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Society of Neurosurgical Anesthesia and Critical Care Newsletter

The 25th Annual Meeting of SNACC, October 17, 1997

SYMPOSIUM PRECEDING THE MEETING: ALPHA$_2$ AGONISTS IN NEUROANESTHESIA

The symposium was chaired by Adrian Gelb, M.B., Ch.B. (London, Ontario, Canada). The symposium began with a presentation entitled "The Pharmacology of Alpha$_2$ Agonists" by Mervyn Maze, M.B. (Palo Alto, CA, U.S.A.). Pekka O. Talke, M.D. (San Francisco, CA, U.S.A.) followed with the discussion "Clinical Experience with Alpha$_2$ Agonists and Potential Role in Neuroanesthesia." The final presentation, by William E. Hoffman, Ph.D. (Chicago, IL, U.S.A.) covered "Alpha$_2$ Agonists and Cerebral Ischemia." The dinner symposium was sponsored by SNACC and made possible by an unrestricted educational grant from Abbott Laboratories. Clonidine, an imidazole, is a selective alpha$_2$ adrenergic agonist (200:1 alpha$_2$:alpha$_1$) used to treat hypertension. The alpha$_2$ agonist effects of clonidine include sedation, anxiolysis, analgesia (not reversed by naloxone), MAC reduction, attenuation of stress-induced sympathoadrenal responses, bradycardia, and hypotension. Dexmedetomidine (dex) is an imidazole compound superselective for the alpha$_2$ agonist (1600:1 alpha$_2$:alpha$_1$). Dex has been shown to decrease MAC of halothane by over 95% in animal studies. Three subtypes of alpha$_2$ receptors have been described. Recently, the alpha$_2$A adrenoreceptor subtype has been demonstrated to be responsible for the hypnotic response to dex in rat locus ceruleus.

Certain effects of alpha$_2$ agonists, including sedation, anxiolysis, and a decrease in MAC without causing respiratory depression, make these compounds an attractive adjuvent for clinical neuroanesthesia. Clonidine has been used effectively to attenuate hemodynamic changes associated with headholder placement in neurosurgical patients. Dex was recently shown to have no effect on lumbar cerebrospinal fluid pressure in patients with normal intracranial pressure; cerebral perfusion pressure was, however, decreased when compared to the placebo group. Further investigation of this drug is needed to assess the potential utility of dex in the clinical neurosurgic setting.

Several studies have demonstrated that tissue catecholamines increase during cerebral ischemia, suggesting a role for the sympathetic nervous system in ischemic injury. In a rat model of incomplete ischemia, anesthetics (eg, propofol, ketamine) improved neurologic outcome when compared to nitrous oxide/narcotic control. This improvement was unrelated to changes in CBF or CMRO^2. Ganglionic blockade in the same model decreased catecholamine levels and improved outcome, while an infusion of exogenous catecholamines reversed the protective effects. Administration of clonidine and dex have been shown to improve neurologic outcome in rats subjected to incomplete cerebral ischemia.

A likely mechanism of the protective effect of alpha$_2$ agonists involves the control of sympathetic outflow by excitatory cholinergic receptors and inhibitory alpha$_2$ receptors. In support of this hypothesis, physostigmine increased catecholamine levels and worsened outcome in the incomplete ischemia rat model, while dex attenuated these changes. In conclusion, sympathetic nervous system stimulatory effects during hypoxic ischemia are involved in unanesthetized brain injury. Alpha$_2$ agonists improve outcome in this setting by decreasing sympathetic tone.

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THE 1997 MEETING

The 25th Annual Meeting of the SNACC was held on October 17, 1997 at the San Diego Marriot Hotel Marina in San Diego, California. This year's program was organized by Patricia H. Petrozza, Vice Present of SNACC.

After opening remarks by SNACC's President, William L. Young, M.D., the educational session commenced with an invited lecture by Donald W. Marion, M.D. (Pittsburgh, PA, U.S.A.) entitled "Cerebral Blood Flow and Autoregulation Following Head Injury - What Do We Know? What Does It Mean for Patient Care?" After a brief overview of the scope of traumatic brain injury (TBI) and the concept

of secondary brain injury aggravated by focal or regional cerebral ischemia, Dr. Marion reviewed the post-traumatic abnormalities in cerebral blood flow following severe traumatic brain injury and the relationship of cerebral blood flow to cerebral blood volume. The issue of hyperventilation therapy for the treatment of intracranial hypertension and the associated risk for cerebral ischemia was discussed in terms of recent studies. The concepts of pressure autoregulation and cerebral perfusion pressure were presented in the context of TBI and the impact of current theories on patient management. While in the past the management of severe brain injury emphasized the control of brain swelling, therapy in the 90's emphasizes the preservation and enhancement of functional recovery of the surrounding brain tissue.

In celebration of the Society's Silver Anniversary, a lecture on the history of the genesis of neuroanesthesia in North America was provided by Maurice S. Albin, M.D., M.S.C. (Anes), (San Antonio, TX). Dr. Albin, one of the original organizers and founders of the Neurosurgical Anesthesia Society (NAS), and also its second president, presented a nostalgic outline of the evolution of NAS to SNANSC and ultimately, to SNACC. He then heralded past significant advances and discoveries in the field of the neurosciences with the contributions of numerous neurosurgeons, neuroanesthesiologists, and neuro-intensivists. The uniqueness of the Society was emphasized by the collaborative effort between subspecialties, and the substantial contributions, early on, by female members in leadership roles.

The New Investigator's Award was introduced by Arthur M. Lam, M.D., F.R.C.P.C., President-Elect of SNACC. Entitled, "Quinolinic Acid is Increased in CSF and Associated with Mortality in Human Head Injury," the authors, Sinz, et al, kept with the earlier topic of traumatic brain injury. They described the elevation of CSF quinolinic acid (a tryptophan-derived NMDA agonist produced by macrophage and microglia) in many brain-injured patients which peeked at 72-83 hours after TBI with a higher concentration being associated in non-survivors. This raises the possibility that quinolinic acid may contribute to the pathophysiology of secondary brain injury. Interestingly, the study demonstrated no effect on these levels after 24 hours of therapeutic hypothermia.

The morning concluded with an oral abstract session and simultaneous workshops. Oral presentations moderated by Patrick Ravussin, M.D., (Sion, Switzerland) included basic science studies and clinical research presentations. The findings of M. Scheller, et al, lead to the hypothesis that direct interactions of nitric oxide synthase inhibitors with ligand-gated ion channels in the central nervous system may add to indirect enhancement of anesthesia. H. M. Koenig, et al demonstrated that the inhibition of neuronal nitric oxide synthase reduces transient focal cerebral ischemia associated with neuropathology in diabetic rats. Another study presented by R. Rusa, et al, demonstrated that exogenous estrogen replacement therapy lessens ischemic brain injury in previously oophorectomized female rats.

Three clinical studies were part of the oral presentations, and included an analysis by J. R. Waggoner, III, et al., which determined the efficacy of selective convective brain cooling during normothermic cardiopulmonary bypass in dogs. In a series of positron emission tomography (PET) studies, P.S. Minhas, et al, concluded that in acute brain injury, mild hyperventilation could increase tissue volumes at risk for ischemic injury, and, that these changes may not correlate with significant desaturation as detected by jugular bulb oxymetry. This study generated discussion with regard to issues of CBF vs $CMRO^2$ needs, and whether these findings reflected hypoperfusion or ischemia. M. Szabo, et al, determined prospectively that the prone operative position predisposes to the development of laryngeal edema as evaluated by video laryngoscopy, endotracheal cuff leak, and spirometry. In the patient population studied, the airway edema did not appear to be clinically significant.

Two workshops, popular last year, were offered once again. Marc J. Bloom, M.D., Ph.D., Michael E. Mahla, M.D., Tod B. Sloan, M.D., Ph.D. and James N. Rogers, M.D., led workshop A, "Evoked Potential/EEG." Workshop B, "Transcranial Doppler" was spear-headed by Arthur M. Lam, M.D., F.R.C.P.C., and Christian P. Werner, M.D.

Following lunch, the Walk-Around Poster Discussion with Group Leaders once again generated lively discussion and opportunities to analyze data in a more focused forum. Posters were grouped topically, as follows: cerebral blood flow/physiology; monitoring; clinical neurological science/pharmacology; cerebral blood flow/pharmacology; drugs and techniques; cerebral ischemia/molecular biology; cerebral protection/pharmacology; monitoring/spinal cord; cerebral protection/techniques; cerebral blood flow/TCD. The abstract of all oral and poster presentations were published in the *Journal of Neurosurgical Anesthesiology* 1997;9:378–402.

The afternoon joint session with the American Society of Critical Care Anesthesiologists was a resounding success. The topic, "Subarachnoid Hemorrhage: A Management Update" was selected as a common interest to both Societies, and was moderated by John C. Drummond, M.D., (San Diego, CA). The neurosurgical perspective was provided R. Loch McDonald, M.D., (Chicago, IL) and featured an overview of the pathophysiology and treatment of delayed ischemic deficits after aneurysmal subarachnoid hemorrhage. Michael Diringer, M.D., (St. Louis, MO)

provided the neurological/intensivist perspective by discussing the systemic and cerebral circulatory disturbances following subarachnoid hemorrhage, and extrapolating the pathophysiology to implications for management. The role of invasive neuroradiology after subarachnoid hemorrhage was discussed by Gary Duckwiler, M.D., (Los Angeles, CA) in terms of diagnostic and therapeutic methods. The question and answer period subsequent to these presentations highlighted the value a combined forum for discussion of controversial issues in this area.

The final afternoon session addressed "Clinical Dilemmas in Neuroanesthesia"and was moderated by Patricia Petrozza, M.D. An anesthetic protocol for motor mapping in an adult, using a new technique of patient self-administration of propofol via a patient-controlled delivery system, was outlined by Rosemary A. Craen, M.B., B.S., F.A.N.Z.C.A., (London, Canada). The management of intracerebral hemorrhage was reviewed by Basil Matta, M.B., F.R.C.A., (Cambridge, U.K.), and some of the issues addressed briefly by previous speakers, in terms of the pathophysiology of acute brain injury, were revisited and applied in practical terms to state-of-the-art ICU management. Irene P. Osborn, M.D., (New York, NY) offered insight into the pitfalls associated with the anesthetic care of pediatric patients for Magnetic Resonance Imaging.

Following a highly successful educational symposium, the 25th Annual SNACC Meeting was concluded with a business meeting.

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