Assessing, treating and managing patients with sepsis

Summary
This article outlines the causes, signs and symptoms of systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis and septic shock, the implications and available treatments. The article also highlights a campaign to reduce the incidence of sepsis and reflects on efforts to reduce healthcare-associated infections.

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Introduction
Sepsis affects 18 million people worldwide each year (Slade et al 2003). Nurses in all areas of practice, from the community to acute hospitals, will care for septic patients during their professional careers. However, although it is a common problem, many nurses receive little training in how to identify patients with severe sepsis correctly or about how important early and aggressive treatment is to help prevent the condition deteriorating. There also appears to be confusion over the definition of sepsis, with a variety of terms such as septic, bacteraemia, sepsis, septicemia and septic shock being used interchangeably.

Over the last few decades there has not been a dramatic improvement in survival rates for sepsis (Friedman et al 1998). In recognition of the seriousness of the problem, an international campaign, called the Surviving Sepsis Campaign, which is run mainly by the Institute of Health Improvement (www.ihi.org/IHI/Topics/CriticalCare/Sepsis) is now under way to raise awareness of sepsis around the world and promote evidence-based care. It is hoped that by so doing, the death rate from sepsis can be reduced significantly.
Altered mental state – it is important to involve the patient’s family to assess an alteration.

Hyperglycaemia in the absence of diabetes.

Hypoxaemia – oxygen saturation (SpO2) <93 per cent or PaO2 (partial pressure of arterial oxygen) <9kPa on arterial blood gas (ABG) analysis.

Acute oliguria – urine output <0.5ml/kg/hr and/or raised urea and creatinine.

Coagulopathy – INR (International Normalised Ratio) >1.5, APTT (activated partial thromboplastin time) >60 seconds or platelets <100.

Serum lactate >2mmol/L (Box 1).

Organ dysfunction can also affect the liver – evidenced by abnormal liver function tests (LFTs) and the gastrointestinal tract – evidenced by bowel stasis (ileus).

Septic shock
This is severe sepsis with hypotension (systolic blood pressure below 90mmHg), which does not respond to adequate fluid resuscitation (approximately 1.5-2.0 litres of fluid) (Dellinger et al 2004). Additional signs and symptoms to consider at the bedside when deciding if a patient has sepsis are: a positive fluid balance, an unexplained metabolic acidosis (a blood pH value <7.35 with a bicarbonate level less than 20mmol/L, and a base deficit > –2.5 mmol/L and decreased capillary refill, or mottling of the skin (Levy et al 2001).

Capillary refill time is measured by applying pressure to a fingertip, held at heart level for five seconds, and then letting go while counting how long it takes the finger to return to a pink colour. Normally this happens in less than two seconds.

Time out 1
Reflect on your understanding of systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis, and septic shock. If you had to explain these terms to a junior colleague what would you say? Think about the patients you have cared for with sepsis, what signs and symptoms did they have and what treatment did they receive?

Defining sepsis
Sepsis is a word that nurses use frequently. However, in the past there has been a general lack of consensus about the definition of sepsis. In response to this, in 1991, experts from around the world produced clear and concise definitions for sepsis, severe sepsis and septic shock, and introduced the term systemic inflammatory response syndrome (SIRS) (Bone et al 1992).


Systemic inflammatory response syndrome (SIRS)
This is the body’s response to a variety of insults and is manifested by two or more of the following conditions:

- High or low temperature >38˚C or <36˚C.
- Heart rate >90 beats per minute.
- Respiratory rate > 20 breaths per minute or PaCO2 (partial pressure of arterial carbon dioxide) < 4.3kPa.
- High or low white blood cell count >12,000 or <4,000.

Although the SIRS response is produced by an infection in sepsis, SIRS can also be caused by any major insult to the body, such as burns, acute pancreatitis, myocardial infarction or trauma. Nurses and doctors should consider whether SIRS is caused by an infection or some other insult.

Sepsis
This is a known or suspected infection, accompanied by evidence of two or more of the SIRS criteria mentioned above.

Severe sepsis
This is sepsis (a known or suspected infection with two or more of the SIRS criteria), associated with organ dysfunction, hypotension or poor perfusion. All organs, including the cardiovascular system, lungs, liver, kidneys and brain can be affected. Signs of organ dysfunction are:

- Hypotension – defined as a systolic blood pressure <90mmHg, a mean blood pressure <60mmHg, or a reduction in a patient’s systolic blood pressure of more than 40mmHg from his or her usual measurement in the absence of other causes of hypotension.

- Coagulopathy – INR (International Normalised Ratio) >1.5, APTT (activated partial thromboplastin time) >60 seconds or platelets <100.

- Serum lactate >2mmol/L (Box 1).

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BOX 1

Serum lactate

A raised blood lactate level is also a sign of severe sepsis, even if the patient’s blood pressure is normal. Lactic acid is produced as an end product when the tissues do not receive adequate oxygen resulting in anaerobic metabolism. The lactic acid leads to a raised blood lactate level. This is a similar process to the development of cramp during exercise, caused by a build-up of lactic acid in the muscles, which have to produce energy without oxygen (anaerobic metabolism). The physiological changes in sepsis result in inadequate delivery of oxygen to the tissues and anaerobic metabolism.
seconds. Patients with sepsis often have warm peripheries and a normal or brisk capillary refill of two seconds or less, as a result of vasodilation. However, it is important to note that patients with severe sepsis may also present with cool peripheries and a longer capillary refill time of greater than two seconds. This can result from the reduced pumping force of the left ventricle.

Despite the agreed sepsis definitions, it appears that some medical staff are not aware that these common definitions exist. Of the 1,058 doctors questioned in Poeze et al.'s (2004) study, no more than 17 per cent agreed on any one definition and 83 per cent said it is likely that sepsis is missed frequently. There do not appear to be any published studies that have explored nurses’ knowledge of diagnosing sepsis, but many nurses may not be aware of these common definitions, and may, as a result, fail to recognise that a patient is septic. All health professionals need to know the signs and symptoms to work effectively as a team.

For sepsis to be treated, it should first be recognised using a common agreed definition. This requires nurses to be as familiar with the signs and symptoms of severe sepsis as might be expected with the signs and symptoms of venous thromboembolism, or angina. Many patients can have sepsis and not be particularly ill. This is uncomplicated sepsis of the kind experienced by people with influenza or a chest infection. Such individuals have a known or suspected infection, and two or more of the SIRS criteria, but do not require hospital treatment (International Sepsis Forum (ISF) 2002).

It has been suggested that some individuals may be genetically more susceptible to developing severe sepsis or septic shock than others (ISF 2002). There is no single cause of sepsis and the clinical presentation can vary from uncomplicated sepsis to septic shock with multiple organ failure and death (ISF 2002).

**Time out 2**

Reflect on the scenarios below and consider if these patients have sepsis, severe sepsis, or septic shock and list the reasons for your answer:

1. Mary is 51 years old and has been treated by the GP for a chest infection for the past week. Today she has deteriorated and returned to the GP, who immediately called an ambulance. Her signs and symptoms on arrival to the A&E department are:
   - Respiratory rate: 28
   - Oxygen saturation: 92% on 60% oxygen
   - Pulse: 124 beats per minute
   - Blood pressure: 140/63mmHg
   - Temperature: 35.9˚C
   - White cell count: 9,000.

2. Alan is a 71-year-old man who had a laparotomy seven days ago. The ward nurse looking after him has contacted the critical care outreach team as Alan is scoring 5 on the hospital Early Warning Score. His signs and symptoms are:
   - Respiratory rate: 26
   - Pulse: 105 beats per minute
   - Blood pressure: 89/56mmHg
   - Urine output: 25ml for the past two hours consecutively
   - Drowsy but responds to voice
   - Temperature: 38.4˚C
   - White cell count: 18,000
   - The full blood count shows his platelets are 76.

(Answers on page 64)

**Common causes of sepsis**

Sepsis can result from an infection in various parts of the body and although a lay understanding of sepsis might include reference to blood poisoning positive blood cultures are not needed to diagnose it. About 90 per cent of the cases of sepsis are caused by bacteria (Box 2), but sepsis can also be caused by viruses or fungi, particularly Candida spp. (Cohen et al 2004).

Infection is a major reason for patients to be admitted to hospital and some patients may develop infections while in hospital (Department of Health (DH) 2003). Infections can be found in any system of the body. The most common sources of infection that can lead to sepsis are:

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**BOX 2**

**Bacteria associated with sepsis**

<table>
<thead>
<tr>
<th>Gram-negative</th>
<th>Gram-positive</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Escherichia coli</em></td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td><em>Klebsiella</em> spp.</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td><em>Enterobacter</em> spp.</td>
<td>Alpha and beta-haemolytic streptococci</td>
</tr>
<tr>
<td><em>Pseudomonas aerugi nosa</em></td>
<td></td>
</tr>
<tr>
<td><em>Serratia</em> spp.</td>
<td></td>
</tr>
<tr>
<td><em>Proteus</em> spp.</td>
<td></td>
</tr>
<tr>
<td><em>Bacteroides fragilis</em> (anaerobe) (Edwards 2001)</td>
<td></td>
</tr>
</tbody>
</table>

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Respiratory tract – community-acquired or healthcare-associated (nosocomial) pneumonia.

Intra-abdominal cavity – infection might result from diverticulitis, appendicitis, a perforated bowel, and ischaemic or necrotic bowel.

Central nervous system – such as meningitis.

Genitourinary system – uro-sepsis is an infection which might result from an obstruction in the urinary system, or a catheter-related infection.

Skin – wound infections, cellulitis or necrotising infections of the skin and soft tissues with rapid destruction of tissue, such as necrotising fasciitis.

Intravascular catheters – any invasive catheter, for example, central venous catheters or peripheral cannulae.

Patients whose immune system is compromised are at increased risk of developing sepsis. Those at risk include the very young, whose immune systems are not completely developed, and older people, whose immune systems have become weakened as a result of the ageing process. Also at risk are those who have had a transplant, a splenectomy or those who are being treated with radiation or chemotherapy, and patients with diabetes, cancer, human immunodeficiency virus (HIV) or acquired immunodeficiency syndrome (AIDS) (ISF 2002).

The body’s response to sepsis

In understanding the changes that happen to the body in severe sepsis it is helpful to remind ourselves of the changes that occur during the normal immune response. Cells damaged by infection from bacteria, viruses, chemical agents or trauma produce the same non-specific defensive response of inflammation. Regardless of the cause, inflammation has three basic phases: vasodilation and increased permeability of blood vessels; emigration of phagocytes; and tissue repair (Tortora and Grabowski 2000).

Vasodilation brings more blood to the damaged area, and the increased permeability allows phagocytes and antibodies to pass out from the circulation. The clotting cascade is also activated. Clot formation is part of the normal immune response, and may be the body’s attempt to confine any invading organisms to one area of the body (Ahrens and Vollman 2003). This inflammatory response and activation of the clotting system are designed to repair damaged tissues and prevent further damage.

Inflammation is a normal physiological response to a variety of insults, and normally the body restricts the inflammation to the local site of the infection. Severe sepsis, however, produces an exaggerated, excessive, inflammatory response throughout the body. A variety of inflammatory mediators such as histamine, prostaglandins and cytokines, including tumour necrosis factor (TNF), and interleukins, are released into the circulation. These cause widespread vasodilation. Blood vessels widely dilate causing the blood pressure to plummet (Figure 1). The capillaries become more permeable allowing fluid to leak out of the circulation. This produces hypovolaemia which can lower the blood pressure further. The coagulation system also becomes activated and small blood clots (microthrombi) form in the small blood vessels (Figure 2). These thrombi interfere with blood flow to the tissues and organs and,
£1,232 (NICE 2004). This cost burden appears set to rise as the incidence of sepsis in the future is estimated to grow by 1.5 per cent per year (Angus et al 2001). This is because of the ageing population, new types of surgery, and medical technology such as central venous catheters, mechanical ventilators and haemofiltration, all of which put patients at risk of sepsis.

Antibiotic resistance may also be a contributing factor (Angus et al 2001).

Recovery from critical illness can be a long and slow process. Critical care patients with severe sepsis are among the most sick and are, therefore, most likely to experience the longest periods of recovery. Montuclard et al (2000) found that in a group of older ICU survivors one year after discharge, 26 per cent had difficulty transferring from chairs or beds, 23 per cent had difficulty bathing and 15 per cent had difficulty toileting.

Any efforts to diagnose severe sepsis as early as possible and deliver prompt and aggressive treatment may mean that patients are less sick when they arrive in ICU with fewer failing organs, possibly resulting in a shorter stay and a less protracted recovery.

The Surviving Sepsis Campaign

The high death rate from severe sepsis has prompted a joint response from the many intensive care societies around the world. In October 2002, the Surviving Sepsis Campaign was officially launched by the European Society of Intensive Care Medicine, the Society of Critical Care Medicine and the International Sepsis Forum. This is an international effort to bring about rapid improvement in the standard of care of patients with severe sepsis. The main aims of the campaign are (Dellinger et al 2004):

- Increase awareness of sepsis, severe sepsis and septic shock among healthcare staff and the general public.
- Develop evidence-based guidelines for the management of severe sepsis.
- Ensure that the guidelines are put into practice to create a global standard of care for patients with sepsis.

Sepsis

The mortality rate from severe sepsis is high at 28-50 per cent (Angus et al 2001), and there are about 21,000 cases each year of severe sepsis in England and Wales (National Institute for Clinical Excellence (NICE) 2004). Sepsis is very common and, because it is often under recognised, the incidence may be even higher than presently recorded. A patient who dies of sepsis as a result of pneumonia, may have his or her cause of death recorded as pneumonia rather than multiple organ failure as a result of severe sepsis or septic shock (Ahrens and Vollman 2003).

The management of patients with severe sepsis is expensive for the NHS. These patients often require prolonged stays in an intensive care unit (ICU), and multiple supportive therapies, such as mechanical ventilation, renal support (haemofiltration), and respiratory therapy rotational beds. They account for 46 per cent of all ICU bed days, and the average cost per day of ICU in the UK in 2002 was £1,232 (NICE 2004). This cost burden appears set to rise as the incidence of sepsis in the future is estimated to grow by 1.5 per cent per year (Angus et al 2001). This is because of the ageing population, new types of surgery, and medical technology such as central venous catheters, mechanical ventilators and haemofiltration, all of which put patients at risk of sepsis.

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〉 Reduce the mortality from sepsis worldwide by 25 per cent in the five years following the publication of evidence-based guidelines in 2004.

It is essential that nurses are aware of the evidence-based guidelines as their role is crucial to the success of the campaign. Nurses are in an ideal position to identify the first signs of a patient developing sepsis, and the sooner treatment begins the less likely the condition is to spread and result in organ dysfunction or failure (Ahrens and Tuggle 2004).

**Treating severe sepsis**

When treating trauma patients the first hour following injury is known as the ‘golden hour’ because the treatment given or not given during that time can have a significant impact on their survival, and how ill patients ultimately become in the hours and days that follow (American College of Surgeons (ACS) 1997). Prompt treatment with oxygen and intravenous fluids prevents secondary injury to organs as a result of hypoxia and hypotension, thus reducing mortality and morbidity. Patients with severe sepsis share an early window of opportunity in which aggressive treatment can influence survival and the severity of illness (Ahrens and Tuggle 2004).

To be able to treat patients in the ‘golden hour’ following the onset of severe sepsis, we must first be able to recognise it confidently. Patients with severe sepsis or septic shock will need to be cared for on high-dependency units (HDUs) or ICUs. Studies have shown that patients admitted to ICU from the wards are often not referred early enough and sometimes receive suboptimal care on the ward which can increase their mortality (McQuillan et al 1998). Vincent et al (2002) suggest that some patients with sepsis are recognised late and are not treated appropriately before transfer to ICU.

Any patient on a general ward or in a primary care setting whose condition is causing concern or who triggers an Early Warning Score, a method of identifying patients at risk of critical illness developed by critical care outreach teams (McArthur-Rose 2001), should be assessed initially using the airway, breathing, circulation, disability (A, B, C, D) approach (University of Portsmouth 2003). This systematic approach ensures that life-threatening problems are assessed and managed in order of importance. It is based on the rationale that the patient’s airway should always be assessed and managed before anything else because an obstructed airway will kill the patient before a problem with breathing (B) or circulation (C). After the airway has been managed the patient’s breathing should be assessed before circulation.

In March 2004 the Surviving Sepsis Campaign produced comprehensive guidelines on the management of patients with severe sepsis (Dellinger et al 2004).

**Initial treatment within one hour of diagnosis**

- Perform baseline observation of vital signs: respiration rate, oxygen saturation (SpO₂), capillary refill time, heart rate, and blood pressure. Consider the patient’s level of consciousness – is he or she alert or drowsy? Level of consciousness is represented by the D of the ABCD approach and refers to disability of the central nervous system.
- Give supplemental oxygen therapy, via a face mask, to achieve SpO₂ >95 per cent.
- Obtain blood specimens: lactate, full blood count, urea and electrolytes, glucose, liver function tests, clotting screen and blood cultures (Box 3).
- Administer intravenous broad spectrum antibiotics. In severe sepsis antibiotics should be given within one hour of diagnosis (Dellinger et al 2004). Giving prompt antibiotic therapy may reduce mortality by 10-15 per cent, compared with patients in whom antibiotic therapy is delayed (Wheeler and Bernard 1999).
- Any septic patient who has a lactate level above 4mmol/L should be considered to have severe sepsis even if his or her blood pressure is within his or her normal range.
- If systolic blood pressure is <90mmHg, or >40mmHg lower than the patient’s normal blood pressure, administer intravenous fluid challenges. A fluid challenge is usually 500ml given over five to ten minutes (University of Portsmouth 2003).
- If the patient remains hypotensive consider insertion of a central venous catheter, and the continuation of intravenous fluid challenges to achieve a central venous pressure (CVP) of 8-12mmHg. Patients who do not respond to fluid challenges and remain hypotensive after having 1.5-2.0 litres may require vasoconstricting drugs, such as noradrenaline (norepinephrine).

**BOX 3**

**Blood cultures**

Blood cultures should be taken before antibiotics are given to identify any micro-organisms that may be in the blood. Blood cultures should be taken from a peripheral vein and from any invasive catheters that the patient may have in situ, such as a central venous catheter. Blood cultures, however, are not always positive in patients with severe sepsis. Cohen et al (2004) suggest that care should be taken when obtaining blood cultures to prevent them becoming contaminated and giving a false positive result.
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- Insert a urinary catheter with an hour urometer that allows the hourly urine volume to be measured – and obtain a urine specimen.

### Ongoing management of severe sepsis
- Observe the patient – perform half-hourly observations of vital signs and hourly measurement of urinary output.
- Source control – every patient with severe sepsis should be examined to establish a source or a focus of infection that is causing the sepsis. If a source is identified prompt action should be taken to control or eliminate it (Dellinger et al. 2004). Surgery for obstructed bowel or ischaemic and necrotic bowel, debridement of infected or necrotic tissue, drainage of an abscess and removal of an infected intravenous catheter are all examples of source control. Source control should be carried out after initial resuscitation, as it can prevent further organ damage and improve the patient’s chances of survival.
- Consider referral and transfer of the patient to a HDU or ICU.

### Time out 5

Veronica had a laparotomy for Crohn's disease two days ago. Today she has severe abdominal pain. Her observations are:
- **Respiratory rate:** 32
- **Pulse:** 137 beats per minute
- **Blood pressure:** 90/46mmHg
- **Urine output:** 20-22ml/hr for two hours
- **Temperature:** 38.6°C

Does this patient have severe sepsis? What immediate treatment should be given? The doctor on the ward is a newly qualified pre-registration house officer and asks your advice on how to treat the patient.

Some hospitals have introduced patient group directions to enable nurses to administer fluid boluses in certain situations, such as hypotension and low urine output. Reflect on the risks and benefits of this, and how it might help in the treatment of septic patients on the wards or in primary care.

### New therapies

In the past five years new therapies have emerged that have been shown to increase the chances of survival from severe sepsis significantly.

**Early goal-directed therapy** Early goal-directed therapy is a form of early and aggressive resuscitation for patients with severe sepsis based on the findings of a research study carried out in the US by Rivers et al (2001). The study showed that if patients with severe sepsis admitted to the accident and emergency department were given aggressive resuscitation, including measurement of central venous oxygen saturations in the first six hours of diagnosis, their chances of survival were increased by 16 per cent (Rivers et al. 2001).

The early goal-directed therapy protocol should be used in patients who have severe sepsis and remain hypotensive (systolic blood pressure <90mmHg) despite being given 1.5-2.0 litres of intravenous fluids in the form of fluid challenges. The protocol has been shown to improve survival dramatically in this group of patients. Patients are given intravenous fluid challenges to achieve a mean blood pressure >65mmHg. If this cannot be achieved after receiving up to two litres of fluid a noradrenaline (norepinephrine) infusion is commenced.

A special central venous catheter is inserted (Figure 3) that can measure the oxygen saturations of venous blood, in the vena cava where the tip of the catheter sits (Figure 4). The normal value for central venous oxygen saturations is approximately 75 per cent. Central venous oxygen saturations represent the balance between the oxygen delivered to the tissues and the oxygen consumed by the tissues. The normal saturation of venous blood returning to the right side of the heart is approximately 75 per cent because the tissues usually consume, or extract, approximately 25 per cent of the oxygen delivered. Some patients with severe sepsis will have central venous oxygen saturations much less than 75 per cent, indicating that the tissues are being starved of oxygen and to compensate are extracting more oxygen than normal (Ahrens and Tuggle 2004).

Rivers et al (2001) aimed to keep the central venous oxygen saturations >70 per cent to ensure the tissues and organs received enough oxygen. If a patient’s saturations were <70 per cent they were given a blood transfusion and/or dobutamine, which increases the contractile force of the heart muscle and improves the delivery of oxygen to the tissues and organs.

Although it would not be possible to carry out early goal-directed therapy fully in a ward area, it is possible to identify promptly patients with severe sepsis who would be eligible for early goal-directed therapy, and give antibiotics and fluid boluses, while patient transfer is organised. Nurses in the ward area can help to ensure that antibiotics are commenced without delay, blood tests and blood cultures are obtained and that the patient’s vital signs are monitored carefully observing for any signs of deterioration. Ward nurses also have a key role in informing the critical care outreach team of these patients. Patients eligible for early goal-directed therapy are those whose systolic blood pressure remains <90mmHg despite fluid boluses, or who have a lactate level >4mmol/L (Rivers et al. 2001).
Activated protein C

Activated protein C is a naturally occurring protein made by the body and is both an anticoagulant and an anti-inflammatory. It promotes fibrinolysis and inhibits thrombosis as well as reducing inflammation by blocking the release of cytokines. It would be the perfect remedy to counteract the multiple small thrombi and widespread inflammation that characterise severe sepsis, but the body’s ability to convert protein C to activated protein C in severe sepsis is impaired because sepsis diminishes the ability to produce thrombin which is necessary for this process (Yan et al 2001).

A randomised controlled trial, known as the Prowess trial (Bernard et al 2001), compared the use of activated protein C with a placebo treatment in patients with severe sepsis/septic shock. The results showed that patients treated with activated protein C were more likely to survive. The death rate in the activated protein C group was 6 per cent lower than in the group who did not receive it.

Activated protein C is known as drotrecogin alfa (activated) and has been evaluated by NICE (2004). It is now recommended ‘for use in adult patients who have severe sepsis that has resulted in multiple organ failure (that is two or more major organs have failed) and who are being provided with optimal ICU support’ (NICE 2004).

Activated protein C may potentially increase the risk of bleeding, and may be contraindicated in certain patients. Activated protein C is a relatively expensive treatment. The cost for a 70kg patient is £4,905 (NICE 2004). However, although expensive, treatment with activated protein C may lead to a reduced length of stay in ICU. The average cost per day to keep a patient in ICU was estimated at £1,232 in 2002 (NICE 2004).

Reducing the incidence of sepsis

Some patients develop severe sepsis from infections that they acquire while in hospital. One in ten NHS hospital patients are affected by healthcare-associated infections (HCAIs) each year (DH 2003) and the National Audit Office (2000) estimates the cost of these at £1 billion per year. The most common of these infections are urinary infections as a result of indwelling urinary catheters and pneumonia.

The DH’s (2003) document Winning Ways states that evidence-based countermeasures to reduce HCAIs are not being implemented effectively in the majority of hospitals. Nurses must make continued efforts to play an active part in reducing the number of HCAIs as this can potentially lead to fewer patients developing sepsis in hospital.

Ahrens and Tuggle (2004) suggest that it may also be beneficial to raise the public’s awareness of the Surviving Sepsis Campaign and the warning signs of developing sepsis, in
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the same way that the public is made aware of the signs of a heart attack and urged to seek prompt medical care. This may help to achieve the main aim of the campaign which is to reduce mortality.

Conclusion

Sepsis is a common condition and can be fatal. A good knowledge of the signs and symptoms of sepsis, SIRS, severe sepsis and septic shock is the key to prompt recognition. Everyone, suspected of having severe sepsis should have blood taken for a serum lactate level. In patients with severe sepsis, early aggressive treatment and adherence to evidence-based guidelines can help to save lives. In addition, efforts to reduce HCAIs can aid in the reduction of the incidence of severe sepsis NS

References


