Definition: shock from The Penguin Dictionary of Psychology

1
A clinical syndrome that accompanies disruption of the oxygen supply to tissues, particularly brain tissues. Shock, to some extent, accompanies every injury although it is generally detectable only when there has been a major trauma, such as serious injury, surgery, an overdose of certain drugs or an extremely strong emotional experience.

2
The result of passing an electric current through the body. Severe shock in sense 2 can produce shock in sense 1. See also SHOCK THERAPY.

Summary Article: SHOCK, TREATMENT OF
From Encyclopedia of Medical Devices and Instrumentation

INTRODUCTION
Assessment and treatment of shock is based on understanding of circulatory system physiology and cellular metabolism. Shock is defined as inadequate supply of oxygen and nutrients due to inadequate capillary perfusion to the cells. This definition is not always correct, as in severe shock some cells cannot metabolize oxygen even with adequate perfusion. In addition, the removal of metabolites is equally important or even more crucial over time, since accumulated metabolites will cause cell injury.

Deficient capillary perfusion triggers a host of metabolic changes in every organ and tissue, which affects whole body homeostasis and circulation. Ideally, one should evaluate the cellular metabolism, but this assessment can be done only indirectly by measuring the acid–base balance in the form of blood gas and pH. This arterial or venous blood test does not give any information about the regional metabolic alterations. For evaluation of the circulatory function, indirect measurements are used including heart rate, blood pressure, and urinary output. Invasive hemodynamic monitoring devices, such as the pulmonary artery catheter are also used for cardiac function assessment. To effectively reverse shock requires monitoring of the pathophysiologic state of hypoperfusion. Various devices are used for this state to be monitored directly or indirectly, continuously and intermittently, so that the patient response to therapy can be determined and treatment adjustments made.

ETIOLOGY
There are three common types of shock: loss of blood volume (hypovolemic shock), abnormal blood distribution (e.g., septic shock), and cardiac pump failure (e.g., cardiogenic shock). In practice, these three types of shock do not occur independently, but are frequently mixed with a similar final pathway, irrespective of the circulatory or cardiac state that may have been the precipitant (1).

Heart
The critical event in the development of cardiogenic shock is severe impairment of heart muscle contractile performance. Cardiac performance is primarily determined by four factors: preload, afterload, strength of contraction, and heart rate. Preload is defined as the force exerted on
myocardium at the end of ventricular filling. If the filling is inadequate (low preload), cardiac output will be reduced. This finding is best exemplified by extracardiac obstruction, such as pericardial tamponade (blood in the pericardial sac that surrounds the heart) or by the reduction of venous return to the heart caused by high thoracic pressure (such as occurs with a pneumothorax, which is air under tension between the ribs and lung). Afterload is the resistance to emptying of the ventricles with myocardial contraction after the opening of the pulmonary and aortic valves. A rapid increase in afterload as in valvular stenosis leads to decreased volume of blood ejected from ventricles and decreased cardiac output. Contractility is most affected by loss of heart muscle as a result of myocardial infarction (heart attack). In the acute setting of myocardial ischemia, loss of at least 40% of left ventricular heart muscle results in severe depression of cardiac performance and shock.

A similar picture may also result from myocarditis (an inflammation of the heart muscle) and with prolonged cardiopulmonary bypass during cardiac surgery. In addition, myocardial stunning may occur following reversible myocardial ischemia. If the heart rate is too fast as in ventricular dysrhythmias, this may compromise ventricular filling and decrease cardiac output. If the rate is too slow, cardiac output may be insufficient, and shock may ensue. Both high and low heart rates are common complications of myocardial infarction (1,2,3).

**Loss of Volume**

Hypovolemic shock is caused by a reduction in intravascular circulating volume to a point where compensation is no longer possible, by constriction of venous capacitance vessels, to maintain cardiac filling. The loss of circulating volume may result from hemorrhage, dehydration, or leakage from the circulation into the body tissues. Trauma and gastrointestinal bleeding are common causes of rapid blood loss and lead to a reduction in preload and cardiac output. However, the oxygen \(O_2\) carrying capacity of blood is not severely impaired except in massive blood loss causing a decrease in hemoglobin concentration. Dehydration results in intravascular volume reduction with an increase in \(O_2\) carrying capacity, since the number of red blood cells per unit of volume will increase, but the cardiac output is decreased.

**Abnormal Distribution**

Maldistribution of blood flow occurs with widespread vasodilation usually caused by infectious agents (septic shock) but vasodilation with low systemic vascular resistance may be caused by other mechanisms including endocrine disease, anaphylaxis (severe systemic allergic reaction), and neurogenic shock.

**PATHOPHYSIOLOGY**

The underlying result of shock of any etiology is hypoperfusion at the cellular level due to an inability to provide the cell with adequate oxygen, glucose, and other nutrients necessary to maintain normal functions. Consumption is calculated by the Fick equation, where cardiac output = \(O_2\) consumption % (arterial-mixed venous \(O_2\) content). If the cardiac output is low, the tissue blood flow is reduced.

Hypoperfusion and hypovolemia due to blood loss decrease the blood pressure and cardiac output. With the loss of up to 15% of total blood volume, no detectable changes in heart rate and blood pressure may be found because venous capacitance vessel constriction compensates for hypovolemia and maintains cardiac filling pressures. If fluid loss is 15–30% of blood volume, that is, 750–1500 mL in a 70 kg male, heart rate will increase tending to maintain cardiac output. Pulse pressure (the difference
between systolic and diastolic blood pressure) decreases due to a rise in vascular resistance mediated by circulating catecholamines. The systemic vascular resistance (SVR) increases by constriction of arterioles, which tends to maintain an adequate arterial pressure. However, an increase in SVR raises arterial pressure, but unless it is accompanied by an increase in cardiac output, it has no effect on tissue blood flow. The relationship between flow and pressure is as follows: flow = pressure/resistance, and can be applied to the entire circulatory system, or to a single organ or even an electric circuit (Ohms law).

If the heart produces a constant flow per unit time (cardiac output), then the tissue blood flow (perfusion) will be inversely proportional to the vascular resistance. Therefore, in the case of shock, capillary perfusion should be globally reduced. However, this is not the case, as different organs have variable blood flow and oxygen utilization. The kidneys have high blood flow and little O₂ extraction, whereas the heart has relatively low blood flow (because it is contracting) and high O₂ extraction (Fig. 1) (4). In shock, vasoconstriction occurs preferentially in certain regions (skin, muscle, viscera), diverting the blood flow from these high vascular resistance organs to more vital organs with low resistance, such as heart and brain. If blood loss continues beyond 30–40% of total blood volume, the compensatory mechanisms fail and the blood pressure will fall, accompanied by marked tachycardia, changes in mental status, and decreased urinary output (1).

In the first stage of shock, arterioles are constricted and the amount of oxygen available to tissues may be insufficient for their metabolic needs. The global oxygen consumption will decrease after O₂ extraction from hemoglobin has been maximized (from a normal of 25% of the O₂ carriage to up to 70%). When O₂ consumption falls below a critical level, local metabolism becomes anaerobic, which leads to an increased production of lactic acid by the cells. Later on, this metabolic acidosis causes relaxation of the precapillary sphincters while the postcapillary venules are still constricted. Therefore, the capillaries become overfilled with blood and the hydrostatic pressure increases such that there will be loss of plasma into the interstitial space, further depleting the circulating volume.

Hemoconcentration occurs and the blood viscosity is increased, causing slowing of the blood flow through the microcirculation. A vicious circle is established, where slow flow causes an increase in viscosity, which in turn leads to a decrease in flow, and so on.

Venous oxygen saturations of blood leaving various organs are shown on the left side and blood flow expressed in mL/min and as a % of total cardiac output (Qt) are shown on the right side of this figure. Note that the heart and brain with low blood flow relative to their oxygen requirements (e.g., coronary blood flow and

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carotid artery) have low venous oxygen saturations. In comparison, an organ such as the kidney has high blood flow, but little oxygen requirement and contributes relatively more to the final mixed-venous $O_2$ saturation of 75% in the pulmonary artery than does the much smaller venous blood flow from the heart and brain. For this reason, a normal pulmonary artery oxygen saturation is not a good indicator of adequate shock resuscitation of the brain or heart.

In the latter stages, capillaries are filled with a sludge of red blood cells, which impairs the local flow. In addition, there is further reduction in tissue exchanges by shunting of blood through arteriovenous anastomoses so the functional flow is nearly zero. Therefore, cells lack $O_2$ and anaerobic metabolism will proceed to a point where cells are no longer able to survive. If a significant number of cells die, the organ function will be compromised. The lung is the first organ to fail, approximately 3–7 days after the primary shock event. The condition that results is called adult respiratory distress syndrome (ARDS) and is characterized by an increase in physiologic shunting and refractory hypoxemia. Kidney involvement is apparent within 2–5 days with acute renal failure due to ischemic tubular necrosis, followed by electrolyte disturbances and metabolic acidosis. Clearance of creatinine by the kidney approximates glomerular filtration rate and when this falls <25 mL/min, there is early onset of renal failure that is still potentially reversible (5). If liver failure occurs, the first sign is jaundice, but the most significant evidence of failure is abnormal hepatic metabolism, with impaired protein synthesis and an inability to process available energy substrates. Gastric hemorrhage may occur as a late manifestation and is usually precipitated by coagulopathy, a common event in shock. The immunologic response is depressed leading to an increased susceptibility to infections. The syndrome in which all these events occur—multiple organ system failure—is a terminal event common to all types of shock (3).

**SHOCK ASSESSMENT**

Assessment and management of a patient in shock are accomplished simultaneously. Since evaluation of cellular metabolism cannot be done directly, the physician must rely on surrogate clinical findings such as blood pressure (BP), heart rate, skin temperature, urinary output and mental status, and on data obtained by using various monitoring devices.

Measurement of BP is routine in shock patients. The systolic blood pressure measure (SBP) is not a good indicator of blood loss, as up to 30% of circulating volume may be lost without any change in SBP. Instead the diastolic blood pressure (DBP) is more sensitive and is usually elevated in shock due to peripheral vasoconstriction. Therefore, mean arterial pressure defined as MAP = DBP + 1/3 pulse pressure (SBP-DBP) seems to be more useful for BP monitoring. Blood pressure measured by auscultation is inaccurate in patients with low peripheral blood flow. Invasive measurement using an intra-arterial catheter inserted into radial, brachial, axillary, or femoral arteries is more precise and provides continuous monitoring and easy access for blood gas and pH analysis.

Change in heart rate occurs with increasing blood loss, values > 100/min in adults are detectable before any change in SBP. Pulse pressure is usually low due to increased DBP. Another clinical monitor is capillary refill time, which is the time for return of blood flow after compression of the nail beds until blanching occurs. It is prolonged > 2 s in severe peripheral vasoconstriction. Skin temperature correlates well with peripheral blood flow and a difference of 3–6 °C between toe and rectal temperature reflects severe peripheral vasoconstriction (2). The ability of commonly used clinical parameters to quantify acute hemorrhage is shown in Fig. 2 (6). Base deficit, mean arterial pressure, serial hemoglobin, and serum lactate are related to blood loss.
Preload is assessed by measuring central venous pressure (CVP), which correlates with right atrial pressure, with a catheter inserted in the superior vena cava via the jugular or subclavian veins. Normal CVP is 5–12 cm of H\textsubscript{2}O and values <5 are generally found in hypovolemic states and indicate the need for assessment of reserve cardiac function by rapid fluid administration together with assessment of changes in CVP. However, CVP does not always correlate with fluid requirement because an increase in pulmonary vascular resistance (hypoxia, acidosis, increased intrathoracic pressure) may be associated with high CVP, reflecting right ventricular failure, even when there is considerable blood loss.

Commonly used clinical and laboratory values compared to quantity of acute hemorrhage. Graphs show predicted versus actual blood volume reductions for six representative parameters. Solid black lines represent an individual animal (n = 10). Gray bar represents the ideal in which predictions equal actual blood volume reductions. For mean arterial pressure, predictions at large volume hemorrhage were more accurate than predictions with small volume bleeds. Models such as heart rate and lactate both showed significance variability before hemorrhage among animals and flat slopes (e.g., mixed-venous \textit{PCO}_2) indicated fixed-volume predictions regardless of actual degree of hemorrhage. (Reproduced with permission from Waisman Y et al. J. Appl. Physiol. 1993; 74: 410–519.)

The possible responses to rapid (50 mL/min for 5 min) administration of a fluid is shown schematically. The vertical axis shows the change in cardiac filling pressure (could be either CVP or PAQP) in response to fluid. Time is on the horizontal axis. Fluid infusion ceases after 5 min and the filling pressures are remeasured (and cardiac output measured if available). Fluid challenge in outcomes 3A and 3B should be repeated until filling pressures increase and remain elevated for 5 min after ceasing fluid infusion (outcome No. 2). (Reproduced with
**Fluid Challenge**

Reserve cardiac function is assessed by means of a fluid challenge until CVP pressures are elevated at least 2 mmHg above the baseline for 10 min after fluid infusion ceases. There are four possible outcomes when 250 mL boluses of fluid are given over 5 min (Fig. 3). Outcome No. 1: Filling pressures rise with the challenge and continue to rise even after fluid infusion ceases. If cardiac output is measured (see below), there is no increase with elevation of filling pressures and the heart has limited reserve function and is not able to deal with the increased fluid load by increasing contractility by the Frank–Starling mechanism (this mechanism describes the property of heart muscle increasing its contraction in proportion to fiber length, up to a maximum point when contractility decreases and cardiac failure occurs). Further fluid infusion (when this response occurs) is expected to produce cardiac failure. The therapeutic indication this response dictates is to restrict fluid infusion and reduce myocardial depressant agents. If the trend continues, inotropic agents are required to increase cardiac contractility and reverse myocardial depression. Outcome No. 2: Central venous pressure or pulmonary capillary wedge pressure (PCWP) rise 3–4 mmHg, but then falls to a level 2 mmHg above baseline, indicating myocardial contractility is adequate for the increase in cardiac preload. Management should be to infuse fluids and maintain cardiac filling pressures within this range for optimum cardiac output and oxygen transport for the prevailing vascular tone. In outcome No. 3, filling pressures either rise briefly (3A) or do not rise at all (3B) with the 250 mL fluid challenge indicating that the patient has considerable reserve cardiac function and a greater fluid load could be tolerated. If the patient has low urine flow, has evidence of poor tissue perfusion, such as acidosis or low mixed venous oxygen tension, inotropic agents, or diuretics should not be given. Rather, fluid infusion should continue at the same rate until outcome No. 2 is obtained. In fact, because filling pressures have not increased in outcome No. 3, it is unlikely that myocardial fiber length would increase and, therefore, cardiac contractility would not have changed by the Frank–Starling mechanism. Outcome No. 4 is seen in ~5% of cases and results in a fall in filling pressures when fluid is infused rapidly. The two most likely explanations are either that rapid fluid infusion has a vasodilator effect on peripheral vasculature and reduces left ventricular afterload, increasing cardiac output, or alternatively, it may be related to a reduction in heart rate seen when fluid is infused in the hypovolemic patient. The decreased heart rate allows more time for myocardial perfusion, which occurs mostly during diastole or cardiac relaxation. Cardiac output increases and the Frank–Starling function curve shifts to the left due to improve myocardial oxygenation (7).

**Cardiac Catheterization**

Catheterization of pulmonary artery (PA) is a method of hemodynamic monitoring that can be used to assess right and left ventricular function as well as quantitate the proportion of blood shunting and to calculate tissue delivery and O₂ extraction. Catheterization is usually reserved for chronically ill patients with heart disease or shock refractory to conventional therapy, because it requires an invasive flow directed, balloon tipped catheter to be floated in the blood stream through the right side of the heart into the pulmonary artery. Such heart catheterization has complications including infection and cardiac rhythm irregularities. It provides data on PA pressure, PCWP, vascular resistances, cardiac output (commonly by a thermodilution technique), and also allows blood sampling from PA, for measurement of mixed-venous saturation (SvO₂) and calculation of intrapulmonary shunting of blood.

**Equations**

Normal SvO₂ is ~75% (Fig. 1), and it reflects the ratio between oxygen delivery [arterial O₂ content
(CaO₂ × cardiac output (Qt)) and oxygen extraction [ratio of O₂ consumption (VO₂) and O₂ delivery]. Consumption is calculated by the Fick equation, where cardiac output = O₂ consumption % (arterial mixed-venous O₂ content). If the cardiac output is low, the tissue blood flow is reduced.

Blood gas and pH analysis give a rough estimate of oxygen utilization and cellular metabolism by calculation of bicarbonate and base deficit. A low arterial pO₂ (hypoxemia) may accompany shock and be found before any clinical sign due to ventilation–perfusion inequality and increased venous admixture by shunting (see above). The causes of hypoxemia include hypoxic hypoxemia (low inspired O₂), anemic hypoxemia (low hemoglobin for carrying O₂) stagnant hypoxemia (low cardiac output for delivery of O₂), and histotoxic hypoxemia (poisoning of the enzyme systems used to offload O₂ from hemoglobin at the tissues). Blood lactate is an indicator of tissue hypoperfusion and anaerobic metabolism and is elevated in patients with low cardiac output. Lactate is a clinically useful marker of the amount and duration of shock. Sustained reduction in elevated lactate is an important clinical marker of recovery. Arterial pH can be high or normal despite metabolic acidosis, because of low pCO₂ due to increased respiratory rate and alveolar ventilation, common in patients with low cardiac output.

Real-Time Noninvasive Measures of Systemic Perfusion

Sublingual capnometry (measurement of sublingual pCO₂-PSICO₂) is a new technique for assessment of systemic perfusion failure. It is based on elevated pCO₂ in tissues with low blood flow due to intracellular buffering of hydrogen ions by bicarbonate. Elevated PSICO₂ correlates well with increased blood lactate and low mean arterial pressure (MAP), markers of tissue hypoperfusion. PSICO₂ has the advantage of prompt indication and continuous monitoring of tissue flow reversal, unlike lactate whose clearance presents significant delay. A threshold value of 70 mmHg for PSICO₂ has been identified that is predictive of both the severity state and survival. A PSICO₂ > 70 mmHg is highly predictive of circulatory failure whereas readings <70 mmHg are highly predictive of survival (9). A similar technique to sublingual capnometry is gastrointestinal tonometry, which measures gut mucosal pCO₂. Calculation of intramucosal gut pH is possible (pHi) using the Henderson–Hasselbach equation: pHi = 6.1 + log (HCO₃⁻/α* tonometer pCO₂), where α is the solubility of CO₂ in plasma (α = 0.03) Values of pHi <7.32 define mucosal hypoperfusion and are associated with high mortality rates (10).

Brain Perfusion

Brain perfusion monitoring is technically difficult and inaccurate. Clinically, signs of confusion, altered sensorium, agitation, and decreased consciousness give a rough idea about cerebral hypoperfusion. Jugular venous oxygen saturation (SjvO₂), transcranial cerebral oximetry, and brain tissue oxygen tension (PbtO₂) monitoring are the methods of monitoring brain oxygenation. Measurement of SjvO₂ is performed using a catheter inserted in the jugular bulb, the upper part of the internal jugular vein. Continuous monitoring of venous saturation without blood sampling is possible by using intravenous oximetry. This type of device has been used in patients with head injury and during anesthesia for neurosurgery. It provides only global brain oxygenation monitoring and is susceptible to errors. Cerebral oximeters using near-infrared spectroscopy seem to be a promising alternative. They can evaluate regional ischemia, hemoglobin saturation, and even concentration of oxygenated and reduced hemoglobin. However, these monitors can only assess trends, where each patient is their own control. There are no normative data for comparison and the boundaries of monitored brain tissue cannot be precisely defined. Brain tissue oxygen tension is measured with small catheters inserted directly into the brain tissue during a craniotomy or via a burr hole. These catheters measure pO₂, pCO₂, pH, and temperature. Some studies suggest a normal value for PbtO₂ of ~35 mmHg, whereas cerebral ischemia...
is usually defined as a $P_{btO_2} < 8 \text{ mmHg}$. These data were obtained in patients with traumatic brain injury, but their usefulness should be confirmed by further studies (11,12). Extra cellular glutamate and aspartate measures (obtained by microdialysis) are closely related to outcome after head injury (13). These markers were also related to the type of head injury and suggest that excitatory amino acids play a role in the evolution of brain injury.

**SHOCK MANAGEMENT**

Treatment of shock should be directed to its underlying cause. However, establishing an exact diagnosis can be time consuming, so management is focused on simultaneously stabilizing the patient and proceeding with diagnostic tests to identify the cause of shock.

For cardiogenic shock, the goal is an increase in cardiac output by acting to change preload, afterload, contractility, or heart rate. Therapy is tailored using information provided by a PA catheter. Various drugs are given to increase contractility and to relieve pulmonary congestion. In unresponsive cases, additional measures may be considered, such as urgent myocardial revascularization in acute myocardial infarction, intraaortic balloon counterpulsation, and anatomic defects repair (ruptured valves). In extracardiac compression, relieving the pressure of pericardial tamponade by pericardial puncture or insertion of a chest tube for increased intrathoracic pressure due to pneumothorax is the treatment of choice, when these are the causes of impaired cardiac filling and decreased cardiac output.

In case of septic shock (the most common form of distributive shock), large quantities of fluids are administered to fill the vascular bed and maintain perfusion pressure. Cardioactive drugs are used only if cardiac output declines. At the same time steps are taken to identify and control the source of infection (3).

Hypovolemic shock requires initial rapid expansion of circulating volume. Fluid resuscitation is initiated with administration of crystalloid or colloid solutions through large-bore intravenous lines. Rapid infusion devices can be used to pump large amounts of fluids in <10 min. A potential adverse effect resulting from resuscitation with large amounts of fluid, when using rapid infusion devices, is a drop in body core temperature. Levels <35 °C are associated with impaired coagulation and depressed cardiac contractility (14). Covering the patient with inflatable warming blankets can prevent this complication, but the most effective method for rewarming is an extracorporeal countercurrent warmer through femoral artery and vein cannulation, which can elevate temperature ~6 °C in <30 min. During fluid infusion, hemodynamic parameters are continuously monitored and signs of instability (persistent SBP <90 mmHg) imply there is ongoing blood loss or shock has not been reversed. Classically, a hemoglobin (Hb) level <10 g/dL with continuous loss requires blood transfusion, but recent studies have demonstrated that this level can be as low as 7 g/dL without adverse effects in the majority of the population (15). Those with cardiac or cerebrovascular disease should be transfused at higher hemoglobin concentrations.

**THE FUTURE OF SHOCK DIAGNOSIS AND MANAGEMENT**

Future trends in shock diagnosis and management include identification of mediator’s released in shock states and blockage of the release of harmful mediator factors while facilitating release of those with benefits. The field of proteomics, defining protein expression with shock, will provide many future treatment and diagnostic opportunities. Genomics may identify some individuals or disease states susceptible to adverse outcomes from shock. These future findings could lead to improved outcome,
particularly from septic shock, which has a high mortality and morbidity.

See also Blood Pressure Measurement; Cardiopulmonary Resuscitation; Peripheral Vascular Noninvasive Measurements.

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