Title: BARBITURATE VS. HALOTHANE IN CEREBROVASCULAR THROMBOEMBOLIC EPISODES.

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Introduction. Barbiturate anesthesia is widely employed during neurosurgery because it apparently reduces cerebral metabolism and oxygen demand, thereby minimizing neuronal damage associated with impaired cerebral circulation. Anesthesia with halothane is less popular because of cerebrovascular dilation and subsequent increases in intracranial pressure. To further clarify the potential value of these anesthetics during neurosurgery, their effects were compared on stimulus-evoked cortical responses following thromboembolic occlusion of the middle cerebral artery (MCA).

Methods. In 19 adult mongrel cats, general anesthesia was induced with hexobarbital and maintained with halothane 1-1.5% (HAL). Twelve additional cats were anesthetized with pentobarbital sodium 35 mg/kg i.v.(PB), supplemented to maintain light surgical anesthesia. Mean arterial pressure, end-tidal blood and anesthetic gas concentrations, ECG and rectal temperature were monitored continuously. After placing the cat in a stereotaxic frame, the MCA was exposed. Stimulating electrodes were placed in the pad of the right forepaw, while recording electrodes were positioned on the exposed left somatosensory cortex. In 8 cats, a bipolar electrode was placed in the ventral posterolateral nucleus (VPL) of the thalamus to record the afferent volley inducing the cortical somatosensory evoked potential (SSEP). Changes in the EEG and somtosensory evoked potential (SSEP) were quantified by means of 2 analog peak detectors. SSEP was defined as the peak-peak voltage difference detected during 2-18 msec post-stimulus. Maximum EEG amplitude was the peak-peak voltage difference during 400-480 msec post-stimulus. Stimulation rate was 1 Hz. Blocks of 100 consecutive responses were stored in digital form on magnetic tape for off-line processing. Embolization was produced by repeated brief clamping of the middle cerebral artery with a special clip. Following establishment of baseline EEG and SSEP values, the MCA was occluded repeatedly for 5-7 min at 30-60 min intervals. The process continued until 1) EEG and SSEP indicated an irreversible loss of cerebral function, or 2) the pulmonary and/or hemodynamic instability of the animal made further data collection inappropriate.

Results. Six HAL, but no PB cats, lost cortical responses during exposure of the MCA in a manner suggestive of spontaneous occlusion. Only 2 HAL recovered responses after 2 successive clampings, compared to 7 in the PB group. Although no HAL cats recovered after 3 clampings, in the PB group recovery was seen in 50% of the animals after 3 clampings, 42% after 4, 25% after 5 and 17% after 6 clampings. Ten HAL cats evidenced signs of spontaneous functional loss, indicative of clot formation. Just one such episode occurred in the PB group. Surprisingly, recordings from the thalamic electrode indicated that occlusion of the MCA suppressed responses at this site in 2/5 animals tested.

Discussion. The results indicate that cats anesthetized with halothane are much more susceptible to vascular accidents induced by manipulation of the MCA than are cats anesthetized with pentobarbital. The latter anesthetic appears to protect against clot/spasm formation under these conditions. This finding suggests that barbiturate anesthesia may be preferrable to halothane during cases involving surgical manipulation of the cerebral vessels. The ability of the occluded MCA to suppress VPL responding in some animals may have been due to a variable distribution of the artery or of the embolization. Additional investigation with dye distributiion and/or histological techniques is needed to explain this interesting observation.