Developments in the management of patients with sepsis


Summary
Sepsis is associated with a high incidence of mortality and morbidity, however with appropriate strategies for monitoring and early targeted intervention mortality rates decline. This article examines the basic pathophysiology of the inflammatory response to tissue injury and the effects this has on circulation. A partnership of the Society of Critical Care Medicine, the European Society of Intensive Care Medicine and the International Sepsis Forum has developed the surviving sepsis strategy, which includes the sepsis six treatment pathway and early goal-directed therapy incorporating the six-hour care bundle.

Aims and intended learning outcomes
This article aims to provide the reader with an increased understanding of the causes, signs and symptoms of sepsis and strategies such as the Surviving Sepsis Campaign, which aims to reduce the incidence of the condition. After reading this article you should be able to:

- Identify the differences between sepsis, severe sepsis and septic shock.
- Describe the pathophysiology of the inflammatory process.
- Explain the epidemiology of sepsis.
- Describe the sepsis six treatment pathway in the management of a patient with severe sepsis.
- Reflect on how the sepsis six treatment pathway could be implemented in clinical practice.

Introduction
Sepsis is a condition characterised by a systemic inflammatory response syndrome (SIRS) and the presence of infection. Chinese Emperor Sheng Nung, circa 2737 BC, presented one of the earliest descriptions of inflammation and described the herbal medicines available to treat it. Today, inflammation, sepsis and SIRS are increasing in incidence around the world (Angus et al 2001). Colloquially known as ‘blood poisoning’, sepsis is associated with a high mortality rate, however appropriate treatment and management of the condition can reduce mortality rates (Angus and Wax 2001, Alberti et al 2002, Martin et al 2003).

Angus et al (2001) identified an increase in the incidence of sepsis over time, which is expected to continue to rise as a result of an increasing ageing population, underactive immune systems arising from chemotherapy, organ transplantation, complex co-morbidities such as heart disease and diabetes, human immunodeficiency virus and drug-resistant invading organisms.

In England, Wales and Northern Ireland the incidence of patients admitted to intensive care units with severe sepsis has increased by more than 1.5% per year since reliable data started to be collected in 1996 (Harrison et al 2006). The rise in incidence has led to an increase in hospital-related
deaths from severe sepsis, from 23 deaths per 100,000 of the population in 1996 to 30 deaths per 100,000 of the population in 2003 (Harrison et al 2006). More people die from sepsis than from lung, bowel or breast cancer (Dellinger et al 2008), costing Europe approximately £6 billion per year (Bone et al 1992, Angus et al 2001). Precise mortality figures are difficult to determine with absolute accuracy as the primary disease is predominately documented as the cause of death and not the subsequent sepsis.

The Surviving Sepsis Campaign (Dellinger et al 2008) aims to build awareness of sepsis, improve diagnosis, increase the use of appropriate treatment, educate healthcare professionals, improve post-intensive care unit care, develop guidelines for care and facilitate data collection for the purposes of audit and feedback. Promoting early detection and intervention with goal-directed therapy and adopting the sepsis six strategy in the management of severe sepsis can help to reduce the mortality rates associated with the condition (Dellinger et al 2008).

**Defining sepsis**

Sepsis is a global term that is often misused. Patients who develop signs of circulatory collapse that are not attributable to another cause are often thought to be experiencing sepsis. Sepsis is an acute and severe disease associated with high mortality. The European Society of Intensive Care Medicine launched the Barcelona Declaration, which calls for an increase in the awareness of sepsis and the effects it has on individuals and society. The aim of the campaign was to reduce the sepsis mortality rate by 25% within five years (Bone et al 1992, Silva et al 2006).

Sepsis is a term that is given to a systemic inflammatory response that arises from an infective process, which may be caused by bacterial, viral or fungal invasion that provokes an inflammatory response (Box 1). Alternatively, an inflammatory response can arise from a non-infective process commonly known as systemic inflammatory response syndrome (SIRS). Examples of this may be severe trauma, surgery, complications related to adrenal insufficiency, myocardial infarction, burns or acute pancreatitis. The inflammation arises as a result of either tissue ischaemia or tissue damage. It is important to be aware that the causative agents can arise from non-infective causes as well as infection.

As an alternative to SIRS, the term signs and symptoms of infection (SSI) is being promoted by the Surviving Sepsis Campaign (Dellinger et al 2008). While the majority of patients present with an infective cause of sepsis there is a danger in using SSI to describe the condition in that attention may be distracted from non-infective causes. The term SIRS will be used in this article to indicate a non-infective cause. **Severe sepsis** Severe sepsis occurs when there is induced organ dysfunction. This arises from hypoperfusion resulting in tissue hypoxia. **Septic shock** The consequence of unresolved severe sepsis is multiple organ failure and death from septic shock (Figure 1). Septic shock is defined as sepsis-induced hypoperfusion that persists despite adequate fluid resuscitation. Where there is early, targeted intervention the development of severe sepsis and septic shock is averted and organ dysfunction and death does not occur.

**Epidemiology**

The incidence of sepsis has increased over time and is predicted to increase further (Angus and Wax 2001). There are many reasons for this. Patients...
Learning Zone: Infection Control Focus

with sepsis who experience subsequent deterioration are often high risk because of co-morbidities, increased age, compromised immune systems or recent invasive medical procedures. However, it is difficult to gain absolute figures on the incidence of sepsis because of the different contributing factors and the constantly changing evolution of disease pathology. A pan-European study showed that more than one third of adult patients in intensive care units developed sepsis; 25% of these individuals had sepsis on admission to the intensive care unit having developed it on the wards (Vincent et al 2006). In the UK, the incidence of deaths related to sepsis has increased from 23 to 30 cases per 100,000 of the population between 1996 and 2003 (Harrison et al 2006). These patients account for 46% of all intensive care unit bed days and 33% of all hospital bed days (Intensive Care National Audit and Research Centre 2003).

Time Out 1

Identify five symptoms of infection and describe how each symptom may arise.

The inflammatory response

Inflammation can be described as redness, heat, swelling, pain and loss of function (Jenkins et al 2007). The body is under constant attack from invading organisms and has mechanisms for combating this invasion. Examples of natural barriers to infection are the skin, eyes, nasal passages, lungs and gastrointestinal tract. Unless the skin is damaged invading organisms are unable to gain access. The lungs have the mucociliary escalator that removes debris and bacteria, the eyes have tears containing the enzyme lysosome, which engulfs bacteria and viruses, the nose has nasal hairs, which filter debris and bacteria, and the stomach has acid, which destroys invading organisms (Smith et al 2004). These host defence mechanisms are constantly working. However, these defence mechanisms can be breached at any time provoking an inflammatory response (Jenkins et al 2007).

An inflammatory response is desirable and normal. It enables survival from infection and protects against further injury. For example, a winter dose of influenza results in the body creating an inflammatory response that mobilises the immune system to combat the invading organism and creates an adverse environment for the invading organism. Over time the invading organism is overcome and the body’s environment returns to normal and the individual regains health. A simple equation can summarise how sepsis affects the individual (Box 2).

Wherever there is a mechanism of injury the body will respond appropriately to ensure survival of the individual, however this response can also develop into a disease process.

The body’s response to the mechanism of injury is complex. When there is either an invasion of foreign material such as bacteria or damage to tissue such as ischaemia or muscle damage an immune response is provoked (Jenkins et al 2007). Initially the immune response is triggered by a humoral activity where the circulating antibodies react to the invading organism. These antibodies stick to the bacteria marking them for destruction. Cell-mediated immunity occurs, involving the activation of macrophages, especially neutrophils, to destroy the marked bacteria (Jenkins et al 2007).

The first stage of the inflammatory response promotes blood flow to the site of injury through vasodilation. This creates the first classic sign of inflammation, redness (erythema). The increased blood flow also results in the area feeling hot, the second symptom of inflammation. As part of the inflammatory process the micro-capillaries allow fluid to leak out of the circulation into the interstitial space. This fluid delivers white blood cells and proteins to the site of injury which attack the invading organism and promote growth and repair (Jenkins et al 2007). This results in the third classic symptom of inflammation, swelling. Swelling stretches the tissues creating pain – the final symptom of inflammation. Pain encourages the individual to rest the affected area limiting further damage. In the normal inflammatory response, the damage that occurs to the endothelium of the microcirculation enables fluid to leak out and activates the clotting cascade. The endothelial damage promotes clot formation in the microcirculation at the site of injury preventing debris from macrophage action and the invading organisms entering the systemic circulation. At the appropriate time the clot is dissolved and blood flow is restored. Cessation of blood flow combined with excessive inflammatory-induced swelling results in a marked reduction in blood supply to the area (Jenkins et al 2007).

Box 2

Development of sepsis

<table>
<thead>
<tr>
<th>Mechanism of injury (Infection of tissue)</th>
<th>Body’s response (Inflammation)</th>
<th>Disease (Sepsis/systemic inflammatory response syndrome/signs and symptoms of infection)</th>
</tr>
</thead>
</table>

Nursing Standard
The inflammatory process is self-contained by feedback mechanisms that increase then limit inflammation, promote the leakage of fluid into the interstitial space from the capillary bed and promote coagulation followed by anticoagulation. These inherent checks and balances permit the body to remain in homeostasis (Jenkins et al 2007). Homeostasis occurs continuously in the body mostly without the individual realising it and certainly without the individual feeling severely ill. When an individual starts to display symptoms of illness and physiological deterioration, the inflammatory process is no longer localised to the site of injury and is having a systemic effect. At this point a person can be described as having sepsis or SIRS (Levy et al 2003).

**Time out 2**

What are the early presenting symptoms of sepsis or SIRS? Review the strategies that you might implement to detect these symptoms early in the disease process. List the criteria for diagnosing sepsis.

Similar to other acute illnesses one of the first symptoms of sepsis is an increase in respiratory rate. This arises from subtle changes in acid-base balance, which excite the medulla oblongata to stimulate respiration. The acid-base changes occur as a result of cellular hypoxia from a reduction in oxygen supply to cells, resulting from tissue hypoperfusion. This results in anaerobic cellular respiration, the consequence of which is the production of lactate causing a reduction in pH, known as acidosis (Morton and Fontaine 2009). Septic shock is sometimes referred to as distributive shock, which describes accurately the effects of the disease process on the movement of fluid around the different compartments of the body (Morton and Fontaine 2009). The early symptoms of systemic vasodilatation can be observed – the patient appears red and flushed with a full and bounding pulse and a rapid capillary refill. This systemic response results in an increase in peripheral circulation and the permeability of the micro-vasculature creating a major loss of volume from the core circulation. The consequence of this is the derangement of organ functioning (Jenkins et al 2007). The effects on organ dysfunction are easily detected. For example, early signs may include an increase in respiratory rate, an increase in heart rate and an altered mental state. A reduction in urine output will occur, but the evidence for this may be delayed if the patient is not catheterised. An initial rise in diastolic pressure followed by a drop in blood pressure and alterations in temperature are late symptoms of a lack of oxygen supply to the tissues (Morton and Fontaine 2009).

**Time out 3**

Describe the differences between sepsis, severe sepsis and septic shock.

Septic shock is the term given to the profound effects that the systemic inflammatory response has on oxygen delivery to the tissues. It needs to be treated urgently before irreparable organ damage.

The body titrates cardiac output to the metabolic demands, but aims to maintain a cardiac output of 4-8 litres per minute (Morton and Fontaine 2009). During the early phases of sepsis and because of systemic vasodilatation, the venous return and stroke volume (the amount of blood ejected by the ventricle during contraction) fall, resulting in a reduced cardiac output. To compensate the body will immediately raise the heart rate and increase levels of adrenaline to improve cardiac output and increase vascular tone (Jenkins et al 2007). In response to reduced blood flow, the renin-angiotensin-aldosterone system is activated, increasing vascular tone and conserving fluid by reducing urine output.

As the disease process develops the combined effect of loss of venous return and loss of circulating volume results in significant reduction in cardiac output and blood flow. This in turn results in an inadequate oxygen supply to the tissues, cellular hypoxia and organ dysfunction. The patient may start to display symptoms of deterioration many hours before sepsis becomes detrimental to the individual’s health. The consequences of missing these early symptoms are that when the patient becomes acutely ill he or she will have had compromised oxygen supply to the tissues and organ dysfunction for many hours, which will lead to organ failure and severe sepsis. The criteria for diagnosing severe sepsis are presented in Table 1.

**Table 1**

Criteria for diagnosing severe sepsis

<table>
<thead>
<tr>
<th>General variables</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in body temperature</td>
<td>&gt;38.3°C or &lt;36°C</td>
</tr>
<tr>
<td>Heart rate</td>
<td>&gt;90 beats per minute</td>
</tr>
<tr>
<td>Tachypnoea</td>
<td>&gt;20 breaths per minute</td>
</tr>
<tr>
<td>Mental state</td>
<td>Altered mental state</td>
</tr>
<tr>
<td>Significant oedema or positive fluid balance</td>
<td>20ml/kg over 24 hours</td>
</tr>
<tr>
<td>Hyperglycaemia (in the absence of a diagnosis of diabetes)</td>
<td>Plasma glucose &gt;77mmol/l</td>
</tr>
</tbody>
</table>

(Dellinger et al 2008)
occurs. It refers to shock that is unresponsive to intravascular volume replacement. Septic shock can lead to refractory shock where blood pressure does not respond to vasoactive drugs and this will rapidly lead to death (Annane et al 2005).

Treatment

Prevention is important in managing and reducing the incidence of sepsis. Asepsis, preventing contamination of vulnerable patients, is an important strategy and there are various national strategies (Department of Health (DH) 2003) in place to reduce the incidence of cross-infection in the healthcare system. Removal of invasive intravenous and urinary catheters that are no longer needed is an important element. Implementing simple procedures such as employing a non-touch technique will help to reduce the complications that can result in sepsis, and the nurse is at the centre of these strategies. Early mobilisation reduces the risk of hypostatic pneumonia and improves circulation. Where mobilisation is not possible, optimising respiratory function with incentive spirometry and physiotherapy will help. Maintaining adequate hydration and nutrition are effective strategies that can limit the risk of sepsis from hospital-acquired infection. Where prevention fails, early intervention is imperative in preventing further deterioration (Rivers et al 2001, Levy et al 2003). Early warning scoring systems are also known as modified early warning systems (MEWS) or track and trigger systems are now widely used in hospitals. However, they are not currently in use in the community setting or in nursing homes, where they may also be helpful. MEWS have a positive effect on clinical outcome (Subbe et al 2001, 2003) in a variety of patient groups by identifying those at risk of clinical deterioration (Goldhill and McNarry 2004).

It is recommended that the sepsis six treatment pathway is implemented within the first six hours of identifying the septic patient (Dellinger et al 2008). Delays in implementing fluid resuscitation will result in further patient decline, hastening tissue hypoperfusion and resulting in enduring organ failure.

The sepsis six treatment pathway

The sepsis six treatment pathway comprises the following elements, each of which is discussed in turn (Surviving Sepsis Campaign 2007):

- Oxygen therapy.
- Fluid resuscitation.
- Measurement and monitoring of urine output.
- Blood cultures.
- Antibiotic therapy.
- Lactate, haemoglobin and routine blood monitoring.

A care bundle is designed to ensure that all the tasks are carried out at a specific time and place. It is a collection of interventions, each one of which is based on identified best knowledge or practice as recommended and supported by the DH (NHS Modernisation Agency 2004).

Once the symptoms of deterioration or organ dysfunction have been detected the following steps need to be implemented as soon as possible and within six hours for the remainder of the care bundle. Where compliance with the care bundle has been good there has been a reduction in patient mortality (Gao et al 2005).

Step 1: oxygen therapy

Missing from the six-hour care bundle are recommendations for oxygen therapy in the management of severe sepsis, however this is included in the sepsis six guidelines (Robson and Daniel 2008). All acutely ill patients should receive high-dose oxygen via a non-rebreathing mask to normalise oxygen saturation of haemoglobin during the acute phase of illness, aiming for an oxygen saturation within normal limits (McQuillan et al 1998). Following this, the focus of sepsis management is on restoration of the failing circulation.

Step 2: fluid resuscitation and central venous pressure monitoring

Early goal-directed therapy

On identifying a deteriorating patient, Rivers et al (2001) recommended the implementation of an early goal-directed therapy strategy in the management of sepsis. The first element of this is fluid resuscitation. Patients should receive fluid resuscitation as soon as hypoperfusion is identified and treatment should not be delayed. Rivers et al (2001) recommended administration of fluid resuscitation to achieve a central venous pressure (CVP) of 8-12cmH₂O. In ventilated patients, to reflect the changes in intrathoracic pressure resulting from positive pressure ventilation, a higher CVP target of 12-15cmH₂O is recommended (Bendjelid and Romand 2003).

Fluid resuscitation

Fluid resuscitation should follow the fluid challenge principle where fluid is continued so long as there are improvements in blood pressure, heart rate and urine output (Vincent and Gerlach 2004). Dellinger et al (2008) recommended administering one litre of crystalloid or 300-500ml of colloid over 30 minutes, with a more rapid rate and increased volumes if there are significant signs of
hypoperfusion. Where there are no indications of improvement in blood pressure or where there is evidence of cardiac insufficiency or a documented history of heart failure then the volume of fluid should be reduced or administered more cautiously (Dellinger et al 2008).

The Rivers et al (2001) study recommends the use of CVP monitoring, however this is rarely available on general wards. The Comprehensive Critical Care document (DH 2000) recommends ‘critical care without walls’ and advocates that the resources required for the treatment and management of acutely ill patients should be made available on occasions where patients require aggressive treatment and close monitoring in the short term. With the advent of critical care outreach, understanding and application of such procedures should be enhanced and achievable.

**Blood transfusion** Rivers et al (2001) transfused patients to maintain a haematocrit ≥30%, and the Surviving Sepsis Campaign recommend administering blood products to a target haemoglobin of 7.0-9.0g/dl (Dellinger et al 2008). This may seem lower than the normal haemoglobin level, but consideration needs to be given to balancing the oxygen-carrying capacity of the blood and blood viscosity. The higher the haemoglobin level the higher the viscosity.

**Blood pressure monitoring** Early goal-directed therapy recommends aiming for a mean arterial pressure of ≥65mmHg (Rivers et al 2001). Measuring and recording this value is assisted by automated non-invasive blood pressure monitoring equipment, where is it calculated automatically and displayed.

**Venous oxygen saturation** It has been recommended that central venous oxygen saturation should be measured as an indication of tissue oxygen delivery and consumption. It should be maintained at ≥70% (Rivers et al 2001). Venous oxygen saturation is the same as arterial oxygen saturation, only the blood originates from the central venous system. Venous saturation can only be monitored by sampling blood gases from a central venous catheter, thus strengthening the argument for inserting these catheters as soon as possible.

**Inotrope and vasoactive support** The use of an inotrope to support cardiac output has been recommended (Rivers et al 2001). Rivers et al (2001) used dobutamine at a maximum dose of 20μg/kg/min. However, this should only be used following fluid resuscitation to target levels and in patients where cardiac insufficiency is demonstrated by a low cardiac output. All inotropes and vasoactive drugs have the capacity to cause harm to patients and should be used with specialist advice and knowledge. However, where there is evidence of targeted fluid administration with persistent hypotension a vaspressor such as noradrenaline should be used (LeDoux et al 2000, Hollenberg et al 2004). A vaspressor is a drug that causes a rise in blood pressure by causing constriction of the blood vessels. An alternative vaspressor, dopamine, may be used to support blood pressure, but controversy exists regarding the use of low-dose dopamine to support renal perfusion and is not currently recommended (Bellomo et al 2000).

**Step 3: urine output** Although there is a delayed response to fluid resuscitation, Rivers et al (2001) recommended that urine output should be monitored closely. This should be maintained at ≥0.5ml/kg/hour. The insertion of a urinary catheter with an appropriate measuring bag is required to accurately measure urine output in patients.

**Steps 4 and 5: blood cultures and antibiotic therapy** During insertion of new (<48 hours) intravenous catheters, such as central venous catheters and peripheral catheters for rapid fluid resuscitation, blood cultures should be taken. In addition, the recommendation is to take blood cultures from all existing central catheters that have been in situ for more than 48 hours (Dellinger et al 2008). Other potential sites of infection, for example, sputum, urine and wounds, should also be cultured. Cultures should be taken before the administration of antibiotics to prevent sterilisation of blood cultures, which can occur a few hours after the first antibiotic dose. Following sampling and culturing, broad-spectrum antibiotics should be administered (Dellinger et al 2008). This should be achieved within one hour of recognition of severe sepsis or septic shock. Each hour of delay results in a significant increase in mortality (Kumar et al 2006).

**Step 6: lactate measurement** An elevated lactate measurement is an important indicator of the need to start treating the patient with sepsis (Rivers et al 2001). In sepsis, lactate rises when the supply of oxygen to the cells cannot meet the demand for normal aerobic respiration. However, the absolute measurement can lack accuracy owing to variations in blood flow to tissues during poor tissue perfusion. Treatment for septic shock should commence at a lactate level of ≥4mmol/l (Rivers et al 2001, Dellinger et al 2008). Other laboratory investigations should be performed as indicated, including routine urine and electrolytes, haemoglobin and liver function tests.

**Time out 4**

List the components of the six-hour care bundle and reflect on how you might implement these in practice.
The remainder of this article focuses on the strategy that should be implemented in the 24 hours following diagnosis of severe sepsis or septic shock. This relates specifically to those patients in intensive care and is only covered briefly.

**The 24-hour care bundle**

**Glucose control** It is noted that hyperglycaemia arising from insulin resistance without a previous history of diabetes can occur with critical illness, leading to an increase in mortality. It is recommended that patients who display raised levels of blood glucose following severe sepsis or septic shock should receive intravenous insulin therapy to reduce levels to <150mg/dl (8.3mmol/l) (Dellinger et al 2008).

**Corticosteroid therapy** This should only be considered if response to fluid resuscitation and vasoactive support has been poor. The definitive evidence for the use of corticosteroids has not been proven, although there is some evidence to suggest that corticosteroids can reverse the symptoms of shock (Sprung et al 2008).

**Lung protection strategies** In patients receiving intermittent positive pressure ventilation, the principle of lung protection in sepsis follows the recommendations of the Acute Respiratory Distress Syndrome Network trial (Slutsky and Ranieri 2000). These recommendations include reduction in tidal volume to 6ml/kg body weight with permissive hypercapnia (Slutsky and Ranieri 2000). Maintaining ventilation plateau pressures <30cmH₂O (Slutsky and Ranieri 2000) with the use of positive end expiratory pressures helps to prevent lung collapse at end expiration (Dellinger et al 2008).

Patients who require high levels of ventilation can be positioned prone. The evidence regarding the success of prone ventilation is that it appears to improve oxygenation but not mortality (Lammin et al 1994, Stocker et al 1997, Jolliet et al 1998, Gattinoni et al 2001, Guerin et al 2004). Controversy exists about when in the disease process to use the prone position, how long the patient should be left in this position, when the patient should be turned back to the supine position and whether the procedure should be repeated in the event of deterioration in ventilation.

To prevent aspiration pneumonia in ventilated patients the head of the bed should be elevated to 30-45° (van Nieuwenhoven et al 2006). There are also recommendations that weaning protocols should be in place (Dellinger et al 2008). These include spontaneous breathing trials, targeted sedation scoring and restricted use of paralysing agents, early extubation so long as the patient’s fraction of inspired oxygen (FiO₂) requirements could be met using a face mask and early tracheostomy for failed extubation.

**Recombinant human activated protein C**

Protein C is synthesised by the liver. Once activated (APC) it is one of the major inhibitors of the clotting system. The inflammatory response in severe sepsis is linked to activation of the clotting cascade. In a large, multicentre, randomised, controlled trial, recombinant APC was shown to have anti-inflammatory properties and improved survival in patients with sepsis-induced organ dysfunction (Bernard et al 2001). It is recommended that this treatment is offered so long as the patient meets the criteria for administering the drug and that there are no contraindications.

Other areas for consideration are stress ulcer and deep vein thrombosis prophylaxis and renal replacement therapy.

**Conclusion**

Sepsis is associated with a high incidence of mortality and morbidity and nurses need to have an understanding of the causes, signs and symptoms of the condition to be able to provide early and appropriate care and treatment. Strategies such as the Surviving Sepsis Campaign have helped to raise awareness of the problem and aim to reduce the death rate associated with sepsis.

The care bundles described offer a systematic way of identifying the deteriorating patient, applying simple but effective strategies outside the high-care area. More intensive treatment can be offered in high-care areas and again simple strategies can help to reduce the effects of sepsis. **NS**
References


Sepsis
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- You might like to read the article to update yourself before attempting the questions.

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