to deficient clotting factors in crystalloids and banked packed red blood cells (PRBCs). Early administration of component therapy during massive transfusion (fresh-frozen plasma [FFP] and platelets) approaching a 1:1 ratio of PRBC/FFP appears to improve survival. In extreme emergencies, type-specific or O-negative packed red cells may be transfused. Following severe and/or prolonged hypovolemia, inotropic support with norepinephrine, vasopressin, or dopamine may be required to maintain adequate ventricular performance *but only after* blood volume has been restored. Increases in peripheral vasoconstriction with inadequate resuscitation lead to tissue loss and organ failure. Once hemorrhage is controlled and the patient has stabilized, blood transfusions should not be continued unless the hemoglobin is <~7 g/dL. Studies have demonstrated an increased survival in patients treated with this restrictive blood transfusion protocol.

Successful resuscitation also requires support of respiratory function. Supplemental oxygen should always be provided, and endotracheal intubation may be necessary to maintain arterial oxygenation. Following resuscitation from isolated hemorrhagic shock, end-organ damage is frequently less than following septic or traumatic shock. This may be due to the absence of massive activation of the inflammatory innate immune response and consequent nonspecific organ injury and failure.

TRAUMATIC SHOCK

Shock following trauma is, in large measure, due to hemorrhage. However, even when hemorrhage has been controlled, patients can continue to suffer loss of plasma volume into the interstitium of injured tissues. These fluid losses are compounded by injury-induced inflammatory responses, which contribute to the secondary microcirculatory injury. Proinflammatory mediators are induced by DAMPs released from injured tissue and are recognized by the highly conserved membrane receptors of the TLR family (see "Inflammatory Responses" above). These receptors on cells of the innate immune system, particularly the circulating monocyte, tissue-fixed macrophage, and dendritic cell, are potent activators of an excessive proinflammatory phenotype in response to cellular injury. This causes secondary tissue injury and maldistribution of blood flow, intensifying tissue ischemia and leading to multiple organ system failure. In addition, direct structural injury to the heart, chest, or head can also contribute to shock. For example, pericardial tamponade or tension pneumothorax impairs ventricular filling, whereas myocardial contusion depresses myocardial contractility.

TREATMENT TRAUMATIC SHOCK

Inability of the patient to maintain a systolic blood pressure ≥90 mmHg after trauma-induced hypovolemia is associated with a mortality rate up to ~50%. To prevent this decompensation of homeostatic mechanisms, therapy must be promptly administered.

The initial management of the seriously injured patient requires attention to the "ABCs" of resuscitation: assurance of an *airway* (A), adequate ventilation (*breathing*, B), and establishment of an adequate blood volume to support the *circulation* (C). Control of ongoing hemorrhage requires immediate attention. Early stabilization of fractures, debridement of devitalized or contaminated tissues, and evacuation of hematomata all reduce the subsequent inflammatory response to the initial insult and minimize damaged tissue release of DAMPs and subsequent diffuse organ injury. Supplementation of depleted endogenous antioxidants also reduces subsequent organ failure and mortality.

CARDIOGENIC SHOCK

See Chap. 326.

COMPRESSIVE CARDIOGENIC SHOCK

With extrinsic compression, the heart and surrounding structures are less compliant, and therefore, normal filling pressures generate inadequate diastolic filling and stroke volume. Blood or fluid within the poorly distensible pericardial sac may cause tamponade (Chap. 288). Any cause of increased intrathoracic pressure, such as tension pneumothorax, herniation of abdominal viscera through a diaphragmatic hernia, or excessive positive-pressure ventilation to support pulmonary function, can also initiate compressive cardiogenic shock while simultaneously impeding venous return and preload. Although initially responsive to increased filling pressures produced by volume expansion, as compression increases, cardiogenic shock recurs. The window of opportunity gained by volume loading may be very brief until irreversible shock recurs. Diagnosis and intervention must occur urgently.

The diagnosis of compressive cardiogenic shock is most frequently based on clinical findings, the chest radiograph, and an echocardiogram. The diagnosis of compressive cardiac shock may be more difficult to establish in the setting of trauma when hypovolemia and cardiac compression are present simultaneously. The classic findings of pericardial tamponade include the triad of hypotension, neck vein distention, and muffled heart sounds (Chap. 288). Pulsus paradoxus (i.e., an inspiratory reduction in systolic pressure >10 mmHg) may also be noted. The diagnosis is confirmed by echocardiography, and treatment consists of immediate pericardiocentesis or the creation of an open subxiphoid pericardial window. A tension pneumothorax produces ipsilateral decreased breath sounds, tracheal deviation away from the affected thorax, and jugular venous distention. Radiographic findings include increased intrathoracic volume, depression of the diaphragm of the affected hemithorax, and shifting of the mediastinum to the contralateral side. Chest decompression must be carried out immediately and, ideally, should occur based on clinical findings rather than awaiting a chest radiograph. Release of air and restoration of normal cardiovascular dynamics are both diagnostic and therapeutic.

SEPTIC SHOCK

See Chap. 325.

NEUROGENIC SHOCK

Interruption of sympathetic vasomotor input after a high cervical spinal cord injury, inadvertent cephalad migration of spinal anesthesia, or devastating head injury may result in neurogenic shock. In addition to arteriolar dilation, venodilation causes pooling in the venous system, which decreases venous return and cardiac output. The extremities are often warm, in contrast to the usual sympathetic vasoconstriction-induced coolness in hypovolemic or cardiogenic shock. Treatment involves a simultaneous approach to the relative hypovolemia and to the loss of vasomotor tone. Excessive volumes of fluid may be required to restore normal hemodynamics if given alone. Once hemorrhage has been ruled out, norepinephrine or a pure α-adrenergic agent (phenylephrine) may be necessary to augment vascular resistance and maintain an adequate MAP.

HYPOADRENAL SHOCK

(See also Chap. 406) The normal host response to the stress of illness, operation, or trauma requires that the adrenal glands hypersecrete cortisol in excess of that normally required. Hypoadrenal shock occurs in settings in which unrecognized adrenal insufficiency complicates the host response to the stress induced by acute illness or major surgery. Adrenocortical insufficiency may occur as a consequence of the chronic administration of high doses of exogenous glucocorticoids. In addition, recent studies have shown that critical illness, including trauma and sepsis, may also induce a relative hypoadrenal state. Other, less common causes include adrenal insufficiency secondary to idiopathic atrophy, use of etomidate for intubation, tuberculosis, metastatic disease, bilateral hemorrhage, and amyloidosis. The shock produced by adrenal insufficiency is characterized by loss of homeostasis with reductions in systemic vascular resistance, hypovolemia, and reduced cardiac output. The diagnosis of adrenal insufficiency may be established by means of an ACTH stimulation test.