The following represents the proceedings from the Second International Symposium on Central Nervous System Monitoring, held in Gmunden, Austria, September 8 and 9, 1989. As was the first Symposium, this meeting was eclectic, with many different disciplines represented. In addition, the Symposium consisted partly of tutorials and partly of scientific papers. Thus, it offered a unique educational and scientific experience. These abstracts offer only a taste of that experience, and the interested reader should contact the individual authors.

For the American reader, the abstracts offer a glimpse of what is going on in other countries, particularly European countries, in this important area. It is obvious that the Europeans have accumulated a vast amount of valuable clinical experience, with a wide variety of central nervous system monitoring techniques.

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CENTRAL NERVOUS SYSTEM MONITORING FOR DIAGNOSIS AND CONTROL OF THERAPY

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Within the intensive care unit, central nervous system monitoring primarily serves to identify intracranial complications and to control the success of therapy. During surgery, cerebral monitoring not only permits anesthesia regulation, but, with interventions in the brain or the cerebral circulation, it also permits the assessment of the effect of certain measures and a selective avoidance of lasting damage.

Therefore, monitoring in the intensive care unit and the operating theater will require many different kinds of variables. In the intensive care unit, the identification of anything that can occupy intracranial space is paramount; the most important variables will therefore be intracranial pressure and cerebral perfusion pressure. Electrophysiology and, for example, transcranial Doppler sonography are useful for the assessment of cerebral vasospasm. In the operating area, the focus is on the use of monitoring with computer-assessed electroencephalographic (EEG) and selected evoked potentials for the earliest identification of an ischemic or directly pressure-related danger to the brain, again supplemented by transcranial Doppler sonography.

Monitoring of intracranial pressure should preferably be effected by minimally invasive, miniature sensors directly implanted intracranially. Compared with ventricular pressure measurement, which is limited to use with special indications (cerebrospinal fluid drainage, for example), the above method is less prone to artifact production and cannot cause infections. The intracranial pressure sensor by Hellige, constructed with a cavity resonator, has been shown to be reliable in comparative tests but is difficult to implant. More simple to implant, but less exact, are the microcatheter-type miniature sensors (Gaeltic, Philips) and, more recently, intra-parenchymal fiberglass pressure recording by means of the Camino-pressure system. A computer-supported evaluation is also meaningful because of the wavelike pressure dynamics (long-term graphs, histogram statistics), used also for perfusion pressure calculation, in
which pressure recording in the temporal artery proved useful as a reference of carotid pressure. Pressure recording allows diagnosis for operation, from decompression trepanation to the regulation of conservative therapy.

For our purposes, the currently available electrophysiologic monitoring was found to be suboptimal. We therefore developed a special eight-channel EEG computer system with integrated evoked potentials. This supplied useful intraoperative information, particularly in cerebrovascular neurosurgery. For example, intraluminal shunts, implantation of extracranial bypasses, and media-thromboendarterectomy, which was made safe only by the use of this method, were all regulated by means of computer-supported EEG and evoked potential analysis. Since the introduction of this system 2 years ago, no lasting neurologic deficits have appeared after cerebrovascular neurosurgery (carotid stenoses, vertebral stenoses, intracranial vascular stenoses, bypass surgery).

The transcranial Doppler can also be used for continuous monitoring with a fixed flat probe. In the intensive care unit, this permits testing of therapy effects with vasospasm and is an important aid in the determination of cerebral death. In the operating theater, continuous flow regulation complements electrophysiologic monitoring, particularly in carotid artery surgery.

### INFLUENCE OF ANESTHETICS ON BLOOD FLOW VELOCITY OF CEREBRAL CIRCULATION

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Owing to continuing technical developments in the area of cardiopulmonary function, monitoring perioperative patient safety has attained high levels. Central nervous system monitoring, however, has lagged, partly owing to a lack of acceptance of suitable methods. This, in turn, was due to difficult development, invasiveness, problems of interpretation, and high costs.

The inclusion of transcranial Doppler sonography in perioperative anesthetic monitoring has opened up new opportunities for the assessment of the reactivity of cerebral vessels to CO₂, despite some reservations about the method. The method's advantages are obvious: quick, noninvasive, regionally specific, continuous, applicable anywhere, and repeatable at will. In contrast, one must consider the important disadvantage: it is velocity that is being measured.

Before clinical use in anesthesia, at least two problems need to be solved: (1) To what extent does the Doppler-sonographic signal represent known alterations in cerebral perfusion under diverse anesthesia conditions? and (2) Is it possible to draw conclusions regarding autoregulation and reactivity of cerebral vessels to CO₂ by means of this method?

With regard to the first question, we examined a total of 45 patients in three groups, each receiving a different anesthetic agent regimen: modified neuroleptic analgesia (midazolam/alfentanil), ataral analgesia (ketamine/midazolam), or thiopental. In each case, recordings were undertaken within the area of the middle cerebral artery at three points in time with the patient supine: before premedication in a waking state, 20 minutes after preanesthetic medication (Thalamonal 0.03 ml/kg and atropine 0.01 mg/kg), and during anesthesia steady state after intubation and during controlled ventilation (PcO₂ = 36 mm Hg).

The following results were determined: mean flow velocity in all patients was 51.6 cm/s, flow velocity decreased with increasing age, mean flow velocity increased after preanesthetic medication (+11%), and, in all of the anesthetic procedures mean flow velocity reflected the changes in cerebral perfusion.

To clarify the question of cerebral reactivity to CO₂ and autoregulation, we studied the reaction of the vMCA to CO₂ alterations and to increases in arterial mean pressure in an additional 7 patients during 1 vol% isoflurane (60% N₂O/40% O₂ in steady state, 30 minutes after induction of anesthesia). The results demonstrated largely retained autoregulation and reactivity to CO₂ when isoflurane was 1 vol% or less.

Apart from the already established potential for the application of transcranial Doppler sonography in neurology and neurosurgery, this method provides a new potential for the monitoring of cerebral O₂ supply in postoperative anesthetic care, particularly in combination with already established monitoring systems.

### ASSESSMENT OF THE PROTECTIVE EFFECTS OF ANESTHETIC AGENTS

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Although the precise mechanism subserving the state we define as "anesthesia" has yet to be clarified, it is clear that, in simple terms, "anesthetic" agents induce unconsciousness by primarily suppressing cortical function. This is evident by alterations in cortical electrical activity and, since function is depressed, by decreases in the requirement for oxygen and the utilization of glucose. Since the demand for energy substrates is usually decreased in the anesthetized individual, it has been suggested that those anesthetic agents that suppress cerebral metabolism could have a role in protecting the brain in those pathologic situations in which the supply of energy substrates is reduced (or the demand increased). Although this appears to be a reasonable hypothesis, it is probably an oversimplification of a complex issue, particularly since anesthetic agents have "vascular" as well as "metabolic" effects.

Propofol is an anesthetic agent that is effective when administered as a single injection, as intermittent boluses, or in an infusion. It is known to decrease the cerebral metabolic rate for oxygen and, consequently, could—at least theoretically—have "protective" properties. The present communication will consider this possibility by characterizing the effects of propofol noted in a number of investigations (experimental and clinical) designed to examine a variety of clinically relevant situations.

The investigations demonstrate that propofol decreases metabolic demand (alterations in spontaneous electrical activity; dose-dependent decreases in cerebral metabolism) and can decrease to lower values of mean arterial pressure, the threshold at which extracellular potassium concentration increases in a model of incomplete global ischemia. However, the effect of propofol per se was less clear in a model of focal ischemia (middle cerebral artery occlusion). In patients without intracranial pathologic evidence of disease, propofol has been shown to suppress spontaneous cerebral electrical activity, and the effect, if any, of this property is currently being investigated in patients undergoing cardiopulmonary bypass. Obviously, it will not be possible to compare directly each of the studies. However, results of such a "package" of studies using a single agent may throw some light on the question of whether anesthetic agents have protective effects as far as the brain is concerned.