Critical Care: A System-Oriented Approach

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Neurological Dysfunction

Etiology and Diagnosis

Brain dysfunction presents as an alteration in mental function (altered mental status) that spans the spectrum from delirium to coma. The most common manifestations are disorientation and agitation, characterized by disorganized thinking, deficits in short-term memory, and auditory hallucinations. The etiology is usually listed as multifactorial [potential contributing factors are listed in Table 10.1]. Establishing a diagnosis is difficult (often made by exclusion) and requires a careful history and focused neurological examination.

Computed tomography of the head to establish an anatomical abnormality is indicated in patients with focal neurological findings, in patients at risk for stroke or intracranial hemorrhage, or in those with a history of trauma. Lumbar puncture to obtain cerebral spinal fluid should be reserved for patients with a history and physical examination suggestive of meningitis.

Treatment

It is helpful to objectively document neurological status with a simple scoring system. One example is the modified Ramsay sedation scale (Table 10.2). Regular use of this scale minimizes interobserver variance and facilitates communication regarding neurological status and titration of sedatives.

The treatment of brain dysfunction is supportive. Anxiolytics, typically benzodiazepines given intravenously, are used to treat agitation. In a randomized, prospective multicenter study comparing intermittent i.v. lorazepam to continuous i.v. midazolam, the two agents were found to be equally safe and effective and without significant differences in their effects on hemodynamic profiles. A prospective, randomized, multicenter trial found that an infusion of continuous i.v. propofol was equally safe and effective but had a significantly shorter duration of action when compared to continuous i.v. midazolam. As neither benzodiazepines nor propofol offer analgesia, the additional use of a narcotic (morphine or fentanyl) is particularly helpful in those patients experiencing postoperative pain or discomfort from invasive devices.

Another type of agent used to treat agitation is i.v. haloperidol, especially in combination with benzodiazepines, for the treatment of delirium, hypervigilance, and paranoia ("ICU psychosis").

Prognosis

The prognosis for brain dysfunction in the critically ill is usually excellent. In the absence of acquired anatomical abnormalities (e.g., stroke or trauma), the mental status changes associated with critical illness almost always improve as MODS resolves and neuroactive drugs are discontinued.

Cardiovascular Dysfunction

Etiology and Diagnosis

Failure of the cardiovascular system presents as hypotension, variably defined. Widely accepted criteria include systolic blood pressure (SBP) less than 90 mmHg or mean arterial blood pressure (MAP) less than 60 mmHg. The pathophysiology of hypotension can be easily understood by analyzing the determinants of blood pressure. If Ohm’s law, pressure = flow × resistance, is applied to blood flow, one approximation is

\[
\text{MAP} = \text{HR} \times \text{SV} \times \text{SVR}
\]
Cerebrovascular event (stroke)
Catecholamine excess associated with the neurohumoral response
Ethanol or drug (benzodiazepine) withdrawal
Septic encephalopathy
Neurotoxin accumulation secondary to renal dysfunction

Asleep, unresponsive to loud auditory stimulus, glabellar tap, or prodding
UTO Chemically paralyzed

4 Asleep or drowsy, responds to loud auditory stimulus or light glabellar tap
3 Awake, responds to commands only
2 Awake, cooperative, oriented, and tranquil
1 Awake, anxious, agitated, or restless

Level Findings

<table>
<thead>
<tr>
<th>Level</th>
<th>Findings</th>
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<tbody>
<tr>
<td>1</td>
<td>Awake, anxious, agitated, or restless</td>
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<tr>
<td>2</td>
<td>Awake, cooperative, oriented, and tranquil</td>
</tr>
<tr>
<td>3</td>
<td>Awake, responds to commands only</td>
</tr>
<tr>
<td>4</td>
<td>Asleep or drowsy, responds to loud auditory stimulus or light glabellar tap</td>
</tr>
<tr>
<td>5</td>
<td>Asleep, unresponsive to loud auditory stimulus or glabellar tap, but responds when prodded</td>
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<tr>
<td>6</td>
<td>Asleep, unresponsive to loud auditory stimulus, glabellar tap, or prodding</td>
</tr>
<tr>
<td>UTO</td>
<td>Chemically paralyzed</td>
</tr>
</tbody>
</table>

As SV is determined by myocardial preload, afterload, and contractility, then

\[
\text{MAP} = \frac{\text{HR} \times \text{preload} \times \text{afterload} \times \text{contractility}}{100}
\]

In other words, MAP is proportional to heart rate and some measure of myocardial preload, afterload, and contractility. The conclusion is that hypotension can only result from a decrease in one of these four determinants: heart rate, preload, afterload, or contractility.

The clinical (bedside) assessment of patients who present with shock is notoriously unreliable. Of the four determinants of blood pressure—preload, afterload, contractility, and heart rate—only the latter can be determined at the bedside with a reliable degree of accuracy.

Because of ambiguities and the critically ill, unstable nature of patients in shock, hemodynamic data obtained from invasive indwelling monitors can be particularly useful, both diagnostically and therapeutically (Chapter 17). Indwelling arterial catheters permit continuous real-time measurement of systemic arterial pressures and easy access to the arterial circulation for blood gas measurements. Venous catheters placed in a central vein permit assessment of central venous and right atrial pressures, two measures of right-ventricular preload. The balloon-tipped, thermodilution pulmonary artery (PA) catheter provides access to mixed venous blood, permits an estimation of left ventricular preload in the “wedged” position, and provides data allowing the clinician to estimate right- and left-ventricular contractility (cardiac index, stroke volume, right ventricular ejection fraction). Cardiac rhythm, conduction, and perfusion abnormalities are assessed routinely on a 12-lead ECG tracing. Transthoracic and, more recently, transesophageal echocardiography can be used to determine valvular and regional myocardial function and an estimation of filling pressures in patients with evidence of primary heart dysfunction or endocarditis.

**Treatment**

The goals of pharmacological therapy for shock mirror those of the endogenous response to stress, that is, optimization of myocardial preload, contractility, afterload, and heart rate.

As the most frequent etiology of shock in the perioperative period is hypovolemic or septic shock, administration of i.v. fluids to increase preload is a safe, logical first step. The goal is to “load” the ventricles to optimize stroke volume and thereby cardiac output (Starling relationship); this can be attained typically by increasing filling pressures to 12 to 18 mmHg. The usual initial bolus dose of i.v. fluids (IVF) is 10 to 20 ml/kg. Both crystalloid and colloid (hetastarch, albumin) solutions are effective, but balanced salt solutions are the most cost-effective initial choice because of their low cost and physiological concentrations of salts [large volumes can be infused with minimal changes in serum electrolytes].

Infusion of blood products is reserved for specific therapy of anemia or coagulopathy (see Chapter 10).

Once preload has been optimized, distributive forms of shock are treated with agents that increase afterload [Table 10.3]. Doses of the alpha-adrenergic receptor agonists noradrenaline, epinephrine, phenylephrine, or dopamine are titrated to maintain blood pressure at an arbitrary level, typically a mean arterial pressure (MAP) of 60 to 65 mmHg.

For shock secondary to myocardial failure, a number of pharmacological agents are available to increase cardiac contractility [see Table 10.3]. The drugs used most commonly are dobutamine, dopamine, and phosphodiesterase inhibitors such as amrinone.

A heart rate of approximately 90 bpm is regarded as optimal in patients with shock. Lower heart rates decrease oxygen demand (thereby protecting the myocardium) but do so at the expense of cardiac output. Dopamine and dobutamine can be titrated to increase heart rate and cardiac index; atropine and isoproterenol are used in the setting of bradycardia emergencies. When preload is adequate, heart rates higher than 90 bpm may increase cardiac index but also increase oxygen demand and the possibility of myocardial ischemia. Heart rates greater than approximately 140 bpm in adults are associated with inadequate diastolic filling and resultant decreased cardiac index.

Historically, resuscitation endpoints have include normalization of vital signs and indicators of organ perfusion [resolution of oliguria, hyperlactatemia, and acidemia]. Other endpoints such as supranormal oxygen activity, subcutaneous tissue oximetry, and gastric tonometry remain controversial or investigational.

**Prognosis**

Shock, especially refractory shock requiring vasopressor therapy, is a robust predictor of increased mortality. Persistent hyperlactatemia is also accurate, suggesting that prolonged tissue hypoperfusion may play a role in determining outcome.