Autoregulation of cerebral perfusion

This article has been translated from WikiSkripta; ready for the **editor's review**.

Autoregulation of brain perfusion in general

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

$$CPP = MAP - ICP$$

Mechanism of autoregulation of cerebral perfusion

Self-regulation consists in the fact that **increasing systemic pressure causes compensatory** in the CNS **vasoconstriction**, **in the event of a decrease** in systemic pressure, sufficient CNS flow is maintained by **vasodilatation** of the cerebral blood stream. Cerebral perfusion drops sharply when the CPP value drops below a critical value (usually 50 torr). Hypoperfusion, ischemia, and ultimately brain death occur. The **most stable perfusion** of the CNS is in the range of **CPP 50–160 torr**. At values > 160 torr, on the other hand, CNS flow rises rapidly, there is a breakdown of the blood-brain barrier with the subsequent development of cerebral edema and bleeding in case of rupture of cerebral vessels.

- **In newborns and infants**, the MAP itself ranges from 40-50 torr. Therefore, in this age category, the most stable CNS perfusion is achieved in the range of 40–80 torr.
- CNS circulation is also characterized by a minimal influence on vasomotility through catecholamines due to the presence of the blood-brain barrier.

Self-regulation failure

In the case of impaired autoregulation of cerebral perfusion, 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 **recordings of the MAP and ICP curves over time** we obtain approx. 40 consecutive correlations (i.e. at the same moment in time we determine the intersection of the MAP value from the X axis and the ICP value from Y axis). ***Positive values of the index indicate a loss of autoregulation of cerebral perfusion**. In practice, PRx results correlate well with transcranial Doppler sonography.

An increase in cerebral blood flow in case of autoregulation disorder is accompanied by a decrease in cerebral perfusion pressure and is assessed by the '*Mx index*:

$$Mx = Vic/CPP$$

- ***Vic*** = blood flow velocity in a. carotis interna measured by transcranial ultrasonograph.
- ***CPP*** = cerebral perfusion pressure, which can be determined from mean arterial pressure, intracranial pressure and CVP values.

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

The CPP value should not fall below 50 torr (6.6 kPa), **in neonates and infants < 40 torr**.

Intracranial pressure

The physiological value of ICP during spontaneous breathing is **5-20 torr** (0.33-2.66 kPa), in children we tolerate values < 15 torr, in newborns and infants < 10 torr. In the recommendations for intensive care of adults, **intracranial hypertension is then defined** as **ICP > 20 torr** (> 2.66 kPa). The CVP value is not reported in some formulas. Compared to MAP values, it is essentially negligible.

Intracranial pressure is determined by the pressure of brain tissue, MMM and blood on the cranial skeleton. The occurrence of intracranial hypertension results from the fact that the brain, its vessels and MMM are stored in a relatively rigid calva. An increase in any intracranial compartment will cause both an increase in ICP and at the same time a decrease in other parts of the intracranium. The connection between individual compartments on the influence of ICP is discussed in the so-called **the Monroe-Kellie doctrine**: the sum of the components contributing to the resulting ICP (ie, brain parenchyma, cerebrospinal fluid, and blood) should be constant. The **increasing ICP** that accompanies the increase in brain compartment volume is **not linear**!

⚠ **Monroe-Kellie doctrine: the intracranial volume (approx. 1700 ml) consisting of brain tissue (80%), blood in cerebral vessels (10%) and cerebrospinal fluid in the cerebral ventricles and subarachnoid spaces (10%) is unchanging and due to high water content and incompressible.**

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

Venous blood represents 5% of the intracranial volume, MMM also 5%, brain parenchyma then 90% of the volume. **When auroregulation is impaired**, cerebral perfusion **pressure changes** in the area of the arterial bed are **adversely reflected in changes in intracranial pressure**. When the CPP drops below 50 torr, the regulatory mechanisms decompensate. **An increase in intracranial pressure** is **accompanied by** a dramatic **decrease in CPP**. Establishing normal ratios 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-mentioned pressure and perfusion changes are accompanied by brain edema, which is a non-specific reaction of brain tissue to nox.

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