a significant reduction in the incidence of hypertension was observed. The methodology of a continuous quality improvement program can be applied to a range of anesthetic problems.

The final session of the symposium was devoted to clinical case discussions open to the entire forum.

The Eleventh International Symposium on Anesthesia and Intensive Care will be held in Israel from November 3–6, 1998. For information, registration, and abstract forms, contact Nathan Weksler, M.D., Division of Anesthesiology, Soroka Medical Center, P.O. Box 151, Beer Sheva, Israel; or the Symposium Secretariat, P.O. Box 29041, Tel Aviv, Israel, 61290.

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Society of Neurosurgical Anesthesia and Critical Care.

San Diego, California, October 17, 1997.

The Society of Neurosurgical Anesthesia and Critical Care (SNACC) held its 25th annual meeting on October 17, 1997, at the San Diego Marriott Hotel and Marina, San Diego, California. The program was prepared and organized by Patricia H. Petrozza, M.D., Vice President of SNACC.

After opening remarks by William L. Young, M.D., President of SNACC, the educational session commenced with an invited presentation by Donald W. Marion, M.D., Associate Professor of Neurological Surgery and Director of the Brain Trauma Research Center at the University of Pittsburgh Medical Center. Dr. Marion began his lecture with a brief overview of the scope of traumatic brain injury (TBI) and introduced the concept of secondary brain injury aggravated by focal or regional cerebral ischemia. To celebrate the Society's silver annual meeting, Maurice S. Albin, M.D., M.S.C., lectured on the history of neuroanesthesia in North America. As one of the original organizers and founder and the second president of the Neurosurgical Anesthesia Society (NAS), Albin provided a personal history of the evolution of NAS, to the Society of Neurosurgical Anesthesia and Neurological Supportive Care (SNANSC), which in 1986 was renamed the Society of Neurosurgical Anesthesia and Critical Care (SNACC) in recognition of the importance of critical care medicine to patients with severe neurologic dysfunction.

Each year, SNACC recognizes research by new investigators in the field of the neurosciences. This year's New Investigator's Award was awarded to E. H. Sinz *et al.* for the study entitled, "Quinolinic Acid Is Increased in CSF and Associated with Mortality in Human Head Injury." An oral abstract session and simultaneous workshops on transcranial Doppler (TCD) monitoring and evoked potential and EEG monitoring concluded the morning program.

This year the American Society of Critical Care Anesthesiologists was invited to participate in an afternoon joint session which proved to be a resounding success. The theme, "Subarachnoid Hemorrhage: A Management Update," was selected as a platform for discussion of controversial issues common to both our Societies and was effectively moderated by John C. Drummond, M.D.

A surgical perspective was provided by R. Loch McDonald, M.D., Ph.D., from the University of Chicago, who outlined the pathophysiology and development of cerebral vasospasm after aneurysmal rupture and subarachnoid hemorrhage (SAH). The many possible etiologies theorized for this arterial narrowing and delayed ischemic deficits underlie the established and evolving treatments for vasospasm, which include the use of calcium-channel antagonists, induced hypertension, hypervolemia and hemodilution, and investigational therapies (Tirilazad, a steroid derivative, and fibrinolytic agents such as tissue plasminogen activator [t-PA] used for intracisternal clot lysis).

A neurology and intensivist perspective was provided by Michael Diringer, M.D., from the Washington University School of Medicine, who discussed the controversies surrounding hemodynamic augmentation in the management of vasospasm. Acute abnormalities of cardiac rhythm and function were reviewed, and it was suggested that these disturbances could contribute to compromised perfusion. Impaired sodium and intravascular volume regulation after subarachnoid hemorrhage suggest two possible disturbances of sodium and water homeostasis requiring opposing fluid therapies. A "salt wasting" syndrome leads to volume contraction and hyponatremia and should be treated with large volumes of saline, whereas inappropriate antidiuretic hormone secretion (SIADH) should be treated with free water restriction, or if necessary, hypertonic saline.

Finally, the role of invasive neuroradiology after SAH was reviewed by Gary Duckwiler, M.D., from the University of California at Los Angeles. The Guglielmi detachable coils (GDC) have recently been introduced for the management of aneurysms. Typically, patients who are considered high surgical risk or patients with aneurysms that are technically difficult to approach surgically are considered for therapy. Complications associated with GDC therapy and their management were discussed, as well as discussion of the future prospects for this therapy.

The final afternoon session addressed, "Clinical Dilemmas in Neuroanesthesia," moderated by Patricia H. Petrozza, M.D. Rosemary A. Craen, M.D. (Canada) discussed the anesthetic considerations in providing care to patients who require motor mapping for epilepsy surgery and presented a new technique of patient self-administration of propofol using a patient-controlled delivery system. Intracerebral hemorrhage and its management in the critical care setting was discussed by Basil Matta, M.B. (U.K.). Therapeutic ICU management emphasizes the maintenance of oxygenation and cerebral perfusion and the correction of any coagulopathies. Adjuncts to guiding these therapies include ICP, jugular bulb oximetry, and TCD monitoring and brain PO_2 and microdialysis measurements in ischemia. Irene Osborne, M.D., presented the patient treatment issues involved in providing anesthetic care to children with elevated intracranial pressure presenting for magnetic resonance imaging (MRI).

In addition to the oral presentation of abstracts, the majority of presentations were in poster format, organized in a walk-around poster discussion with group leaders. Abstracts of the scientific papers presented at this meeting have been published in their entirety in the October 1997 issue of the *Journal of Neurosurgical Anesthesiology*, volume 9, number 4. Following is a highlight of some original research presented at the meeting.

Cerebral Blood Flow and Physiology

Endotoxin was shown by O. Tajiri *et al.* to produce a significant reduction in CBF accompanied by vasoconstriction after stable recovery from hypotension, when compared with hemorrhage followed by reinfusion, and previous administration of pentoxifylline restores the endotoxin-induced deterioration of CBF. A. Rebel *et al.* (Germany) demonstrated that vasodilating mechanisms can compensate for increase in blood viscosity, and above a threshold hematocrit, brain oxygen delivery is maintained. J. Marota *et al.* showed by fMRI that although local blood flow changes quickly in response to neural activation (somatosensory stimulation), spatially coupled cerebral blood volume changes occur more slowing and persist beyond the period of activation.

Several studies considered the effects of the newer inhalational agents. Animal studies demonstrated a dose-dependent decrease in cerebral vascular tone by sevoflurane and desflurane with autoregulatory mechanisms preserved at normal concentrations. In human studies, cerebral reactivity to carbon dioxide and autoregulation were shown to be preserved by sevoflurane, the cerebrocirculatory response to changes in carbon dioxide were shown to be more predictable in patients with intracranial mass lesions anesthetized with sevoflurane as compared to isoflurane, and sevoflurane exhibited less direct cerebral vasodilation than other volatile agents. The newer opioid remifentanil was shown to maintain CO_2 reactivity, although at a lower level than that shown with isoflurane, and to decrease cerebral blood flow in a dose-related fashion. Adenosine was shown to produce an early increase in CBF, in contrast to nicardipine, which uniformly increased CBF throughout grey matter.

Cerebral Ischemia and Cerebral Protection

The neuroscience community continues to strive to better define the cellular and molecular mechanisms of cerebral ischemia. The roles of NOS activity in focal ischemia, protein kinase C activity and expression after ischemia, AMPA/kainate receptor antagonist improvement in O_2 balance during cerebral ischemia, intercellular adhesion molecule-1 (ICAM-1) expression after spinal cord ischemia, $\beta 2$ integrin Mac-1 in neutrophil infiltration and necrotic cerebral cell death after transient focal cerebral ischemia, and glutamate transporter activation as a major cause of sodium accumulation were presented and discussed.

The cerebral protective effects of various anesthetic agents were also presented. In a spinal cord injury model, white matter, but not grey matter, spinal cord blood flow was reduced during anesthesia with fentanyl and isoflurane, but maintained during anesthesia with ketamine. Isoflurane in a dose producing electroencephalograph (EEG) burst-suppression reduced excitotoxic injury produced by cortical injection of NMDA. The neuroprotective effects of the barbiturates were compared and suggest that thiopental protects against brief anoxic damage by delaying membrane depolarization and preserving protein synthesis, methohexital provided greater protective effect compared with thiopental and pentobarbital, and pentobarbital and propofol do not differ in neurologic or histologic outcome, in respective studies. The role of estrogens in neural protection was the focus of several studies. Exogenous estrogen replacement therapy lessens ischemic brain injury *via* vascular mechanisms in previously oophorectomized female rats, estrogen provides perfusion-independent neuroprotection, and estrogen increases cyclic GMP in selected brain homogenates, but not in the microvascular fractions. Inhibition of neuronal nitric oxide synthase reduced transient focal cerebral ischemia associated neuropathology in diabetic rats, and selective, convective brain cooling during normothermic cardiopulmonary bypass in dogs was demonstrated.

Clinical Neurologic Sciences and Critical Care

Several studies considered cerebral hemodynamics in head injury patients. Phenylephrine-induced hypertension therapy in head-injured rats did not attenuate brain edema, reduce tissue volume, or improve neurologic outcomes. In head-injured patients treated with propofol infusion, a significant improvement of impaired cerebral autoregulation was shown. A series of positron emission tomography (PET) studies demonstrated that in acute brain injury, mild hyperventilation could increase volumes at risk for ischemic injury, and these changes may not correlate with significant desaturation as detected by jugular bulb oximetry. Hypertonic saline treatment (23.4% and 3%) effectively treated refractory intracranial hypertension or cerebral edema and led to earlier extubation in complex spine surgery. Prone operative position was shown to predispose to the development of laryngeal edema.

Pharmacology

The role of NO in anesthetic mechanisms was probed in two studies that demonstrated an indirect enhancement of anesthesia by interactions of NOS inhibitors with ligand-gated ion channels and an increase in anesthetic potency of ketamine after NOS inhibition by a possible interaction with the NMDA-NO-signal transduction pathway. Nicardipine infusion was shown in one study to be just as effective as nitroprusside for controlling blood pressure in neurosurgical patients perioperatively. Children on chronic anticonvulsant therapy were shown to demonstrate resistance to rocuronium, a nondepolarizing muscle relaxant with rapid hepatic metabolism, whereas the effect of cisatracurium, which is metabolized by Hofmann elimination, were not changed in these patients. Propofol reduced the episodes of postoperative nausea and vomiting compared to isoflurane, and ondansetron reduced nausea and vomiting only in the propofol group in one study.

Monitoring Techniques

Transcranial Doppler was used to test autoregulation and CO_2 reactivity during carotid endarterectomy (CEA), and near-infrared spectroscopy (NIRS) measurements in CEA patients correlates with drops in TCD middle cerebral artery velocities during severe intraoperative cerebral ischemia. Intraoperative EEG changes appeared to relate to the degree of postoperative cerebral dysfunction in one study. Jugular bulb venous oxygen saturation ($SjvO_2$) did not correlate with brain tissue oxygen measurements in head-injured patients during hyperventilation in another study. In a study of the electrocorticographic effects of sevoflurane and isoflurane in patients with seizure disorders, sevoflurane increased spike frequency more than isoflurane. The potential value of using electromyography (EMG) for intraoperative monitoring during spine procedures was evaluated in two studies.

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