Brain perfusion autoregulation

Brain perfusion autoregulation in general

The brain is supplied with blood through the right and left *a. carotis communis* (anterior circulation) and vertebral arteries (posterior circulation). Cerebral autoregulation is the **ability to maintain a constant blood flow to the brain as systemic blood pressure changes**. Autoregulation is determined by the relationship between cerebral perfusion (= *cerebral blood flow*, CBF) and cerebral perfusion pressure (= *cerebral perfusion pressure*, CPP):

$$CPP = MAP - ICP$$

Mechanisms of cerebral perfusion autoregulation

The self-regulation itself consists in the fact that when the system pressure increases, **vasoconstriction occurs compensatively** in the CNS, and **when the system pressure decreases** sufficient CNS flow is maintained by **vasodilation** of the cerebral flow. Brain perfusion drops sharply when the CPP falls below a critical value (usually 50 torrs). Hypoperfusion, ischemia, and eventually brain death occurs. **The most stable CNS perfusion** is in the range **CPP 50–160 torr**. At values > 160 torrs, on the other hand, CNS flow rises rapidly, and the blood-brain barrier fails with the subsequent development of cerebral edema and bleeding during cerebral vascular rupture.

- **In newborns and infants** MAP alone is in the range of 40-50 torr. In this age group, therefore, the most stable CNS perfusion is achieved in the range of 40-80 torr.
- CNS circulation is also characterized by minimal effects of vasomotor activity via catecholamines due to the presence of the blood-brain barrier.

Autoregulation disorder

When cerebral perfusion autoregulation is impaired, an increase in arterial pressure leads to an increase in intracranial pressure and, conversely, a decrease in arterial pressure leads to a decrease in intracranial pressure. The situation is evaluated by the so-called **PRx index** (*pressure–reactivity index*), which expresses the relationship between **MAP** (mean arterial pressure) and **ICP** (intracranial pressure):

$$PRx = MAP/ICP$$

- **We obtain the index** by obtaining about 40 consecutive correlations **from the MAP and ICP curve recordings over time** (ie at the same time we determine the intersection of the MAP value from the X-axis and the ICP from the Y-axis). ***Positive index values indicate a loss of cerebral perfusion autoregulation**. In practice, PRx results correlate well with transcranial Doppler sonography.

The increases in blood flow to the brain in the event of a disorder of autoregulation is accompanied by a decrease in perfusion brain pressure and is assessed by the **Mx index**:

$$Mx = Vic/CPP$$

- ***Vic*** = blood flow rate in the a. carotis interna measured by transcranial ultrasonography.
- ***CPP*** = perfusion brain pressure, which can be determined from mean arterial pressure, intracranial pressure and CVP.

$$CPP = MAP - (ICP + CVP)$$

The CPP value should not fall below 50 torrs (6,6 kPa), **in newborns and infants <40 torrs**.

Intracranial pressure

The physiological value of ICP in spontaneous breathing is **5–20 torr** (0,33–2,66 kPa), in children we tolerate values < 15 torrs, and in newborns and infants < 10 torr. In adult intensive care guidelines, **intracranial hypertension is defined** as **ICP > 20 torrs** (> 2,66 kPa). The CVP value is not reported in some samples. In relation to the MAP values, it is essentially negligible.

Intracranial pressure is determined by the pressure of brain tissue, MMM and blood on the cranial skeleton. The development of intracranial hypertension results from the fact that the brain, its vessels and MMM are stored in a relatively rigid calf. Enlargement of any intracranial compartment will cause both an increase in ICP and a decrease in other parts of the intracranium. **The so-called Monro-Kellie doctrine** speaks about the connections of individual compartments to the influence of ICP : the sum of components involved in the resulting ICP (ie brain parenchyma, cerebrospinal fluid and blood) should be constant. **However, the increase in ICP**, However, the increase in ICP **is not linear**!

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ICP is **affected** in this order: **venous blood < MMM < brain parenchyma**.

Venous blood represents 5% of the volume of the intracranium, MMM also 5%, and the brain parenchyma then 90% of the volume. When cerebral perfusion **autoregulation is affected, pressure changes** from the arterial circulation **are adversely reflected in changes in intracranial pressure**. When the CPP falls below 50 torrs, the regulatory mechanisms decompensate. **The increase in intracranial pressure** is **accompanied** by a dramatic **decrease in CPP**. Establishing normal conditions is very difficult, but can be achieved by inducing cerebral vasoconstriction and increasing MAP. **The goal of treatment is to normalize both CPP and ICP**. The above pressure and perfusion changes are accompanied by cerebral edema, which is a non-specific response of brain tissue to noxus.

Links

Related articles

- Intracranial hypertension

References

- HAVRÁNEK, Jiří: *Intrakraniální hypertenze.*