Inhalation Anesthesia (Paediatrics)

In newborns and small children, inhalation anesthetics have **more significant adverse cardiovascular effects** . In contrast to older children and adults, overdosing, especially during induction of anesthesia, may cause slight bradycardia with a drop in blood pressure or even cardiac arrest due to a rapid rise in alveolar concentration and subsequently blood concentration. The arrhythmogenic effects of halothane in higher concentrations are also more pronounced in this age group. An important role is played by the rapid rise of the partial pressures of these substances in the blood.

Halothane

Currently, it is no longer used in human anesthesia in our country. In children, it was mainly replaced by sevoflurane , which has more favorable pharmacological properties.

Introduction to anesthesia usually took place calmly and carefully, its depth could be regulated quickly and easily. It was often combined with nitrous oxide in the inhalation mixture, but an alternative was also the administration of halothane alone with oxygen.

Isoflurane

Isoflurane stinks and **can lead to coughing, laryngospasm, breath holding and even hypoxemia** during induction of anesthesia . Therefore, this substance is not suitable for inhalation introduction. But even in children, we can use isoflurane **as a primary inhalation anesthetic after intravenous introduction** . To avoid respiratory effects, it is recommended to increase the concentration slowly. Isoflurane causes a **more pronounced depression of breathing** compared to halothane, so we should ventilate children in a controlled manner during anesthesia. The effects on pressure are matched at comparable concentrations of halothane, but the heart rate is stable, that is, bradycardia usually does not occur after isoflurane.

Sevoflurane

Introduction to anesthesia is very quick, as is awakening. Cardiovascular depressant effects are less pronounced compared to other volatile anesthetics. Sevoflurane **is not arrhythmogenic** and does not sensitize the myocardium to exogenous adrenaline . Like all volatile anesthetics, sevoflurane leads to a dose-dependent decrease in arterial pressure and peripheral vascular resistance. The negative inotropic effect is dose dependent.

Sevoflurane **does not irritate the respiratory tract** , so the substance is suitable for induction of anesthesia by inhalation. The respiratory effects of sevoflurane are analogous to other inhalation anesthetics, i.e. it suppresses breathing. Sevoflurane also **relaxes skeletal muscles** and potentiates the effect of non-depolarizing myorelaxants. Like other volatile anesthetics , **it can cause malignant hyperthermia** . In patients with intracranial hypertension, a further increase in intracranial pressure must be expected. Sevoflurane most likely does not have hepatotoxic effects.

In the initial phase, we use an inspiratory concentration of 7–8%, to maintain anesthesia then 1.5–3%, with N 2 O then 0.5–3%. Very high concentrations (8% by volume) allow, after pre-filling the system with gas and fresh anesthetic, a very rapid inhalation induction with a few breaths and loss of consciousness within one minute. The combination with opioids makes it possible to reduce the concentration of sevoflurane. If spontaneous breathing is maintained, the concentration of sevoflurane is gradually reduced. The patient usually falls asleep within 40-60 seconds after the initial inhalation. Halothane with an inspiratory concentration of 4 vol% has a comparably short onset time, but there are greater defensive movements and less cardiovascular stability.

In general, children wake up 2x faster after sevoflurane anesthesia than after halothane, if inspiratory concentrations are maintained until the end of the procedure. But if, as is usual in practice, the concentrations of volatile anesthetics are reduced well in advance of the end of the operation, the differences in awakening times between the two substances are very small. **During rapid awakening after sevoflurane, we observe restlessness, agitation and early perception of pain** in the awakening phase in a considerable number of children , which often requires sedatives, and thus the advantage of rapid awakening is lost. Postoperative nausea and vomiting occur equally frequently after halothane and sevoflurane.

Nitrous oxide (paradise gas, N 2 O)

Nitrous oxide is often used **to supplement general anesthesia** . It enhances the effects of other anesthetics and thus reduces their required dose. Its very small potency is the reason why it **is not used as a monoanesthesia** . The supply of just nitrous oxide mixed with oxygen is not capable of putting the patient under general anesthesia. Its **effect begins** relatively **quickly** . Otherwise, when the supply is stopped, the gas flows quickly again from the brain into the blood, is exhaled and the patient wakes up.

The maximum inspiratory concentration of nitrous oxide is 70%. Clinically, **concentrations** between **50-70% are most often** administered . The combination of volatile inhalation anesthetics with nitrous oxide makes it possible to administer their lower concentrations. Lower concentrations of inhaled anesthetics lead to fewer cardiovascular

and respiratory effects and enable faster awakening. In addition, nitrous oxide complements the administration of benzodiazepines , opioids and muscle relaxants in balanced anesthesia .

The effects of nitrous oxide **on the circulation are very small** in people with cardiac health (negative inotropic effect), **the respiratory effect** is either **none** or represents a slight depression of breathing. Nitrous oxide **does not affect the function of the liver, kidneys, intestines and has no myorelaxant effects** . Clinically important and **dangerous is the diffusion of N2O into closed body spaces filled with gas** – depending on the expansion of the said space, its volume or pressure in the given hollow space will increase. The higher the alveolar concentration of nitrous oxide, the faster the diffusion into closed gas-filled body cavities. For this reason, the **administration of N2O in patients with strictly contraindicated with pneumothorax** . If an air embolism is suspected N2O supply must be stopped immediately. Also of practical importance is the fact that the sealing cuff of a tube or cannula filled with air canreach 2-3 times its original volume by diffusion of N2O, so that the air pressure in the cuff will increase dangerously. Filling the sealing cuff with nitrous oxide can prevent the mentioned risk and trauma to the trachea.

Nitrous oxide has a relatively **good analgesic effect** and is basically used to supplement inhalation and intravenous anesthetics. Its most important advantage is that it makes it possible to reduce their dosage, thereby significantly reducing their side effects. The disadvantage is its relatively low anesthetic capacity, diffusion into body cavities with gaseous content, risk of increased intracranial pressure in patients with limited intracranial compliance.

Links

Related Articles

- Anesthesia
- Anesthesia (pediatrics)
- Neuroleptics (pediatrics)
- Benzodiazepines (pediatrics)
- Intravenous anesthetics (pediatrics)
- Opioids (pediatrics)

Source

- HAVRÁNEK, Jiří: *Inhalation anesthesia* .