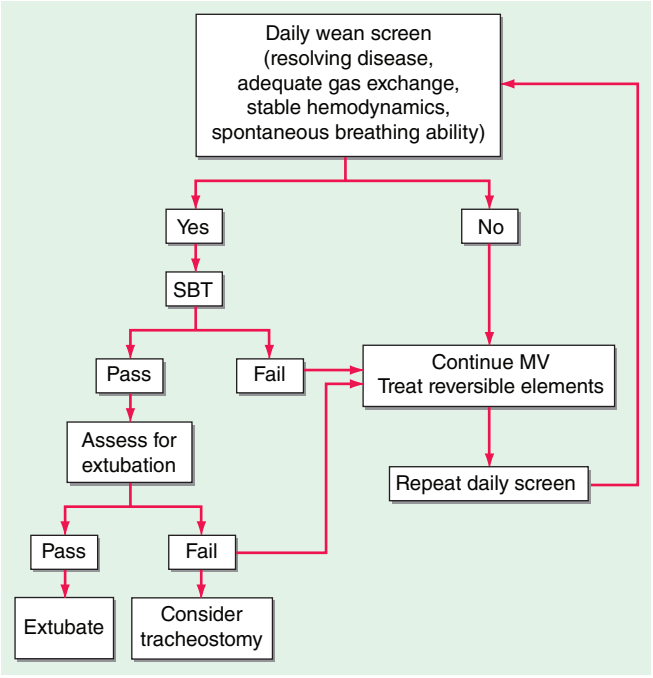


**FIGURE 323-2 Flow chart to guide the daily approach to management of patients being considered for weaning off mechanical ventilation (MV).** If attempts at extubation fail, a tracheostomy should be considered. SBT, spontaneous breathing trial.

secretions, and is alert enough to follow commands. In addition, other factors must be taken into account, such as the possible difficulty of replacing the tube if that maneuver is required. If upper airway difficulty is suspected, an evaluation using a "cuff-leak" test (assessing the presence of air movement around a deflated endotracheal tube cuff) is supported by some internists. Despite all precautions, ~10–15% of extubated patients require reintubation. Several studies suggest that NIV can be used to obviate reintubation, particularly in patients with ventilatory failure secondary to COPD exacerbation; in this setting, earlier extubation with the use of prophylactic NIV has yielded good results. The use of NIV to facilitate weaning in respiratory failure of other etiologies is not currently indicated.

**Prolonged Mechanical Ventilation and Tracheostomy** From 5% to 13% of patients undergoing MV will go on to require prolonged MV (>21 days). In these instances, critical care personnel must decide whether and when to perform a tracheostomy. This decision is individualized and is based on the risk and benefits of tracheostomy and prolonged intubation as well as the patient's preferences and expected outcomes. A tracheostomy is thought to be more comfortable, to require less sedation, and to provide a more secure airway and may also reduce weaning time. However, tracheostomy carries the risk of complications, which occur in 5–40% of these procedures and include bleeding, cardiopulmonary arrest, hypoxia, structural damage, pneumothorax, pneumomediastinum, and wound infection. In patients with long-term tracheostomy, complex complications include tracheal stenosis, granulation, and erosion of the innominate artery. In general, if a patient needs MV for more than 10–14 days, a tracheostomy, planned under optimal conditions, is indicated. Whether it is completed at the bedside or as an operative procedure depends on local resources and experience. Some 5–10% of patients are deemed unable to wean in the ICU. These patients may benefit from transfer to special units where a multidisciplinary approach, including nutrition optimization, physical therapy with rehabilitation, and slower weaning methods (including SIMV with PSV), results in successful weaning rates of up to 30%. Unfortunately, close to 2% of ventilated patients may ultimately become dependent on ventilatory support to maintain life. Most of these patients remain in chronic care institutions, although some with strong social, economic, and family support may live a relatively fulfilling life with at-home ventilation.



# SECTION 2 SHOCK AND CARDIAC ARREST

## 324 Approach to the Patient with Shock

Ronald V. Maier

*Shock* is the clinical syndrome that results from inadequate tissue perfusion. Irrespective of cause, the hypoperfusion-induced imbalance between the delivery of and requirements for oxygen and substrate leads to cellular dysfunction. The cellular injury created by the inadequate delivery of oxygen and substrates also induces the production and release of damage-associated molecular patterns (DAMPs or "danger signals") and inflammatory mediators that further compromise perfusion through functional and structural changes within the microvasculature. This leads to a vicious cycle in which impaired perfusion is responsible for cellular injury that causes maldistribution of blood flow, further compromising cellular perfusion; the latter ultimately causes multiple organ failure (MOF) and, if the process is not interrupted, leads to death. The clinical manifestations of shock are also the result, in part, of autonomic neuroendocrine responses to hypoperfusion as well as the breakdown in organ function induced by severe cellular dysfunction (Fig. 324-1).

When very severe and/or persistent, inadequate oxygen delivery leads to irreversible cell injury, only rapid restoration of oxygen delivery can reverse the progression of the shock state. The fundamental approach to management, therefore, is to recognize overt and impending shock in a timely fashion and to intervene emergently to restore perfusion. Doing so often requires the expansion or reexpansion of intravascular blood volume. Control of any inciting pathologic process (e.g., continued hemorrhage, impairment of cardiac function, or infection) must occur simultaneously.

Clinical shock is usually accompanied by hypotension (i.e., a mean arterial pressure [MAP] <60 mmHg in previously normotensive persons). Multiple classification schemes have been developed in an attempt to synthesize the seemingly dissimilar processes leading to shock. Strict adherence to a classification scheme may be difficult from a clinical standpoint because of the frequent combination of two or more causes of shock in any individual patient, but the classification shown in Table 324-1 provides a useful reference point from which to discuss and further delineate the underlying processes.

### PATHOGENESIS AND ORGAN RESPONSE

#### MICROCIRCULATION

Normally when cardiac output falls, systemic vascular resistance rises to maintain a level of systemic pressure that is adequate for perfusion of the heart and brain at the expense of other tissues such as muscle, skin, and especially the gastrointestinal (GI) tract. Systemic vascular resistance is determined primarily by the luminal diameter of arterioles. The metabolic rates of the heart and brain are high, and their stores of energy substrate are low. These organs are critically dependent on a