Society of Neurosurgical Anesthesia and Neurologic Supportive Care

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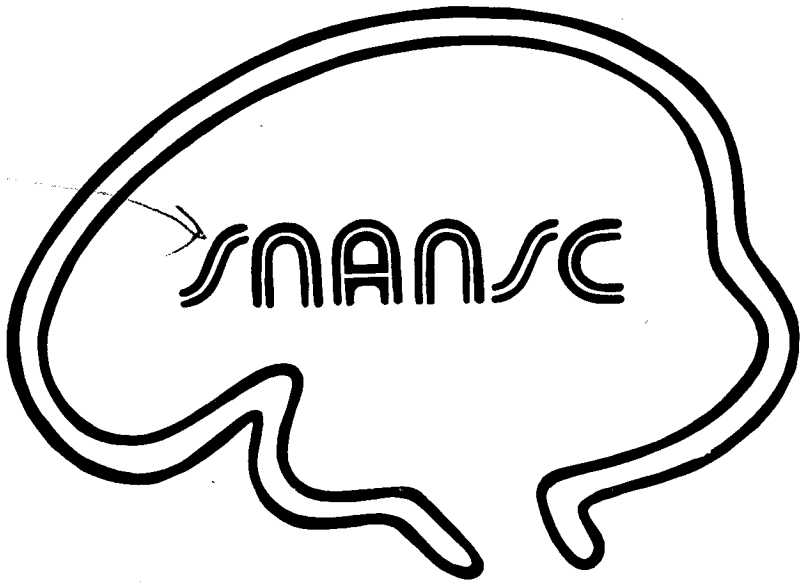

1986 Spring Newsletter

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The President's Corner

Probably the big news for this letter is that the annual meeting program has been finalized. I hope you'll agree that it looks like another winner, but if you feel that an area of your particular interest has been omitted, let me or Wayne know and we'll try to include it next year.

If you are looking at that May 1 abstract deadline wondering whether to submit your American Society of Anesthesiologist paper to the Society of Neurosurgical Anesthesia and Neurologic Supportive Care as well, let me encourage you to do so. The American Society of Anesthesiologist supports the concept of prior presentation of papers to "small subspecialty societies for peer review," and that includes us. Over the years I certainly feel that I've gotten far more constructive input by presenting my work to 200+ colleagues at the Society of Neurosurgical Anesthesia and Neurologic Supportive Care than I have from talking in a small conference room at the American Society of Anesthesiologist meeting.

In addition to gathering together the ingredients for another outstanding annual meeting, Wayne Marshall also has done yeoman service traveling the vacation outposts of the USA looking for the optimal location for our first annual "Fun in the Sun" midwinter neuroanesthesia conference in 1987. At press time it looks like this outstanding CME event will be taking place at Marriott's fabulous Casa Marina resort in Key West, Florida. For those of you who can't make it to the Annual Meeting in Las Vegas, this will be an ideal opportunity to saturate on everything new in clinical neuroanesthesia skills. More information will follow in the summer newsletter.

Another innovation is the Society's first Educational Forum included in this newsletter. David Smith has done a fine job of discussing the issue of glucose control in neurosurgical patients, a topic of ongoing interest and controversy. Hopefully, these columns can become a regular feature of the Newsletter and will serve to expand on the clinical care debates that are frequently covered during the Annual Meeting panel discussions.

If you are reading this newsletter and are not a member of the Society, let me urge you to join by contacting our headquarters in Richmond. In addition to the quarterly newsletter and a reduced registration fee for the Annual Meeting, the Society also distributes an annually updated comprehensive bibliography of articles pertinent to the neurosciences and maintains a yearly revised summary of neuroanesthesia and neurological critical care fellowship opportunities throughout North America. To quote from a letter I recently received from Neal Kassell, "This little society accomplishes more than any large organization I've ever belonged to."

Wishing you all a warm, sunny and productive Spring season.

Bob

Robert F. Bedford, M.D.

1986 ANNUAL MEETING

JANSSEN DISTINGUISHED LECTURER ANNOUNCED

The Society is pleased to announce that this year's Janssen Distinguished Lecturer will be Dr. Alan I. Faden, Professor and Vice-Chairman of The Department of Neurology at the University of California, San Francisco, VA Medical Center. Dr. Faden has been at the forefront of research pertaining to the effects of endogenous opioids and opiate antagonists in a variety of neuropathological processes. He will speak to us on *"Opioids, Opiate Antagonists, and CNS Injury"*, certainly a topic of major clinical and research interest to members of our Society.

Although Janssen has been a major supporter of our Society for many years, we are particularly happy to have received their commitment on behalf of our Distinguished Lecturer Program for this year's and future annual meetings.

1986 ANNUAL PROGRAM FINAL

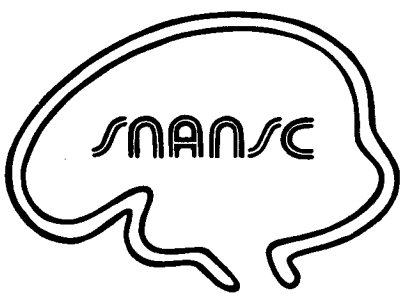
Enclosed with this newsletter is the final outline for the 1986 annual Society of Neurosurgical Anesthesia and Neurologic Supportive Care meeting to be held at the Las Vegas Hilton, Las Vegas, Nevada, Thursday, October 16, 1986. On behalf of the Society, I wish to thank all participants for their efforts.

Wayne Marshall, M.D.

1987 WORKSHOP ON NEUROANESTHESIA

Plans for the 1987 workshop on neuroanesthesia are nearing completion. The dates for the meeting are February 19-22, 1987. The venue is the Marriott Casa Marina Hotel in Key West, Florida. Travel arrangements, a full program, and further details will be sent to you shortly.

Wayne Marshall, M.D.



The Forum

The following is a permanent column of the Newsletter collated and edited by David S. Smith, M.D., Ph.D. Responses are encouraged and will be printed as feasible.

Editor

INTRODUCTION

The Forum is an experiment, an attempt to create an informal exchange of information and ideas of interest to those engaged in the perioperative management of patients with neurosurgical disease, head injury, or spinal cord injury. If it proves useful or of interest to the membership, The Forum will appear in each issue of the Society of Neurosurgical Anesthesia and Neurologic Supportive Care newsletter.

The Forum will provide informal comment on various issues that are current or controversial. Its format will be varied but aimed at the level one might expect from discussion with a knowledgeable associate over lunch or in the operating room.

The first offering of The Forum concerns the use of intraoperative glucose. The editor solicits the readers' comments concerning this offering and your reaction to the concept of The Forum. He would also like to solicit your contributions for future editions of The Forum. Suggestions for future topics or specific questions you would like discussed are also welcome.

Note that The Forum is not peer reviewed and reflects only the opinion of the author. The opinions expressed are not the official views of the Society of Neurosurgical Anesthesia and Neurologic Supportive Care or its officers.

INSTRUCTIONS FOR CONTRIBUTORS

Contributions to The Forum should be short (about 2-8 double spaced pages). They may be on any topic that might be of interest to the membership of the Society of Neurosurgical Anesthesia and Neurologic Supportive Care. Discussion of interesting or unusual cases or interesting approaches to problems are encouraged. Key references and sources for further information should be cited, but exhaustive bibliographies avoided. Contributions should be typed and submitted in duplicate. They may contain a table, but no figures. Correspondence concerning material appearing in The Forum will also be published as space permits. Contributions selected for publication will be edited for clarity and length, but they will not undergo formal peer review.

Correspondence and contributions should be sent to The Forum, c/o David S. Smith, M.D., Department of Anesthesia, Hospital of the University of Pennsylvania, 7 Dulles Building, 3400 Spruce Street, Philadelphia, PA 19104.

INTRAOPERATIVE GLUCOSE

David S. Smith, M.D.
Department of Anesthesia
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There is substantial experimental evidence suggesting that the administration of glucose prior to an episode of hypoxia or ischemia will exacerbate subsequent brain damage; this evidence is consistent over a wide range of experimental animals and experimental situations of ischemia and hypoxia. For example, Siesjo's group used a rat model of incomplete ischemia consisting of bilateral carotid ligation and hypotension for 30 minutes followed by restoration of blood pressure and flow for 90 minutes. In rats pretreated with glucose, there was failure to recover brain high energy metabolites,[1] increased histologic damage[2] and poorer regional cerebral blood flow in the recovery period[3] compared to animals given the equivalent volume of saline. Blood glucose in the treated animals was high, about 800 mg/dl, just prior to the onset of ischemia.[1] Pulsinelli, et al,[4] used 4 vessel occlusion in rats to produce ischemia and found markedly increased brain damage in animals given glucose in doses that produced blood levels of about 300 mg/dl compared to 168 mg/dl in saline treated controls. Siemkowitz and Hansen,[5] using a model of complete ischemia by neck compression in rats, found that glucose pretreatment produced an exacerbation of damage. Ginsberg and collaborators[6,7] had similar findings in cats subjected to incomplete ischemia. Most recently, Lanier, et al,[8] studied complete ischemia in monkeys and found that glucose administration, even in the absence of frank hyperglycemia, led to increased brain damage and that the degree of damage was proportional to the blood glucose level.

Evidence for the same phenomenon in man is less clear. Melamed[9] noted that hyperglycemia occurred in acute stroke, and that the degree of glucose elevation was related to the severity of the stroke. Patients in coma after stroke had high blood glucose levels, and hospital mortality was higher in these patients. Pulsinelli, et al,[10] noted, in a retrospective study, a relationship between admission blood glucose and severity of stroke. In a prospective study by the same group, 13 patients (76%) with admission blood glucose less than 120 mg/dl had good neurologic outcome while 8 patients (57%) with admission glucose above 120 mg/dl had poor neurologic outcome.[10] Though suggestive, these differences were not statistically significant. Finally, Longstreth and Inui[11] did a retrospective review of patients after cardiac arrest and found definite correlations between neurologic outcome and blood glucose. Good prospective studies involving the perioperative period are lacking but would be difficult to obtain, considering the degree of insult that appears to be required to see the augmentation of brain damage by glucose. However, the marked and constant findings in animals has led some to suggest that patients at risk for intraoperative ischemia not be given glucose.[12] This would include patients for carotid endarterectomy and cerebral aneurysm clipping. One might also include patients for brain tumor resection, considering the fact that brain retractor pressure may compromise cerebral perfusion.

One problem with this recommendation is the possibility that withholding glucose in a fasted patient may lead to intraoperative hypoglycemia, which in itself may cause brain damage. Recently,

THE FORUM Cont'd

Sieber, et al,[13] reported that craniotomy patients given about 1 liter of 5% glucose over 4 hours developed blood glucose levels of about 230 mg/dl, while patients given saline had blood glucose levels of about 160 mg/dl. They also noted that hypoglycemia did not occur in any of the patients studied (16), findings consistent with others that have studied blood glucose levels in the operating room. A need for intraoperative glucose may exist for unstable diabetics, diabetics on oral hypoglycemic agents, and those recently on hyperalimentation, but the typical patient may not require intraoperative glucose, at least for operations of 4 hours or less. Thus, my associates and I have joined others in using non-glucose containing solutions during operations on patients at risk for intraoperative ischemia. There does not appear to be a major risk for withholding glucose, and the possibility exists that some patients may benefit.

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1985 ANNUAL MEETING REPORT

The following is a summary of the 1985 Society of Neurosurgical Anesthesia and Neurologic Supportive Care meeting in San Francisco October 11, 1985, submitted by David S. Warner, M.D., Assistant Professor, Department of Anesthesia, University of Iowa.

The 13th Annual Meeting of the Society for Neurosurgical Anesthesia and Neurologic Supportive Care was held at the San Francisco Hilton on October 11, 1985. The meeting focused on several areas of current interest including 1) research advances in cerebral physiology and protection, and 2) intraoperative electro-physiologic monitoring as related to neuroanesthesia.

In response to the current interest concerning glucose metabolism during cerebral ischemia two reports were given. Lanier et al. (Mayo Clinic) studied the effects of infusion of modest doses of dextrose prior to reversible complete ischemia in pig-tailed monkeys by measuring neurologic and histopathologic outcome. Plasma glucose was not significantly elevated when compared to control animals receiving lactated ringers, but brain injury was more severe in those animals receiving dextrose. A correlation was found between plasma glucose levels and extent of injury, although all animals had glucose values less than 250 mg/dl. This data suggests that preoperative administration of dextrose containing solutions, while not necessarily causing hyperglycemia, may worsen post-ischemic neurologic outcome. In another report, Kofke et al. (Penn State, Hershey) analyzed brain metabolites during 1 MAC (end-tidal) anesthesia in the rate. Brain energy stores were similarly preserved for enflurane, halothane, and isoflurane. Plasma and brain glucose levels, however, were greater in the isoflurane anesthetized group suggesting that cerebral lactic acidosis and brain damage could be worse if isoflurane is given in the setting of anaerobic metaboslism.

Relevant to the effects of anesthesia on intracellular metabolism, Litt et al. (San Francisco) reported on the interaction between halothane and cerebral high energy phosphates during hypercarbia. As measured by NMR spectrometry, hypercarbic rats ($PaCo_2$ = 200 mmHg) were shown to have a two-fold increase in phospocreatinine when halothane (0.5%) was present despite adequate arterial oxygenation and mean arterial pressure. ATP was unaffected by halothane while ADP was reduced by 50% suggesting that halothane may interfere with the regulation of ATPase.

Technological advances in electroencephalographic (EEG) monitoring have allowed increased intraoperative availability of this modality to anesthesiologists. Dr. Grundy (Gainesville) presented information concerning the basic theoretical concepts of processed EEG signals including effects of physiologic changes and anesthetics on the EEG. In this rapidly evolving field, the role of the anesthesiologist in interpreting intraoperative EEG monitoring during cases where this service is traditionally provided by a neurologist remains undefined and bore discussion. It was also pointed out that EEG monitoring by the anesthesiologist in hospitals where this is not routinely performed, specifically during carotid endarterectomies, would constitute an improvement in patient care. An example of the value of application of this modality was presented by B. Kim (Yale). During lateral rotation of the head necessary for placement of jugular venous catheters or surgical positioning, cortical electrical activity was monitored by a five-lead processed EEG. Fifteen out of 28 patients showed significant decreases in alpha and beta spectral activity. Although no patient demonstrated post-operative neurologic deficits, these changes may have represented subclinical cellular hypoxia.

Paralleling the EEG, evoked potential monitoring is also becoming available for routine use in the operating room and critical care units. Dr. Samra (Galveston) reviewed considerations for pur-

1985 ANNUAL MEETING REPORT — *Continued*

chasing such equipment by the anesthesiologist including standards recommended by the American Enicephalographic Society. Other important factors to be considered include service contracts, on-site installation and owner training, the systems performance record at other centers, and cost. Both Dr. Grundy and Dr. Samra, respectively, emphasized the value of testing several different machines in the operating room prior to making a purchase.

Two presentations dealt with application of evoked potential monitoring. Steinberg et al. (London, Ontario) produced focal cerebral ischemia by middle cerebral artery occlusion in cats comparing somatosensory evoked potentials (SEP) with histological outcome. A striking association was found between SEP and neuronal ischemic changes suggesting that alterations in SEP provide a reliable indication of severe focal ischemia. Cottrell et al. (Brooklyn) addressed the risk of quadraplegia during surgery in the sitting position. Cord function was monitored by SEP and motor evoked potentials (MEP) in seated anesthetized monkeys with cervical hyperflexion. They found that while SEP may be useful in monitoring dorsal column function, damage to spinal cord ventral tracts may be unobserved unless MEP is also monitored.

Isoflurane induced deliberate hypotension was the subject of two papers. Alan Artru (Seattle) spoke on research evaluating the effects of combined hypocapnia and isoflurane induced hypotension on cerebral blood flow (CBF). Previous work has indicated a potentiation of the hypocapnic vasoconstrictor response by 1 MAC isoflurance. This allows the possibility that the even larger doses of isoflurane used to produce deliberate hypotension may further augment hypercapnic vasoconstriction which would be deleterious under conditions where CBF is already at nearly critical levels. Artru confirmed a persistent partial preservation of cerebrovascular responsiveness to hypocapnia ($PaCO_2$ = 20 mmHg) during isoflurane-induced hypotension (mean arterial pressure = 50 mmHg), but found CBF to remain above critical thresholds with this combination (39.4 ± 4.9 ml· 100 gm^{-1}· min^{-1}). These results imply that hypocapnia may be used to decrease cerebral blood volume to improve operative conditions during isoflurane-induced hypotension for neurosurgery. In a similar study Van Aken et al. (Munster) evaluated the responsiveness of the cerebral circulation during isoflurane-induced alterations in mean arterial pressure (MAP) in baboons. CBF, as measured by $Xe,^{133}$ was assessed after stepwise decreases in mean arterial pressure (10%, 20%, 40%, and 50% reduction from control) and at 100 mins after withdrawal of isoflurane. At each step, CBF during angiotensin induced hypertension was also assessed. The investigators found low dose isoflurane (0.5%) to be associated with a modest decrease in CBF which remained unchanged when challenged by angiotensin. With MAP at 50% of control (isoflurane -1.4%), CBF was near control values and became markedly elevated when angiotensin was administered. This apparent inhibition of autoregulatory function by high dose isoflurance was shown to persist until at least 100 mins after the isoflurane was discontinued.

Two papers dealt with cerebral metabolic protection. Based upon previous findings that the EEG and cerebral metabolic rate of oxygen consumption are depressed by isoflurane, Warner et al. (Lund) administered isoflurane to produce EEG burst suppression in the rat. Combination bilateral carotid arterial occlusion and systemic hypotension produced reversible forebrain ischemia for 10 mins. Seven days later, brains from surviving rats were formalin fixed for quantitative histopathologic analysis. No difference was seen in number of irreversibly injured neurons in isoflurane treated rats versus untreated controls. Hartung and Cottrell (Brooklyn) hypothesized that discrepancies in the literature concerning protective effects of barbiturates during hypoxia are due to the presence or absence of nitrous oxide in the experimental model. Mice were exposed to hypoxic gas mixtures, with and without nitrous oxide after pretreatment or no pretreatment with thiopental. The survival times were recorded. Pretreatment with thiopental significantly increased survival time but the effect was completely abolished by adding 50% nitrous oxide to the hypoxic gas mixture. The data therefore suggest a negative synergistic action between thiopental and nitrous oxide with respect to cerebral protection.

The Resident Research Essay Award was presented to Rosemary Hickey, M.D. (San Antonio) for work comparing the autoregulation of the brain and spinal cord. Alterations in mean arterial pressure were generated in thiopental anesthetized rats with either neosynephrine or trimethaphan. No statisically significant difference was seen between brain and spinal cord blood flow as measured by the radioactive microsphere technique for pressures within the autoregulatory range (60-120 mmHg). Outside of this range changes in brain and spinal cord blood flow paralleled changes in perfusion pressure. The authors concluded that the autoregulatory capability of the spinal cord is very similar to that of the brain and speculated about anesthetic effects on spinal cord blood flow under conditions of spinal cord injury.

Two papers were presented in respect to anesthesia and intracranial pressure (ICP)/ Katz and Artru (Seattle) evaluated the efficacy of diazepam and hyperventilation in blocking ketamine induced increased in ICP. Cerebral blood volume (CBV) was assessed using gamma scintillation counting of circulating ^{131}RISA in dogs with normal intracranial compliance. While ketamine alone increased CBV and ICP, pre- or post-treatment with either diazepam or hypocapnia blunted this response. It appears that hypocapnia or diazepam minimizes the ketamine-induced increase of ICP by causing cerebral vasoconstriction and a decrease of CBV. Minton et al. (Charlottesville) studied the effects of succinylcholine on ICP in humans with brain tumors. Succinylcholine administered alone significantly increased ICP by a mean of 5 mmHg. When succinylcholine administration was preceded by a 100% neuromuscular twitch depression induced by vecuronium, however, a significant increase in ICP from succinylcholine was not seen. The authors concluded: 1) succinylcholine may be expected to induce a marked increase in ICP in some pa-

1985 ANNUAL MEETING REPORT — *Continued*

tients with intracranial mass lesions; and 2) increases in ICP by succinylcholine appear to be due to an effect at motor nerve terminals.

The Society also enjoyed a special lecture by Dr. Charles Wilson (San Francisco) titled Transphenoidal Surgery for Pituitary Adenomas. In conclusion Dr. Albin (San Antonio) presented a tribute to Dr. D. G. McDowall, a recently deceased, world-class neuroanesthetist. The Society will reconvene for its 14th annual meeting at the Las Vegas Hilton, October 16, 1986.

POSITIONS AVAILABLE

An anesthesiologist is being sought by the Department of Anesthesiology of the Saint Louis University Medical Center, Saint Louis, MO. The department is currently enjoying an increase in neurosurgical cases and is seeking an anesthesiologist with a strong background in neuroanesthesia, board eligible or certified, and a fellowship in neuroanesthesia. Teaching and research are a large part of the position. Facilities for research are available. Send C. V. to John F. Schweiss, M.D., Professor and Chairman, Section on Anesthesiology, St. Louis University School of Medicine, 1325 S. Grand Blvd., St. Louis, MO. 63104.