Brain Perfusion and Oxygenation

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KEYWORDS

• Brain perfusion and oxygenation • Oxygen consumption
• Cerebral metabolic rate of oxygen • Cerebral blood flow • Neuromonitoring

KEY POINTS

- Maintenance of brain perfusion and oxygenation is of paramount importance to patient outcome with various types of brain injuries (traumatic, ischemic, and hemorrhagic).
- Historically, monitoring of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) has been the mainstay of neuromonitoring techniques used at the critical care bedside to monitor brain perfusion and oxygenation.
- Within recent years, other neuromonitoring techniques have been studied and developed to provide information concerning brain perfusion and oxygenation.

PHYSIOLOGY OF BRAIN PERFUSION AND OXYGENATION

Cerebral Metabolism

Cerebral metabolism is supported by the constant supply of oxygen and glucose.[1,2] Oxygen consumption by the brain accounts for approximately 20% of the total body oxygen consumption.[3] The cerebral metabolic rate of oxygen ($CMRO_2$) averages 3 to 3.8 mL/100 g/min. Neuronal electrical activity is supported through the generation of ATP, which requires approximately 60% of the total brain oxygen consumption.[1] Most of the ATP produced in the body is the result of oxidative phosphorylation occurring in the mitochondria of the cell, which is otherwise known as aerobic metabolism.[4] In cerebral metabolism, aerobic metabolism depends on the continuous presence of oxygen and glucose.[1,4]

Glucose is the primary source of energy in the brain.[1] This energy promotes maintenance of the gradient of ions across the cerebral cell membrane and transmission of electrical impulses.[3] The brain consumes glucose at the rate of 5 mg/100 g/min, mostly through aerobic metabolism. A continuous supply of glucose is necessary to maintain cerebral function.[1] Hypoglycemia can cause seizures and loss of consciousness.[5] Hyperglycemia can accelerate cerebral acidosis, which promotes cell damage

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and can exacerbate brain injury.[1] Without the continuous presence of oxygen, aerobic metabolism changes to anaerobic metabolism, using the glucose present to produce ATP through the breakdown of glucose to pyruvate to lactate, which results in metabolic acidosis.[2,4]

If brain tissue perfusion is interrupted, with resulting hypoxia, ATP stores are reduced. Under most circumstances, if (cerebral blood flow CBF) is not reestablished within 3 to 8 min, irreversible cellular injury to cerebral tissue occurs.[1]

Decreased CBF results in hypoxia and ischemic injury to the cell. Potassium exits the cell and calcium enters the cell, causing cellular acidosis and necrosis of the cell. Glutamate, a neurotransmitter, is released by ischemic cells. Glutamate promotes the movement of sodium and calcium into the cell, thereby causing cellular edema. Inflammatory mediators (such as prostaglandins and leukotrienes) accumulate as a result of the ischemia and necrosis and cause cellular edema. Biochemical events, such as free radical production, caused by ischemia damage the cell membrane through peroxidation. These processes lead to cellular edema and cell death.[2]

CBF

As previously stated, the brain requires a constant perfusion to ensure the delivery of oxygen necessary for cellular metabolism.[1,6] Oxygen delivery to the brain is accomplished through normal CBF. On an average, the normal CBF is 54 mL/100 g/min in an average brain weighing 1400 g. In other words, normal CBF averages 756 mL/min.[3]

In traumatic brain injury, irreversible tissue damage occurs when the CBF threshold decreases to 15 mL/100 g/min. In ischemic stroke, irreversible tissue damage occurs when the CBF threshold decreases to 5 to 8.5 mL/100 g/min.[7] In general, cerebral impairment is associated with a CBF between 20 and 25 mL/100 g/min.[1]

Determinants of CBF

ICP is maintained through a mechanism known as the Monro-Kellie doctrine. The components of ICP are the brain, blood, and cerebrospinal fluid. If a component increases owing to injury, insult, or disease, then another component must decrease to maintain a stable ICP.[8] The normal ICP, in general, ranges between 5 and 10 mm Hg.[9] The general goal is to maintain the ICP less than 20 mm Hg.[6]

Autoregulation in the brain is a process whereby the CBF is maintained through vasodilation and vasoconstriction of cerebral arterioles to promote a constant CPP. Vasodilation occurs if the systemic blood pressure decreases to allow an increase in CBF. Vasoconstriction occurs if the systemic blood pressure increases, thereby decreasing CBF to prevent hyperperfusion.[3,6] Several factors influence autoregulation. Hypoxia, hyperthermia, acidosis, and hypoventilation cause vasodilation (increase in CBF). Hypothermia, alkalosis, and hyperventilation cause vasoconstriction (decrease in CBF).[9] Cerebral autoregulation can be impaired by insults such as trauma, hypoxemia, and hypercapnia.[7] When brain tissue is damaged by such insults, autoregulation is impaired and cerebral ischemia can result.[9]

Cerebral autoregulation directly affects CBF, thereby preserving CPP over a wide range of mean arterial blood pressure (60–160 mm Hg).[1,3,6] CPP is the difference between the mean arterial pressure (MAP) and the cerebral venous pressure, which is approximated by the ICP.[1,9] More clearly stated, CPP = MAP − ICP.[10] Guidelines for the management of severe traumatic brain injury promote the maintenance of CPP between 50 and 70 mm Hg. Cerebral ischemia may be a greater threat at levels less than 50 mm Hg. The development of adult respiratory distress syndrome is a greater risk at levels greater than 70 mm Hg.[11]