process.

Drug therapy

Optimum drug therapy should be established before definitive rehabilitation is commenced. This will include relief of bronchoconstriction now recognised as a component of variable severity, intermittent antibiotics for infection and diuretics when peripheral oedema is manifest. Supplemental oxygen is given for respiratory failure. A number of new products are under study, vasodilator agents for pulmonary hypertension, mucolytics and anti-oxidants, alpha,-antitrypsin replacement, anti-inflammatory drugs and respiratory agonists, particularly almitrine bismesylate.

For relief of bronchoconstriction beta,-adrenergic drugs are administered through a variety of devices. The dose is adjusted for age, severity of disease and individual response. A willingness to use much higher doses than formerly, particularly through nebulisers, is apparent. Oral preparations are generally more helpful in children than adults. Newer long-acting β,-agonists are under evaluation. Tolerance is consistently good, adverse reactions are principally muscle tremor and tachyarrhythmia. Ipratropium bromide, a parasympathetic blocking drug, has useful additional effect in many patients and is perhaps the first choice in severe emphysema. Theophyllines are effective but associated with a wider spectrum of side effects. Oral corticosteroids benefit a subgroup of patients who must be defined by therapeutic trial. Long-term maintenance therapy should on current evidence be restricted to positive responders. Long-term inhaled corticosteroids remain under active evaluation. Individual exacerbations of bronchial infection should be treated with short courses of antibiotics such as broad spectrum penicillins, oxytetracycline or cephalosporins. The policy of self administration of reserve antibiotics for exacerbations has generally worked well. Continuous cycles of antibiotics, particularly in the winter months, are seldom indicated except for bronchectasis and cystic fibrosis.

Influenza infections have a devastating effect in COPD resulting in high mortality. Preventive vaccination remains imperfect. Killed vaccines only should be used. Attenuated organisms regain their virulence in COPD. Killed vaccines are 50–60% effective against infections caused by drift of surface H and N antigens but much less so against substantial antigen changes (or shift) responsible for pandemics. Coryzal side effects are common. Pneumococcal, staphylococcal and haemophilus vaccines are of limited benefit and remain the subject of much research. Of the newer medicament pulmonary vasodilator drugs for pulmonary hypertension cannot yet be recommended, the indications for mucolytics and anti-oxidants remain contentious and alpha,-antitrypsin replacement or enhancement is feasible through aerosol though expensive, but of unproven benefit in COPD.

Of the respiratory stimulants Almitrine bismesylate, a chemoceptor agonist, is the most interesting. Taken orally it increases Pao 2 and reduces Paco 2 in hypoxic COPD. Recommended dose schedules are now much lower than previously suggested. Effects of low dose Almitrine on long-term survival and clinical benefit remain to be established.

Domelitory oxygen therapy

Chronic respiratory failure, manifested by hypoxaemia and in some instances also hypercapnia, is associated with two broad categories of chronic pulmonary disease, obstructive airways disease (COPD) and restrictive disorders due largely to lung fibrosis or neuromuscular failure. Special techniques of low flow oxygen administration are necessary for severe hypoxaemic COPD. Oxygen supplementation for restrictive disorders generally demands higher flow rates; since hypoventilation is frequently a significant cause of oxygen lack, ventilatory support is also mandatory. The latter has generally worked in restrictive diseases but is not the subject of this report. Ventilatory support for severe respiratory failure in obstructive airways disease is more difficult to apply but is detailed below.

Oxygen supplementation for COPD patients in the home is supplied through cylinders, the oxygen concentrator or from liquid oxygen sources. Patient connection is by mask, nasal prongs and occasionally more invasively through catheters inserted into the
nasopharynx or trachea. Oxygen is given to relieve hypoxaemia and certain types of breathlessness. Before administration physiologic evidence of hypoxaemia should be obtained. Evidence of response should be obtained by measuring arterial blood gases or oxygen saturation. There are three forms of treatment, 1) long-term domiciliary oxygen (LTOT) 2) portable oxygen and 3) short-burst intermittent therapy.

**Long-term domiciliary oxygen (LTOT)**

Long-term domiciliary oxygen is the term applied to continuous oxygen supplementation in the home throughout the 24 hour day.

The minimum daily treatment period is 15 hours, it should include the night. A better survival response is achieved with near 24 hour daily periods. The absolute indications are a stable PaO₂ breathing air of 7.3 kPa (55 mmHg) or less with an FEV₁ <1.5 litres and FVC <2.0 litres. Many countries have relaxed the criteria in various ways, PaO₂ up to 8.7 kPa (65 mmHg) if haemacrit is elevated (>55%), oedema witnessed, or there is severe nocturnal or exercise hypoxaemia despite daytime normoxaemia. None of these additional features have been shown to be associated with an adverse prognosis, so firm recommendation to treat in such instances cannot yet be made.

Stability of hypoxaemia is critically important as life long therapy is not intended for patients merely recovering from short term hypoxaemia such as follows an infective exacerbation. The minimum period of assessment is three weeks with limits of change of PaO₂ of ± 0.5 kPa (4 mmHg) at or below PaO₂ of 7.3 kPa (55 mmHg). If PaO₂ is unstable as defined, then longer periods of assessment are mandatory.

The most convenient administrative device is the biprong nasal cannula. Flow rate should be prescribed after tests to achieve a PaO₂ of at least 8.7 kPa (65 mmHg) have been undertaken but will generally be between 1.5 and 2.5 litres per minute. Hypercapnia will often prove a limiting factor to attainable flow rate, and should not be permitted to rise by more than 1.3 kPa (10 mmHg). Reservoir cannulae and phased inspiration devices conserve gas but are more applicable to liquid than compressed oxygen sources. Nasopharyngeal and transtracheal catheters are helpful for the few patients refractory to cannula therapy. Transtracheal catheters in addition conceal the therapeutic device in self conscious patients. They should be supplied by experienced centres as complication rates can otherwise be high. Masks are generally poorly tolerated for long periods and have proven less suitable for LTOT.

**Portable oxygen**

Portable oxygen is provided by light weight gas cylinders or by small cryogenic vessels containing liquid gas. The former have to be repeatedly filled from a stationary cylinder and provide less walking distance. The latter enable longer periods of use but are more expensive and need a special delivery system for the liquid gas. Portable oxygen is given, 1) to relieve exercise hypoxaemia and dyspnœa enabling greater walking distances and 2) to extend the daily period of LTOT. Neither of these benefits have been clearly demonstrated but there is useful supportive evidence. The usual recommended flow rate is 4 l/min. Double blind laboratory tests against air breathing should define benefit before prescription. About 25% of severe COPD patients can expect to improve by reduced breathlessness (>10% improvement on visual analogue scales) and/or increased walking distance (>10% baseline standardised 6 minute test levels).

**Short burst intermittent therapy**

In some countries cylinders are provided in the home for short period use through a simple face mask for the relief of breathlessness, particularly after exercise. Use varies widely from several times per day of a few minutes duration, to once or twice per month. Such use is largely of placebo value, is expensive and cannot possibly relieve the consequences of long-term hypoxaemia. Recent double-blind studies have demonstrated some relief of dyspnœa in about a third of individuals. Surprisingly not all were hypoxaemic. At the present time the following recommendation is made. Short burst oxygen can be given in COPD after double-blind tests against air breathing have demonstrated at least a 10% repeated reduction of breathlessness, measured on visual analogue scales. More research is clearly needed.

**Expectation of benefit of long-term domiciliary oxygen (LTOT)**

LTOT is administered in hypoxaemic COPD to improve survival, sleep quality, neuropsychiatric disorder, quality of life and mobility and to reduce haematocrit, pulmonary hypertension and oedema.

Improvement of survival is clear and ideally doubles survival period or adds five years of life. Oxygen therapy corrects hypoxaemia, not hypercapnia but fails to arrest the progression of airway disease. Much less benefit has been recorded for neuropsychologic impairment, quality of life and mobility. Patient compliance with this demanding therapy has not been fully studied in relation to the outcome variables. The benefits of LTOT in terms of survival are demonstrated only in those with stable daytime hypoxaemia. Therapy should not be commenced too late as benefits are small in pre terminal disease.

**Home ventilatory support**

Long-term domiciliary oxygen (LTOT) fails to arrest the decline of PaO₂. In advanced hypercapnic respiratory failure mechanical ventilatory assistance, either
mandatory or elective, has been studied. The goals are to 1) extend life, 2) enhance quality of life 3) reduce morbidity 4) improve physical and physiological function and 5) be cost beneficial, in particular to decrease hospital admissions. It should be considered for those patients with frequent decompensations of respiratory failure to prevent a fatal episode, in those who fail to wean after an acute exacerbation and where \( P_{aco_2} \) cannot be maintained over 7.0 kPa (52 mmHg) without excessive hypercapnia (10.0 kPa or 75 mmHg).

There are a number of methods, of ventilatory support; positive pressure ventilation through a tracheostomy or by nasal or facial mask and negative pressure ventilation by cuirasse, shell or poncho around the chest and abdomen. Treatment may be intermittent, nocturnal or continuous. Long-term results are unclear and hampered by lack of control studies. The use of tracheostomy is associated with poor outcome, 8% survival at 10 years in one study. Non invasive nasal intermittent positive pressure ventilation is a new technique under evaluation in hypoxaemic COPD. Devices may be volume or pressure controlled. BIPAP (expiratory and inspiratory positive airway pressure) nasal ventilation machines, a derivative of CPAP (continuous positive airway pressure) devices provide a novel type of pressure supported ventilation. The machines are connected to the patient through a specially fitted nasal mask. The aim is to use ventilatory support only at night leaving the patient undisturbed in the day. The most suitable patients will be those developing persistent elevation of \( P_{aco_2} \) (>8 kPa, 60 mmHg) an LTOT. Although some successes are recorded many patients find it difficult to accomodate the machines for more than a few hours. The respective indications for volume dependent and pressure controlled ventilators in COPD remain to be defined.

The potential for ventilatory support in acute exacerbations of respiratory failure in COPD is considerable. Preliminary results of external negative pressure ventilatory techniques are disparate. In COPD tolerance for more than 2 hours is difficult but improvement of blood gases, dyspnoea and respiratory muscle strength have been recorded. Nasal intermittent positive pressure ventilation (NIPV) using methods described above offers an alternative approach. It may well find a useful place but is still under evaluation.

The use of home ventilatory support in COPD remains a research procedure. Successful transfer of a patient from hospital to home requires all the resources of a well organised and trained home care team.

**Home respiratory care**

Home care is the provision of health services in the patient’s home rather than in hospital or other institutional setting. The aim is to provide the best possible lifestyle for the individual at considerable saving in cost. Successful home care will depend upon a clear assessment of disease severity, firm understanding of therapeutic benefits, careful patient selection for differing aspects of the rehabilitation programme, properly organised services using trained personnel and equipment of defined specification, maintenance, alarm and breakdown schedules. Recognition and therapy for personal and family psychosocial impairment is mandatory. Home care in COPD has advanced principally in the provision of LTOT. Irregular interpretation of admission guidelines to LTOT has increased the health care burden in some countries without clear patient benefit. The home care team is best supervised by a physician with the help of a General practitioner. Nursing staff and other members of the home care team are variously provided by state, domiciliary, charitable, commercial and agency organisations with equally variable quality of training. Only in the United States have respiratory home care therapists a defined training programme but even so, still compete with a multitude of other home care providers.

In Europe, job descriptions should be agreed for respiratory home care therapists and appropriate training programmes commenced. Such personnel will combine duties of nurse, physiotherapists and social worker in this special area and have a working knowledge of the equipment employed.

**Acknowledgement:** The working group are grateful for the support of the Clinica del Lavoro Foundation, Pavia, Italy for making the preparation of this document possible.

**References**

**Rationale of pulmonary rehabilitation**


**Selection of patients**


**Evaluation of rehabilitation therapy**

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Mechanisms of exercise limitation


Exercise prescription


Inspiratory muscle training


Breathing retraining


Chest physiotherapy


Smoking cessation


Nutrition


Patient education and psychosocial support


Drug therapy


Oxygen therapy


Home mechanical ventilation


Home care


