Title: INTRACRANIAL PRESSURE RESPONSES FOLLOWING RAPID INDUCTION OF HYPOTENSION WITH TRIMETHAPHAN

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Introduction. Studies on cerebral hemodynamics subsequent to rapid trimethaphan induced hypotension indicate that significant changes may occur in brain electrical activity, and possibly in cerebral blood flow. It has also been noted that a transient increase in brain retraction pressure occurs during experimental retraction following rapid induction of hypotension with trimethaphan. This increase in pressure appears to be the result of brain swelling and therefore may have significant implications in the closed cranium. This study has been designed to evaluate the effect of rapid induction of hypotension by trimethaphan on intracranial pressure (ICP).

Methods. Changes in ICP as a function of hypotension induction rate were evaluated in 15 mongrel dogs anesthetized with pentobarbital. The ECG, arterial and central venous pressures were monitored with appropriate transducers and the animals ventilated to maintain normal blood gases. The animals were moved into the prone position and their heads fixed into a stereotactic frame. The left temporal muscle was reflected and a small burr hole was made over the temporal cortex. The dura was incised and a subarachnoid bolt was positioned for continuous monitoring of ICP. Mean arterial pressure was reduced with trimethaphan to 55mmHg in Group I within 5 min, in Group II between 5 and 10 min, and in Group III between 10 and 15 min. There were five animals in each group. The ICP time courses for each group were subjected to a polynomial regression analysis, and a correlation coefficient calculation. A t statistic was also generated for the correlation coefficient with $p<0.05$ taken as significant.

Results. The ICP time course in Group I shows that ICP and arterial pressure initially fall concomitantly. Immediately following arterial pressure plateau, ICP appears to rebound and reach pressures as high as 20mmHg within 2 min. Maximum ICP increase occurs 4 minutes after arterial pressure plateaus and reaches a mean pressure of 15.5 ± 4.4mmHg ($p<0.05$ compared to control values 6.0 ± 2.7mmHg). Following this rebound, ICP gradually returns to normal and remains unchanged even when arterial pressure is permitted to return to normal. In Groups II and III, no significant changes in ICP time course are noted following hypotension induction.

Discussion. Rapid induction of hypotension with trimethaphan (1-5min, Group I) appears to produce a significant, transient increase in ICP. This increase in pressure may be mediated by a rapid change in brain blood volume secondary to cerebrovascular dilation. Recent reports have indicated that trimethaphan may have, in addition to its ganglionic affect, a direct dilatory action on capacitance vessels and this finding is consistent with our observations. Furthermore, in brains where cerebral compliance has been substantially taken up by a mass lesion, ICP increases following rapid hypotension induction may reach intolerable limits.