**TABLE 330-1 NEUROLOGIC DISORDERS IN CRITICAL ILLNESS**

| Localization Along Neuroaxis | Syndrome |
|---|---|
| **Central Nervous System** | |
| Brain: Cerebral hemispheres | Global encephalopathy |
| | Delirium |
| | Sepsis |
| | Organ failure—hepatic, renal |
| | Medication related—sedatives, hypnotics, analgesics, $H_2$ blockers, antihypertensives |
| | Drug overdose |
| | Electrolyte disturbance—hyponatremia, hypoglycemia |
| | Hypotension/hypoperfusion |
| | Hypoxia |
| | Meningitis |
| | Subarachnoid hemorrhage |
| | Wernicke's disease |
| | Seizure—postictal or nonconvulsive status |
| | Hypertensive encephalopathy |
| | Hypothyroidism—myxedema |
| | Focal deficits |
| | Ischemic stroke |
| | Tumor |
| | Abscess, subdural empyema |
| | Intraparenchymal hemorrhage |
| | Subdural/epidural hematoma |
| Brainstem/cerebellum | Mass effect and compression |
| | Basilar artery thrombosis |
| | Intraparenchymal hemorrhage |
| | Central pontine myelinolysis |
| Spinal cord | Mass effect and compression |
| | Disk herniation |
| | Epidural hematoma |
| | Ischemia—hypotension/embolic |
| | Epidural abscess |
| | Trauma |
| | Myelitis |
| **Peripheral Nervous System** | |
| Peripheral nerve | |
| Axonal | Critical illness polyneuropathy |
| | Neuromuscular blocking agent complications |
| | Metabolic disturbances, uremia, hyperglycemia |
| | Medication effects—chemotherapeutic, antiretroviral |
| Demyelinating | Guillain-Barré syndrome |
| | Chronic inflammatory demyelinating polyneuropathy |
| Neuromuscular junction | Prolonged effect of neuromuscular blockade |
| | Medication effects—aminoglycosides |
| | Myasthenia gravis, Lambert-Eaton syndrome, botulism |
| Muscle | Critical illness myopathy |
| | Cachectic myopathy |
| | Acute necrotizing myopathy |
| | Thick-filament myopathy |
| | Electrolyte disturbances—hypokalemia/hyperkalemia, hypophosphatemia |
| | Rhabdomyolysis |



cell death occurs without cerebral edema and therefore is often not seen on brain imaging. At present, interventions for prevention and treatment of apoptotic cell death remain less well defined than those for ischemia. **Excitotoxicity and mechanisms of cell death are discussed in more detail in Chap. 444e.**

**Cerebral Perfusion and Autoregulation** Brain tissue requires constant perfusion in order to ensure adequate delivery of substrate. The hemodynamic response of the brain has the capacity to preserve perfusion across a wide range of systemic blood pressures. Cerebral perfusion pressure (CPP), defined as the mean systemic arterial pressure (MAP) minus the ICP, provides the driving force for circulation across the capillary beds of the brain. *Autoregulation* refers to the physiologic response whereby cerebral blood flow (CBF) is regulated via alterations in cerebrovascular resistance in order to maintain perfusion over wide physiologic changes such as neuronal activation or changes in hemodynamic function. If systemic blood pressure drops, cerebral perfusion is preserved through vasodilation of arterioles in the brain; likewise, arteriolar vasoconstriction occurs at high systemic pressures to prevent hyperperfusion, resulting in fairly constant perfusion across a wide range of systemic blood pressures **(Fig. 330-1)**. At the extreme limits of MAP or CPP (high or low), flow becomes directly related to perfusion pressure. These autoregulatory changes occur in the microcirculation and are mediated by vessels below the resolution of those seen on angiography. CBF is also strongly influenced by pH and $Paco_2$. CBF increases with hypercapnia and acidosis and decreases with hypocapnia and alkalosis because of pH related changes in cerebral vascular resistance. This forms the basis for the use of hyperventilation to lower ICP, and this effect on ICP is mediated through a decrease in both CBF and intracranial blood volume. Cerebral autoregulation is a complex process critical to the normal homeostatic functioning of the brain, and this process may be disordered focally and unpredictably in disease states such as traumatic brain injury and severe focal cerebral ischemia.

**Cerebrospinal Fluid and Intracranial Pressure** The cranial contents consist essentially of brain, cerebrospinal fluid (CSF), and blood. CSF is produced principally in the choroid plexus of each lateral ventricle, exits the brain via the foramens of Luschka and Magendie, and flows over the cortex to be absorbed into the venous system along the superior sagittal sinus. In adults, approximately 150 mL of CSF are contained within the ventricles and surrounding the brain and spinal cord; the cerebral blood volume is also ~150 mL. The bony skull offers excellent protection for the brain but allows little tolerance for additional volume. Significant

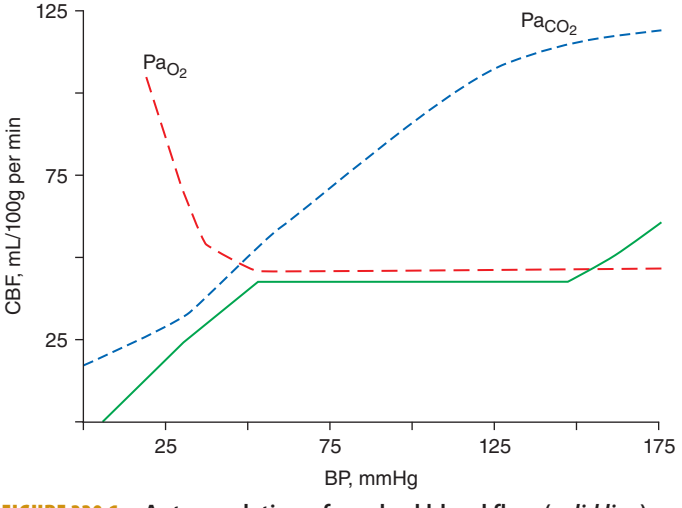


**FIGURE 330-1 Autoregulation of cerebral blood flow (*solid line*).** Cerebral perfusion is constant over a wide range of systemic blood pressure. Perfusion is increased in the setting of hypoxia or hypercarbia. BP, blood pressure; CBF, cerebral blood flow. *(Reprinted with permission from HM Shapiro: Anesthesiology 43:447, 1975. Copyright 1975, Lippincott Company.).*