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Effect of sevoflurane on cerebral perfusion pressure in patients with internal hydrocephalus

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Abstract

Background. Due to its confirmed neuroprotective properties, sevoflurane is one of a few anaesthetics used for neuroanaesthesia. Its effects on the cerebral and systemic circulations may be of particular importance in patientswith intracranial pathology. This study aimed to evaluate the effect of sevoflurane at concentrations lower than 1 MAC on cerebral perfusion pressure (CPP) in patients with internal hydrocephalus.

Methods. The study was conducted on14 patients with internal hydrocephalus, who underwent ventriculo-peritoneal shunt implantation. After inserting the catheter into the lateral cerebral ventricle, sevoflurane, at 1.1 and 2.2 vol%, was initiated at two successive 15-minute intervals. The intracranial pressure (ICP) was continuously measured; special attention was focused on the values prior to and at the end of each observation period. The following parameters were monitored: mean arterial pressure (MAP), CPP, heart rate, end-tidal CO_2 concentration, core body temperature, and the inspiratory and end-expiratory concentrations of sevoflurane.

Results. The HR and MAP decreased during successive observation intervals compared to baseline values. Likewise, the CPP decreased from 75.6 ± 2.8 mm Hg to 72.2 ± 2.6 mm Hg to 70.2 ± 0.8 mm Hg. The baseline value for ICP was 16.3 ± 0.6 mm Hg and increased to 17.7 ± 0.8 and 18.9 ± 0.5 mm Hg during the next observation periods.

Conclusions. Sevoflurane administered ata concentration below 1MAC to patients with internal hydrocephalus increases the ICP and decreases the MAP, which leads to adecrease in CPP. The CPP decrease is more dependent on depressing the systemic circulatory system than an increased ICP.

Key words: volatile anaesthetics, sevoflurane; intracranial pressure; cerebral perfusion pressure; internal hydrocephalus

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Sevoflurane, one of a few anaesthetics of used for neuroanaesthesia, beneficially affects the factors that determine intracranial homeostasis and exerts neuroprotective effects, which has been confirmed in experimental and clinical studies [1]. The effects of sevoflurane on the cerebral and systemic circulations are particularly relevant in patients with concomitant intracranial pathology.

The present study aimed to assess the effect of sevoflurane ≤ 1 MAC on the cerebral perfusion pressure (CPP) in patients with internal hydrocephalus.

METHODS

The study design was approved by the Bioethics Committee for Scientific Research. Fourteen patients with internal hydrocephalus, 6 females and 8 males, aged 58.5 ± 8.4 years who underwent ventriculo-peritoneal shunt implantationin the Department of Neurosurgery, Medical University of Gdańsk, were included in the study. In all of the patients, internal hydrocephalus developed 2–3 weeks after intracranial aneurysm clipping. On admission, the Glasgow Come Scale (GCS) scores for the patients ranged

Table 1. Values of the parameters monitored*

Parameter	Concentration of sevoflurane		
	0	1.1 vol%	2.2 vol%
MAP (mm Hg)	92.0 ± 2.5	89.8 ± 2.6**	89.1 ± 0.9**
ICP (mm Hg)	16.3 ± 0.6	17.7 ± 0.8**	18.9 ± 0.5**
CPP (mm Hg)	75.6 ± 2.8	72.2 ± 2.6**	70.2 ± 0.8**
Heart rate (min^{-1})	52.5 ± 1.4	49.2 ± 1.7**	47.0 ± 1.6**
Core temperature (°C)	37.2 ± 0.4	37.1 ± 0.3	37.2 ± 0.4

*Data presented as a mean ± SD; **$P < 0.05$ compared to the baseline value

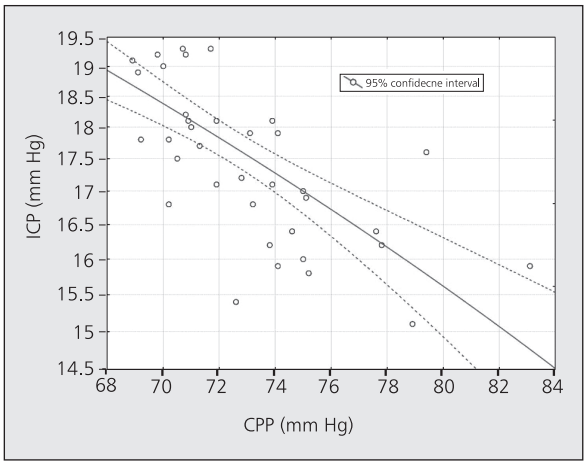

Figure 1. Correlation between CPP and ICP

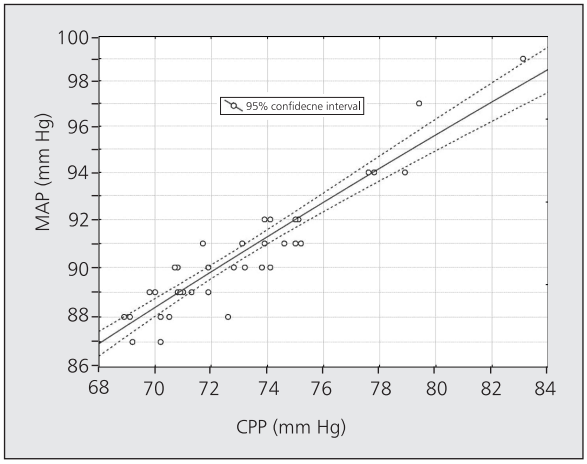

Figure 2. Correlation between CPP and MAP

from 11 to 12. Patients with additional diseases were excluded from the study.

The patients were premedicated with i.m. midazolam 0.1 mg kg^{-1}. Anaesthesia was induced with propofol 1 mg kg^{-1}, fentanyl 5 mg kg^{-1} and atracurium 0.4 mg kg^{-1}. The lungs were mechanically ventilated in a circle system with a mixture of air and oxygen at a 1:1 ratio; the end-tidal pressure of CO_2 was 38–40 mm Hg. Anaesthesia was maintained with a continuous infusion of propofol and atracurium atdoses of 2.0 and 0.3 mg kg^{-1} h^{-1}, respectively. An arterial cannula was inserted into the left radial artery for all of the patients. The following parameters were monitored: mean arterial pressure (MAP), intracranial pressure (ICP), cranial perfusion pressure (CPP), heart rate (HR), end-tidal CO_2 concentration (E_TCO_2), inspiratory and end-expiratory concentrations of sevoflurane and core body temperature. The ICP was measured using the catheter introduced into the anterior horn of the lateral ventricle coupled with the Viggo Spectramed transducer (Viggo, Sweden). The zero level for both transducers was determined at the height of the external auditory meatus. Once the baseline ICP was measured, sevoflurane was administered at concentrations of 1.1 and 2.2 vol% during two consecutive 15-minute time intervals. Special attention was focused on the pre-sevoflurane value for the monitored parameters and on the values recorded at the end of each successive exposure period.

Statistical analysis was performed using GraphPad InStat 3.10 for Windows (GraphPad Software Inc., USA) and Statistica for Windows 9.1 (Statsoft Inc., USA). The data are presented as the means ± SD. The distribution of data was evaluated using the Kolmogorov-Smirnov test. Depending on the type of distribution, the Tukey-Kramer or Dunn's test was applied. Data correlations were assessed using the Pearson test. $P < 0.05$ was considered to be statistically significant.

RESULTS

The average values for the E_TCO_2 are consistent with the assumptions. The use of sevoflurane at inspiratory concentrations of 1.1 and 2.2 vol% resulted in end-tidal pressures of 0.88 ± 0.04 and 1.91 ± 0.05 vol%, respectively. The HR and MAP decreased with successive observation periods compared to the baseline values (Table 1).

The baseline value for the ICP was 16.3 ± 0.6 mmHg and increased with eachsuccessive observation period. Moreover, a decrease in the CPP was observed with each successive observation periodcompared to the baseline value (Table 1).

A very strong correlation was found between the CPP and ICP ($P < 0.05$, r = –0.71) and between the CPP and MAP ($P < 0.05$, r = 0.94) (Figs 1, 2).

DISCUSSION

Post-haemorrhagic hydrocephalus (PH) is a common complication of a subarachnoid haemorrhage (SH) caused by an intracranial aneurysm rupture; approximately 20% of SH patients are affected [2–4]. Depending on the timing for the development of symptoms, post-haemorrhagic hydrocephalus is classified as acute, occurring during the first 72 hours after subarachnoid haemorrhage, or chronic, developing after 14 days. The pathomechanism for chronic hydrocephalus following a SH is complex, multifactorial and not fully elucidated. One of the theories regards a change in the cerebrospinal fluid (CSF) flow [5–7], which can be caused by the mechanical blockage of CSF outflow from the ventricular system of the brain by blood products (clots formed by adhesion) at the level of the Sylvian aqueduct or the Magendie or Luschka foramen [8, 9] or impaired CSF absorption by the arachnoid granulations [10]. In chronic PH, the major pathology is caused by dysfunction of the granulations due to fibrosis [11] induced by inflammationof the granulations and meninges in response to blood products [10, 12], which eventually leads to disrupted CSF absorption. The amount of CSF produced ranges between 0.35 and 0.4 mL min^{-1} [13]. Impaired flow and reabsorption quickly lead to an increased ICP. The baseline ICP in our population substantially exceeded the normal value. Experimental studies have demonstrated that 1-MAC sevoflurane inhibits the production and increases the resistance of cerebrospinal fluid absorption and does not the affect ICP [14], which was confirmed by other authors [15]. Therefore, the increased ICP observed in our study likely resulted from an increase in the cerebral blood flowand cerebral circulating blood volume caused by sevoflurane, which was emphasised in another study [16].

Experimental and clinical studies have revealed that sevoflurane does not affect or slightly increases the heart rate [17]. The decreased heart rate in our patientsappears to have been caused by an increased intracranial pressure. According to Agrawal and colleagues [18], who analysed the causes of bradycardia in neurosurgical patients, one of the causes is a disturbed volume-pressure relationship.

The effect exerted by sevoflurane on the cardiovascular system is the outcome of its influence on cardiac output, peripheral vascular resistance and the autonomic nervous system [17]. In experimental studies, sevoflurane, similar to isoflurane, causes a dose-dependent reduction in the MAP attributable to decreased vascular resistance [19]. In healthy volunteers anaesthetised with 1.2-MAC sevoflurane, a 30% decrease in the MAP was observed [17].

The main objective of an anaesthesiologist during intracranial surgery is to provide appropriate conditions for the operating field, maintain a proper CPP and oxygenation of the nervous tissue. The effects of anaesthetics on the cardiovascular system are essential for maintaining a proper CPP. Its value is determined by the difference between the MAP and ICP, with 70 mm Hg considered to be critical [20]. Our analysis of a correlation among the CPP, ICP and MAP indicates that the systemic circulation component has a greater impact on cerebral tissue perfusionthan the other variables.

The study limitations resulting from the use of propofol should be stressed. Choosing this anaesthetic is based on its properties and on the data reported by other authors [21]. Propofol in clinical doses only slight modulates cerebral blood flow and does not affect the ICP, autoregulation of the cerebral circulation or CO_2 reactivity in the cerebral vessels [22].

CONCLUSIONS

1. Using sevoflurane at ≤ 1 MAC in patients with internal hydrocephalus increases the ICP and reduces the MAP, which leads to a decrease in the CPP.
2. A decrease in the CPR is moredependent on depressing the systemic circulatory system than an increased ICP.

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