The author reviews the pathophysiology of septic shock conditions, specifically endotoxin or gram-negative septic shock. Emphasis is placed on the anesthetic management of the patient intraoperatively as well as preoperative assessment. The choice of anesthetic techniques will be discussed along with specific pharmacological agents that have been researched in the area of treatment for septic shock conditions.

In recent years much attention has been focused on the care of the traumatized patient. As a result more trauma-oriented centers and educational programs have developed, contributing to the decreasing mortality rate of initial trauma. However, of the patients that do survive the initial injury, many of them succumb to other complications, one of which is sepsis with subsequent septic shock.

Fry and associates, in their study of 553 emergency surgical patients, demonstrated that "multiple system organ failure" (MSOF) remains a principal cause of death after major operative procedures and/or severe trauma, and is primarily due to infection.1

With more trauma patients surviving and undergoing surgery, it is important that the anesthetist is knowledgeable of the pathophysiologic sequelae of sepsis and its effects, especially to the renal, cardiovascular, respiratory and clotting systems.2 Successful maintenance and improvement of these systems is probably the biggest challenge presented to the anesthetist managing the care of such high risk patients.

Many causes of septicemia in man have been identified or insinuated (Table I). The causative bacteria can be gram-negative or gram-positive, with about 70% of the cases of septic shock due to gram-negative bacteremia.3 Since gram-negative bacteremia is responsible for the majority of septic shock conditions, its manifestations will be reviewed exclusively.

Endotoxin is present in the cell wall of gram-negative bacteria and is released from the cell wall upon the death of the bacteria. It is believed that the gut may be the major source of circulating endotoxin, and its entry occurs when the permeability of the intestinal wall is increased by ischemia, chemical irritation or circulating vasoactive agents.4 Septic shock due to the cellular factor of gram-negative bacteria is termed endotoxin shock.5

Once the endotoxin enters into the circulation, it is presumed that it inflicts direct injury to capillaries, thereby altering their permeability, with resultant loss of plasma from the intravascular space. This in turn decreases the effective circulating blood volume, venous return and cardiac output leading to decreased tissue perfusion. The lack of oxygen to the tissues for utilization leads to anaerobic metabolism and local acidosis. The tissues are further subjected to anoxia by the release
of catecholamines also thought to be a result of endotoxin in the circulation. The release of catecholamines from the adrenal medulla and the postganglionic sympathetic nerve endings causes an intense vasoconstriction in arterioles and venules which leads to stagnant anoxia in the microcirculation.5

It has been accepted that septic shock occurs in two phases. The first phase is the early or hyperdynamic phase.3 This phase is manifested by warm moist skin, thought to be due to vasodilation caused by the endotoxin. Tachypnea, fever chills, and confusion are also present. There is a lowered peripheral resistance and high cardiac output in the early phase of the disease.10 The vasodilation of this phase is also caused by the endotoxin acting as an antigenic stimulus to the release of such vasoactive substances as histamine and bradykinin.8

Reversal of endotoxin-induced hypotension in animals treated with naloxone suggests that endorphins may play a role in the manifestations of septic shock.8

Further, in a study with human septic shock patients, naloxone given intravenously resulted in increased blood pressure, cardiac output and decreased systemic vascular resistance. This study then suggests that endorphins may contribute to the hypotension of sepsis and therefore naloxone may be of value in the treatment of septic shock.7

In the late or hypovolemic phase, vasoconstriction replaces vasodilation.8 Decreasing cardiac output leads to significant cellular anoxia with worsening lactic acidosis. Due to the intense vasoconstriction, cold and cyanotic skin is observed. The patient exhibits progressive lethargy and possibly coma. Renal perfusion is decreased with oliguria or anuria, rising blood urea nitrogen (BUN) and serum potassium levels.

Hematologic variations develop as the shock worsens. There is typically a decrease in the platelet count, prolonged prothrombin and activated partial thromboplastin times, with elevations in concentrations of fibrin degradation products, mirroring disseminated intravascular coagulopathy (DIC).8

With increasing interstitial pulmonary edema and resultant arterial hypoxemia, intubation with mechanical ventilation may need to be instituted. This measure may also decrease the actual work of breathing and therefore the strain on an already compromised myocardium.

Along with the increased work load on the myocardium to sustain adequate cardiac output, myocardial depressant factor (MDF) has been found in the circulation causing depression of myocardial contractility. One study described this in man during the condition of septic shock.8

The pathophysiologic effects of endotoxin shock can be much more extensive than portrayed above, however, that is not within the scope of this article. Rather, the anesthetic management of these patients as they present in various stages of shock will be focused upon.

Anesthesia for shock patients

In order to formulate an individualized plan of care for each patient, it is important that the surgeon and anesthetist communicate freely. If the patient is not facing life-threatening consequences without immediate surgery, it may be advantageous to delay surgery and allow the anesthetist time to further assess the patient's preoperative needs. This added assessment may be helpful to insure optimum functioning of organ systems intraoperatively.

The phase of the shock syndrome should be ascertained, as the therapy and supportive measures may vary for each phase. If the patient has deteriorated to the late or hypovolemic phase, volume expansion and repletion is required to insure adequate circulating volume. This may not be so important in the early phase if the patient is already normovolemic, with a high or normal cardiac output.

The author believes that central venous and pulmonary artery cannulation may yield extremely valuable information in preoperative and perioperative monitoring.

Measurements of central venous pressure reflect the right side of the circulation. It can be used to assess the adequacy of vascular volume and in-

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<p>| Table I |</p>
<table>
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<tr>
<th>Causes of septicemia</th>
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<tr>
<td>Trauma</td>
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<td>GU tract instrumentation</td>
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<td>Generalized skin infection</td>
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<td>Contamination of:</td>
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<td>Anesthetic drugs</td>
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<td>Intravenous catheters</td>
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<td>Immunosuppression</td>
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<td>Extensive burns</td>
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directly the adequacy of ventricular filling, although it may not be reliable in patients with severe left ventricular disease.\(^8\)

Cannulation of the pulmonary artery enables the determination of cardiac output and pulmonary capillary wedge pressures as well as allowing access for true mixed venous blood. Pulmonary capillary wedge pressure provides for an indirect measurement of left atrial pressure, which helps to estimate left ventricular filling pressure. True mixed venous blood can be used in the calculation of intrapulmonary shunting.\(^9\)

If these lines are already present when the patient presents to surgery, then previous baseline measurements will provide additional information for detecting unfavorable trends during the anesthetic.

Fever should be lowered preoperatively if possible to reduce oxygen consumption. Temperature monitoring should be done continuously during the course of the anesthetic administration. If fever increases or reoccurs, every effort should be made to reduce it.

The anesthetist should closely monitor urinary output as this may reflect adequacy of renal perfusion and give clues to approaching oliguria or anuria. This will necessitate a urinary catheter with frequent urine measurements at least every hour if not more often. Close monitoring of urinary output will also be useful for evaluating the effects of any administered diuretics.

Arterial catheters should probably be present in most patients with this condition to provide the anesthetist with moment-to-moment changes in arterial pressure. These catheters also provide access for arterial blood gas determinations. Serial measurements will enable the need for mechanical ventilation to be instituted if carbon dioxide retention or arterial hypoxemia is evident. If pulmonary edema is a significant problem then positive end expiratory pressure may be helpful in improving arterial oxygen tension. Intubation with mechanical ventilation may be indicated if the patient is tachypneic and/or dyspneic preoperatively, as this will lessen the work of breathing and decrease oxygen consumption. It has been noted that early institution of mechanical ventilation resulted in improved survival of patients with septic shock.\(^10\)

Platelet counts, prothrombin and activated partial thromboplastin times, fibrinogen levels and fibrin degradation product levels should probably be evaluated to allow for early detection of disseminated intravascular coagulopathy, a grave finding in these patients. It was noted previously that these levels were altered in DIC.

Despite all therapy given, cardiac output may still be significantly decreased, necessitating the use of inotropic drugs. The primary drugs employed are digitalis, dopamine and isoproterenol. Dopamine now appears to be the inotropic drug of choice for these patients. Distribution of blood to the kidney and mesentery makes it more useful.\(^2\)

The anesthetic choice and technique may be different for each patient; however, if the site of surgery permits, local or regional anesthesia is the technique of choice.\(^2\) In patients with septic shock, blocks should be performed with minimal doses of the anesthetic agent, since myocardial depression may ensue from absorption of local anesthetic drugs. This has been documented in at least one case by intraoperative measurements.\(^11\)

Septicemia, bacteremia and shock have been listed as absolute contraindications for spinal anesthesia according to Lund and Cwik.\(^12\) Also, further hypotension may be manifest by the sympathetic ganglionic blockade produced by spinal anesthesia. If this technique is used, it would be advisable to have large-bore intravenous lines present and inotropic therapy available. As mentioned before, dopamine may be the drug of choice.

Where regional or local anesthesia is deemed inappropriate, general anesthesia may be necessary. However, the anesthetist may want to avoid moderate to deep levels of potent inhalational agents, since some heart failure is often present during septic shock.\(^2\) With an increase in pulmonary vascular resistance reported from anesthesia with nitrous oxide,\(^13\) the anesthetist may also want to avoid this agent.

In a study of 18 patients in bacteremic shock, Stanley and Reddy demonstrated that the fentanyl-oxygen technique may show promise in the anesthetic management of these patients. All 18 patients had systolic blood pressure readings of less than 90. Fentanyl in doses of 100-300 \(\mu\)g/min were given until the patients were unresponsive. After the administration of an additional 250 \(\mu\)g of fentanyl, intubation was performed. Increments of 150-250 \(\mu\)g were given for systolic blood pressure readings of more than 120 mmHg and pulse rates greater than 125/minute, or if sweating and tearing developed. The researchers noted no cardiovascular depression; there were slight increases in arterial pressure, stroke volume, cardiac output and systemic vascular resistance intraoperatively. Heart rate was decreased slightly.\(^14\)

Wong and Jenkins suggest that in the early stages of septicemic shock, ketamine might be a useful agent to use if anesthesia is required. In the study, cats subjected to experimentally induced
endotoxin shock were given 5 mg/kg of ketamine. The animals' hemodynamic parameters of cardiac output, arterial pressure and heart rate returned towards, although not quite to, normal.  

Steroid therapy has been a controversial in the treatment of septic shock conditions. The Food and Drug Administration recently reviewed the indications for the use of corticosteroids for septic shock, particularly treatment with methylprednisolone sodium succinate (MPSS). As a result of this review, septic shock was deleted as an indication for the use of high doses of this drug. Firm evidence as to the benefit of high doses of MPSS in these patients obviously was not noted.  

In contrast, Sibbald and associates reported on a study of septic patients whereby after the administration of a massive dose of corticosteroid there was a prompt decrease in pulmonary capillary leakage in most patients. Other data in this study strongly suggests that possibly the earlier the dose of corticosteroid is administered, the more effective it will be in stopping capillary leakage.  

Conclusion  
In the moribund patient, little or no anesthetic may be needed but with the knowledge of the effects of endotoxin on the various organ systems, the anesthetist can concentrate on the maintenance and/or improvement of the hemodynamic parameters of the patient intraoperatively.

REFERENCES  

AUTHOR  
Brenda M. Brancheau, RN, received her diploma in nursing from St. Vincent Hospital School of Nursing, Toledo, Ohio in 1976. She practiced nursing for more than five years at Riverside Hospital in Toledo, two of which were spent in the Emergency Department. She is enrolled in the St. Vincent Medical Center School of Anesthesia for Nurses. At the time this paper was written, she was a senior in that program.