Title : EFFECT OF BLOOD PRESSURE ON SPINAL CORD BLOOD FLOW IN DOGS

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## INTRODUCTION

Although the effect of changes in arterial blood pressure on cerebral blood flow has been extensively studied, surprisingly few studies examined spinal cord blood flow (SCBF) responses to changes in arterial pressure. We report the effects of induced hypo- and hypertension on canine SCBF.

## METHODS

Eighteen dogs were divided into 3 groups of six. The effects of hemorrhagic hypotension were investigated in Group 1, trimethaphan-induced hypotension in Group 2, and norepinephrine-induced hypertension in Group 3. All dogs were induced with thiopental and maintained on 0.5% halothane in a 60% $N_2O$-$O_2$ mixture. Ventilation was controlled to maintain arterial $P_{O_2}$ and $P_{CO_2}$ within normal limits. A femoral artery was cannulated for measuring arterial pressure and sampling. Packed cell volume was measured by micro-hematocrit method and the result used in determining $\lambda$ (tissue/blood partition coefficient). Naso-pharyngeal temperature was maintained between 37 and 38°C. The required cord segments ($T_{12}/T_{13}$) were exposed by dorsal laminectomies with the dura left intact. In all groups, control measurements were obtained at normotension, and measurements were then repeated after a new blood pressure level had stabilized for 10 minutes.

In Group 1, graded hypotension was produced by bleeding the dogs via the arterial cannula. In Group 2, hypotension was induced by trimethaphan, and in Group 3, hypertension was produced by infusing norepinephrine. $^{133}Xe$ was injected directly into the spinal cord, and its clearance measured by a scintillation counter coupled through a ratemeter to a linear recorder. The clearance curve was transposed onto semilog paper for calculation of $T_{\frac{1}{2}}$. SCBF (ml/100 g/min) was calculated from

$$\frac{\lambda \times \log_e 2 \times 60}{T_{\frac{1}{2}}} \times 100$$

Arterial blood gases were measured by standard electrodes.

## RESULTS

Group 1: Gradual decrease of arterial pressure (bleeding) from 150 to 60 torr did not significantly alter flow. Below 60 torr, flow decreased with pressure.

Group 2: With trimethaphan, an initial increase in flow occurred. Flow then returned to control values until a mean pressure of 50 torr was reached. Further reduction in pressure decreased flow.

Group 3: SCBF progressively increased with rise in mean pressure from 150 to 210 torr.

## DISCUSSION

This study demonstrates that at normal $P_{O_2}$ and $P_{CO_2}$, the spinal cord blood vessels of anesthetized dog exhibited a well-functioning autoregulation between mean pressures of 50 and 150 torr. Autoregulation was evident with lower mean pressures with trimethaphan, presumably due to the vasodilator effect of the drug. Autoregulation has also been shown in the canine cerebral circulation. Autoregulation allows normal functioning of brain and spinal cord energy metabolism in the face of decreased arterial pressure. This may assume clinical importance in situations where deliberate hypotension is used to decrease intraoperative blood loss, e.g. spinal fusion operations. Siesjö *et al.* (1) found no significant change in high energy phosphates in rat brain until arterial pressure was decreased to 35 torr. The mechanism responsible for spinal (or cerebral) vascular autoregulation is still unknown. Both a metabolic and a myogenic hypothesis have been forwarded. Further studies are required to elucidate the responsible mechanism.

## REFERENCE

1. Siesjö BK, *et al*: Energy metabolism of the brain at reduced cerebral perfusion pressures and in arterial hypoxaemia. In Brain Hypoxia. JB Brierley, BS Meldrum, Editors. London, Heinemann, 1971, pp. 79-93.