Reports of Scientific Meetings

Bryan E. Marshall, M.D., Editor

The Society of Neurosurgical Anesthesia and Neurologic Supportive Care

The Society of Neurosurgical Anesthesia and Neurologic Supportive Care met for its sixth annual meeting in Chicago on Friday, October 20, 1978, on the eve of the 1978 Annual Meeting of the American Society of Anesthesiologists.

The one-day meeting consisted of mini-symposia on protection of the brain and edema control, neurophysiologic monitoring of the brain, induced hypotension, and cerebral vasospasm. A special lecture on the cellular basis of neuroleptic and anesthetic action was given. The session was concluded by presentation of free papers.

The panel entitled, "Brain Protection and Edema Control," was moderated by Harvey Shapiro (San Diego). John D. Michenfelder (Rochester) outlined results of research on cerebral metabolic effects of ischemia using animal models. He summarized evidence for the protective role of barbiturates in focal and global cerebral ischemia based on metabolic depressant effects of barbiturates.

James E. Cottrell (New York) reviewed literature concerning etiology and classification of cerebral edema. Anesthetic management of patients on the upswing of the intracranial compliance curve must avoid increase in intracranial pressure (ICP). Increased ICP is partly treated by decreasing brain water content with osmotic diuretics and recently, loop diuretics. Dr. Cottrell's data show a transient increase in ICP after a bolus dose of mannitol (1 g/kg) while no such effect was observed when furosemide (1 mg/kg) was used. In addition, a direct cerebral effect of furosemide to inhibit active and inactive cellular chloride transport prevents astroglial swelling, decreasing capillary impedance.

Harvey Shapiro discussed control of ICP and cerebral edema by use of barbiturates when mannitol and steroids do not suffice. Barbiturates are a valuable adjunct in treating high ICP because of cerebral vasoconstriction.

A minimal protective effect by barbiturates after global ischemia was reported by Derek Bruce (Philadelphia), but significant protection of the brain was found after focal ischemia in children. A blood barbiturate level adequate to control ICP is best maintained by bolus injection of pentothal, 5 mg/kg, followed by continuous infusion of 2–3 mg/kg/h and added boluses of 3 mg/kg.

Philip Seeman (Toronto) delivered a special lecture, "Cellular Basis of Neuroleptic and Anesthetic Actions." The effectiveness of both local and general anesthetics is proportional to their oil–water partition coefficients, which reflect the cellular membrane–interstitial fluid partition coefficients. The degree of anesthetic action assessed in an experimental model of a single neuron action potential is directly dependent on the concentration of anesthetic molecules in the neuronal membrane, or rather, on the percentage of space occupied by the molecules of the anesthetic, since the necessary concentration is indirectly proportional to the molecular weight of the anesthetic. It appears that 0.3 per cent of membrane volume has to be taken up by cellular membrane expansion, which is reversible by the recompression at high atmospheric pressure. Anesthetics also fluidize cellular and intracellular vesicular membranes containing transmitters, which may facilitate transmission. It is difficult to explain the mode of action of neuroleptics since the concentration at which they are effective in treating psychotic patients is below that necessary to achieve an action-potential block. Indirect evidence suggests that these agents act by interfering with dopaminergic receptors. Studies of Seeman's group using radioligand receptor assay proved that neuroleptics specifically bind to dopaminergic receptors at clinical concentrations.

James R. Harp (Philadelphia) introduced and conducted the panel on neurophysiologic monitoring of the brain.

Richard R. Greenberg (Richmond) reviewed his experience in using continuous neurophysiologic monitoring of comatose patients. With multimodality evoked potentials the on-line computer-assisted data analysis allows immediate diagnosis of alterations in brain function.

Ty Smith (San Diego) discussed the use of the EEG to monitor brain function. The standard EEG is difficult to interpret and impractical to record. There is a need to simplify and condense EEG information in such a way that it would be possible to monitor constantly and interpret immediately. The many methods presently available include zero crossing technique; cerebral function monitor (CFM), which is commercially available; peak or mean power frequency; density-modulated spectral array (DSA); compressed spectral array. These techniques can detect changes in anesthetic depth, in cerebral perfusion, and possibly in brain temperature. DSA provides the most information most rapidly.

Mayo Clinic experience with CFM was reported by Roy F. Cucchiara. This device is unreliable in detecting hemispheric cerebral ischemia when used with electrodes applied bilaterally. With electrodes unilaterally, the CFM detects hemispheric ischemia, but not as sensitively as the EEG. Anesthetic effects can mimic the effect of ischemia as detected by CFM.

Brian Marshall (Toronto) chaired the panel on induced hypotension and cerebral vasospasm.

Joseph M. Messick (Rochester) reviewed the goals of hypotension in neurosurgery, its indications, physiology, agents used, and monitoring.

James E. Cottrell discussed the methods and agents used in controlled hypotension. His lecture centered on the use of sodium nitroprusside (SNP) and nitroglycerin (NTG). The hazards of SNP include accumulation of cyanide in blood, increased ICP due to cerebral vasodilation, hypothyroidism, diminished platelet aggregation and concentration, and rebound hypertension from increased plasma renin activity. The studies of Dr. Cottrell's group indicate that cyanide

accumulation may be treated by hydroxycobalamin, which combines with cyanide. Tachypylaxis is due to the direct effect of cyanide on blood-vessel sulfhydryl groups, which is later compounded by acidosis. NTG intravenously also increases ICP, and some patients are refractory to its hypotensive effects. Trimethaphan has undesirable side effects from parasympathetic blockade and, in addition, might be directly harmful to the brain.

Frederick Simeone (Philadelphia) reviewed recent experience with cerebral vasospasm. This condition occurs after intracranial hemorrhage and after cerebral-aneurysm clipping. When the vascular lumen is more than 50 per cent constricted, neurologic deficit or death may result. Except for kanamycin and reserpine, which seem to prevent the spasm, most pharmacologic treatments fail. Once spasm has developed, good results have been obtained with expansion of the intravascular volume and sustained artificial hypertension.

In the free papers section, Robert F. Bedford (Charlottesville) reported that lidocaine, 1.5 mg/kg, intravenously, before endotracheal intubation prevented not only systemic blood pressure rise but also the ICP increase that often follows endotracheal intubation. The same effect could be obtained by increasing the induction dose of pentothal from 3 to 6 mg/kg.

Pavel Illner (New York) reported on increased plasma renin activity in anesthetized patients with SNP-induced hypotension. This increase in not due to anesthesia itself, and might be the cause of the rebound hypertension frequently seen after discontinuation of SNP, since renin persists in plasma after SNP has already disappeared, due to its shorter half-life.

PAVEL ILLNER, M.D.
Resident—Anesthesiology
School of Medicine
New York University Medical Center
New York, New York

JAMES E. COTTRELL, M.D.
Professor and Chairman
SUNY at Downstate Medical Center
Brooklyn, New York