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Neurologic Emergencies in the Intensive Care Unit

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Neurologic Emergencies in the Intensive Care Unit

I. INTRODUCTION

This overview focuses on the neurointensive care aspects of stroke, head trauma, subarachnoid hemorrhage, and meningitis. These syndromes present diagnostic and management challenges in the intensive care setting and serve as a humbling reminder of the vulnerability of the central nervous system (CNS), particularly the brain. Despite its ingeniously designed protective shield (ie, the cranium), the brain's complex anatomy and physiology leave little room for compromise.

The neurologic emergencies discussed herein reflect acute abnormalities in blood flow, oxygen delivery, oxygen extraction, blood-brain barrier integrity, and intracranial pressure (ICP). Appropriate management of patients presenting with these conditions requires a sound understanding of intracranial dynamics.

VASCULAR ANATOMY OF THE BRAIN

Blood is supplied to the brain through a network of vessels stemming from 2 pairs of arteries: the vertebral arteries, which supply the posterior portion of the brain, and the internal carotid arteries, which supply the anterior portion (**Figure 1**). The circle of Willis, an anastomosis that joins these 4 arteries, allows a great degree of territorial overlap and compensatory perfusion (**Figure 2**).

The integrity of the collateral circulation dictates the degree of hemodynamic compromise that occurs after vessel occlusion. Although large vessel occlusion does occur, small vessel occlusion is more clinically important and may result from emboli or from vascular catastrophes related to congenital anomalies (ie, arteriovenous malformations, aneurysms).¹

INTRACRANIAL HEMODYNAMICS

Cerebral Blood Flow

The brain is characterized by its ability to autoregulate a constant blood flow despite changes in perfusion pressure. Cerebral blood flow in a healthy brain is approximately 50 mL/100 g brain tissue per minute. If

cerebral blood flow falls to below 18 mL/100 g per minute, irreversible neuronal damage occurs.

Cerebral perfusion pressure (CPP) is an indirect means of measuring cerebral blood flow. CPP is the difference between the systemic mean arterial pressure and the ICP. CPP in a healthy brain is approximately 70 to 100 mm Hg. When autoregulation is intact, the cerebral blood flow remains constant across a broad range of CPPs. When CPP falls below 40 mm Hg, however, autoregulation fails and ischemia occurs. Increased ICP usually results in dramatic reductions in cerebral perfusion, leading to brain ischemia.

A classification scheme has been developed to characterize the pathophysiology of cerebral hemodynamic compromise resulting from arterial occlusion or systemic hypotension.² In stage 0, cerebral blood flow and oxygen extraction are balanced. In stage I of CPP reduction, oxygen perfusion is maintained by increasing intracranial intravascular volume via vasodilatation. In stage II of CPP reduction, vasodilatation is unable to compensate for decreased pressure, and oxygen extraction is therefore increased. Positron emission tomography (PET) has shown that oxygen extraction in the setting of stage II hemodynamic failure may increase to as high as 80% or even 85% (in comparison to a normal level of 33%).^{3,4}

Intracranial Pressure

According to the Monro-Kellie doctrine, the calvaria creates a confined space in which any increase in volume of blood, brain, or cerebrospinal fluid (CSF) that is not compensated for by a reduction in volume of another component results in a dramatic increase in ICP. ICP is normally between 0 and 10 mm Hg. Increased ICP is defined as ICP greater than 20 mm Hg lasting for 5 minutes or longer.

The absence of lymphatic drainage intracranially leaves the cerebral parenchyma vulnerable to expansion from edema. Cerebral edema caused by trauma, infection, expanding blood masses or tumors, or increased vasodilation can result in excessive ICP.

Signs associated with increased ICP include headache, confusion, change in mental status, vomiting,