

Indications for mechanical ventilation

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Mechanical ventilation comprises all types of artificial ventilation in which a mechanical device is used to replace or aid the work normally carried out by the ventilatory muscles. It has been used to treat ventilatory failure since the portable "iron lung" was introduced by Drinker and Shaw in 1929,¹ but only during the Copenhagen poliomyelitis epidemic in 1952 were the skills of anaesthetists and physicians brought together in a major breakthrough that established the life-saving value and simplicity of intermittent positive pressure ventilation.^{2,3} This was an important landmark in the treatment of acute respiratory failure and soon afterwards the benefits of intermittent positive pressure ventilation in the postoperative period were also proved.⁴ Unless otherwise stated, this paper refers to mechanical ventilation as any form of intermittent positive pressure ventilation, applied through an endotracheal tube, with or without positive end expiratory pressure or allowance for spontaneous breaths.

Mechanical ventilation is indicated where established or impending respiratory failure exists, defined as the inability of the breathing apparatus to maintain normal gas exchange. Respiratory failure may be predominantly due to failure of oxygenation (type I) or to an inability to eliminate carbon dioxide (type II or "ventilatory" failure). Type I is usually associated with lung parenchymal disease, alveolar collapse, or an increase in lung water. Type II is generally associated with a lack of ventilatory drive, musculoskeletal disease, or neuromuscular blockade. In principle, mechanical ventilation is indicated predominantly for ventilatory failure (type II).

The vast majority of patients receiving mechanical ventilation do not have pulmonary disease. This is the group undergoing major surgery under general anaesthesia with the aid of neuromuscular blocking drugs. Outside the operating theatre most patients receive mechanical ventilation in the intensive therapy unit. Within non-specialised intensive therapy units (table 1) over half of all ventilated patients have had cardiac, aortic, or other major surgery, rarely needing intermittent positive pressure ventilation for more than 24 hours. The other major groups requiring ventilation are patients with head or chest trauma (<15%) and various forms of poisoning (<8%) and those who are critically ill with severe primary respiratory (<15%) or cardiac (<3%) disease.⁵⁻⁷ A very small group

of patients receive mechanical ventilation at home or in specialised institutions; these will be considered separately in articles 5 and 6 of this series.

A comprehensive list of indications for mechanical ventilation appears in table 2. Indications for mechanical ventilation in anaesthesia, after surgery, in neonates and in organ donation are outside the scope of this review. Mechanical ventilation should be used only when it is strictly necessary as there are many inherent risks.⁸ Indeed, ventilation may unnecessarily prolong the distress of terminal disease and the benefits of its use should therefore be carefully weighed against the disadvantages. The basic "recipe" for setting up intermittent positive pressure ventilation, in a patient without lung disease, appears in table 3.

Benefits of mechanical ventilation

The principal benefit of mechanical ventila-

Table 1 Underlying pathological conditions of patients admitted to non-specialised intensive therapy units in three surveys

	No of patients	% of total
<i>A Pontoppidan et al⁵ (1972): patients admitted to intensive therapy units over nine consecutive years (1961-70) requiring mechanical ventilation; mortality 35-40% in the first five years, then 10-20%</i>		
Cardiothoracic surgery	239	25.9
Neurosurgery including head trauma	69	7.5
Other surgery including trauma	243	26.3
Neuromuscular disease	71	7.7
Poisoning	73	7.9
Primary respiratory disease	121	13.1
Cardiac failure	12	1.3
Other	95	10.3
Total	923	
<i>B Petty et al⁶ (1975): 1877 patients admitted to intensive therapy units over 10 consecutive years (1964-74) who received ventilation for more than 24 hours; survival rate was 75.2%</i>		
Postoperative	1009	53.8
Neurosurgical	115	6.1
Trauma	168	8.9
Poisoning	145	7.7
Primary respiratory disease	250	13.3
Total	1877	
<i>C Knaus et al⁷ (1989): 3884 consecutive patients admitted to intensive therapy units in 12 United States hospitals, of whom 1886 (49%) received mechanical ventilation</i>		
Postoperative	1315	70
Others	571	30

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Table 2 List of possible indications for mechanical ventilation

Routine anaesthesia
Cardiothoracic and abdominal surgery and neurosurgery
Prolonged surgery and surgery requiring prone position
Surgery in frail patients or those with cardiac disease
Clinical investigations (radiology, tissue biopsy) requiring temporary immobility
Postoperative management
Major surgery of the heart or the great vessels
Abdominal distention, debility, or electrolyte imbalance
Pre-existing lung disease, respiratory muscle weakness, kyphoscoliosis, myasthenia gravis, morbid obesity
Respiratory disease (parenchymal or airway)
Pneumonia, asthma, lung contusion
Acute exacerbation of chronic bronchitis, emphysema
Adult respiratory distress syndrome, hyaline membrane disease, cystic fibrosis
Chest wall disease
Trauma with flail segment, ruptured diaphragm
Chest wall burns, kyphoscoliosis
Neuromuscular disease
Polyneuritis, Guillain-Barré disease, Lambert-Eaton disease
Myasthenia gravis, myopathies, paralyzing poisons
Central nervous system impairment
Drug overdose: narcotics, anaesthetics, barbiturates
Trauma, meningoencephalitis, tumours, infarction
Brain oedema, raised intracranial pressure
Intracranial bleed, status epilepticus, tetanus, rabies
Central hypoventilation
Cardiovascular disease
Cardiac arrest, severe shock—sepsis or other causes
Left ventricular failure—pulmonary oedema
Neonatal conditions
Severe prematurity
Severe bronchopulmonary dysplasia
Central hypoventilation syndrome
Increased metabolism and carbon dioxide production precipitating ventilatory failure in patients with pre-existing disease
Organ donation

Table 3 Basic "recipe" for setting up mechanical ventilation in an adult patient without pulmonary disease and with a normal metabolic rate

AIRWAY
Access via oral or nasal cuffed endotracheal tube or cuffed tracheostomy tube
VENTILATOR
Set tidal volume (VT) at 6 ml/kg body weight
Set respiratory rate (RR) at 12–14 breaths/min; minute volume (= VT × RR) should be 80–90 ml/kg body weight
Set ratio of inspiratory:expiratory time to 1:3; peak inflation pressure should not exceed 30 cm H ₂ O
Provide humidification of inspired gas mixture
Set oxygen concentration at 30–60%
Set alarms for
ventilator disconnection
peak inspiratory pressure > 30 cm H ₂ O
fractional inspired oxygen (FIO ₂) < 0.25 or > 0.6
PATIENT
Ensure analgesia and sedation: mandatory if patient is paralysed (neuromuscular blockers only when strictly necessary—that is, tetanus, head injuries)—use opiates and/or benzodiazepines
Monitor effects of intermittent positive pressure ventilation on circulation and gastric distension
Check blood gas tensions regularly (2–4 hourly) and after changing any of the ventilator settings:
adjust minute volume according to arterial carbon dioxide tension
adjust FIO ₂ and positive end expiratory pressure according to arterial oxygen tension
Institute basic nursing care for the unconscious patient:
regular routine observations, turning on bed, mouth wash
regular check for bilateral breath sounds and expansion of both lungs—risk of endobronchial intubation, pneumothorax, accumulation of secretions
regular check for state of consciousness, need of pain relief, and sedation
Chest radiograph on alternate days to check for:
position of endotracheal tube
pleural effusion
alveolar collapse or consolidation

tion is the control gained over the airway and over the work of breathing. The ventilator replaces the work of exhausted or temporarily inadequate respiratory muscles. The ability to remove secretions from upper airways (by simple suction or aided by fiberoptic bronchoscopy) may be advantageous and additional measures, such as positive end expiratory pressure or effective aerosol delivery, may be instituted in certain patients. Ventilation also allows large doses of narcotic analgesics or neuromuscular blocking agents to be used where clinically indicated (in tetanus, for example). Table 2 lists clinical circumstances in which mechanical ventilation may be of benefit.

Risks and side effects of mechanical ventilation

THE RISKS

Some dangers of mechanical ventilation apply to all patients. Effective, long term intermittent positive pressure ventilation cannot be established without securing a sealed connection with the airway via an endotracheal or tracheostomy tube; the insertion of this tube, however, requires either general or local anaesthesia with its attendant risks.

Anaesthesia

The risks of the anaesthesia needed for endotracheal intubation include myocardial depression caused by general or local anaesthetics; aspiration of gastric contents; a further fall in arterial oxygen tension (PaO₂), especially if intubation is difficult; an idiosyncratic reaction to anaesthetic drugs; and reflex worsening of bronchoconstriction after tracheal intubation or suction of secretions. These risks are not substantially reduced if a topical local anaesthetic is used before intubation of the trachea.

Sedation and paralysis

Intermittent positive pressure ventilation through a nasal or an orotracheal tube is poorly tolerated without sedation and often requires paralyzing drugs. The ideal sedative should be very short acting and be given by constant intravenous infusion, and should have minimal side effects, especially on the circulation. None of the available sedatives is devoid of side effects: the opiates are complicated by tolerance and paralysis of the gut (with consequent delay in absorption) and their prolonged respiratory depressant effects delay weaning from the ventilator. Barbiturates and chlormethiazole present similar problems and also cause myocardial depression. The benzodiazepines often require increasing dosage because of tolerance and this causes prolonged depressant effects, lasting for days after the last dose, on the central nervous system. Of the established anaesthetics, only two recently introduced drugs, currently being tested for long term sedation, have potential as sedatives for the intensive therapy unit—propofol,⁹ an intravenous anaesthetic, and isoflurane,¹⁰ a volatile inhala-

Table 4 Critical values* of physiological variables widely accepted as part of the criteria for administering mechanical ventilation to adult patients (normal ranges in parentheses)

VENTILATORY MECHANICS		
Tidal volume (VT; ml/kg)	<3	(5-7)
Respiratory rate (breaths/min)	>35	(12-20)
Minute ventilation (l/min)	<3 or >20	(6-10)
Vital capacity (VC; ml/kg)	<10-15†	(65-75)
FEV ₁ (ml/kg)	<10	
Max inspiratory pressure	> -20-25†	(-75--100)
VD:VT ratio	>0.6	(0.25-0.4)
GAS EXCHANGE		
PaO ₂		
FIO ₂ = 0.6	<8 kPa	
PA-aO ₂		
FIO ₂ = 1.0	>46-60†‡ kPa	3.3-8.6
PaCO ₂	>6.7-8†§ kPa	4.6-6.0
CIRCULATORY VARIABLES		
Cardiac output	<2	l/min or
Cardiac index	<1.2	l/min/m ²

*The values in this table summarise those appearing in the current publications.^{5-7 19 20 22} Consequently they are only approximate and VT, VC, and FEV₁ are not given in relation to age and sex.

†Range of published values.^{7 19 22}

‡After at least 10 minutes of continuous FIO₂ = 1.0.

§In patients without metabolic acidosis or chronic hypercapnia.

VD—dead space; FIO₂—fractional inspired oxygen; PaO₂—arterial oxygen tension; PaCO₂—arterial carbon dioxide tension; P_{(A-a)O₂}—alveolar-arterial oxygen difference.

tion anaesthetic. They are both short acting agents, with no cumulative effects, and are relatively free of cardiovascular and respiratory effects at sedative doses. There is a wide choice of suitable neuromuscular blocking drugs: pancuronium, vecuronium, and atracurium have minimal side effects and the last two are sufficiently short acting to allow rapid regulation of the state of paralysis. All intensive therapy unit staff should be aware that neuromuscular blocking agents have no sedative effects and that patients may be awake and paralysed if sedation is not prescribed. Another danger of paralysis is the

Table 5 Considerations in initiating or withholding ventilation

(a) Clinical factors relevant to the decision to initiate or withhold ventilation in a patient with acute respiratory failure

- The conscious patient's acceptance of the treatment; degree of permanent mental impairment or other permanent, severe disability
- History of previous admissions to intensive therapy unit and outcome of previous episodes of intermittent positive pressure ventilation
- Is the underlying disease reversible?
- Likelihood of successful weaning from the ventilator
- Multiple organ failure?

(b) Factors influencing the decision to initiate ventilation in acute respiratory failure

- Rapidly worsening physiological variables
- Evidence of heart failure—fall in blood pressure, rise in heart rate, fall in urine output, etc
- Presence of severe dyspnoea and sweating
- Prominent use of accessory muscles, paradoxical movement of the abdomen
- Inability to expectorate secretions
- Severe fatigue of respiratory muscles—usually heralded by upward trends in respiratory rate and arterial carbon dioxide tension
- Increasing confusion, restlessness, and exhaustion

inability of the patient to make spontaneous breathing efforts should there be an accidental ventilator disconnection.

Equipment failure

The risks of equipment failure include accidental disconnection of the ventilator, undetected leaks or malfunction of the endotracheal tube, all leading to alveolar hypoventilation; barotrauma to the lungs if high inflation pressures are applied to the airway in error, leading to pneumothorax or subcutaneous emphysema; tracheal burns if heated humidifiers are used; oxygen toxicity if FIO₂ is above 0.6 for a prolonged period.

Hyperinflation

Hyperinflation not associated with equipment failure occurs in patients with severe acute bronchospasm or chronic airflow limitation with increased functional residual capacity. Intermittent positive pressure ventilation may lead to very high intrathoracic pressures, with adverse effects on cardiac output and increased risk of pneumothorax.

Risks from the cardiovascular effects

Mean intrathoracic pressure is raised by intermittent positive pressure ventilation, especially where positive end expiratory pressure is used, causing a fall in cardiac output. These effects are unimportant in the relatively fit patient¹¹ undergoing elective surgery but may not be tolerated in the severely ill patient with hypovolaemia or with increased airways resistance.

CARDIOVASCULAR EFFECTS OF INTERMITTENT POSITIVE PRESSURE VENTILATION

Not all the effects of intermittent positive pressure ventilation on the cardiovascular system are adverse. They result from the rise in intrathoracic pressure, especially if positive end expiratory pressure is used,¹² and are mediated through direct mechanical interference with the heart,¹³ through indirect reflexes of the autonomic nervous system, and through hormone release or changes in blood gases. The predominant direct adverse effects of intermittent positive pressure ventilation on the right heart are a reduction in venous return (preload) and an increase in pulmonary vascular resistance (afterload). The direct effects on the left heart are less pronounced and less well established, the widely held view being that both pulmonary venous return (preload) and afterload decrease. This effect on left ventricular afterload is due to a fall in ventricular transmural pressure because of the increase in intrathoracic pressure (this also applies to the right ventricle).¹⁴ This mechanism provides a form of "assistance" to ventricular work, which may be beneficial in cardiac failure.^{13 15} The reflex responses are complex, depending on multiple neural and chemical feedback loops.¹⁶ The neural reflexes are mediated initially by the vagus nerve; stronger reflexes include the whole sympathetic system,¹⁷ affecting vascular resistances and circulating catecholamines. The reflexes originate from lung and atrial stretch receptors

and from the arterial baroreceptors and chemoreceptors (the latter only if arterial carbon dioxide tension (P_{aCO_2}) falls or oxygen tension (P_{aO_2}) rises in response to intermittent positive pressure ventilation). The humoral reflex response to intermittent positive pressure ventilation includes an increase in antidiuretic hormone and renin-angiotensin and a decrease in atrial natriuretic peptide, which may be partly responsible for the sodium retention seen in ventilated patients¹⁸; the changes in catecholamines are partly mediated by P_{aCO_2} changes. The pattern of circulatory changes is variable: the predominant effect is a decrease in both cardiac output (typically by 25%) and arterial blood pressure, an increase in heart rate, and a slight increase in systemic vascular resistance; right and left atrial pressures increase in relation to atmospheric pressure (transmural pressures *decrease*). This pattern is often modified by blood gas changes, such as the improvement in oxygenation usually associated with intermittent positive pressure ventilation; large changes in P_{aCO_2} cause the largest changes in circulatory variables because of the powerful effects of carbon dioxide on the sympathetic system. In addition to cardiovascular effects, the increased intrathoracic pressure also hinders the venous drainage from the head, which may be important when intracranial pressure is raised.

Criteria for initiation of mechanical ventilation

Despite the risks listed above, patients developing acute severe respiratory failure secondary to reversible conditions should be ventilated as a life saving measure. Examples are pneumonia, asthma, neuromuscular syndromes, head or chest trauma, pulmonary oedema secondary to heart failure, poisoning, and septic shock. In nearly all cases the indications for mechanical ventilation are clearcut and ventilation is needed for only a short time (under 48 hours); there are circumstances, however, where the indications are less clear.^{19 20}

Firstly, it is important, but may be difficult, to decide whether mechanical ventilation is the most appropriate treatment for the patient. In the adult respiratory distress syndrome, for example, it is debatable whether mechanical ventilation with positive end expiratory pressure should be started before the onset of type II respiratory failure. Although this is routine in many centres, the available evidence suggests that early institution of intermittent positive pressure ventilation does not alter the course or final outcome.²¹ In general, intermittent positive pressure ventilation without respiratory failure is rarely indicated except after major surgery or in the management of raised intracranial pressure.

A second circumstance in which the decision to ventilate is difficult is in acute respiratory failure associated with terminal malignancy, advanced AIDS, or severe chronic airflow limitation, because it is often impossible to predict whether the underlying disease will

allow sufficient recovery for successful weaning from the ventilator. Published figures from various centres indicate that the mortality of these patients while ventilated is very high^{7 22} and associated with substantial human and material costs.²³ A decision to withhold mechanical ventilation in these patients should be made by a senior physician who first takes into account the present wishes of the fully conscious patient or the known wishes of the unconscious patient. In the unconscious patient the views of the closest relatives and of the staff directly concerned should also be considered. Where there is doubt, it is ethically more acceptable to withdraw treatment later than to withhold it in the moment of crisis.^{13 14} All possible measures should be used in these patients to postpone the need for ventilation—for example, the use of doxapram if there is inadequate respiratory drive, continuous positive airway pressure if the main problem is airway collapse, and nasal intermittent positive pressure ventilation, which may be well tolerated for short periods, obviating the need for endotracheal intubation.²⁴

It may be equally difficult to define the precise moment when ventilation should be started. Despite 70 years of worldwide experience with mechanical ventilation, there are no exact criteria on which to base a decision.¹⁹ In the past 25 years, however, guidelines have evolved from a consensus of opinion among physicians and anaesthetists. Table 4 shows a set of physiological variables with a range of critical values that have been proposed by several authors. These critical values, however, are empirical, merely representing the accumulated experience of clinicians.^{7 19 22} The decision to ventilate therefore rests firmly on the clinical findings. For example, having rapidly worsening respiratory variables is more important than exceeding a single critical value; fatigue and exhaustion cannot be quantified and should be judged by an experienced clinician. It is generally accepted, however, that reaching any of the critical values in table 4 is associated with terminal respiratory failure unless mechanical ventilation is instituted.¹¹ Table 5 lists the clinical factors which, complemented by the variables of table 4, influence the decision to start mechanical ventilation.

Ideally, severe respiratory failure should be anticipated and the decision to ventilate should be made before more than one of the critical values of table 4 is exceeded. Severe dyspnoea, restlessness, and exhaustion are in themselves good indicators for initiating ventilation when the underlying clinical condition is not expected to improve within one to two hours. Patients with impending acute respiratory failure need frequent and expert monitoring within an intensive care setting so that the decision to ventilate can be made at the appropriate time. Admission to the intensive therapy unit should therefore be arranged before mechanical ventilation is actually needed because a delay in the decision to ventilate may trigger a sequence of irreversible events, including multiple organ failure and cerebral oedema. Furthermore, the risks

associated with sedation and endotracheal intubation increase as the clinical state of the patient deteriorates. The state of the patient is too often allowed to deteriorate too far in the general medical or surgical ward before being admitted to the intensive therapy unit. On the other hand, the usual consensus among physicians and surgeons, because of the high costs and the fierce competition for beds, is that admission to the intensive therapy unit is not warranted until intermittent positive pressure ventilation is essential. This deadlock, with obvious disadvantages to the patients, can be resolved only if clear policies for admission to the intensive therapy unit are established among the senior clinicians concerned. Scoring systems that help to predict the probability of survival of severely ill patients⁷ may help in defining such admission policies.

High risk patients

Some clinical conditions deserve separate discussion because of their special risks and difficulties.

ACUTE ASTHMA OR ACUTE EXACERBATIONS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Respiratory failure is accompanied by chest hyperinflation, impaired diaphragm function, tachypnoea, and considerable use of accessory muscles. When intermittent positive pressure ventilation is applied inflation pressure may be very high (above 40 cm H₂O) with a very steep upward slope with each inflation, indicating very low chest compliance. The high intrathoracic pressures may have a substantial effect on the circulation, causing a fall in cardiac output. The risk of pneumothorax is high. When intermittent positive pressure ventilation is indicated in the exhausted, rapidly deteriorating patient it should not initially be aimed at correcting PaCO₂; values up to 9 kPa are acceptable. Tidal volume should be small (under 0.6 l) and the ratio of inspiratory to expiratory time must be low (less than 1:4), allowing maximum time for the lungs to deflate in expiration²⁵; a high oxygen concentration (at least 0.75) should be used to maintain oxygenation during the short period of life threatening respiratory failure. Saturated humidification of inspired gases, warmed to body temperature, is essential. The use of positive end expiratory pressure is contraindicated.

PATIENTS AT SPECIAL RISK FROM BAROTRAUMA

A proportion of patients with chronic respiratory failure have bullous emphysema, and intermittent positive pressure ventilation considerably increases the risk of pneumothorax. Positive end expiratory pressure is again contraindicated and inflation pressures should not exceed 40 cm H₂O with a maximum inspiratory:expiratory ratio of 1:3, even if the patient remains underventilated (oxygenation should be ensured by raising FIO₂). The patient should be closely monitored for signs of tension pneumothorax (sudden increase in inflation pressure, tachycardia, and fall in blood pressure) and equipment should be ready for the

insertion of a chest drain. When bullae burst and drains are inserted the problems of bronchopleural fistulas may supervene.

PATIENTS WITH STIFF LUNGS

Stiff lungs are usually the result of chronic interstitial disease or the adult respiratory distress syndrome secondary to cardiovascular shock, acid aspiration, or trauma. Intermittent positive pressure ventilation is used to replace the excessive work demanded of the respiratory muscles during the acute phase; high inflation pressures are needed, with positive end expiratory pressure if necessary to maintain oxygenation, so long as cardiac output is not severely compromised. In terms of peripheral oxygen delivery, a normal cardiac output with a PaO₂ of 6 kPa is better than half the normal cardiac output with a PaO₂ of 12 kPa. Low PaO₂ values are acceptable if the haemoglobin content of the blood is normal and cardiac output can reflexly rise above normal (haemodynamic monitoring and oxygen delivery will be discussed in article 3 of this series). The high inflation pressures sometimes necessary in these patients lead to other problems, including pneumothorax and the difficulty of maintaining an adequate seal of the endotracheal tube without overinflating the cuff and damaging the trachea. It is impossible to predict accurately whether the respiratory muscles will be able to cope with chronically stiff lungs after the acute phase has passed.

LUNG SURGERY

Intermittent positive pressure ventilation should be avoided after lung surgery that includes bronchial resection because of the high incidence of bronchopleural fistula originating at the bronchial stump. If respiratory failure is due to lack of central drive doxapram could be tried before intermittent positive pressure ventilation is considered; narcotic analgesics should be avoided and pain relief by local analgesia is useful. If ventilation is necessary because of temporary respiratory failure endobronchial intubation of the intact side should be considered. If endobronchial intubation is not possible maximum inflation pressure, through a normal endotracheal tube, should be kept below 20 cm H₂O and positive end expiratory pressure should be avoided.

Special mechanical ventilation techniques

The "nuts and bolts" of ventilators and modalities of mechanical ventilation were reviewed in the first article (October, p753). Intermittent positive pressure ventilation, with or without positive end expiratory pressure, is the modality of mechanical ventilation best suited for anaesthesia, postoperative ventilation, cardiopulmonary resuscitation, and most non-surgical conditions treated in the intensive therapy unit. Modifications of the original technique, which allow spontaneous or assisted breaths, are often used in the intensive therapy unit. The problems of weaning and ventilatory assistance will be dealt with in article 4. Two

special techniques merit further discussion because of their potential application in difficult cases.

HIGH FREQUENCY VENTILATION

High frequency ventilation provides small inflations at rates of over 60 breaths/min (usually 200–300 breaths/min) by means of a special ventilator. The technique can maintain adequate gas exchange while achieving lower peak inflation pressures than conventional intermittent positive pressure ventilation. The mean airway pressure, however, is the same or higher than with the latter for equivalent alveolar ventilation, which reduces the possible advantages of high frequency ventilation to a few special circumstances.²⁶ There are so far unresolved problems with the equipment, such as humidification of inspired gases and monitoring of airway pressures during high frequency ventilation. It has been suggested that the technique has advantages over conventional intermittent positive pressure ventilation in the treatment of persistent bronchopleural fistula²⁷ and respiratory failure²⁸ associated with cardiac failure; both claims, however, have been disputed.^{29 30}

DIFFERENTIAL VENTILATION

Differential ventilation allows ventilation of each lung with different gas mixtures or different pressure-time settings. It requires either a double lumen endotracheal tube or two cuffed bronchial tubes. Two synchronised ventilators are also needed. Experience with this technique is limited; the published evidence is based on case reports in patients with unilateral lung disease and on experimental animal work. Reported uses of differential ventilation include the treatment of patients with persistent bronchopleural fistula, unilateral bullae, and conditions in which copious secretions from one lung are affecting the function of the normal lung. If a high oxygen concentration or high inflation pressures are needed because of unilateral disease the "good" lung can be spared the risk of oxygen toxicity or excessive inflation pressures by differential ventilation.³¹

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