

REPORT OF SCIENTIFIC MEETING

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Society of Neurosurgical Anesthesia and Critical Care and American Society of Critical Care Anesthesiologists

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The 15th Annual Meeting of the Society of Neurosurgical Anesthesia and Critical Care was held in cooperation with the American Society of Critical Care Anesthesiologists at the Atlanta Marriott Marquis October 8, 1987. A diverse spectrum of topics relevant to the practice of neuroanesthesia and critical care was presented with emphasis centering on care of the patient with head trauma, the pathophysiology and pharmacology of cerebral ischemia, and the problems of venous air embolism as related to sitting position craniotomy.

HEAD TRAUMA

The widespread use of Glasgow Coma Scale (GCS) has facilitated the accumulation of a large body of data useful for predicting outcome after severe head injury. Conclusions drawn from these data were reviewed by Graham Teasdale, M.R.C.P., F.R.C.S. (Glasgow, Scotland). He reported that residual deficits from head injury consist primarily of cognition and personality changes, while physical abnormalities persist in only 20% of patients. Long-term outcome still remains difficult to predict in the acute stage of injury, although 50% of patients remaining vegetative 1 month after injury will die within a year. Approximately 2/3 of those with less severe injury persisting at 1 month will show significant improvement over the same period. There is probably no time, however, during the first year after injury that final outcome can be predicted, except that no patients remaining vegetative at 3 months have shown significant improvement. Of the functions ascertained by the GCS (*i.e.*, eye opening, best verbal response, and best motor response), best motor response has the highest predictive value. Dr. Teasdale emphasized that predictions based on the GCS should be made on a patient-by-patient basis, and that scores be communicated not as a sum of scores for the three categories, but, rather, as specific scores for each function. Other factors not necessarily related to outcome (*e.g.*, endotracheal tube, or an infant unable to follow commands) may skew the prognosis, inappropriately, to the negative side.

Care of acutely head-injured patients was also discussed. Steven Allen, M.D. (Houston, TX), reviewed the fact that hypoxemia can lead to an increase in CBF and, thus, ICP. He pointed out that about 40% of head-injured patients have hypoxemia ($Pa_{O_2} < 60$ mmHg) on hospital admission that cannot be attributed to injury of other organ systems. This tendency toward hypoxemia progresses during the first 48–72 h after injury. It has been suggested that this hypoxemia may be due to neurogenically mediated bronchiolar constriction leading to loss of hypoxic pulmonary vasoconstriction. Because these hypoxemic episodes tend to be unpredictable and transient, continuous monitoring of oxygenation (*e.g.*, pulse oximeter) is indicated in the first 48–72 h after injury to allow rapid diagnosis and institution of therapy.

Andrew Kofke, M.D. (Hershey, PA), stressed the impor-

ance of maintaining cardiovascular stability in the acutely head-injured patient. Hypertension, particularly in patients with disrupted autoregulation, may lead to increases ICP. Antihypertensive agents with vasodilatory properties (*e.g.*, sodium nitroprusside, hydralazine, and calcium antagonists) may increase ICP, and should be avoided. Rather, drugs which act on the autonomic nervous system (*e.g.*, propranolol, labetalol, esmolol) should be used. To emphasize this concept, Bunegin *et al.* (San Antonio, TX) presented data from dogs with non-compliant brains showing that esmolol is effective in reducing blood pressure while having no effect on ICP. These data, combined with a report from Gibson *et al.* (Rochester, MN), who found esmolol to be effective in controlling hypertension during emergence from neuroanesthesia in humans, suggest that esmolol may find an important role in neuroanesthetic pharmacology. For pharmacologic treatment of hypotension, Dr. Kofke suggested use of alpha agonists (*e.g.*, phenylephrine), which appear to have less effect of CBF than beta agonists. Along with these considerations, Guy Clifton, M.D. (Richmond, VA), reported that head-injured patients are hypermetabolic and have increased caloric requirements (especially if posturing is present). Such patients require a minimum of 0.2 g of nitrogen/kg/day to avoid increased morbidity from malnutrition. With respect to ICP monitoring, Miller *et al.* (Jackson, MS) reported additional evidence that ICP measurements obtained contralateral to a lesion may not reflect ipsilateral increases in pressure.

CEREBRAL ISCHEMIA

Post-ischemic hypoperfusion (PIH) is a well-documented sequel to global cerebral ischemia. Two papers reported on attempts to manipulate this phenomenon pharmacologically. Olympio *et al.* (Winston-Salem, NC) recognized that the potent vasoconstrictor thromboxane A_2 (TxA_2) is elevated after ischemia, and that PIH is improved by inhibiting TxA_2 release. They reasoned that blockade of TxA_2 activity might have a similar effect, and administered a TxA_2 receptor antagonist (SQ29,548) to dogs sustaining global ischemia. However, no improvement in PIH was seen, suggesting that either SQ29,548 does not cross the blood-brain barrier, or that TxA_2 levels were sufficiently high to overcome the competitive antagonism. Similarly, Petrozza *et al.* (Winston-Salem, NC) failed to improve PIH with a kappa opioid receptor antagonist thought to effect calcium channels.

Kirsch *et al.* (Baltimore, MD) evaluated the role of free radicals in reperfusion injury. Utilizing electron spin resonance spectroscopy, a fivefold increase in free radical signals was detected during reperfusion when compared to values obtained during ischemia in rats. Similarly, free radical mediated lipid peroxidation was not elevated until perfusion was restored. They found that this increase in lipid peroxidation could be inhibited by pre-ischemic administration of superoxide dismutase, a free radical scavenger.

Lactic acidosis has also been implicated in worsening outcome from cerebral ischemia. Zapp *et al.* (Hershey, PA) evaluated the effects of inhalational anesthetics on the relationship between plasma glucose and brain lactate levels resulting from 5 min of decapitation ischemia. Increasing concentrations of

isoflurane, halothane, and enflurane (0.5, 1.0, and 1.5 MAC) all led to an increase in pre-ischemic plasma glucose with isoflurane having the greatest effect. This isoflurane-induced increase in plasma glucose was associated with a larger increase in brain lactate levels during ischemia when compared to halothane or enflurane anesthesia. The authors suggested that this isoflurane-induced increase in lactate may be detrimental during conditions of cerebral ischemia.

A final mechanism of ischemia was explored by Boening *et al.* (Brooklyn, NY). While the effects of inhibiting calcium influx at the cellular level have been extensively studied, the effects of inhibiting sodium influx induced by anoxia have not. Using an incubated hippocampal slice preparation allowing detection of neuronal electrophysiologic viability, sodium influx was inhibited by tetrodotoxin (TTX). After demonstrating inhibition of depolarization by TTX during normoxia, anoxia (5 min) was induced. The TTX was then washed out. Partial post-anoxic recovery of function was achieved in TTX-treated slices, as opposed to no recovery in untreated controls, suggesting sodium channel blockade to be a potential avenue for anti-ischemic therapy.

Several papers evaluated the efficacy of various anesthetic agents in improving outcome from ischemic injury. Baughman *et al.* (Chicago, IL) administered low and high doses of methohexital, midazolam, or etomidate to rats before delivering a reversible, severe, or moderately severe hemispheric insult. Neurologic recovery was then evaluated. When ischemia was moderately severe, all three drugs in the low-dose range improved outcome when compared to N₂O-anesthetized controls. At higher drug doses, the protective effect of methohexital and midazolam was reduced but persistent, while the protective effect of etomidate was abolished. Protective effects were uniformly absent if ischemia was severe, and this was independent of drug or dose of drug. This information is consistent with previous reports suggesting that protection will be afforded by cerebral metabolic rate depressants during mild-moderate ischemia, but will be absent with severe insults.

Gelb *et al.* (London, Ontario) administered intravenous lidocaine (0.05 mg/kg) to cats before middle cerebral artery (MCA) occlusion, and then continued a lidocaine infusion during 3 h of occlusion and for 3 h of reperfusion. Treated animals had a significantly reduced infarct size compared to untreated controls, demonstrating a protective benefit from lidocaine during focal ischemia in this model. This effect correlated with partial preservation of sensory evoked potentials (abolished in untreated controls) and improved blood flow in grey matter peripheral in the infarct. Milde *et al.* (Rochester, MN), using a MCA model in the pigtail monkey, compared deep isoflurane and thiopental anesthesia, but were unable to distinguish outcome after a 5-h occlusion period as a function of anesthetic agent.

AIR EMBOLISM

Venous air embolism (VAE), such as can occur during sitting position craniotomy, continues to be studied with respect to etiology, detection, and therapy. Oliver *et al.* (Rochester, MN) developed a swine model allowing study of paradoxical air embolism (PAE) where air entering the right heart crosses over to the left-sided circulation. A balloon atrial septostomy was created, and right (RAP) and left (LAP) atrial, as well as

pulmonary capillary wedge (PAWCP), pressures were monitored. Air was infused iv (0.27 ml/kg/min) under different ventilatory conditions with intracardiac air detected by trans-esophageal echocardiography. They found the incidence of PAE to be the same during spontaneous or positive pressure ventilation, and unaffected by the presence or absence of PEEP. They also noted that PAE can occur when LAP > RAP, and that PACWP did not accurately reflect LAP during air embolism. It was concluded that any one mode of ventilation cannot be recommended for reducing PAE, and that monitoring of PACWP will not assist in predicting probability of PAE when VAE occurs. Russell *et al.* (Hershey, PA) found emission nitrogen spectrometry (as used in pulmonary function labs) to be superior to mass spectroscopy for detection of VAE in the dog. Bowdle *et al.* (Seattle, WA) introduced a new multi-orificed catheter developed for rapid aspiration of VAE. This catheter (9 French), designed to serve also as an introducer sheath for a pulmonary artery (PA) catheter, is 25 cm in length, allowing EKG placement directly into the right atrium (RA). When tested in sitting dogs, this sheath was far more effective in retrieving air than was the proximal port of the PA catheter. They also answered a long-standing question. When using EKG to place a multi-orificed RA catheter, from which orifice does the EKG signal arise? By placing a single-orificed CVP catheter through the multi-orificed sheath and recording EKG simultaneously from both catheters, the tip of the single-orificed catheter was located at the midpoint between the proximal orifice and tip of the introducer when both signals showed equal P-wave deflection. Thus, the multi-orificed signal must come from the mid-point between the proximal orifice and the tip of catheter. Finally, Spies *et al.* (Chicago, IL) evaluated a perfluorocarbon emulsion (PFE) for treatment of VAE in swine. Because PFE has enhanced solubility for both O₂ and N₂ and improved rheology over blood, PFE may allow dispersion of air emboli. Anesthetized swine received 0.5 ml/kg/min of air iv for 3 min. Half the animals were treated with PFE, and the remainder received an equal volume of hetastarch. Hemodynamic values were recorded throughout. More rapid recovery of blood pressure, dp/dT, and systemic vascular resistance were seen in the PFE group, although this group also had higher PA pressures. Autopsy inspection showed hetastarch-treated pigs to have large air bubbles localized in the right heart, while, in the PFE animals, small frothy bubbles dispersed throughout the pulmonary vasculature were noted. Although outcome was not assessed, this study suggests that further studies may find value for PFE in the treatment of severe air embolism.

The meeting concluded with the installation of Philippa Newfield, M.D., as president. This was followed by a business meeting. The 16th Annual Meeting for the Society of Neurosurgical Anesthesia and Critical Care will be held at the San Francisco Hilton on October 7, 1988. Abstract and membership information can be obtained from the Society office, P.O. Box 11083, Richmond, Virginia 23230.

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