GENERAL MEDICAL CARE ON THE NEUROMEDICAL INTENSIVE CARE UNIT

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The role of an intensive care unit is to maintain a patient's normal physiological homeostasis while actively treating the underlying cause of any physiological derangement. Modern neurological intensive care evolved from the neurorespiratory units established in the 1950s to treat patients with respiratory failure caused by poliomyelitis. Thus there has been greater emphasis on the longer term care of the paralysed patient than in the conventional general medical intensive care unit. This review attempts to give an overview of the management of patients treated in the neuromedical intensive care unit (NICU) and is directed towards the general neurologist rather than the intensivist. More detailed texts of neurological and general intensive care are available.[1–4] Discussion will be targeted towards a number of areas:

- respiratory system
- cardiovascular system
- alimentary system
- nosocomial infection and infection surveillance
- anticoagulation
- patient comfort.

RESPIRATORY SYSTEM

Patients with acute neurological disorders require tracheal intubation and ventilation because of the development of acute respiratory insufficiency or because they are unable to protect their upper airway from obstruction as a consequence of impaired consciousness or bulbar weakness.[3] The latter predisposes to pulmonary aspiration of saliva and food that cannot be cleared by the patient because of an inadequate cough secondary to poor diaphragmatic and anterior abdominal wall musculature. Bronchopneumonia often results.

Respiratory insufficiency and failure is a common manifestation of a wide variety of neurological disease. Central nervous system (CNS) and brainstem diseases cause respiratory depression characterised by central disorders of the respiratory pattern, including hyperventilation, irregular, ataxic or cluster breathing, hiccup, and recurrent apnoea. These may result in inadequate oxygen delivery and hypercapnia, culminating in respiratory arrest. Neuromuscular disease is discussed elsewhere in this supplement and is characterised by symptoms of progressive respiratory impairment or the development of respiratory failure.

Whatever the aetiology, the resultant respiratory muscle weakness leads to decreased tidal and minute volumes, atelectasis, and ventilation/perfusion imbalance, all of which contribute to hypoxaemia and hypercarbia. The development of respiratory symptoms may be relatively insidious (for example, in motor neurone disease) or sudden (for example, Guillain-Barré syndrome).

How should the general neurologist monitor patients with neurological respiratory insufficiency? The most useful and reproducible test on the general ward is the measurement of the forced vital capacity (FVC) (the largest volume that can be expired slowly after a maximal inspiration) using a spirometer. The normal value is approximately 70–75 ml/kg. Serial measurements often indicate whether respiratory muscle function is stable or deteriorating and are especially important in fluctuating diseases such as myasthenia gravis. It is important to note that by the time arterial blood gas tensions have become abnormal respiratory muscle function is often severely compromised.

There are a number of indications for tracheal intubation and mechanical ventilation and these are listed in box 1.

Airway management

In severe neurological illness it is essential to establish an adequate airway rapidly, as acute hypoxaemia and hypercapnia may exacerbate the severity of the insult. Initial management of intermittent airway obstruction involves the placement of an oral or nasal airway. Any patient who is unable to protect or maintain a patent airway requires tracheal intubation. Apart from providing a route

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Box 1: Indications for tracheal intubation

- Respiratory failure (that is, Pa_{O_2} < 8 kPa (60 mm Hg) breathing room air) with or without hypercarbia
- A decreasing forced vital capacity (FVC) in the presence of bulbar dysfunction
- Bulbar failure (in order to protect the airway from pulmonary aspiration)
- Encephalopathy/coma
- To provide control of Pa_{CO_2} in patients with raised intracranial pressure
- Cardiovascular instability (for example, shocked patient)

for mechanical ventilation and the application of continuous positive airways pressure (CPAP), tracheal intubation may be required to secure and maintain a clear airway, protect the lungs from aspiration, and to allow control of bronchial secretions with tracheal suction.

Many patients find it difficult to tolerate oral endotracheal tubes. This applies particularly to those who are fully conscious and require prolonged intubation. In these cases the nasal route may be preferred since it is more comfortable, secure fixation of the tube is more easily achieved, and proper mouth care can be undertaken. However, nasal intubation is contraindicated in patients with adjacent facial or skull fractures and may be associated with bacteraemia secondary to sinus infection or otitis media and meningitis if there is a leak of cerebrospinal fluid (CSF).

The nature of neurological respiratory failure often dictates a prolonged period of mechanical ventilation. When this is the case, the orotracheal tube should be replaced by a tracheostomy as soon as possible. Tracheostomy allows greater patient comfort, easier nursing management (including suction), and the possibility of oral nutrition and speech, as well as aiding weaning from mechanical ventilation by reducing respiratory dead space. Increasingly, surgical tracheostomy is being replaced by a percutaneous procedure which may be carried out in the intensive therapy unit.

Mechanical ventilation

Mechanical ventilation of the lungs[5 6] may be delivered using intermittent negative pressure ventilation or intermittent positive pressure ventilation. The former is delivered using devices such as tank ventilators (iron lungs) and cuirass ventilators; apart from in a few specialised units, these devices are rarely used nowadays.

Intermittent positive pressure ventilation (IPPV) has evolved extensively since the original "bellows" concept used in neurorespiratory units during the 1950s poliomyelitis epidemics; this evolution has been accompanied by an ever increasing terminology. However, a simplified guide to the most common modes of ventilation is given below.

During *controlled mechanical ventilation* the ventilator delivers a preset tidal volume at a preset respiratory rate, independent of the patient's respiratory effort. Thus the patient is completely dependent on the ventilator. It is used for patients with severe neurological disorders who are unable to breathe or who have received neuromuscular blocking agents. In *intermittent mandatory ventilation* a mandatory minute volume is preset and delivered by the ventilator, but the patient is allowed to breathe spontaneously from a gas source between ventilator breaths. As the patient weans, the proportion of minute ventilation delivered by the ventilator is reduced, usually by decreasing the respiratory rate. Coordination of the positive pressure ventilation by the ventilator so that it does not coincide with a spontaneous breath is known as *synchronised intermittent mandatory ventilation (SIMV)*. *Inspiratory pressure support* is a refined form of assisted ventilation in which the patient's spontaneous breath is augmented with supplementary gas flow. It requires the patient to generate a negative pressure before augmentation occurs (that is, the patient needs to make an inspiratory effort). *Inspiratory volume support* is a variation of inspiratory pressure support in which the ventilator automatically monitors the lung properties and modifies the inspiratory pressure support in order to deliver a predetermined tidal volume. *Positive end expiratory pressure (PEEP)* is an adjunct to IPPV and maintains a positive pressure during expiration (that is, instead of airway pressure returning to atmospheric at end expiration, there is a variable positive pressure). This results in splinting of the small airways and alveoli, preventing collapse. It improves oxygenation and reduces pulmonary shunt. *Continuous positive airway pressure (CPAP)* is similar in principle to PEEP; CPAP is the application of positive airway pressure throughout all phases of spontaneous ventilation. It is a useful mode for weaning patients from mechanical ventilation.

Weaning

Weaning of patients from mechanical ventilation may start as soon as respiratory muscle function returns; the patient needs to be able to maintain adequate oxygenation, a normal respiratory rate, and appropriate spontaneous tidal volumes with minimal mechanical support. In addition, the patient needs to maintain a patent airway, to initiate cough and gag reflexes, and to clear secretions independently. Conditions for successful weaning include the absence of severe infection, adequate nutrition, absence of electrolyte disturbance (especially potassium, magnesium, and phosphate), minimum sedation, and arterial blood gas tension near premorbid levels on reasonable inspired oxygen concentrations. Sophisticated ventilatory modes discussed above aid weaning, but many units prefer to wean by allowing patients to breathe spontaneously off the ventilator for increasing periods solely with CPAP.

Pulmonary complications

Pulmonary complications are common in NICU patients. The most frequent manifestations are acute hypoxaemic respiratory failure caused by ventilation–perfusion mismatch from atelectasis, aspiration, pulmonary oedema or pulmonary embolus.

Nosocomial pneumonia

Nosocomial or aspiration pneumonia[7] is usually caused by gastric and oesophageal colonisation with aerobic bacteria and repeated small volume aspiration during sleep, especially with impaired pharyngeal reflex (box 2). It usually develops subclinically and fulminant aspiration syndrome is rare. There may be no symptoms before the development of transient hypoxaemia or infiltrates. Other patients may present with dyspnoea, tachypnoea, tachycardia, and hypoxia with or without pyrexia. Severe infection with a large intrapulmonary shunt will result in hypoxaemia, hypovolaemia, and hypotension. Pulmonary artery pressure may increase because of hypoxaemic vasoconstriction, and pulmonary oedema can develop secondary to acid induced lung damage. Chest *x* ray usually shows right lower lobe infiltration, atelectasis, and air bronchogram with bilateral, diffuse infiltrates. Intensive physiotherapy and nursing support are essential, and mechanical ventilation may be indicated if progressive infiltration abnormalities are shown. Appropriate broad spectrum

Box 2: Risk factors for nosocomial infections

- Age over 70
- Mechanical ventilation
- Colonisation of the oropharynx pharynx
- Use of H_2 receptor antagonists
- Underlying chronic lung disease and reintubation

empirical antibiotic treatment with a cephalosporin and aminoglycosides are used, although vancomycin is preferred if *Staphylococcus aureus* pneumonia is suspected. For aerobic infections a cephalosporin and metronidazole are indicated.

Ventilator associated pneumonia

Ventilator associated pneumonia may develop at least 48 hours after intubation. It is associated with retrograde colonisation of the oropharynx caused by gastro-oesophageal reflux and possibly also cross colonisation from contaminated hospital personnel or equipment. In addition many mechanically ventilated patients are immobile and cannot expand the distal airways with sighs, deep breaths, and coughs. It is usually caused by *Pseudomonas aeruginosa* infection.

Adult respiratory distress syndrome

Adult respiratory distress syndrome (ARDS)[8] is a form of non-cardiogenic pulmonary oedema characterised by reduced respiratory compliance and increased work of breathing and V/Q mismatch, leading to an increased shunt and hypoxaemia.

Pulmonary vascular resistance is raised and the chest *x* ray shows bilateral patchy "cotton wool" infiltrates, typically peripherally. ARDS may follow any major illness, sepsis or surgery but is common following aspiration or septicaemia. Multiorgan failure may occur and is a common cause of death. The management is largely supportive with nutrition, deep vein thrombosis (DVT) prophylaxis, and prevention of infection. Ventilatory support is required with oxygen therapy and PEEP. Other forms of ventilatory support have also been used.

Neurogenic pulmonary oedema

Neurogenic pulmonary oedema[9] may develop after an acute injury to the brain or brainstem in patients with intracranial pathology, including subarachnoid haemorrhage, cerebral emboli, cerebral tumours, status epilepticus, and raised intracranial pressure (ICP). Neurogenic pulmonary oedema always appears soon after the onset of the neurological event and should be distinguished from severe aspiration, ARDS, and multiple pulmonary emboli. The patient may present with excessive sweating, hypertension, tachypnoea, and frothy sputum. Chest *x* ray shows diffuse pulmonary infiltrates, but the diagnosis depends on showing hypoxaemic respiratory failure with a normal pulmonary artery wedge pressure in the absence of a cardiac cause. The management depends on the recruitment of collapsed alveoli with PEEP to correct the pronounced ventilation–perfusion mismatch.

Pulmonary embolism

Patients with neurological disease are susceptible to DVT, particularly if they are immobilised or have paralysis. This may lead to pulmonary embolism[10] with mechanical obstruction of a pulmonary artery or arteriole caused by a blood borne thrombus. The presentation is often variable and pulmonary emboli may be silent and difficult to diagnose. Smaller emboli may cause few haemodynamic effects or may result in infarction of a section of lung tissue if collateral blood flow is inadequate. Massive pulmonary emboli cause rapid respiratory and cardiovascular collapse and death. The treatment of pulmonary embolism depends upon its size. Cardiopulmonary resuscitation may be necessary following a massive embolus with the patient in shock from acute cor pulmonale, justifying aggressive treatment with thrombolysis. Supportive treatment includes correcting hypoxaemia, intravenous fluid replacement, inotropic drugs, and analgesia. Mechanical ventilation may be necessary if the patient cannot maintain adequate oxygenation. Anticoagulation is usually begun with unfractionated heparin, the dose being titrated against the activated partial thromboplastin time. In ischaemic stroke, dosage should not be adjusted to lower levels for fear of haemorrhagic conversion as an inadequate dose increases mortality from pulmonary emboli. Warfarin administration should begin 48 hours after the start of heparin and continue for six months thereafter.

CARDIOVASCULAR SYSTEM

Regarding the cardiovascular system, the principle function of the NICU is the monitoring and maintenance of arterial blood pressure and blood volume to ensure adequate organ perfusion.[11 12]

Mean arterial pressure is the product of cardiac output and systemic vascular resistance (SVR). In turn, cardiac output is the product of stroke volume and heart rate. It therefore follows that in order to maintain organ perfusion, stroke volume (affected by venous return to the heart and myocardial contractility), heart rate, and SVR must be kept at normal levels. From the above, it will be apparent that hypotension may be the result of an inadequate circulating blood volume, poor myocardial contractility, a decreased SVR or a combination of these factors. Monitoring of these variables with central venous or pulmonary artery catheters, or devices using Doppler principles, help untangle the explanation of hypotension and allows appropriate treatment. It must be stressed that low blood pressure is often caused by a combination of factors—for example, sepsis causes decreased myocardial contractility as well as a decreased SVR.

Hypovolaemia

Hypovolaemia is an important factor in many patients with critical neurological illness and, in particular, acute intracranial events. The choice of fluid replacement depends on a number of factors. If the patient is anaemic (haemoglobin < 8 g/dl), transfusion of red cells should be considered, especially if the patient has a history of ischaemic heart disease. If the patient's haemoglobin is above this value, blood volume may be increased by the use of colloids or crystalloid solutions. The former include blood products, hetastarches, and gelatin derivatives. These solutions are favoured by many as they stay in the intravascular space for a longer period, lower volumes are required for correction of the decreased blood volume, and they result in less peripheral oedema. However, colloids are expensive, carry the risk of anaphylaxis, and the absence of clotting factors may precipitate a coagulopathy. The alternative is crystalloid solution. Generally, in neurointensive care practice, 0.9% sodium chloride solution is favoured over glucose or lactate containing solutions which have the potential to increase intracellular glucose concentrations and thus increase the risk of ischaemic damage.

Decreased myocardial contractility

Decreased myocardial contractility may be the result of many factors including sepsis, hypoxaemia, hypercarbia, acidosis, cardiac disease, electrolyte disturbance, and drugs (for example, most intravenous anaesthetic agents used for sedation of patients on NICU). Inotropic drugs such as noradrenaline (norepinephrine), adrenaline (epinephrine), and dobutamine are used to improve contractility and therefore cardiac output. Similarly, a low SVR may be multifactorial—sepsis, pyrexia, hypoxaemia, and various drugs may contribute. The ensuing hypotension requires correction by the use of vasoconstricting drugs such as noradrenaline while ensuring an adequate circulating blood volume.

Hypertension

Hypertension[13 14] is a common sequelae of acute neurological events but its treatment requires careful consideration, even though a high mean arterial pressure (MAP) may predispose to rebleeding after subarachnoid haemorrhage, worsening of cerebral oedema, extension of haemorrhage, or haemorrhagic transformation of an infarct. However, it is probably detrimental to attempt to control acute hypertension because patients with raised ICP require an elevated MAP to maintain an adequate cerebral perfusion pressure, and any fall in cerebral blood flow may lead to global ischaemia. However, in the absence of raised ICP, persistently raised MAP in a normally normotensive patient does justify treatment. Commonly used drugs in the acute situation include intravenous labetalol, hydralazine, and sodium nitroprusside. Longer term treatment includes oral β adrenergic receptor antagonists, angiotensin converting enzyme inhibitors, and calcium channel antagonists. It is reasonable to lower the blood pressure as needed to protect against end organ dysfunction, while keeping it somewhat higher than the patient's baseline. Treatments are indicated if the mean arterial blood pressure is higher than 130 mm Hg or cerebral perfusion pressure is higher than 85 mm Hg.

Arrhythmias

Cardiac arrhythmias are associated with aneurysmal subarachnoid haemorrhage, head injury, acute ischaemic or haemorrhagic stroke, status epilepticus, Guillain-Barré syndrome, and brain death. In the acute phase of neurological illness pharmacological management of cardiac arrhythmias is not usually indicated because many of these manifestations are brief and treatment may have a detrimental effect or reduce blood pressure to unwanted levels. The most common morphological electrocardiographic (ECG) changes in acute CNS events are prolonged QTc intervals, ST segment sagging, and deeply inverted T waves (box 3).

Sinus bradycardia is associated with sympathetic blockade and increased vagal tone. It occurs in Guillain-Barre syndrome and with acute lesions in the posterior fossa, particularly with rapidly progressing tentorial herniation. Progressive sinus bradycardia, particularly following tracheal suction, are an indication of potential heart block and cardiac arrest. It may be treated with anticholinergic drugs (for example atropine), β adrenergic receptor agonists or cardiac pacing.

Box 3: Electrocardiograph changes which occur after ischaemic stroke and subarachnoid haemorrhage

- Prolonged QTc interval
- T waves deeply inverted
- ST segment sagging (I, aVL)
- Prominent U waves
- Short or long PR interval
- Large, peaked T waves (cerebral T waves) are rare

Sinus tachycardia is extremely common in acute neurological illness and may be associated with hypoxaemia, hypercapnia, and neuromuscular blockade. It may also occur as a compensatory mechanism in anaemia, hypovolaemia, and pulmonary emboli. If prolonged, the rate should be slowed as myocardial oxygen consumption is directly related to rate and myocardial ischaemia may develop, especially with coexisting coronary artery disease. Treatment is usually directed at the cause. β Adrenergic receptor blockers may be required if the patient is at risk of myocardial ischaemia.

Atrial fibrillation with a rapid ventricular response is common, as either a cause or a consequence, in acute stroke. The ventricular responses may be fast, resulting in inadequate ventricular filling and reduced stroke volume and cardiac output. Cardiac failure, hypotension, and systemic emboli from intra-atrial thrombus can result. Drug treatment is aimed at reducing atrioventricular node conduction and ventricular rate. Digoxin may be used, but β blockers, verapamil, and amiodarone are also valuable and DC cardioversion may be necessary.

Atrial flutter may compromise ventricular filling and increase the risk of ischaemia. The diagnosis is established by carotid massage or a single dose of verapamil. The treatment is aimed at restoring sinus rhythm using DC cardioversion or antiarrhythmics.

Atrioventricular block is most commonly seen in Guillain-Barré syndrome. In Mobitz II second degree and third degree heart block the ventricle fails to respond to atrial stimulation. In these circumstances cardiac pacing is necessary as there may be rapid progression to cardiac arrest.

Ventricular tachycardia is common in subarachnoid haemorrhage and may predispose to the development of torsade de pointes, ventricular flutter, and ventricular fibrillation. Treatment depends on the blood pressure. Antiarrhythmic drugs may be needed, but electrical cardioversion is indicated if ventricular tachycardia causes hypotension, and pacing may be necessary.

ALIMENTARY SYSTEM

Protein catabolism is common in patients nursed on the ITU and is often caused by an inadequate supply of calories and an increased energy and oxygen consumption associated with the stress response to injury and sepsis. The resultant generalised muscle loss also affects respiratory muscles and adds to the difficulties in weaning from mechanical ventilation.

Enteral feeding via a nasogastric tube should be started as soon as possible as this decreases catabolism, provides protection against peptic ulceration, and maintains intestinal integrity, thus decreasing the occurrence of bacterial translocation (see nosocomial infection). Total parenteral nutrition should only be used if enteral nutrition fails or is impossible to use. Enteral feeding may also be administered via a percutaneous gastrostomy when prolonged support is required; it is particularly valuable when there is a severe bulbar or pseudobulbar palsy such as in motor neurone disease.

Gastrointestinal (GI) bleeding[15] is common in NICU patients, but is usually minor and manifests itself as chronic anaemia in the presence of positive faecal occult blood. This mild bleeding is usually abolished by the use of proton pump

inhibitors (for example, omeprazole) or H_2 receptor antagonists (for example, ranitidine) and discontinuation of drugs that may be contributing (such as non-steroidal anti-inflammatory drugs). More major GI haemorrhage requires resuscitation and endoscopic investigation along standard lines.

Disorders of GI motility are almost invariably present in NICU patients. Constipation is a result of opioid drug administration, immobility, and a lack of fibre in some enteral feeds. Treatment with oral or rectal laxatives is usually necessary. Diarrhoea has many causes but infection (including that by *Clostridium difficile*), malabsorbtion, bowel ischaemia, and diarrhoea associated with enteral feeds are the most important. Treatment depends on identification of the aetiology. Ileus presents with a distended and silent abdomen associated with a failure to absorb enteral feed. It is common in patients with neurological conditions affecting autonomic function—for example, high cervical lesions, Guillain-Barré syndrome—but may be secondary to medication, meningitis, and spinal cord infarction. Paralytic ileus may present as a failure to absorb enteral nutrition, vomiting, absence of flatus, distension, no visible peristalsis, and loss of bowel sounds. It is associated with pain, distension, and tympani. Abdominal *x* ray reveals gas filled loops of small intestine. It usually resolves spontaneously but may progress to acute colonic pseudo-obstruction (Ogilvie's syndrome) and lead to perforation. Paralytic ileus is managed by discontinuing opiates/anticholinergic agents, the placement of nasogastric and rectal tubes, and adequate hydration. It may be helped by prokinetic agents such as metoclopramide and erythromycin.

NOSOCOMIAL INFECTION AND INFECTION SURVEILLANCE

Sepsis (and the systemic inflammatory response to sepsis) remains the major cause of organ failure and death in the intensive care unit, being either directly or indirectly responsible for 75% of all deaths.[16 17] Common sites of infection include the urinary tract, respiratory tract (especially ventilator associated pneumonia), and vascular cannulae (catheter related sepsis). This is particularly associated with internal jugular and subclavian catheters, but peripheral catheters also carry a considerable risk. Thus placement of intravenous catheters requires meticulous aseptic technique and regular changing of lines.[18] It is important to ensure that the tips of catheters that have been removed are cultured. Catheter related infections are usually caused by coagulase negative staphylococci (*Staphylococcus epidermidis*) or *Staphylococcus aureus*, and treatment is empiric using vancomycin and cephalosporins.

Most nosocomial infections seen in the NICU are endogenous, caused by colonisation of the patient's GI tract by pathogenic bacteria which then translocate through the intestinal mucosa to reach distant sites by haematogenous spread. Although Gram negative organisms such as *Escherichia coli, Klebsiella*, and *Pseudomonas* species have been traditionally responsible, Gram positive organisms (*Streptococcus* and *Staphylococcus* species) are increasingly common, especially when associated with vascular cannulation. Fungi may also be implicated.

The importance of infection control in the NICU cannot be overstated. This involves the isolation of the infected patient whenever possible, meticulous staff hygiene (hand washing before and after each patient contact, aseptic techniques for invasive procedures, etc), early identification and treatment of infection by the routine sending of blood, urine, sputum, etc, for culture, use of disposable equipment and, most importantly, joint daily ward rounds between microbiologists and the ICU team.

Treatment of nosocomial infection, with or without septicaemia, requires resuscitation and the administration of appropriate antibacterial drugs in adequate doses for an appropriate period. The reader is referred to more detailed accounts.[1–4 19 20]

Box 4: Causes of agitation and restlessness on the NICU

- Pain
- Anxiety
- Confusion
- Sleep deprivation
- Sepsis
- Drugs
- Drug withdrawal
- Metabolic (hypo-, hyperglycaemia, hyponatraemia, uraemia, hepatic)
- Respiratory (infection, hypoxaemia, hypercapnia)
- Cardiac (low output state, hypotension)

ANTICOAGULATION

Pulmonary embolus remains an important cause of death in patients who are critically ill on the ICU. A hypercoagulable state may result from a primary disorder (for example, protein C or S deficiency, antithrombin III deficiency or activated protein C resistance) or secondary to malignancy, sepsis, protein loss, disseminated intravascular coagulation diabetes mellitus, immobility or circulating inhibitors (for example, lupus anticoagulant). Prophylaxis against venous thrombosis is essential and all patients should wear graduated compression stockings and have regular active or passive leg exercises. Low molecular weight heparin is routinely administered; it decreases the incidence of DVT without haemorrhagic side effects, and is more easily monitored than unfractionated heparin.

PATIENT COMFORT

Agitation is common in the NICU and has a variety of causes (box 4). It may manifest as discomfort, pulling at intravenous and bladder catheters, tracheal and nasogastric tubes, shouting, aggressive behaviour, extreme restlessness, and confusion. Pain is particularly common and often unrecognised because of confusion and the difficulties with communication in the aphonic or paralysed patient. Examination may show clues such as profuse sweating, sustained tachycardia, blood pressure fluctuations, and dilated pupils.

Most patients will require sedation but there is a natural reluctance to sedate patients with an evolving CNS disorder. The first line of management is to reassure and calm the patient in a quiet environment ensuring a normal diurnal cycle. Next there should be careful nursing and treatment of the underlying causes, including positioning, splinting, bed cages, catheterisation, and physical treatments. Nonetheless sedative drugs are important and must, when indicated, be used to reduce pain, distress, and anxiety and to aid toleration of tracheal tubes, IPPV, tracheal suction, and physiotherapy. Patients with respiratory failure caused by neurological disease often require long periods of mechanical ventilation

on the NICU and it is not desirable to keep such patients continuously sedated throughout their stay. Furthermore, assessment of their condition and subsequent weaning is impossible with sedation present. However, while the patient has an orotracheal tube in situ or during periods of cardiorespiratory instability or raised ICP, sedation may be essential.

A number of intravenous agents are available. Propofol,[21 22] a diisopropylphenol, is extensively used for short term sedation of critically ill adult patients. Its pharmacokinetic properties allow rapid wakening after discontinuation of its infusion, even in patients with poor renal or hepatic function. It has also been used successfully in the treatment of refractory status epilepticus. Its use is often accompanied by hypotension which may require the introduction of inotropic agents such as noradrenaline. Benzodiazepines have been extensively used in the ICU setting; water soluble midazolam is the drug of choice as it is metabolised to inactive compounds and has a shorter half life (two hours) than other members of the group and has an amnesic effect. Total doses of propofol or midazolam can be reduced by the concomitant use of opioid drugs such as fentanyl and morphine. The use of such sedative and opioid drugs often allows mechanical ventilation without having to resort to neuromuscular blocking drugs.[23]

Pain

Pain may be a prominent feature of many neurological conditions seen in the NICU. This needs to be recognised and treated accordingly. Combinations of opioid and non-opioid drug treatment, including agents directed towards neuropathic pain (for example, amitriptyline, gabapentin), may be required. Opioid analgesics are commonly used in the NICU. These provide effective analgesia, induce euphoria, and aid toleration of IPPV. Side effects of opiates include hypotension, nausea and vomiting, and decreased gut motility; however, the fear of addiction is vastly exaggerated. Intravenous administration is more easily controlled and predictable. Drugs include morphine, fentanyl (both of which are associated with accumulation of metabolites), and the short acting agent alfentanyl. Naloxone reverses opioid toxicity but does not produce long lasting effects. Non-opioid analgesics include aspirin and other non-steroidal anti-inflammatory drugs such as diclofenac; these are increasingly used in the NICU as they have a minimal effect on the level of consciousness. The most important side effect is gastric toxicity. Codeine is an alternative, weaker analgesic.

Communication

Speech is often possible even in patients who are mechanically ventilated via a tracheostomy. It requires specialist speaking valves and, more importantly, the expertise of an experienced speech therapist. Where speech is impossible, other communication aids may be appropriate.

Sleep

Sleep deprivation almost invariably occurs in patients nursed in the NICU. Factors include disruption of day/night cycles, environmental factors such as noise, and patient factors such as fear. The disruption is associated with agitation, confusion, and the development of ICU psychosis. Furthermore, sleep deprivation has a direct effect on respiratory muscle function, catabolism, and the immune response. It is essential to re-establish a normal pattern of sleep by the use of hypnotic drugs (for example, benzodiazepines, sedative tricyclic antidepressants, zopiclone). The use of melatonin has proved useful in resistant insomnia.[24]

CONCLUSION

Neurological intensive care is now a subspecialty of both neurology and intensive care. The NICU requires a multidisciplinary approach to the management of critically ill patients. The intensivist and the neurologist work together in a closely linked team including nurses and therapists, and considerable input from various medical specialities is required. Close attention to communication, daily nursing care, physical therapy, and infection control will ensure the best outcome.

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