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Title : SPINAL CORD BLOOD FLOW DURING INDUCED HYPOTENSION: COMPARISON OF NITROPRUSSIDE AND TRIMETHAPHAN

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Introduction

The response of the spinal cord circulation to agents used for inducing hypotension is not well documented. Since induced hypotension is commonly employed during surgical procedures, it appeared important to investigate the effects of hypotensive drugs on spinal cord blood flow (SCBF).

Methods

This study was crried out on 12 mongrel dogs weighing between 11 and 16 kg. Anesthesia was induced with thiopental sodium, 25 mg/kg, and endotracheal intubation was accomplished after muscle paralysis with succinylcholine, 40 mg, intravenously. Nitrous oxide, 70 percent, oxygen and intermittent fentanyl were used to maintain anesthesia. Muscle paralysis was produced with pancuronium, 0.1 mg/kg, approximately every 2 hours. Ventilation was controlled to maintain arterial P_{CO_2} within normal limits. Catheters were placed in the femoral artery and inferior vena cava for pressure monitoring, blood sampling, and administration of fluids and drugs. A standard dorsal laminectomy was performed to expose T_{12} and T_{13} segments with the dura intact. ^{133}Xe was introduced into the cord by direct injection and SCBF was calculated from its clearance curve, which usually resolved into two components, slow and fast.

The dogs were divided randomly into two groups of six. In Group 1 the effects of nitroprusside (NP) hypotension were assessed, whereas in Group 2 the effects of trimethaphan (T) hypotension were determined.

Results

Control blood pressure and SCBF were not significantly different between the two groups. Hypotension was produced easily in all animals. During the induction of hypotension with NP, SCBF increased significantly ($P<0.02$) from 18.7 to 26.8 ml/min/100 g. It returned to its starting value when mean arterial pressure (MAP) reached 60 to 65 mm Hg; thereafter it decreased with further reductions in MAP. In dogs rendered hypotensive with trimethaphan, there was no significant change in SCBF until a MAP of 60 mm Hg was reached. Further decreases in MAP were associated with parallel reductions in SCBF.

Discussion

These results demonstrate that nitroprusside-induced hypotension was associated with an increase in SCBF, which then returned to control values when a MAP of 60 to 65 mm Hg was reached. With trimethaphan, however, SCBF remained relatively constant and in the normal range between MAPs of 60 to 125 mm Hg. Reductions in MAP below 60 mm Hg were accompanied by similar decreases in SCBF regardless of the drug employed. These findings suggest that nitroprusside disturbed, while trimethaphan preserved, autoregulation of SCBF.

Spinal cord circulation seems to behave in a manner similar to that of the cerebral circulation. Turner *et al.*[1] have shown that administration of NP in man was associated with a significant increase in mean intracranial pressure and cerebral blood flow when MAP was decreased moderately. When MAP had decreased to 70 percent of pre-NP values, mean intracranial pressure returned to baseline values, and thereafter decreased steadily with further decreases in MAP. They also found that trimethaphan produced no significant changes in ICP. Ivankovich *et al.*[2] have demonstrated the vasodilating properties of NP on cerebral vessels, following either a bolus injection or the continuous infusion of the drug in goats.

It thus appears that NP disturbs autoregulation of SCBF, whereas trimethaphan does not affect it. The reason for this is not immediately apparent. It may well be related to differences in the mechanism of action: NP is a vascular smooth muscle dilator whereas T is a ganglionic blocker. Moreover, the more rapid onset of action of NP compared to T may also be a factor.

References

1. Turner JM, Powell D, Gibson RM, McDowall DG. Intracranial pressure changes in neurosurgical patients during hypotension induced with sodium nitroprusside or trimethaphan. Br J Anaesth. 1977; 49: 419-425.
2. Ivankovich AD, Miletich DJ, Albrecht RF, Zahed B. Sodium nitroprusside and cerebral blood flow in the anesthetized and unanesthetized goat. Anesthesiology. 1976; 44: 21-26.