THE MANAGEMENT OF SEPSIS

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DEFINITIONS OF 'SEPSIS' AND 'SYSTEMIC INFLAMMATORY RESPONSE SYNDROME'

Patients are often described as being "septic" or having "septic shock". These terms are used in a variety of ways by different doctors and in 1992 'sepsis' and several new terms were formally defined:

1. Systemic inflammatory response syndrome (SIRS) replaced the previous term 'sepsis syndrome'. This is the body's response to a variety of severe clinical insults. It is characterised by the presence of two or more of the following features:

- Temperature >38ºC or <36ºC
- Heart rate > 90/min
- Respiratory rate > 20/min or $PaCO_2$ <4.3kPa
- White cell count > 12 x 10^9/l

2. Sepsis is defined as SIRS in response to infection.

3. Severe sepsis is sepsis associated with:

- organ dysfunction (altered organ function such that normal physiology cannot be maintained without support)
- hypotension (systolic blood pressure < 90mmHg or a reduction of > 40 mmHg from the patient's normal in the absence of other causes of hypotension)

- organ hypoperfusion (revealed by signs such as lactic acidosis, oliguria, acute alteration of mental status).

4. Septic shock describes sepsis with hypotension despite adequate fluid resuscitation.

5. Multiple organ dysfunction syndrome (MODS) describes a state where dysfunction is seen in several organs.

In this article the term SIRS is used. The clinical appearance of a patient with SIRS resulting from infection (sepsis) or other causes (such as burns or pancreatitis) is similar. However there will be differences in the management of the different underlying problem. The initial approach to looking after these patients is similar.

INITIAL ASSESSMENT AND MANAGEMENT

Initial management of a critically ill patient includes:

- Immediate assessment of the airway, breathing and circulation
- A brief history
- A limited examination of the relevant systems of the body.
- A secondary assessment after stabilisation of the patient including a more thorough history, detailed examination by system and appropriate investigations.

Initial Management

Airway and breathing. Respiratory failure is common and may develop at any stage so repeated

Induction and intubation in critically ill patients

Anaesthesia for intubation and ventilation of critically ill patients is hazardous and often poorly tolerated. Consider the following points:

- A trained assistant or second anaesthetist should be present.
- Never leave a hypoxic patient unattended. Give high concentrations of oxygen whilst preparing equipment.
- Obtain wide-bore intravenous access (14G or 16G cannulae). In shocked patients attempt to improve intravascular filling pre-induction, using clinical signs such as heart rate, BP and capillary refill time to guide fluid therapy.
- A patient with severe sepsis / SIRS will have some degree of haemodynamic compromise and induction of anaesthesia will often result in severe hypotension. Induce slowly using small doses of i/v anaesthetic agents. Ketamine, etomidate or diazepam may provide greater haemodynamic stability, although in practice thiopentone may be used provided it is given carefully.
- Respiratory reserve may be poor - preoxygenate for three minutes via a tight-fitting mask and reservoir bag. Patients who are dyspnoeic may require respiratory assistance during this phase.
- Rapid sequence induction and intubation with application of cricoid pressure should be used. Avoid suxamethonium in patients at risk of hyperkalaemia.
- Expect the patient to become hypotensive post induction. This may respond to an infusion of 500 - 1000mls of crystalloid or colloid, but often iv vasopressors are required. Suitable drugs include ephedrine 6 - 9mg iv, metaraminol 2 - 4mg or epinephrine (adrenaline) 1:10 000 in 0.5 - 1ml doses.
- After induction either continue with an anaesthetic or consider another form of sedation to facilitate mechanic ventilation. Frequently a combination of midazolam and morphine are used given either by infusion or intermittent boluses. Neuromuscular blocking drugs may be used but are frequently unnecessary in patients who are critically ill.
- This is a convenient time to pass a nasogastric tube and urinary catheter.

assessments are necessary. A depressed conscious level is the most common cause of airway obstruction. Patients with inadequate airway reflexes should be nursed in the recovery position and if possible intubated and mechanically ventilated.

A clear airway does not indicate effective breathing. Failure of gas exchange may be caused by lung parenchymal problems (pneumonia, lung collapse, pulmonary oedema), failure of the mechanics of ventilation (pneumothorax, haemothorax, airway rupture) or reduced respiratory drive (encephalopathy).

Respiratory failure is suggested by signs of respiratory distress including dyspnoea, increased respiratory rate, use of accessory muscles, cyanosis, confusion, tachycardia, sweating. The diagnosis is made clinically but may be confirmed by pulse oximetry and arterial blood gases. Patients with a depressed conscious level may not react normally to hypoxia and signs of respiratory failure may be difficult to detect. Patients with inadequate ventilation, gas exchange or both require ventilatory support. This usually necessitates intubation and mechanical ventilation although in some patients gas exchange and oxygenation can be improved by the application of continuous positive airway pressure (CPAP) by face mask or non-invasive ventilation.

Circulation. Tachycardia and hypotension are almost universal findings in the septic patient and result from a number of cardiovascular problems. In early sepsis, and in patients who have been partially or fully fluid resuscitated, the low blood pressure and high heart rate are associated with a high cardiac output and a low peripheral vascular resistance with warm peripheries and bounding pulses. In contrast, patients who have not been significantly resuscitated or have presented late in the course of their illness have a low cardiac output and high systemic vascular resistance. These patients are peripherally cold, sweaty, with weak, thready pulses and they need urgent resuscitation. Many patients present with an unclear or mixed clinical picture. However resuscitation aims to restore circulating volume, cardiac output and reversal of hypotension.

Initially infuse i/v crystalloid or colloid rapidly guided by the clinical response. In a peripherally warm, vasodilated patient with a high cardiac output several litres of crystalloid may be needed to establish adequate intravascular filling. In patients with a mixed or unclear clinical picture, clinical assessment may be difficult. Administering large volumes of fluid to patients with known cardiac disease or myocardial dysfunction related to their acute illness is a problem. In these patients insertion of a central venous catheter will help by measuring the central venous pressure (CVP) to guide fluid resuscitation and to provide a route for infusion of vasopressors or inotropes. A one-off reading of CVP may be misleading but following a trend of measurements and their response to fluid challenges is helpful - *see Update in Anaesthesia No 12.* Urine output should be charted hourly.

History. The primary insult may be self-evident (eg trauma, burns, recent surgery) or more difficult to diagnose (eg pancreatitis, gynaecological sepsis), particularly in unconscious patients.

Examination. The appearance of the patient is variable; they may appear well, warm and well-perfused with bounding pulses or may be cold, vasoconstricted and peripherally cyanosed. The 'warm' and 'cold' patients represent two ends of a spectrum of presentations. The examination will reflect the degree of their illness, their state of intravascular hydration and may reveal the underlying cause.

When looking for an underlying source of infection consider:

- *Central nervous system:* Global (sleepiness, confusion, agitation, coma) or focal (localised abnormality of movement or sensation) neurological dysfunction suggesting meningitis, encephalitis, cerebral malaria or abscess.
- *Respiratory system:* Mucopurulent discharge from the respiratory tract, dyspnoea, lung consolidation or pleural fluid collection.
- *Gastrointestinal tract:* Abdominal pain with guarding and rigidity suggesting peritoneal irritation.
- *Vaginal discharge* or history of termination suggest gynaecological sepsis
- *Skin:* Purulent skin wound, signs of inflammation (redness, pain, swelling, heat) or petechial rash (meningococcaemia).
- The patient with SIRS may have a number of other, non-infective diagnoses. Consider myocardial infarction, pulmonary embolism, diabetic ketoacidosis,

poisoning or drug overdose, eclampsia, cerebrovascular event.

- In some patients the diagnosis is unclear at this stage and treatment has to continue along "best guess" lines.

SECONDARY ASSESSMENT

After the initial assessment and resuscitation, the patient should have a secure airway, adequate ventilation, and cardiovascular resuscitation should have commenced. These need to be rechecked regularly. The priorities during the next phase are:

- Fill in the gaps in the patient's acute and past medical history.
- Perform a full physical examination by system.
- Perform relevant investigations (see below).
- Communicate with the other teams involved in the patient's management (e.g. general surgeons for intra-abdominal infection, gynaecologists for gynaecological sepsis).
- Continue resuscitation.

Perform investigations to confirm or clarify problems that are clinically evident, or to look for complications that are likely in each particular clinical setting. Investigations will be governed by the availability of these tests in each centre and the time available. For example, for a septic patient with abdominal signs in a centre with no access to radiological facilities, diagnostic laparotomy may be the definitive investigation (and treatment). Table 2 shows common initial investigations.

Haematological	FBC (raised WCC common, may be reduced in overwhelming sepsis) Coagulation screen Thick and thin blood film (malarial parasites) Confirm sickle cell / thalassaemia status
Biochemistry	Sodium, potassium, urea, creatinine Glucose (usually increased in SIRS) Amylase (raised in pancreatitis, ischaemic bowel, perforated bowel) Liver function tests Cardiac enzymes if infarct likely CPK in crush injuries
Arterial blood gas	Respiratory function Acid-base balance
ECG	To exclude cardiac causes of hypotension or to differentiate sinus tachycardia from arrhythmia
Chest Xray	To confirm clinical findings in chest (e.g. acute pneumonia), to investigate underlying lung disease and confirm the position of an endotracheal tube and central venous line
Microbiological	To confirm the presence of infection - Samples depend on history and examination. A 'septic screen' may be required in difficult cases. ● Blood cultures (ideally three sets during pyrexial episodes). ● Sputum (protected catheter specimens or broncho-alveolar lavage may be available for intubated, ventilated patients). ● Mid-stream urine (MSU) or catheter specimen of urine (CSU). ● CSF where indicated via lumbar puncture. ● Wound swabs from any suspected sites (including old cannula sites). ● High vaginal swab. ● Stool for ova, cysts and parasites. ● Deeper infection may be clinically or radiologically evident. Samples may be amenable to percutaneous aspiration or sent after surgical drainage or debridement.

Monitoring is not dependent on expensive equipment, but it requires the continuous presence of trained nursing staff. Clear documentation aids the assessment of subtle changes in the patient's clinical state. Patients with severe SIRS / sepsis should have observations recorded hourly. Record body temperature, pulse, blood pressure, urine output, CVP, respiratory rate and SpO_2 (if available). Accurate fluid balance is essential - insensible losses may be very significant in hot climates. Ideally measure the patient's temperature centrally (rectal or nasopharyngeal). Other sites include the axilla or mouth. Of these, the axilla is the least accurate but most convenient; whilst the rectal route is the most accurate but least convenient. Always use the same site.

TREATMENT OF THE UNDERLYING PROBLEM

Clearly this depends on the nature of the initial insult and may be straightforward (oxygen, antibiotics and chest physiotherapy for a lobar pneumonia), or complex involving different specialities (orthopaedics, general and plastic surgery for major trauma). Infection is common, either as cause, or as a secondary complication. Treatment of infection may involve:

Antibiotic therapy. The initial antibiotic prescription is a 'best guess', and will depend on the clinical picture of the patient, local patterns of antibiotic resistance and the local availability of antibiotics. It should be broad enough to cover the most likely pathogens, but not so broad as to encourage antibiotic resistance. The advice of a local microbiologist or infectious diseases specialist is valuable.

Surgical debridement. Pus-filled cavities (abscess, empyema), necrotic tissue, infected tissue or gross tissue contamination (open wounds, peritonitis) cannot be treated by antibiotics alone and must be treated surgically at the earliest opportunity. The surgical team should assess the patient as soon as possible. Anaesthesia for these patients is discussed later in this article.

THERAPEUTIC STRATEGIES FOR PRESERVING ORGAN FUNCTION

Organ failure results from inadequate organ oxygenation due to poor perfusion. Strategies to maintain or restore organ function are **general**, aimed at improving delivery of oxygen and nutrients to all tissues, or **organ-specific** (e.g. the kidney and gut).

Improving Oxygen Delivery

Oxygen delivery to the tissues, (DO_2) is defined as: DO_2 = cardiac output x haemoglobin level x oxygen saturation

Each of these three factors should be optimised to improve oxygen delivery.

Cardiac output. In SIRS the cardiac output may be low, high or normal. Whilst cardiac output at normal or supranormal levels is required to maintain oxygen delivery, maintenance of blood pressure itself is also important to ensure perfusion pressure is adequate (eg filtration at the kidney). Although most organs are capable of some autoregulation, this mechanism cannot always compensate for the circulatory disturbance in sepsis. This is why a vasodilated patient with a high cardiac output needs intervention to elevate their blood pressure.

The main treatments for maintaining cardiovascular function are correction of hypovolaemia with fluid therapy, inotropes and vasopressor agents.

- Correction of hypovolaemia (fluid therapy). Vasodilation causes blood to pool in the periphery, and abnormal capillary permeability results in fluid leak into the tissues. These changes decrease the relative blood volume (by vasodilation) and absolute blood volume (by capillary leak) causing a fall in the preload of the heart and therefore a decrease in cardiac output. Monitor progress clinically: a satisfactory response to fluid therapy is suggested by a falling heart rate, increase in the blood pressure, decrease in the capillary refill time and improvement in organ function. A central venous catheter may be helpful if the clinical picture is hard to interpret or mixed (co-existent cardiac disease).

Clear benefits of colloid over crystalloid have not been demonstrated, but crystalloid redistributes rapidly into the whole extracellular volume (about 14 litres in a 70 kg man) and so larger volumes must be given for intravascular resuscitation. In anaemic patients blood is often required.

- Use of inotropic and vassopressor (vasoconstrictor) agents. If the blood pressure remains low after the patient is judged to be adequately intravascularly filled, the patient either has inadequate myocardial 'pump' function or has a degree of vasodilation which cannot be overcome by fluid therapy alone. Sometimes patients present as clear-

Infusions of inotropic and vasopressor drugs

Adrenaline and Noradrenaline:	Mix 5 mg in 50ml 5% glucose. This gives a mixture of 1 in 10 000 adrenaline. Start at low dose (1 to 5 ml/hr) and titrate to the desired blood pressure Noradrenaline may be available in 4 mg vials - mix 4 mg in 40ml 5% glucose and use as above.
Dobutamine and Dopamine:	Multiply the patient's weight in kg by three. Make this number of milligrams of dobutamine or dopamine up to 50 ml in dextrose or saline. Infusion of this solution at a particular rate in ml/hr gives the same number in mcg/kg/min (e.g. 2ml/hr = 2mcg/kg/min.)

cut examples of one of these two syndromes, but more often patients have a mixture of clinical signs. If the patient appears vasodilated with a hyperdynamic circulation an agent with vasopressor (α adrenoreceptor agonist) properties, such as noradrenaline (norepinephrine) is appropriate to elevate the blood pressure.

If the patient is cool peripherally (has a large core to peripheral temperature difference), has signs of poor organ perfusion and/or a low blood pressure then an agent with more positive inotropic properties is the best choice. Examples are adrenaline (epinephrine), dobutamine or dopamine.

Inotropes should be given through a central venous catheter and direct intra-arterial blood pressure measurement is preferable for accurate, continuous readings. Proposed regimens for use of these drugs are shown below. In practice few of these drugs have 'pure' effects; noradrenaline also has positive inotropic effects via stimulation of β1-adrenoreceptors. Combinations of vasopressor and inotropic drugs are often used.

If the clinical picture is difficult to interpret, other means of investigation are available in some centres. Pulmonary artery flotation catheters (Swan-Ganz catheters) indirectly measure the left atrial pressure, which may be a more accurate measure of intravascular volume status. The saturation of blood sampled from the pulmonary artery gives the mixed venous blood oxygen saturation which can be used to assess adequacy of oxygen delivery. Use of trans-oesophageal doppler is increasing.

Oxygen saturation and gas exchange. The majority of patients with severe sepsis require intubation and ventilation and almost 50 % go on to develop problems with gas exchange. Lung problems associated with SIRS is termed 'Acute Lung Injury' (ALI). 'Acute Respiratory Distress Syndrome' (ARDS) describes the most severe form of ALI. In both cases the lungs become oedematous and damaged and are less able to take up oxygen or eliminate carbon dioxide. ALI may resolve with treatment of the underlying cause of the SIRS, or progress to a stage where lung fibrosis takes place. Steroids may have a role in the treatment of late refractory ALI / ARDS, but are thought to be ineffective in the early stages. Some of the lung damage sustained during critical illness may be due to mechanical ventilation: excessive driving pressure causes over-expansion of and damage to alveoli. In patients with ALI, ventilators should be set at a more protective ventilation strategy in patients with:

- Limitation of plateau pressure to less than 35 cm H_2O
- Use of smaller tidal volumes (up to 8 ml/kg)
- Lower target minute volumes resulting in $PaCO_2$ values higher than normal (so called 'permissive hypercapnia'). This results in a respiratory acidosis which is usually well tolerated, provided the arterial pH does not fall below 7.2.
- Use of pressure-control rather than volume-control ventilation.
- Higher positive end-expiratory pressures (PEEP, 10 or 12 cm H_2O instead of 5 or 6cm H_2O)
- Longer inflation phase with I:E ratio of approximately 1:1, to improve the distribution of gas within the lung.

- Avoid an FiO_2 above 60% if possible - aim for SpO_2 93-95%.

Ventilator-associated pneumonia (VAP) is a frequent complication of ventilation. This is thought to arise from contamination of the respiratory tract by aspiration of material regurgitated from the stomach ('micro-aspiration' around the endotracheal tube cuff). Techniques which are thought to reduce the incidence of VAP include:

- Aseptic technique when suctioning patients
- Nursing the patient semi-recumbent instead of completely flat
- Avoiding the use of proton-pump inhibitors or H_2-antagonists which encourage bacterial growth in the stomach due to loss of acidity. Establishing early nasogastric feeding
- Ensuring that the cuff of the endotracheal tube is correctly inflated
- Avoiding re-intubation or manipulation of the airway circuit

Treatment of anaemia. Recent studies show that transfusion of blood to critically ill patients to maintain a haemoglobin level of greater than 10 g/dl does not alter the patients outcome. With the multiple potential problems associated with blood transfusion, in the absence of ischaemic heart disease, it is reasonable to allow the haemoglobin to remain at 7 to 9 g/dl.

Nutrient supply and hormonal changes in SIRS. Insulin secretion is reduced with the stress of severe illness whilst cortisol and growth hormone secretion both increase. Patients are prone to hyperglycaemia due to the insulin-antagonism of these hormones and drugs such as adrenaline (epinephrine). A slow intravenous infusion of an insulin solution (1 unit per ml) may be required to maintain normal blood sugar levels (5 to 9 mmol/l), but if this is not practical then adequate glycaemic control can be achieved with intermittent subcutaneous injections of insulin. Check the blood sugar at regular intervals.

During a prolonged illness the patient's metabolic requirements will be increased by the effects of fever and infection, and the patient will become catabolic, breaking down their own tissues (especially muscle) to use as metabolic fuel. This process cannot be reversed, but can be limited to some extent by supplying the patient with appropriate quantities of energy (in the form of fat and carbohydrate), nitrogen (in the form of protein, peptides or amino acids), minerals and vitamins. Feeding via the enteral route (e.g. via a nasogastric tube) is preferable; proposed benefits include reduced 'stress' ulceration in the stomach, preservation of bowel mucosal function and reduction of **bacterial translocation** from the bowel lumen into the circulation (see below). Some conditions preclude enteral feeding (recent bowel resection) but other problems may be overcome (e.g. nasojejunal tube for pancreatitis or percutaneous gastrostomy for oesophageal disease). Intravenous nutrition may be used if enteral feeding is not possible, but is expensive, and associated with a number of significant complications (most notably infection).

Organ-specific Strategies

Gastrointestinal tract. The bowel may act as the 'motor' for MODS, by the mechanism of bacterial translocation across damaged mucosa whose integrity has been damaged by hypoxia. As described above, early enteral feeding is the main preventative measure to counter this.

H_2-antagonists (e.g. ranitidine) and proton pump inhibitors (e.g. omeprazole) have been used to reduce mucosal damage in patients who cannot be fed enterally. The disadvantage is that by reducing gastric acidity these drugs allow bacterial overgrowth and may increase the likelihood of ventilator associated pneumonia and bacterial translocation. Sucralfate is a cheaper alternative which gives some mucosal protection without reducing gastric acidity.

Liver. In the acute phase of sepsis (within the first 24 or 48 hours) the liver may be damaged by periods of low blood pressure, reflected in sharp rises in circulating liver enzymes (lactate dehydrogenase and both aspartate and alanine transaminase). With adequate resuscitation this damage is self-limiting and reversible. Maintenance of liver function depends on effective resuscitation, rapid removal of the septic focus, appropriate antibiotic treatment, early nutritional support and the avoidance of further damage. Hepatic damage may cause encephalopathy, coagulapathy and hypoglycaemia.

Kidneys. The ion channels in the tubular epithelium of the renal medulla are energy (and therefore oxygen) dependent and so particularly sensitive to episodes of

hypotension and hypoxia. Up to 65 % of patients with sepsis develop abnormalities of renal function and if renal replacement therapy (haemofiltration or haemodialysis) is required the mortality is as high as 75 %. Indications for renal replacement therapy include, severe or refractory hyperkalaemia, severe metabolic acidosis, low or absent urine output or symptomatic uraemia (e.g. pericardial effusion).

If a patient is oliguric, consider the following:

- **Exclude obstructive causes** - flush the urinary catheter, consider urethral damage in trauma.
- **Fluid resuscitation.** Reduction in blood volume stimulates the release of renin, anti-diuretic hormone and activation of the sympathetic nervous system, reducing the volume of urine produced by the kidney. These effects may be reversed by adequate fluid resuscitation guided clinically and, if necessary, using a central venous catheter.
- **Blood pressure.** The kidney autoregulates the filtration pressure in the glomerulus by altering the resistance of afferent and efferent arterioles. Autoregulation fails if the mean arterial blood pressure falls below about 60mm Hg and urine flow decreases or stops. Correction of hypovolaemia may not restore the blood pressure. Use an inotrope or vasopressor as described above.
- **Nephrotoxic agents.** Stop non-steroidal anti-inflammatory drugs, angiotensin-converting enzyme (ACE) inhibitors and avoid radiographic contrast media. Levels of aminoglycoside antibiotics (gentamicin, netilmicin) and vancomycin should be checked.
- **Diuretics**. Loop diuretics such as frusemide may establish a diuresis but should only be used after optimal restoration of intravascular volume. By inhibiting the active transport of ions in the loop of Henle loop diuretics may offer some protection to tubular cells from hypoxic damage.

If these strategies do not restore urine flow, then acute renal failure has occurred. In the absence of specific nephrotoxic agents the cause is likely to be acute tubular necrosis, which in most cases is reversible. The time to return of renal function is variable (from a few days to several weeks) and in the interim some form of renal replacement is necessary to control hypervolaemia, acidosis, hyperkalaemia and uraemia. The choice is largely dependant on local availability and transfer to another centre may be necessary. Monitor the patient using daily weight, and measurement of electrolytes, urea and creatinine.

MONITORING THE PATIENT'S PROGRESS

Failure to improve or deterioration at any stage should prompt thorough reassessment of the patient (ABC,

Complications following sepsis

Complications of intubation and mechanical ventilation	Pneumonia Pneumothorax Vocal cord damage Tracheal stenosis Difficulty weaning from ventilatory support
Hospital-acquired (nosocomial) infections	Cellulitis at the site of venous or arterial cannulae Cannula-related septicaemia Urinary-tract infections Blood and blood-product infections (HIV, hepatitis B)
Complications secondary to immobility and severe illness	Pressure sores Peripheral nerve palsies Corneal abrasions Pulmonary emboli and/or deep vein thromboses Malnutrition and weakness
Drug-related	Kidney damage (aminoglycoside antibiotics)
Disturbance of sleep / wake cycle	

history, examination etc). Consider whether the original diagnosis is correct, a new diagnosis has evolved, the current treatment is appropriate and correctly instigated, or whether a complication has developed. Signs of deterioration may include:

- Persistent or worsening tachycardia
- Persistently elevated or swinging temperature
- Rising white cell count, C-reactive protein
- Fall in blood pressure, or increased requirement for vasopressor drugs to maintain the same blood pressure
- Deteriorating renal output
- Deterioration in conscious level
- Deterioration in respiratory function

PREVENTING COMPLICATIONS

Patients with SIRS may suffer depression of immune function and many of the procedures performed on intensive care units breach the body's natural defences (e.g. orotracheal intubation, peripheral cannulae and central venous cannulae) and leave the patient prone to secondary infections. These and other complications are listed below.

Prevention of Infection

Medical staff have been implicated in the spread of infectious agents between patients. All staff must wash their hands before and after attending to a patient. Equipment (such as thermometers, stethoscopes, bed pans) should not be shared between patients if possible, but where this is necessary the equipment should be thoroughly cleaned between patients. Staff should protect themselves and their clothes from becoming contaminated with biological material by wearing (ideally disposable) aprons and gloves. Visitors should be discouraged from moving between patients. Patients should be washed daily and never be left in soiled bed linen. Wounds, including drain sites and intravenous cannulae sites, should be inspected, cleaned and dressed at regular intervals. Intravenous cannulae and central lines should be removed as soon as practical. Some units have strict protocols governing the replacement of in-dwelling cannulae after a set number of days, other units replace the cannula when clinically indicated.

Immobility and Severe Illness

Patients immobilised by sedation or severe illness are vulnerable to complications that can be prevented by good nursing care. Pressure damage can be prevented by re-positioning the patient every two to four hours, and by replacing wet linen. Particular attention should be paid to the skin over bony prominences, such as the heels and elbows, by padding these areas with cotton-wool, lint or even sheep's wool. Eye damage can be prevented by taping the eyes shut or application of protective gel. Joint stiffness and peripheral oedema may both benefit from passive movement of both the legs and arms. If there is a severe shortage of nursing staff all of these activities can be carried out by the patient's relatives if they are given the appropriate training and encouragement. In longer stay patients physiotherapy is essential to minimise muscle wasting and maintain good active and passive range of movement. Critical illness neuropathy and myopathy are common complications of SIRS.

ETHICAL ISSUES AND RESOURCE ALLOCATION

There is no predictive scoring system which gives accurate predictions of outcome for individual patients. Survival from an episode of severe SIRS/sepsis is dependent the patient's age, previous health and the time delay before the onset of medical intervention, as well as the appropriateness and quality of medical care. Few countries have limitless resources, and so difficult decisions face all intensive care doctors when deciding between the potential benefits for one critically ill patient and need for provision of healthcare to several less critically ill patients.

APPENDIX

Anaesthesia for the Septic Patient

The surgical drainage of abscess cavities, laparotomies, debridement of infected wounds or amputation of gangrenous limbs may be central to the successful treatment of a patient with severe sepsis. Surgery and anaesthesia is often required, even in patients in poor clinical condition.

Pre-operative Preparation

The time taken to improve a patient's condition before surgery must be balanced against the urgency to surgically treat the underlying problem. Recent studies

have shown that the outcome from surgery in these high risk patients is improved if the patient's condition is 'optimised' preoperatively. When surgery can be delayed (even for a few hours), attempts should be made to resuscitate the patient to ensure adequate oxygen delivery, cardiac output and blood pressure. This is often easiest done in theatre, recovery or ICU. In a few patients immediate surgery is lifesaving and should be carried out as soon as practical (eg necrotising fasciitis). In these patients preparation time is limited but initial resuscitation (airway, breathing and circulation) should be completed and continuing resuscitation carried out during anaesthesia. Common problems in the perioperative period include anaemia, hypotension, coagulation disturbance, electrolyte disturbance (particularly hyper or hypokalaemia) and acidosis.

Regional or General Anaesthesia?

Physiological stability during anaesthesia is compromised by the combined effects of sepsis, anaesthesia, blood loss and surgical stress. Close monitoring is required because rapid changes in physiological parameters may occur.

When inducing anaesthesia in a septic patient the same considerations which are described in the airway / breathing section of resuscitation apply. Supplementary analgesia may be needed and intravenous or inhalational agents can be used to maintain anaesthesia. Use smaller doses of cardiovascularly active drugs to assess the patient's response. Ketamine anaesthesia is widely used in these high risk patients, although in this situation it may be cardiovascularly depressant and does not protect the airway as effectively as an endotracheal tube. Following induction of anaesthesia there is a reduction in sympathetic tone that often results in hypotension which may need treating by i/v infusion of fluids and a vasopressor.

Neuraxial blockade (spinal and extradural anaesthesia) should only be considered if recent blood tests have shown the clotting to be normal. The haemodynamic effects of a these techniques in the setting of cardiovascular compromise may be devastating and hard to reverse. A further concern is the risk of epidural abscess complicating an epidural haematoma formation. The evidence is not clear but the risk is likely to be increased in patients who are frankly septic. Peripheral nerve blocks or regional infiltration may be used and are very effective at minimising the sympathetic response to a painful stimulus, whilst avoiding the systemic effects of opioids.

Further reading

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