REPORT OF A SCIENTIFIC MEETING

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The Annual Winter Meeting of the Society of Neurosurgical Anesthesia and Critical Care was held on March 14 and 15, 1990, at the Sheraton Waikiki Hotel, Honolulu, Hawaii. Among the topics discussed were management of spinal cord injury, intracranial pressure, and brain trauma. There also was an update on the mechanisms of, and therapies for, ischemic brain injury.

Spinal Cord Injury

The assessment and management of spinal cord injury (SCI) were reviewed by Lawrence Pitts, M.D. (San Francisco, California). According to Dr. Pitts, the level and origin of SCI is initially assessed using historic information, physical examination, and plain roentgenograms. The latter can identify fractures and alterations of alignment of the spinal column, and these abnormal areas can be evaluated further using computerized tomography (CT) and magnetic resonance imaging (MRI) scans. CT may help identify intraaxial hematomas or edema, as well as provide additional information about bone fractures. Optimal information about cord edema, disruption, or hemorrhage is provided by weighted MRI data (*i.e.*, T1- and T2-weighted images). In most instances of surgical intervention after SCI, surgery is undertaken to provide stability of the spine and not to improve cord function. Exceptions to this include patients with progressive neurologic deficits and those with major nerve root involvement from an injury-related compression. In a small number of neurotrauma patients, there may be co-existing head injury and SCI; however, concerns for SCI should not unduly delay initial treatment of head injury.

Brian Andrews, M.D. (San Francisco, California) attributed SCI to a variety of pathomechanisms, including direct disruption of cellular elements within the cord, neurovascular injury, and biochemical abnormalities. Neurovascular injury occurs due to a variety of factors including systemic hypoxia and hypotension and central cord syndrome. Neurovascular injury *per se* may then result in decreased spinal cord blood flow and cord edema. Biochemical injuries may be mediated by fluxes in ionic calcium and iron, thromboxane, arachidonic acid metabolites, and excitatory amino acids. Initial medical therapy of SCI should involve treatment of aberrations of system physiology. Numerous experimental therapies are under investigation; however, none is presently at the stage that it can be recommended clinically.

Respiratory and hemodynamic management of the SCI patient was outlined by Colin MacKenzie, M.D. (Baltimore, Maryland). The most common level of injury, C5–6, is also the most mobile portion of the cervical spine. Injuries at this level will result in a loss of expiratory respiratory function (mediated by thoracic and lumbar nerves) as well as a significant decrement in inspiratory function. Cardiac dysfunction may be common, particularly in patients in spinal shock, because the latter group of patients experiences a relative hypovolemia secondary to functional sympathectomy. There also may be left ventricular dysfunction, as assessed by echocardiography. Thus, SCI patients may have difficulty compensating for alterations in intravascular volume. This is compounded by a potential limitation on the ability to alter heart rate or contractility. Appropriate therapy of hypotension may involve fluid challenge and inotropic support. Exacerbation of left ventricular dysfunction should be anticipated when using volatile anesthetics.

In the panel discussion that followed, all three experts agreed that patients having spinal shock are best managed using a pulmonary artery catheter; however, there was disagreement regarding the relative importance of using intravenous fluids *versus* vasopressors to increase blood pressure.

Intracranial Pressure and Brain Trauma

The patient with abnormal intracranial elastance (dP/dV) may experience large increases in intracranial pressure (ICP) and only small increases in cerebral blood volume (CBV). Thus, inhalational anesthetics, which produce increases in CBV, may also produce increases in ICP. Robert Bedford, M.D. (New York, New York) stated that, in patients having baseline increases in ICP, hyperventilation tends to attenuate the inhalational anesthetic-induced increases in cerebral blood flow (CBF), and presumably CBV and ICP. However, inhalational anesthetics should be used judiciously, particularly in patients with large or rapidly expanding intracranial mass lesions or with evidence of a midline shift of cerebral structures, because these patients may be at the greatest risk for large ICP increases even with small increases in CBV. G. J. Edelman, M.D. (Chicago, Illinois) presented data demonstrating that isoflurane produces larger increases in infratentorial than in supratentorial CBF in rats. Assuming that these data are transferable to humans and that the CBF increases are accompanied by increases in CBV, isoflurane may have different effects in patients with posterior fossa pathology than in those with supratentorial pathology. William Lanier, M.D. (Rochester, Minnesota) reviewed the effect of muscle relaxants on ICP. Muscle relaxants may increase ICP either *via* a histamine-mediated effect (*e.g.*, d-tubocurarine) or a muscle afferent activity (MAA)-mediated effect. Succinylcholine produces increases in MAA that in turn produce cerebral stimulation manifested as EEG activation and increases in CBF and ICP. The cerebral stimulation is not dependent on the presence of muscle fasciculations; however, it can be prevented by pretreatment with subparalyzing doses of metocurine *via* a currently unidentified mechanism.

David Smith, M.D., Ph.D. (Philadelphia, Pennsylvania) discussed fluid management in patients with elevated ICP. According to Dr. Smith, the brain acts as an osmometer, such that in the presence of an intact blood–brain barrier (BBB), brain water content (and thus brain volume) varies inversely with plasma osmolality. In contrast, plasma oncotic pressure has no effect on brain water content. When the BBB is disrupted, bulk flow of water from the vasculature to brain parenchyma no longer depends on Starling forces, but instead, depends on hydrostatic pressure. Thus, in areas of disrupted BBB, neither plasma os-

molality nor oncotic pressure has an effect on brain water content. If decreasing brain water content is a management priority, administration of a hyperosmolar solution is appropriate. Marked hemodilution (*i.e.*, a hemoglobin less than 7 g/dl) may result in a decreased oxygen-carrying capacity of the blood, sufficient to cause secondary increases in CBF and CBV. Dr. Smith stated that hyperglycemia repeatedly has been demonstrated to worsen ischemic neurologic injury caused by a variety of insults. Clinically, if blood glucose is monitored, one can safely delete glucose from intravenous fluids in the perioperative period, even in children, without the risk of adverse effect (*e.g.*, hypoglycemia). In patients having preexisting hyperglycemia, insulin therapy may result in some improvement in risk for ischemic neurologic injury, although the magnitude of the improvement is not clear. Dale Pelligrino, Ph.D. (Chicago, Illinois) reported that blunted cerebrovascular responsiveness in diabetic rats resulted in a suboptimal CBF response to hypoglycemia when compared to nondiabetic rats. This altered CBF response may have been caused by a diabetes-induced autonomic neuropathy. During periods of hypoglycemia, there were more severe abnormalities of somatosensory evoked potentials in diabetic than in nondiabetic rats, presumably because of the influence of the altered vascular responsiveness.

Ischemic Brain Injury

Dennis Choi, M.D., Ph.D. (Stanford, California) reviewed the excitotoxic theory of neurologic injury. This theory predicts that excitatory amino acids (EAA), particularly L-glutamate (Glu), contribute significantly to hypoxic or ischemic neuronal damage. In ischemic brain, there is an accumulation of extracellular Glu (due to both increased release of Glu from—and decreased reuptake into—neurons), and this Glu binds with EAA receptors. During ischemia, the amount of extracellular Glu accumulation is proportional to the severity of the insult. Both *in vitro* and *in vivo* studies have demonstrated that Glu accumulation can serve as a mediator of neuronal injury *via* interaction with EAA receptors. Furthermore, by administering EAA receptor antagonists, one can attenuate Glu-mediated cell injury. Thus, future therapies of ischemic neuronal injury may be directed toward antagonizing the interaction of EAAs such as Glu and their EAA-specific receptors.

Although oxygen free radicals have been postulated as mediating a variety of types of cell injury, William Perkins, M.D. (Portland, Oregon) stated that there is little evidence solidly linking oxygen free radicals to ischemic neuronal injury or demonstrating improvement in outcome produced by free-radical scavengers. Recent advances in free-radical experimentation suggests that because of the kinetics of free radical generation and deterioration, a free-radical scavenging agent must be present at the instant of reperfusion if improvement in postischemic outcome is to occur. This hypothesis is supported by canine studies demonstrating that the 21-aminosteroid oxygen free-radical scavenging agent U74006F consistently improved neurologic functional recovery in a canine model of complete ischemia if the drug was given before, but not immediately after, ischemia.

It has long been recognized that profound hypothermia, when instituted before ischemia, improves postischemic neurologic recovery, presumably because of a reduction in neuronal metabolic requirements during ischemia. Recent data have also suggested improved recovery when modest hypothermia is instituted before or soon after a period of ischemia. This is attributed to effects in addition to metabolic depression. Verna Baughman, M.D., *et al.* (Chicago, Illinois) evaluated the effects of modest postischemic hypothermia (to 31° C) in a rat model of focal cerebral ischemia. When hypothermia was initiated 1 h after the insult, neurologic function assessed at 3 days postischemia was improved. David S. Warner, M.D., *et al.* (Iowa City, Iowa) evaluated the effect of nitrous oxide on outcome from cerebral ischemia in methohexital-treated rats. Methohexital had previously been reported to improve outcome in this model of focal ischemia, presumably through its depression of metabolism. Although nitrous oxide has been reported to increase cerebral metabolism, the addition of nitrous oxide to rats treated with methohexital did not result in a worse ischemic insult compared to that of rats treated with methohexital alone. Ira Rampil, M.D., *et al.* (San Francisco, California) reported that the new inhalational anesthetic desflurane produced EEG evidence of a dose-dependent cerebral depression, similar to that observed during isoflurane anesthesia. They did not identify epileptiform activity as has been reported in dogs given desflurane.

Daniel Cole, M.D. (Loma Linda, California) reviewed the effects of manipulating blood pressure and hematocrit on outcome after focal cerebral ischemia. Hypervolemic hemodilution is intended to improve blood rheology and blood delivery to tissues. However, despite theoretical considerations, implementation of this therapy in patients has been reported to increase the incidence of cerebral edema and myocardial infarction. In experimental preparations, induced hypertension may result in tissue preservation during focal ischemia; however, it is also associated with an increased incidence of cerebral hemorrhage. Therefore, the possible benefits of hemodilution, hypertension, or the combination of the two may be offset by complications induced by these therapies.

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