will be funded by the federal government. Dr. Ward concluded that the challenges for departments will be funding academic time, continuing to provide meaningful clinical education, and maintaining competitive faculty salaries. Furthermore, he noted that these difficult tasks will require innovations in departmental structure.

Charles McClusky, M.D., Professor and Chairperson, Department of Anesthesiology at Texas A&M University, concluded the lecture portion of the program by discussing the qualities of effective leaders. In his discussion, he included quotations about leadership from many great leaders from history. He set forth several maxims that were central to his vision of effective leadership. Dr. McClusky thought a leader should be visible to those he/she leads, should build strong alliances, and should persuade rather than coerce. Further, he expressed his belief that integrity and tenacity were essential attributes of a good leader, along with the ability to make decisions (even if they were subsequently proved to be wrong). He said that good leaders were result-oriented, encouraged innovation, and were good communicators. He concluded with Abraham Lincoln's observation that that, although leaders can face moments of great importance, more commonly leadership is demonstrated by "exerting quiet and subtle influence on a day-to-day basis.

The afternoon session of the program concluded with several workshops. Robert Lagasse, M.D., Associate Professor of Anesthesiology at the Albert Einstein School of Medicine, led a workshop entitled "Teaching Quality Assurance and Peer Review." During the workshop, he reviewed theories and techniques of performance improvement and quality management and then demonstrated how they can be taught in anesthesiology training programs. Meg Rosenblatt, M.D., Assistant Professor of Anesthesiology at Mount Sinai School of Medicine led a workshop entitled "Organizing a Faculty Development Workshop: Preaching to the Unconverted." During her presentation, Dr. Rosenberg reviewed the components of an effective faculty development workshop and discussed ways to implement them. The third workshop, led by Dr. Guyer was an interactive extension of her lecture about recognizing learning disabilities in medical students and graduate physicians and helping the students with these problems to succeed.

Also during this meeting, the second Society for Education in Anesthesia/Duke Prize for Excellence in Anesthesiology Education was presented to Robert Willenkin, M.D., Emeritus Professor of Anesthesiology at the University of Pittsburgh. Dr. Willenkin, who is a founding member of SEA, was honored for his contributions to the advancement of education in anesthesiology. The prize is made possible by a grant from the Department of Anesthesiology at Duke University.

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Society of Neurosurgical Anesthesia and Critical Care. Orlando, Florida, October 16, 1998.

The Society of Neurosurgical Anesthesia and Critical Care (SNACC) held its 26th Annual meeting at the Omni Rosen Hotel, Orlando,

Florida, on October 16, 1998. The program was organized by Jeffery Kirsch, M.D., who made note of several changes in this year's program format which reduced the number of simultaneous events and allowed greater participation in the poster discussions.

After president Arthur Lam's opening remarks, the Society of Neurosurgical Anesthesia and Critical Care educational program began with Dr. Michael V. Johnston, Professor of Neurology and Pediatrics, from Johns Hopkins University, who presented a lecture on "Mechanisms of Brain Injury in Infants and Children. Is Brain Maturity an Important Factor?" Dr. Johnston reviewed the pathogenesis of brain injury and how this insult is manifested differently in the infant compared to the adult. He also presented evidence to demonstrate an age-dependent vulnerability of different brain structures to hypoxia, with similar insults producing different clinical outcomes depending on the age of gestation.

After this presentation, all members attended the walk-around poster discussion session. The majority of research presentations were in this format and divided according to subject matter. Abstracts of all the scientific papers presented at the meeting have been published in the *Journal of Neurosurgical Anesthesiology* (1998; 10(4):257–92).

A summary of some of the scientific research presented follows:

Physiologic Mechanisms of Cerebral Blood Flow

Several studies evaluated the ability of the brain to autoregulate blood flow. DeWitt et al. evaluated myogenic responses to changes in intraluminal pressure in rodent middle cerebral arteries harvested after traumatic brain injury. They found myogenic vasodilatory responses to decreased intraluminal pressure to be reduced within 5 min of brain injury and to persist for at least 30 min afterward. Marota et al. studied cerebral blood volume during autoregulation using functional magnetic resonance imaging sequence and contrast agents and tested the hypothesis that cerebral blood volume was determined passively by cerebral blood flow (CBF) using the hemorrhage-induced hypotension model in a rat. This study showed that neither total nor microvascular cerebral blood volume increased significantly with blood pressure decreases even though CBF decreased significantly. This suggests that cerebral blood volume does not increase with vasodilatation associated with decreased cerebrovascular resistance. Another study evaluated the effects of cyclic guanosine monophosphate on blood-brain barrier permeability and found that its direct application on the cerebral cortex significantly increased blood-brain barrier permeability and suggests that the regulatory effect of nitric oxide on vascular and blood-brain barrier permeability is directly linked to its ability to increase cyclic guanosine monophosphate.

Other studies evaluated the effects of anesthetic agents on CBF. One study evaluated isoflurane's effect on the alteration and desensitization kinetics of the γ -aminobutyric acid A (GABA_A) receptor channel and found that differing concentrations of GABA affect the channel differently in the presence of isoflurane. Another study evaluated the effects of propofol or halothane on hypercapnic cerebral hyperemia using laser Doppler flowmetry and found no effect. However, a study by Lu et al. looked at the effect of sevoflurane and desflurane on the synthesis of nitric oxide and found that 2 minimum alveolar concentration (MAC) sevoflurane increased nitric oxide, but 1.5 MAC desflurane caused no effect. Desflurane had little effect on nitric oxide synthetase within the given dose range and suggests that vasodilation with more than 1 MAC desflurane is not related to the nitric oxide pathway.

Cerebral Blood Flow Clinical Studies

Many of the clinical studies presented this year concerning CBF evaluated remifentanil and its effect compared to other anesthetic agents. Doyle et al. (United Kingdom) compared remifentanil to fentanyl and their effect on hemodynamic changes during carotid endarterectomy and found both to be similar. Likewise, Leary et al. compared remifentanil, alfentanil, and fentanyl for craniotomy and found all three to confer equal intraoperative cardiovascular stability, with remifentanil producing a more-rapid emergence. Möllenberg et al. (Germany) evaluated the effect of remifentanil/propofol versus isoflurane on dynamic cerebral autoregulation and found that the opioidbased anesthetic did not affect cerebrovascular autoregulation compared to 1.5 MAC isoflurane. Likewise, another study found that the addition of varying concentrations of remifentanil to a sevofluranebased anesthetic had no effect on cerebral autoregulation. Several studies evaluated the effect of traumatic brain injury or CBF. Jackson et al. attempted to quantify variations in cerebrovascular reactivity in head-injured patients. This study showed that intracranial pressure monitoring was more responsive to a physiologic challenge than transcranial Doppler flow velocity. Another study by Metz et al. (Germany) determined the accuracy of jugular bulb oxygen saturation as a method to predict CBF in head-injured patients. A significant correlation was found between jugular bulb oxygen saturation and CBF during nonischemic conditions, but this correlation was nonexistent when cerebral ischemia was present. Other studies evaluated cerebral oximetry and evoked-potential monitoring during carotid endarterectomy and the clinical use of cerebral oximetry.

Cerebral Ischemia and Cerebral Protection

Several abstract sessions were dedicated to cerebral ischemia and methods for cerebral protection. Molecular mechanisms of cerebral ischemia were presented. Kimbro et al. evaluated the relation between cortical spreading depression and an ischemic tolerant state in rat brain. Mitogen-activated protein kinase activation was shown to result in new gene transcription in response to neuronal stimulation. This activation may play a role in the development of cortical spreading depression and induced ischemic tolerance. Several other studies evaluated the effect of anesthetic agents on prevention of neuronal injury after focal ischemia. In addition. one study by Kelly et al. noted that isoflurane and pentobarbital, when given to produce burst suppression, prevented neuronal injury to the neurotoxin AMPA. This was similar to NBQX, a specific AMPA antagonist, and was thought to be caused by the ability of the anesthetic agents to antagonize the AMPA receptor. Several other studies showed propofol to have cerebroprotective properties. It reduced infarct size in rats undergoing focal ischemic challenge compared to the awake state. The study of Wilson et al. (Ontario, Canada) showed that propofol prevents and reverses the inhibition of excitatory amino acid uptake in astrocytes subjected to oxidative stress. This property of propofol may inhibit the increase in extracellular glutamate that occurs with ischemia and may be of benefit in delaying the onset of excitatory neuronal death. Other new neuroprotectants, such as 4-phenyl-1-(4 phenyl) piperidine (PPBP), were also studied and presented at this years' meeting. The effect of this drug was found to be time dependent. PPBP attenuates basal and NMDA-evoked nitric oxide production, producing neuroprotection from focal ischemia. Several clinical studies were also presented that evaluated cerebroprotective techniques and what effect the type of

temporary clipping used for aneurysm surgery had on postoperative outcome. This study concluded that no protective technique had a discernible effect on outcome. Another study evaluated the systemic and cerebral hemodynamic changes that occur with mild hypothermia in patients undergoing craniotomy and found that the cardiac index and cerebral oxygenation decreased.

Other studies were presented that evaluated the effect of different therapies for treatment of closed head trauma. The assessment of neurologic injury after liver transplantation and intracranial vascular surgery using \$100 protein determinations were also discussed in this session.

Clinical Neurosciences

Several studies were presented that evaluated anesthetic requirements and anesthetic methods for different neurosurgical procedures. Osborn *et al.* evaluated the use of the intubating laryngeal mask for airway management in neurosurgery patients and Rovira *et al.* (Spain) evaluated the prognostic value of hyperglycemia after subarachnoid hemorrhage and found a blood glucose level of 160 mg/dl to be associated with a poor outcome. It was concluded that this may be used as an outcome prognostic variable. Other investigators presented data concerning the neutral effect of remifentanil on intracranial pressure and the ability of isoflurane to induce paradoxical increases in electroencephalographic bispectral index, which may represent drugspecific central nervous system excitatory phenomena. One study used exhaled pentane, a straight-chain aliphatic hydrocarbon, as a marker for free radical-induced lipid peroxidation and found it to be elevated in patients with cerebral ischemia.

Pharmacology

The prevention of nausea and vomiting after craniotomy has received increased attention, with several studies evaluating both the incidence and the effect of ondansetron on postcraniotomy nausea and vomiting. Other studies evaluated cerebral oximetry during circulatory arrest and cardiac surgery and in head-injured patients. One study measured a novel marker for neurologic injury-neuron-specific enolase—after carotid endarterectomy and found levels to be elevated 24 h after surgery in patients who had neurologic injury. It was postulated that this enzyme marker might be used in the future to detect mild neurologic injury after these procedures. Another study evaluated procalcitonin, a marker for infection, to determine whether aspiration had occurred in head-injured patients. They found procalcitonin to be significantly elevated in patients with clinical signs of aspiration compared to those who did not aspirate. This may be used as a method for early detection of aspiration in unresponsive comatose patients and enable a more rapid initiation of therapy to affect survival.

Monitoring

No effect of clonidine on bispectral electroencephalograpy-guided anesthetic requirements were shown in one study, whereas mild hypothermia did not effect the bispectral index during cardiopulmonary bypass. Other studies of jugular bulb monitoring techniques were presented, including one that showed jugular bulb temperature to be a useful tool for monitoring global brain temperature. Additional stud-

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ies evaluating the effect of isoflurane on transcranial electrical and magnetic motor-evoked responses were also presented.

After the poster discussion session the educational program continued with the presentation of the New Investigators Award. Every year the society recognizes the research of a new investigator. This year G. B. Mackensen *et al.* from Duke University received the award for research involving "Sympathetic Ganglionic Blockade Masks Beneficial Effect of Isoflurane on Histologic Outcome from Near Complete Ischemia in Rats." This study showed an isoflurane anesthetic to have a better neuroprotective effect compared to fentanyl-nitrous oxide because of isoflurane's ability to modulate plasma catecholamine levels that normally increase after an ischemic insult to the brain. This effect was lost when trimethaphan, a ganglionic blocker, was added.

Oral abstract presentations, moderated by Dr. Hans Hennes (Mainz, Germany) closed out the morning session. Four laboratory studies and one retrospective clinical study were presented. Three of the laboratory studies dealt with cerebral injury. The first, presented by Soriano et al. examined P and E selectin-deficient mice and their susceptibility to cerebral ischemia-reperfusion injury. The second presented by R. G. Giffard et al. (Stanford) evaluated the neuroprotective effect of heat shock protein (HSP70) on cellular survival during cerebral ischemia. The third paper presented by Dr. Jevtovic-Todorovic (Washington University, St. Louis) evaluated the effect of prolonged nitrous oxide exposure on rat neurons. The study showed that neurons exposed to anesthetic concentrations of nitrous oxide for 4-8 h had no apparent damage, but the same neurons exposed to similar concentrations of nitrous oxide for 16 h were damaged or destroyed. Future studies to determine the exact threshold times when this toxicity will occur are forthcoming. The final laboratory study was presented by Dr. Himmelseher (Munich, Germany) and dealt with the effect of hypertonichyeroncotic saline on healthy and damaged hippocampal neurons and astrocytes. These studies determined that a 1-h exposure time to hypertonic saline decreased the survival of injured neurons and increased the amount of water space in healthy ones. The final oral presentation presented by Dr. Waggoner (Mayo Clinic) was a retrospective analysis of 1,115 patients and dealt with changing transfusion practices for carotid endarterectomy. This study analyzed the effect these changes had on morbidity and mortality after surgery and found no appreciable increase in the incidence of stroke or myocardial infarction in the group who received less transfusion and whose hematocrit levels were lower compared to a past cohort who were transfused sooner and maintained at higher hematocrit levels.

After a combination lunch and business session the afternoon program began with a pro- con debate moderated by Dr. Donald Prough on the use of hypertonic saline and whether its perioperative use is appropriate in the neurosurgical patient. Dr. Christian Spiss (Vienna)

served as the protagonist and Marek Mirski, M.D., Ph.D. (Honolulu) as the antagonist in this lively debate that emphasized both the positive and the negative aspects of hypertonic saline use during neurosurgery. Although it reduces brain-water content without depleting intravascular volume, its use may produce seizures, hemolysis, CHF, and central pontine myelinolysis, which supports the consensus that hypertonic saline be used with caution when treating cerebral edema.

Additional time was then allotted for poster viewing and discussions with the presenters. Immediately afterward, Dr. Daniel Cole presented a review of synthetic hemoglobin and some of the research regarding these blood substitutes in the treatment of cerebral ischemia. Therapeutic applications of these synthetic hemoglobins were discussed, as well as some experimental evidence regarding the interaction of these synthetic hemoglobins with nitric oxide.

The final afternoon session, moderated by Dr. William Fitch (Glascow, Scotland), addressed "Neuromonitoring. Is the benefit worth the cost?" Dr. Rene Tempelhoff (St. Louis), the protagonist, argued that the development of new technology, although expensive and not always perfect, has in many circumstances improved outcomes to some neurosurgical procedures. He also cited example of how science has been affected throughout history by certain political and economic forces and that these same forces may again be acting against the advancement of science. Dr. Gregory Crosby (Boston), the antagonist, argued that the technology used today may be of limited benefit because it has not be substantiated by good controlled outcome studies. He was concerned that the reliance on monitors for clinical decision-making may transform our judgment and reduce our skills as clinicians. The floor was open for discussion and many viewpoints were considered with a final consensus reached that neurophysiologic monitoring, although not totally proven, is still a useful tool, and further technologic development should be supported.

The educational program concluded with the election of officers, followed by the traditional wine and cheese mixer. Workshops for somatosensory- and motor-evoked potentials, electroencephalographic monitoring, transcranial Doppler, cerebral oximetry, and jugular venous oximetry followed for those who wanted hands-on experience and tips from the experts who routinely use these monitors.

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