REPORTS OF SCIENTIFIC MEETINGS

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American Academy of Pediatrics: Section on Anesthesiology Scientific Session April 27–29, 1990 Seattle, Washington

The Annual Scientific Session of the American Academy of Pediatric Section on Anesthesiology was held in Seattle, Washington, April 27–29, 1990. The Scientific Session consisted of 37 scientific papers presented orally, 10 reports of cases or techniques presented as posters, and a panel discussion.

Postoperative nausea and vomiting were the subject of papers presented by Rosen *et al.* (University of Michigan) and Friesen *et al.* (Denver Children's Hospital). In the study by Rosen *et al.*, children 2–12 yr old experienced less vomiting when anesthesia was maintained with midazolam–alfentanil–N_2O compared to those children in whom anesthesia was maintained with halothane–N_2O–morphine. In the study by Friesen *et al.*, children 2–8 yr old who were given oral transmucosal fentanyl citrate, 15–20 μg/kg and droperidol (50 μg/kg) did not experience significantly less nausea and vomiting than did children who received oral fentanyl without droperidol.

Perioperative dilemmas were addressed in papers presented by Cohen *et al.* (Children's National Medical Center, Washington, DC), Page *et al.* (University of Missouri), and Crawford *et al.* (Hospital for Sick Children, Toronto). Cohen *et al.* observed that adolescents 13–14 yr old showed a higher degree of vulnerability in the preinduction period toward anxiety factors (undressing, embarrassment, loss of control, pain, vomiting, and dizziness) than did adolescents who were younger (11–12 yr old) and older (15–17 yr old). Page *et al.* observed that, compared to children given only atropine, children given chloral hydrate (70 μg/kg) exhibited more satisfactory behavior in the anesthesia room, but the response to the induction of anesthesia was not altered by premedication. Crawford *et al.* concluded that healthy, unpremedicated children who were scheduled for elective surgery may be fasted for only 2 h after 2 ml/kg water without decreasing gastric fluid *p*H or increasing gastric fluid volume. Crawford *et al.* also concluded that in children undergoing cardiopulmonary bypass, preoperative oral ranitidine at 4 μg/kg effectively maintains gastric *p*H above 2.5 throughout surgery and for 12 h into the postoperative period.

Rolf and Coté (Harvard) observed a significant incidence of major hypoxemic events (decrease in Sp_{O_2} to <85% for >30 s) in children monitored without "available" pulse oximetry despite the use of capnography. They concluded that pulse oximetry appears to be far superior to capnography regarding the diagnosis of events leading to hypoxemia. Rolf and Coté also presented data from a laboratory study to suggest that during rapid ventilation, the higher dynamic resistance of small endotracheal tubes may lead to reduction in delivered tidal values and a consequent increase of inspired CO_2. Taylor and Lerman (Hospital for Sick Children, Toronto) presented experimental evidence that the delivery of aerosolized salbutamol through pediatric tracheal tubes is enhanced by administering the drug *via* a no. 19 catheter inserted into the tracheal tube.

Several papers dealt with anesthesia for cardiac surgery. McGowan *et al.* (Yale) presented data from ischemia and reperfused piglet hearts (Langendorff preparation) to suggest that 1) the neonatal myocardium is tolerant to prolonged global ischemia and 2) the mild mechanical dysfunction that results from ischemia is not related to impaired oxidative metabolism or inability to replete high-energy phosphates. Using transcranial duplex sonography in infants, Taylor *et al.* (Hospital for Sick Children, Toronto) observed that cerebral autoregulation is maintained during normothermic cardiopulmonary bypass (CPB) but is pressure-passive during moderate and profoundly hypothermic CPB. Rosen *et al.* (University of Michigan) reported congenital heart surgery cases successfully performed without the administration of blood products using hemodilution to a hematocrit of 15 during bypass. Kleinburg and Lynn (University of Washington) documented stable fentanyl concentrations in children during CPB using either a bubble (Bard) or membrane (Terumo) oxygenator. Lynn *et al.* (University of Washington) found amrinone to be a useful inotropic agent (increased cardiac output), but not a vasopressor (decreased systemic vascular resistance), in children after open heart surgery. Packer *et al.* (The Children's Hospital, Denver) presented data to suggest that desmopressin acetate (DDAVP) may be useful to treat coagulopathy after surgical repair of congenital heart disease. Lagueruela *et al.* (Washington University, St. Louis) showed the presence of a significant delay in bladder temperature relative to core temperature in children during CPB and deep hypothermic cardiac arrest when core temperature changes rapidly.

The complications of pediatric anesthesia were addressed in several presentations. Loftness and Class (The Children's Hospital, Denver) reviewed data of 45,500 pediatric patients and reported that children with ASA physical status of III or greater and those under the age of 6 months had a significantly higher incidence of complications as compared to the overall population. Borland *et al.* (University of Pittsburgh) reviewed 10,040 outpatient pediatric anesthetics and reported that the major medical reason for unscheduled inpatient admission was intractable nausea and vomiting. The group from Pittsburgh also identified 29 cases of pulmonary aspiration in 16,826 children (0.17%) anesthetized over an 18-month period. Risk factors for aspiration included previous esophageal surgery and a history of vomiting during induction. Welborn *et al.* (Children's National Medical Center) presented data to suggest that anemia in former preterm infants may increase the incidence of postoperative apnea. Valley *et al.* (University of North Carolina) reviewed the charts of 116 infants < 9 months old who had undergone inguinal herniography under general or spinal anesthesia and reported that the types of complications in the general anesthesia group tended to be more severe and life-threatening than did those of the spinal anesthesia group. Valley *et al.* also reviewed the charts of 136 pediatric patients (age 1 day to 16 yr) receiving caudal morphine (0.07 mg/kg) and identified several risk factors associated with respiratory depression (age less than 1 yr, use of intra-

operative narcotics, and use of "high" caudal catheters). Salveson *et al.* (University of North Carolina) reported that naloxone infusion (4 $\mu g \cdot kg^{-1} \cdot h^{-1}$) did not affect the incidence of complications after caudal morphine in pediatric patients. Zeligs *et al.* (Long Beach Memorial Medical Center) observed a 40% incidence of vomiting or pruritis, zero incidence of respiratory depression, and excellent analgesia (average duration = 22 h) in 26 pediatric patients (age 2 months to ten yr) given caudal morphine (0.1 mg/kg) and 0.25% bupivacaine (0.5 ml/kg). Morrison *et al.* (The Children's Hospital, Denver) presented data to suggest that intramuscular neurolysis using 5% phenol in water is relatively safe and effective in treating spasticity in children.

Bacsik *et al.* (Harvard) presented an extensive review of the anesthetic implications of stereotactic radiosurgery in children. Ferrari *et al.* (Cornell University) presented two-dimensional echocardiographic data to indicate that ketamine maintains cardiac contractility during induction and maintenance of anesthesia in adriamycin-exposed patients, whereas thiopental does not.

A survey of 2,500 pediatricians (fellows of the American Academy of Pediatrics) was taken by Fisher *et al.* (University of Mayland). The results of this survey indicate that pediatricians remain relatively peripheral to new demands for knowledge of anesthesia-related issues and that their ideal level of preoperative participation contrasts with their low self-esteem in this realm.

The Housestaff Scientific Competition award was presented to Dr. Jeff Leon (The Hospital for Sick Children, Toronto) for his research entitled "Cranial duplex sonography: Do isoflurane and halothane affect the cardiovascular response to carbon dioxide in anesthetised infants and children?" These data indicated that during isoflurane anesthesia, cerebrovascular reactivity to CO_2 was maintained up to 1.0 MAC isoflurane, whereas during halothane anesthesia, reactivity to CO_2 was attenuated. Furthermore, at low levels of CO_2 (20 mmHg), cerebral blood flow velocity varied directly with the halothane concentration up to 1.0 MAC.

A very interesting panel discussion entitled "Easing the way to and from the operating suite: New approaches to sedation and analgesia" detailed the pre- and postoperative management of infants and children scheduled for elective surgery. Don Tyler, M.D. (University of Washington, Seattle) first reviewed the essentials of postoperative pain management, including organizational and multidiscipline considerations and specific techniques for pain control. Linda Jo Rice, M.D. (National Children's Hospital, Washington, DC) reviewed the pharmacology of drugs used in regional anesthesia; indications, contraindications, and complications of regional blocks in detail; and technical aspects of block placement in pediatric anesthesia.

James Diaz, M.D. (Oschner Clinic, New Orleans) completed the discussion with a comprehensive review of premedication techniques in children. He addressed both old and new premedicant/sedative drugs (including midazolam, sufentanil, and eutectic mixture of local anesthetics [EMLA]) delivered through a multitude of routes (including nasal, transmucosal, and transdermal) and concluded with an overview of efficacy and safety considerations.

Abstract presentations resumed with Audenaert *et al.* (University of Kentucky) who reviewed the risk of malignant hyperthermia (MH) in patients with arthrogryposis multiplex congenita. They found no solid evidence relating malignant hyperthermia to arthrogryposis.

Rectal methohexital is a commonly used premedication in children. Daniels *et al.* (Massachusetts General Hospital) investigated the risk of arterial oxygen desaturation during rectal methohexital premedication in 49 healthy infants and children. Two episodes of arterial desaturation to 86 and 90% occurred with the patients in their parents' arms. Desaturation was corrected by repositioning the head.

Daniels *et al.* also explored the difficulties in providing sedation/anesthesia for magnetic resonance image (MRI) scanning. They concluded that capnography and oximetry appear to be essential to minimize the risk of complications during anesthesia coverage in remote areas.

Birmingham *et al.* (Children's Hospital, Chicago) reviewed the anesthetic management of children with bronchogenic cysts. A thorough review of the risks of the use of N_2O and controlled ventilation ensued.

Holzman *et al.* (Boston Children's Hospital) reviewed ten patients with myelodysplasia who experienced anaphylactoid signs consisting of hypotension, flushing, and bronchospasm during an otherwise uneventful anesthetic. The etiology of these signs was unclear but may have been attributable to latex sensitivity. Prophylaxis and treatment of anaphylaxis to latex were discussed.

Two studies investigated the incidence of emesis after strabismus surgery. Pryn *et al.* (Mott Children's Hospital, Ann Arbor) compared acupressure (P6 point) to placebo to reduce the incidence of emesis after strabismus surgery. Acupressure was ineffective. In a second study, Brown *et al.* (Bowman Gray, North Carolina) compared the effectiveness of low-dose droperidol (20 $\mu g/kg$ intravenously) to high-dose droperidol (75 $\mu g/kg$). With the small sample size studied, they found these doses were not significantly more effective than placebo ($P = 0.052$).

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Society of Neurosurgical Anesthesia and Critical Care Annual Meeting October 18, 1990 Las Vegas, Nevada

The 18th Annual Meeting of the Society of Neurosurgical Anesthesia and Critical Care was held at Bally's Hotel, Las Vegas, Nevada, on October 18, 1990. Among the topics discussed were advances in computer-assisted stereotactic neurosurgery, mechanisms of—and therapies for—ischemic brain injury, the effects of anesthetic agents on intracranial pressure (ICP) and cerebral blood flow (CBF), and advances in neurologic monitoring.

Computer-Assisted Stereotactic Neurosurgery

Patrick Kelly, M.D. (Rochester, Minnesota) described a computer-assisted stereotactic neurosurgical system that integrates data from computerized tomography, magnetic resonance imaging, and digitally subtracted angiograms in order to elaborately describe intracranial anatomy. This three-dimensional database is then used to simulate and ultimately select a surgical trajectory that provides access to small, deep-seated, or otherwise difficult to reach intracranial structures, while concomitantly avoiding either neurologically eloquent tissues or vascular structures. With the use of these methods, perioperative morbidity as well as hospital costs have been demonstrated to be less than for conventional craniotomies.

Mechanisms of—and Therapies for—Ischemic Brain Injury

Myron Ginsberg, M.D. (Miami, Florida) reviewed recent research addressing the pathologic mechanisms responsible for ischemic brain injury. His presentation focused on the injurious phenomena that occur during the period of reperfusion. Of the numerous potentially injurious processes that modulate cell damage during reperfusion, Dr. Ginsberg emphasized the role of neurotransmitters. Using a rat model of focal ischemia, Ginsberg and colleagues observed that under certain experimental conditions, histopathologic injury occurred almost exclusively in the striatum. Subsequent investigation of the striatum revealed a 300–500-fold increase in interstitial dopamine during ischemia and a postischemic period of glucose hypermetabolism with decreased CBF. Experimental interventions designed to pharmacologically reduce ischemic dopamine accumulation resulted in a reduction in postischemic neuronal Ca^{2+} uptake and a reduction in neuronal damage. The reduction in neuronal damage was not related to increases in striatal interstitial glutamate, an excitatory amino acid neurotransmitter that is capable of modulating regional cerebral postischemic injury *via* an excitotoxic mechanism.

Dr. Ginsberg also reported that moderate hypothermia (30–34° C) improved postischemic neuronal survival in some animal models, regardless of whether the hypothermia was induced before, during, or after ischemia. This degree of hypothermia did not significantly alter CBF, nor did it alter the rate of decline of high-energy metabolites or the rate of glucose metabolism during ischemia; however, the hypothermia did result in an improved restitution of postischemic oxygen metabolism. Thus, the cerebral protection associated with moderate hypothermia is interpreted as due to a mechanism other than a beneficial relationship in the ratio of cerebral metabolism and blood flow during the period of ischemia. (Note that this hypothesized mechanism differs from the mechanism traditionally used to account for cerebral protection by profound hypothermia. With profound hypothermia, metabolic depression, *per se*, is attributed with providing protection from ischemia.) Dr. Ginsberg concluded that because reperfusion-related injury to the brain is multifactorial, future therapy for ischemic neurologic injury will continue to use traditional physiologic supportive measures, but in addition, may involve combinations of pharmacologic therapies, each directed toward relatively independent mechanisms of injury. It should be noted that currently, most potentially beneficial therapies, including mild hypothermia, are still being experimentally evaluated, and that their effects in humans experiencing cerebral ischemia are unknown.

Daniel J. Cole, M.D. (Loma Linda, California) and colleagues previously demonstrated that increases in systemic arterial blood pressure (i.e., hypertension [HTN]) and/or moderate systemic hemodilution resulted in improved cerebral blood flow after experimental focal cerebral ischemia. However, these therapies had undesirable side effects (e.g., vasogenic edema and cerebral hemorrhage), which increased in severity with increased treatment duration. Dr. Cole presented the results of subsequent experiments examining the effect of a short (15-min duration, beginning 30 min postischemia) or intermediate (90-min duration, beginning with reperfusion) period of postischemic HTN (mean arterial pressure 30 mmHg greater than normal) on postischemic CBF restitution. CBF was significantly improved in the ischemic hemisphere only in the group of animals treated with the short period of postischemic HTN. The authors speculated that postischemic HTN may result in a mechanical opening of collapsed or constricted vessels, washing out cellular aggregates and vasoconstrictive by-products of ischemic metabolism. The absence of CBF improvement in animals treated with the longer period of HTN was attributed to a possible vasogenic edema-producing effect of the therapy.

Several additional studies addressed the biochemical and biophysical events that occur during and after brain ischemia. Using magnetic resonance spectroscopy, Scott Eleff, M.D. (Baltimore, Maryland) described and compared the kinetics of intracellular sodium ($^{23}Na^{+}$) uptake, *p*H changes, and adenosine triphosphate (ATP) depletion during a reversible episode of complete cerebral ischemia. During ischemia, an increase in intracellular sodium closely paralleled but slightly preceded depletion of intracellular ATP. During reperfusion, restitution of extracellular sodium concentrations (12.4 min) significantly preceded recovery of intracellular ATP (16.5 min) and *p*H (38.9 min). Complete recovery of the sodium gradient with only partial recovery of the intracellular ATP suggests that either intracellular ATP is being consumed to reestablish membrane ionic gradients, or that for every ATP hydrolyzed, many sodium ions are extruded from the intracellular space.

Increases in brain glucose concentrations during a period of cerebral ischemia result in a worsened outcome after ischemia. This phenomenon (along with diabetes-induced alterations in the cerebral vasculature) is credited with producing a worse neurologic injury in diabetic patients exposed to brain ischemia (when compared to nondiabetic subjects). As clinicians, we are readily able to measure blood glucose, but not the critical brain glucose concentrations in patients at risk for cerebral ischemia. Roger Hofer, M.D. (Rochester, Minnesota) reported that, in a rat model of diabetes mellitus, brain glucose dynamics differed between diabetic and nondiabetic rats. Specifically, diabetic rats accumulated glucose in the brain, such that for a given blood glucose value, diabetic rats had one-fourth more brain glucose than did nondiabetic rats. This altered relationship between brain and blood glucose in diabetic rats was not affected by either saline or insulin infusion. Dr. Hofer's data suggest that for a given duration of ischemia and at a given concentration of blood glucose, diabetic subjects may have a worse postischemic neurologic injury than may nondiabetics because of the anaerobic metabolism of accumulated brain glucose. In another study in diabetic rats, Dale Pelligrino, Ph.D. (Chicago, Illinois) reported that chronic hyperglycemic diabetes mellitus did not decrease the hyperemic response to cerebral hypoxia or hypercarbia. This study contradicts previous speculation that cerebral vasodilatory reserve is attenuated by chronic diabetes.

Effect of Anesthetic Agents on Intracranial Pressure and Cerebral Blood Flow

Donald Muzzi, M.D. (Rochester, Minnesota) reported human studies of the effect of desflurane on cerebrospinal fluid pressure. Desflurane is a new volatile anesthetic that, like other volatile anesthetics, is a potent cerebral vasodilator. In Dr. Muzzi's study, desflurane was administered to hypocapneic patients (Pa_{CO_2} near 26 mmHg) with supratentorial mass lesions. In this setting, desflurane resulted in a gradual, significant increase in cerebrospinal fluid pressure (from 11 to 19 mmHg over a 45-min period of study). In another clinical study, J. R. Hemstad, M.D. (Seattle, Washington) reported that ICP did not increase after dural closure in patients undergoing craniotomies, regardless of whether N_2O was continued or discontinued. This lack of effect by N_2O was observed in a population of patients that consistently had computerized tomographic evidence of postoperative intracranial air. Thus, routine elimination of N_2O prior to dural closure in an attempt to avoid increases in ICP (due to expansion of extraventricular, intracranial air) may be unwarranted.

Depending on the background anesthetic, sufentanil has been reported either to have no effect or to increase CBF and ICP. Christian Werner, M.D. and colleagues (Hamburg, West Germany and Chicago, Illinois) reported that sufentanil decreased cerebral blood flow velocity, CBF, and cerebral O_2 consumption in parallel in dogs anesthetized with 0.5 MAC isoflurane and 50% N_2O in O_2. F. Lui, M.D. (London, Ontario) reported that *in vitro,* fentanyl, sufentanil, and alfentanil all produced a similar, concentration-dependent contraction of canine basilar artery strips. Alan Artru, M.D. (Seattle, Washington) reported a dose-dependent effect of fentanyl, sufentanil, and alfentanil on cerebrospinal fluid dynamics in dogs, resulting in a variable effect on predicted net cerebrospinal fluid volume. However, when given in low doses, all three opioids favored contraction of cerebrospinal fluid volume because of a decreased resistance to fluid reabsorption. These data, coupled with other recent clinical reports, increasingly support the concept that sufentanil can be used safely as an anesthetic supplement in patients having abnormal intracranial elastance.

Eugene Ornstein, Ph.D., M.D. (New York, New York) reported that, in contrast to other commonly used methods, hypotension (i.e., a 20% reduction in mean arterial blood pressure) induced with the β-adrenergic blocking drug, esmolol, decreased both cardiac output and CBF. Once cardiac output decreased by more than 25%, CBF significantly decreased.

Advances in Neurologic Monitoring

Maurice Albin, M.D. (San Antonio, Texas) reported on the efficacy of transcranial Doppler sonography in detecting intracranial intraarterial emboli during coronary artery bypass graft or valve replacement procedures. Dr. Albin detected intraoperative embolism in 41% of patients having bypass procedures and in 79% of patients having valve replacement procedures. These data support the concept that the published reports of a higher incidence of postoperative neuropsychiatric complications in patients having valve replacement (when compared to patients having bypass grafting) is related to perioperative ischemia produced by emboli to the brain.

Several studies reported the effects of anesthetic techniques on electrophysiologic monitoring. The effects of desflurane on somatosensory evoked potentials (SSEP) was described by Susan Black, M.D. (Chicago, Illinois). She reported that desflurane produced a dose-dependent decrease in SSEP amplitude but did not increase latencies. In patients having normal baseline SSEP, desflurane in concentrations >1.0 MAC permitted reliable monitoring of SSEP. Ramsis Ghaly, M.D. (Pittsburgh, Pennsylvania) reported that hypnotic doses of etomidate, but not of midazolam, preserved the quality of motor-evoked potentials in primates. Also using primates, Tod Sloan, M.D. (San Antonio, Texas) reported that magnetically induced motor-evoked potentials (measured by magnetically stimulating the cerebral cortex and observing the electromyographic activity of the opponens pollicis muscle) were well maintained in the presence of either vecuronium- or atracurium-induced neuromuscular blockade in doses that resulted in less than 80% neuromuscular blockade. Neuromuscular blockade was assessed by observing the electromyographic response of the opponens pollicis to supramaximal stimulation of the median nerve.

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