General care of the ventilated patient in the intensive care unit

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Care of the airway

Patients whose conscious level is impaired often require an artificial aid to maintain a clear airway. An oral (Guedel) airway may be sufficient temporarily, and it allows the passage of a suction catheter alongside. A nasopharyngeal airway is more comfortable, and may permit passage of a fine catheter through the larynx, though this may be traumatic if repeated too often.

An endotracheal tube or tracheostomy is necessary to secure the airway against laryngeal obstruction, to provide a route for artificial ventilation, to allow suction of bronchial secretions, and to protect the lungs against aspiration of pharyngeal and gastric contents. Early problems with endotracheal tubes include misplacement into the oesophagus or a mainstem bronchus, and flexion, extension, or turning of the neck may displace the tube tip. Other complications include aspiration past an incompletely inflated balloon cuff, injuries of oropharyngeal mucous membranes, paralysis and granulomas of vocal cords, and laryngotracheal stenosis. Accidental and self extubation is a risk, especially with young children, and the inflated cuff may damage the larynx as it passes through.

Although the oral route is often easier for intubation, nasal endotracheal tubes have the advantages of avoiding trauma to the mouth and of being more comfortable for the awake patient. They are also longer and narrower than oral tubes, and occasionally present problems with suctioning. They may be associated with paranasal sinusitis, particularly in patients receiving corticosteroids.

The use of plastic materials, standardised connector sizes, and uniform fixation techniques have helped to reduce some of the problems of endotracheal tubes. The introduction of high volume, low pressure cuffs may help to prevent tracheal mucosal damage, though other factors are also important.

Periods of hypotension and sepsis may compromise mucosal blood supply, and frequent changes of tube may damage the larynx. Tracheal mucosal damage is associated with local sepsis.

Ulceration and granulomas of vocal cords occur in many patients after periods of intubation of five to fourteen days and vocal cord paresis may also occur. These complications are associated with laryngeal oedema, and usually resolve in the days or weeks after extubation. Tracheostomy will prevent these lesions.

Suctioning may cause mucosal damage, and ciliary action may be impaired by the frequent use of high vacuum suction apparatus attached to a catheter with a single end placed hole. Pneumothorax may also occur with suctioning, especially in young children. Neonates are liable to develop subglottic oedema and stenosis after intubation. Subsequent hoarseness and inspiratory stridor may be precipitated by an upper respiratory tract infection. In a small minority of cases, reparative surgery may be necessary.

Tracheostomy is necessary for patients requiring long term continuous positive pressure ventilation or continuous positive airway pressure, and those who need long term airway protection or tracheal suctioning. Complications of intubation may also precipitate the decision to provide a tracheostomy. This is preferable for intubation for children, where the risk of self extubation requires constant observation—and a high level of staffing that is not always possible. Once a tracheostomy has been provided, it offers the chance of staged decannulation, with the use of fenestrated tubes and tracheal buttons, and this may permit an early transfer out of the intensive care unit.

The previous policy of carrying out a tracheostomy after a given period of intubation is becoming less popular. Patients with facial injuries may require tracheostomy from the outset and patients who will clearly require intubation for a long time, such as those with the Guillain-Barre syndrome and tetanus, should receive a tracheostomy as soon as possible. Patients with chest trauma requiring continuous positive airway pressure may remain awake with regional analgesia, and find a tracheostomy more tolerable than intubation. On the other hand, with improvements in tube design and attention to risk factors for laryngeal damage, many consider that intubation may be tolerated for longer than was previously thought.

Although the operating theatre provides the best surgical environment, there are risks associated with transferring patients from an intensive care unit. Tracheostomy can be performed in the intensive care unit so long as equipment and trained assistance are available for the surgeon. A minitracheostomy is useful for patients who are unable to tolerate but who do not require continuous positive airway pressure or assisted positive pressure ventilation. It is used as an adjunct to regular physiotherapy. Such procedures can be done at the bedside, with a local anaesthetic only. But they carry many of the risks of
formal tracheostomy, and the potential for haemorrhage should not be underestimated.

Early complications of tracheostomy include pneumothorax and pneumomediastinum, subcutaneous emphysema, iatrogenic haemorrhage, and tube displacement. Aspiration of gastric contents may occur during any airway manoeuvre where the protective reflexes are obtunded. The higher cricothyrotomy approach may produce laryngeal injury, but is not associated with pleural damage. Stomal bleeding is common, though usually not serious. Stomal infection is seen in about 12% of all tracheostomies, and often arises on the fourth or fifth postoperative day.

Later complications of tracheostomy include tube obstruction, aspiration, swallowing dysfunction, and erosion into the oesophagus or the innominate artery. Even if stenosis does not occur, abnormal scar formation and granuloma may occur at the stoma site.

Tracheal stenosis produces stridor in adults only when over 75% of the lumen is obstructed. The most direct method of assessing tracheal stenosis is by fibreoptic bronchoscopy or fibreoptic laryngoscopy, though this carries a risk from the anaesthetic. Lateral soft tissue radiographs of the neck with fluoroscopy are helpful. Maximum flow-volume loops and computed tomography are less sensitive methods of assessment.

All artificial airways require humidification or at least the conservation of exhaled water vapour. Heat-moisture exchangers are adequate for most cases, though high flow rates and tenacious secretions may require a heated humidifier. The use of bacteriostatic materials is now common. Patients with tracheal tubes or tracheostomies are vulnerable to infection because of disrupted local clearance mechanisms, underlying immunosuppression, frequent suctioning, and the microbiological environment of the intensive care unit. Viral infection is easily spread from staff to patients. Contamination of the tracheal tube often precedes pneumonia by two to four days. Pseudomonas aeruginosa is associated specifically with having a tracheostomy.

Sedation and analgesia

Patients in the intensive care unit are exposed to many harmful stimuli. Some are later forgotten, but sensations that are even temporarily unpleasant should be avoided if possible. Among the common problems are anxiety, pain, lack of rest, thirst, tracheal tubes, face masks, nasogastric tubes, urinary catheters, and physiotherapy. Any nursing procedure, such as turning or changes of dressing, are likely to be at the least uncomfortable.

Much can be achieved by careful explanation and appropriate reassurance from intensive care unit staff. Attitudes to the use of sedative drugs have changed in the last few years and there is increasing recognition that sedation, analgesia, and muscle relaxation should be provided specifically where indicated. The use of muscle relaxants is restricted to patients in whom movement is dangerous (particularly with raised intracranial pressure, or where artificial ventilation is difficult, as in severe asthma). Muscle relaxants are not sedatives, and awareness of paralysis is terrifying and should be prevented by the use of sedatives.

Apart from subjective assessments of the adequacy of sedation, several sedation scoring systems have been designed. One system uses simple end points: fully alert, roused by voice, roused by pain, unrousable, paralysed, asleep. Another identifies anxiety or restlessness as a separate category. There are several other systems. Such a system should be used by all staff in an intensive care unit, so that assessments can compare different patients and different drugs.

The simplest method of administering sedatives and analgesics uses repeated bolus doses. This, however, permits peaks and troughs of awareness and pain, which can be avoided by using intravenous infusions. The institution of an infusion should be preceded by a loading dose, account being taken of any cardiovascular or respiratory depressant effects that the drug may have. The rectal route has also been used, and subcutaneous infusions of opiates have been in use in our intensive care unit for several years. Patient controlled sedation and analgesia are also used in some centres.

The ideal sedative (and analgesic) drug has been described as having the following properties: rapid onset and recovery by bolus or infusion; wide therapeutic index; minimal cardiovascular effects; respiratory depression that does not persist; water solubility; lack of irritation to veins; absence of metabolic, immunological, or hypersensitivity reactions; and absence of confusion after the drug is stopped. Such a drug should also be cheap. This is still a tall order, and no drug approaches it. Most intensive care units use a combination of opiates and benzodiazepines, given by bolus injection or infusion, but there has never been a wholly satisfactory alternative to the effective but dangerous drugs etomidate and Althesin. Opiates are widely used, each intensive care unit having its current preference for one or another. All pure opiate agonists are respiratory depressants and antithrombocytic agents, which may be useful; these properties, however, also delay weaning from the ventilator. Vasodilatation is common with opiates, which may be dangerous in the hypovolaemic patient; and gastric emptying and intestinal motility are slowed, which this may delay enteral feeding.

Tolerance to analgesia is common, but addiction is very unlikely if the drug is used for a patient in pain. Relying on opiates alone for sedation is inappropriate in view of their problems, especially in patients with renal impairment, where clearance may be very slow.

Morphine is very useful (and cheap) but has active metabolites. Papaveretum, being half morphine, has similar properties, although its other constituent alkaloids may be more sedative. The short duration of a single bolus dose of fentanyl is due to redistribution rather than clearance, and its elimination half life is
longer than that of morphine; there is therefore no indication for its use in intravenous infusions.52

Alfentanil,47 55-58 with a short elimination half life and a small volume of distribution, is well suited to continuous intravenous use. It is dependent on hepatic metabolism, however, and is expensive. Pethidine has less sedative effect than the other opiates while being an effective analgesic, so it is useful in the awake and spontaneously breathing patient.

The use of regional analgesia is increasing. Epidural opiates do not require such precise placement of an epidural catheter as do local anaesthetics. The risk from respiratory depression is much reduced in an intensive care unit, where constant monitoring and observation should prevent complications. Non-opioid analgesics may be given intramuscularly or rectally. Indomethacin reduces opiate requirements, particularly after operation,53 but may produce gastrointestinal bleeding.

The benzodiazepines are purely sedative, and used alone are inappropriate for patients in pain. They also reduce muscle tone, and promote amnesia. Diazepam has an active metabolite, N-desmethyl diazepam, which has a long elimination half life, and this reduces its usefulness for intravenous infusions.

Midazolam is water soluble and has an elimination half life of two to four hours after a single dose.47 It is particularly useful for intravenous infusion but bolus doses may produce hypertension, especially in the hypovolaemic patient.59 It has a large volume of distribution in some patients, particularly in those with hypoalbuminaemia.60 61 This may explain the apparently unpredictable prolonged effect in patients with multiple organ failure.62 Simultaneous infusion of a combination of alfentanil and midazolam has been recommended,57 59 62 but the mixture of the two drugs in one syringe is therapeutically unsound as each patient has differing requirements for analgesia and sedation. The use of midazolam is common in children.63 64

Chloromethiazole, given by intravenous infusion gives a rapid onset sedation that is easily adjusted, and it is specifically anticonvulsant;55 it contains a large water load, however, and is slow to wear off.66

Chloral hydrate, often given to neonates and infants' by nasogastric tube, reduces opiate and benzodiazepine requirements.

Chlorpromazine, given intramuscularly or rectally, is useful for calming patients suffering from opiate or other drug withdrawal. Its alpha blocking properties may cause problems in hypovolaemic patients.

Ketamine, unlike other sedatives, increases arterial resistance and causes bronchodi- lation. These effects may be useful in hypovolaemic and asthmatic patients respectively. Concurrent use of midazolam may reduce psychological after effects.67 Propofol infusions are being used increasingly in the intensive care unit.68-71 Our policy is to use this excellent but expensive agent for short periods only, particularly when the neurological state has to be examined fre-

quently.

Inhaled sedative agents such as isoflurane have been introduced in some intensive care units,72-74 where scavenging of exhaled gas is possible. The cost of isoflurane used in this way is high, and this has discouraged its widespread application.75

The pharmacological choice for sedation and analgesia in the intensive care unit is large but in the end each unit tends to have its own policy, which medical and nursing staff clearly understand. This is a sensible approach, so long as alternatives are constantly considered.

Non-respiratory monitoring of the ventilated patient

Haemodynamic monitoring is essential when the critically ill patient is ventilated. All patients should have a correctly sited central venous pressure (CVP) line. Central venous pressure measurements are useful in the fluid management of patients who have no appreciable pulmonary hypertension or cardiovascular disease. It is important for the patient not to have a low central venous pressure before assisted ventilation is started as the combination of an inadequate venous return to the right heart and positive pressure ventilation may lead to a catastrophic fall in blood pressure. The alterations in cardiac output during positive pressure ventilation have been ascribed to alterations in preload, with increased intrathoracic pressure causing peripheral translocation of central blood volume.76

The elegant work of Wise et al77 78 and Sylves


ter et al79 has examined many of the factors affecting ventricular performance during mechanical ventilation with positive end expiratory pressure. It is now accepted that where ventilator reserve is limited and positive end expiratory pressure and ventilator settings have to be adjusted these adjustments should be made to optimise oxygen delivery and mixed venous oxygen saturation (which should if possible be over 70%). As with right atrial pressure, measurement of pulmonary capillary wedge pressure requires correct zero recordings and calibration; the measurement should be taken from the "a" wave at end expiration so that the level is not influenced by respiratory pressures.80

Other requirements for accurate measure-

Table 1 Conditions for accurate measurement of pulmonary capillary wedge pressure in the ventilated patient

<table>
<thead>
<tr>
<th>Condition</th>
<th>Measurement</th>
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<tbody>
<tr>
<td>Correct zero and calibration</td>
<td>Measure at end expiration from the &quot;a&quot; wave</td>
</tr>
<tr>
<td>Ensure that the catheter tip is sited below the level of the left atrium</td>
<td>Ensure no interference with pulmonary venous drainage between the catheter tip and left atrium (for example, pulmonary embolus)</td>
</tr>
<tr>
<td>There should be no appreciable gradient between left atrium and left ventricle (for example, mitral valve disease, where large &quot;v&quot; waves may be mistaken for &quot;a&quot; waves)</td>
<td>When the balloon is deflated there should be a distinct pulmonary artery trace</td>
</tr>
<tr>
<td>Should a wedge trace appear the catheter must be withdrawn until the pulmonary artery trace reappears</td>
<td>Should a wedge trace appear the catheter must be withdrawn until the pulmonary artery trace reappears</td>
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ventilated carditis.

Measurement of venous oxygen saturation in the ventilated patient

Table 2: Indications for measuring cardiac output, oxygen delivery, and mixed venous oxygen saturation in the ventilated patient

<table>
<thead>
<tr>
<th>Indication</th>
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<tbody>
<tr>
<td>Raised central venous pressure</td>
</tr>
<tr>
<td>Appearance of pulmonary oedema on the chest radiograph</td>
</tr>
<tr>
<td>Hypotension in the presence of normal central venous pressure and hypervolemic response to volume challenge</td>
</tr>
<tr>
<td>Optimisation of ventilation</td>
</tr>
<tr>
<td>Optimisation of inotropic support</td>
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</table>

In patients with high airway pressures the difference between the pressure transmitted to the catheter tip (wedge pressure) and that transmitted to the left atrium cannot be predicted. In critically ill patients the mean effective pulmonary capillary pressure (measured from the downstroke of the wedge pressure as the point when the rapid decline changes to a slower decline) may be a better measure than pulmonary capillary wedge pressure as any increase in the former may worsen gas exchange.

Cardiac output measurements are usually made by using a pulmonary artery catheter with a thermodilution technique at its tip. The thermodilution technique is as accurate and reproducible as the dye dilution technique, provided that the catheter is placed accurately, the volume and temperature (0°C) of the injectate are accurate, and the injection is always performed at the same point in the respiratory cycle. The derived variable, oxygen delivery, is of particular value for optimising ventilation and can be combined with the measurement of mixed venous oxygen saturation (SVO2) in the pulmonary artery, by blood sampling or the use of a catheter with an optical probe at its tip. Mixed venous oxygen saturation may not be an early predictor of change in cardiac output but it provides an indication of the delicate balance between oxygen delivery and extraction.

Indications for haemodynamic monitoring and measurement of derived variables are listed in Table 2. Recent work has explained the importance of right ventricular performance in patients with acute respiratory failure and how it changes with positive end expiratory pressure. Measurement of right ventricular volume variables has been made possible by the "fast response" thermodilution technique. Bronet and coworkers found that, though in many patients with acute respiratory failure and increasing pulmonary hypertension the right ventricle is dilated and right ventricular ejection fraction decreased, stroke volume was maintained unless there was concomitant disease, such as septic shock or viral myocarditis. Preload augmentation in these patients is clearly important, and it is necessary in respiratory failure to ensure that left atrial pressures are maintained. Positive end expiratory pressure has been found to have two effects on right ventricular function. In most patients it causes unloading of the right ventricle by reducing venous return, and in a few it leads to right ventricular dilatation and a decreased ejection fraction. A further study suggested that the changes in the right ventricle induced by positive end expiratory pressure are probably a function of the initial right ventricular ejection fraction and right ventricular end diastolic volume index.

Nutrition in the ventilated patient

Acute weight loss of 30–40% of the original body weight is usually lethal and maximal physical performance is impaired in healthy individuals who have lost around 10% of body weight. Increased mortality and morbidity correlate closely with an acute loss of body weight of 10–30% of the individual's normal weight.

Clearly the best route for providing nutrition is the gastrointestinal tract. Calories given via the gastrointestinal tract maintain the integrity of the liver and oxidation of nutrients, a process that is imprecisely understood. This route is often not suitable, however, in the ventilated patient because of the nature of the illness and the use of drugs that suppress bowel motility. When bowel absorption is not possible intravenous nutrition may be necessary, but it is important to reintroduce gastrointestinal feeding as soon as possible. When it is reintroduced after several days or weeks of parenteral nutrition, this must be done gradually; diarrhoea may ensue unless the osmolality of the feed is increased slowly over several days.

In the critically ill ventilated patient metabolic and volume normality must be established before nutrition is started (Table 3). When a normal arterial carbon dioxide tension cannot be achieved by adjusting the ventilator settings, the total calories given as glucose should be reduced to cut down carbon dioxide production.

Parenteral nutrition should be given via a designated feeding line. The line is best tunneled subcutaneously and is generally inserted into the subclavian vein by the infraclavicular technique. This method may be complicated in the ventilated patient by a pneumothorax, and should therefore be avoided in patients with poor respiratory function (low arterial oxygen tension). All electrolyte deficiencies must be corrected Acid-base state must be stable Adjust ventilation to ensure a normal arterial oxygen tension Ensure normal calcium, magnesium, and inorganic phosphate concentrations Determine serum B12, folate, and albumin concentrations before starting treatment Cardiac and renal function must be known Tolerance to a glucose load must be known Is there evidence of hepatocellular failure?
tension and saturation despite optimum ventilation and high fractional inspired oxygen, particularly when airway pressures are high. In these circumstances a feeding line may be inserted high into the internal jugular vein and tunnelled in the neck.

Energy requirements for the critically ill have traditionally been established by the use of the Harris-Benedict equation.\(^9\) The total energy expenditure is then calculated by multiplying the basal metabolic rate by a stress factor, which may be augmented by an allowance for physical activity. This method was admirably described by Apelgren and Wilmore,\(^91\) and is still being used in units where the total energy expenditure cannot be estimated by indirect calorimetry (table 4). Cortes and Nelson\(^92\) showed that clinical assessment may overestimate energy expenditure because the apparent degree of illness used as the basis for determining the stress factor is not an accurate guide. Not only is bedside calorimetry useful in more accurately assessing energy expenditure but it may also lead to financial saving. A nomogram for rapid calculation of metabolic requirements has been described.\(^93\) It relies on the assumption that the respiratory quotient (RQ, the ratio of carbon dioxide production to oxygen consumption) is 0·8 and that expired gas can be analysed for carbon dioxide. Carbohydrate has an RQ of 1, fat 0·7, and protein 0·82, however, and lipogenesis (the synthesis of fat from glucose) has an RQ of 8. Carbohydrates given in excess of energy consumption promote lipogenesis, with a consequent increase in carbon dioxide production. In respiratory failure adjustment of the ventilator settings to lower the PaCO\(_2\) may not be possible. In these circumstances, to avoid hypermetabolism related to a high glucose load, the proportion of carbohydrate

table 4 Determination of energy requirements for total parenteral nutrition

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>kcal/day</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>1316</td>
<td>1411</td>
<td>1509</td>
<td>1602</td>
<td>1694</td>
<td>1872</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2 “Stress factor”:\(1\) normal BMR is correction for the disease process stress factor applied to the normal basal metabolic rate.

- Mild Starvation: 0·85–1·00
- Preoperative (no complication): 1·00–1·05
- Cancer\(^\dagger\): 1·10–1·45
- Peritonitis\(^\dagger\): 1·05–1·25
- Severe infection or multiple trauma\(^\dagger\): 1·30–1·55
- Burn\(^\dagger\): 1·50–1·70

3 The basal caloric requirements of stressed patients are not adjusted upward when they are heavily sedated and ventilated, but consider an increase of up to 20% in non-sedated patients.

4 If anabolism and weight gain are the goals, an additional 1000 calories/day may be added.

*Modified from Apelgren and Wilmore.\(^91\)
†Proportional to the extent of disease or injury.

Conversion from traditional to SI units: 1 kcal = 4.184 kJ.

Table 5 Nutritional considerations during weaning

- Observe arterial carbon dioxide tension in relation to carbohydrate load.
- Ensure a non-protein calorie:nitrogen ratio of around 150:1.
- Carbohydrate load should be 40–60% of total non-protein calories, the rest being given as fat.
- Nitrogen load should in general not exceed 14 g daily; if hyperventilation is present consider the possibility that the nitrogen load is excessive.
- Ensure a normal serum phosphate and potassium content.

Observe the fluid balance; do not overload.

Non-nitrogen containing calories should be 40–60%, the rest being given as fat.\(^94\)

The use of fat rather than dextrose as a source of calories has two advantages. The respiratory quotient of fat oxidation is 0·7, compared with 1·0 with dextrose, so carbon dioxide production is lower; and fat, being iso-osmolar and of neutral pH, can be given via a peripheral vein.

The nitrogen requirements are generally assessed by measuring the urinary urea nitrogen loss in the urine.\(^95\)

Nitrogen (N) balance = grams of N - grams of urea N + 4 g IN

The 4 g factor accounts for the unmeasured nitrogen losses in skin and stool. Askanazi and coworkers\(^96\) investigated the respiratory response of patients to increasing protein supply and found that progressive increase enhanced ventilatory drive and minute volume, but that in patients with limited reserve this may lead to respiratory failure in the spontaneously breathing patient. Precise protein requirements in patients requiring ventilatory support remain to be established.\(^97\) Even in the presence of high nitrogen losses the nitrogen load is rarely increased above 14 g daily.

It is important in parenteral feeding to assess electrolyte and fluid balance regularly, with special attention to potassium and phosphate requirements. Hypophosphataemia is known to reduce oxygen transport and energy supply, and when severe may lead to respiratory failure.\(^97\) Vitamins and trace elements should be replaced if feeding is continued for more than five days. Prolonged nutrition is best supplied by a mixture of protein, fat, electrolytes, vitamins, and trace elements made up into a 3 litre bag, prescribed early in the morning and supplied by the pharmacy later in the day. The mixture has to be carefully compiled to ensure compatibility between the different constituents of the feed.

Some studies suggest that ventilator weaning may be facilitated by preceding nutritional support.\(^98,99\) Evidence suggests that respiratory muscle function is diminished in poorly nourished patients. Kelly\(^100\) evaluated initial maximum inspiratory mouth pressure as an index of respiratory muscle function in 51 patients in hospital. Malnourished patients were found to have significantly less inspiratory force than normally nourished patients. The electrolyte content of the diet is particularly important during weaning—see table 5 (weaning is considered in more detail in the next article in this series). Further details on nutrition are sup-
Psychological and sleep disturbances during assisted ventilation
Psychiatric symptoms relating to assisted ventilation will not be manifest while the patient is heavily sedated and ventilation is controlled but are likely to emerge during weaning. It is clearly important to exclude organic dysfunction of the brain and physiological derangements due to blood gas or metabolic abnormalities.

The intensive care environment is recognised as causing stress, as a result of the alien and frightening atmosphere, sleep deprivation, unfamiliar noise, and a feeling of being confined by equipment, for example. Assisted ventilation produces additional stresses, related to awareness of the endotracheal or tracheostomy tube, the discomfort of suction and complicating hypoxia, and the horror of depending on a machine for ventilation.

Gries and Fernsler conducted a survey to assess the causes of stress associated with ventilation in 17 patients, whose ages ranged from 35 to 81 years. Five patients could not recall the period of ventilation and three did not wish to discuss the problem. The others categorised the stresses as shown in table 6. The major complaints were related to restrictions of activity, the awareness and unpleasantness of the tracheal tube, suction, and the process of extubation. Inability to communicate was also frustrating. Some had vivid dreams, probably related to drugs. The more serious psychological problems—"the intensive care syndrome"—include disturbances of cognitive, affective, and perceptual functions. These occur in 12.5-18% of patients, and are likely to be related to metabolic, neurological, or pharmacological factors. Many are related to drug dependence and withdrawal, and are common, and may manifest themselves as aggression, non-recognition of relatives, periods of agitation, and non-cooperation. These changes are most likely to arise in the patient who has been ventilated for a long time or who has previously been dependent on drugs—for example, on benzodiazepines, opiates, or alcohol. Drug withdrawal during weaning from the ventilator may be extremely difficult. Methadone is useful, enabling opiates to be rapidly reduced, and on occasion, where they are considered safe, beta blocking drugs may alleviate the tachycardia and anxiety related to opiate withdrawal. Rectal chlorpromazine given regularly, at the onset of weaning, has a potent calming effect without depressing the respiratory centre.

Gale and O'Shanick discussed preventive psychological interactions for the ventilated patient, which are noted, with modifications, in table 7. Communication problems may in the future be reduced by the use of a word processor by the patient. Patients in an intensive care unit rarely sleep more than a few hours at a time, and frequently do not complete a sleep cycle. Completion of a sleep cycle is essential for maintaining and restoring physical and psychological functions. Experiments in which people are deprived of sleep show that after two to five days subjects become anxious, suspicious, and disoriented, some developing delusions and paranoia. Lack of prolonged sleep may be an important factor contributing to the intensive care syndrome. Weissman and colleagues found that the average length of a sleep period in an intensive care unit was only 24 minutes. The noise level in an intensive care unit is high,

Table 7: Preventive psychological interactions

| Communication problems | Talk to the patient
|------------------------|----------------------
|                        | Provide alphabet, sign, or picture board writing tablet word processor
|                        | Dependence and loss of control
|                        | Allow the patient choice when possible (position in bed, radio station, etc)
|                        | Keep patient informed of progress
|                        | Inform patient of procedures to be undertaken, and the reasons for them
| Fear of death or disability | Explain the ventilator and its alarm systems
|                        | Inform the patient of changes in ventilator settings and the reasons for them
|                        | Allow the patient to express his emotional problems and give support
| Keep discussion and controversies about management away from the bedside | Isolation and fear of strangers
|                        | Establish continuity of care
|                        | Encourage visits by family and close friends
| Sensory alteration | Maintain the patient's orientation with a calendar and clock on the wall, family photographs, and visits by relatives and close friends
|                        | Establish a day-night routine if at all possible
|                        | At night minimise noise
|                        | minimise movement and interference with the patient ensure adequate analgesia and maximise comfort
|                        | During the day provide a daytime environment with, for example, visits television, music, etc.

Table 6: Stressors associated with mechanical ventilation

| Intrapsychical: physiological | Frustration from activity restriction
| Awareness of spontaneous breathing restriction related to ventilation |
| Intrapsychical: psychosocial and cultural | Insufficient explanation and hence misinterpretation of medical condition
| Activity restriction producing a feeling of inability to cope |
| Ventilator dependence |
| Vivid dreams |
| Interpersonal | Awareness of extubation |
| Insufficient explanations |
| Inability to communicate |
| Loss of confidence in and criticism of nursing care |
| Extrapsychical | Unpleasant experiences relating to the tracheal tube |
| Suctioning |
| Extubation |
| Noise—from ventilator or surroundings |

*Modified from Gries and Fernsler.
and has been found to be a major cause of sleep deprivation.\textsuperscript{14,15} Methods whereby a normal sleep cycle can be encouraged are summarised in table 7.

The psychological needs of the ventilated patient have so far received scant attention. Aspects of care include maintaining a normal sleep cycle, minimising noise (particularly at night), awareness of the problems associated with drug dependence and withdrawal, helping and communicating with patients, and endeavouring to establish reorientation and a pleasant and calm environment during weaning.


84 Needham PF, Suer PM. Changes of right ventricular function with positive end expiratory pressure (PEEP) in man. Intens Care Med 1988;14:471-3.


109 Cronin LR, Carrizosa AA. The computer as a communications tool for ventilator and traumecotmy patients in the intensive care unit. Critical Care Nurse 1984; Jan-Feb:72-6.

110 Belitz J. Minimising the psychological complications of patients who require mechanical ventilation. Critical Care Nurse 1983; May-June:42-6.


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