Cerebral function monitor during open heart surgery

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The objective of cardiac surgery is to return the patient to as normal a state as possible. A successful surgical result must be accompanied by the recovery of function of the cardiovascular, pulmonary, renal, and neurologic subsystems. The recovery of the last of these systems has been most difficult to ensure after open heart surgery. Rapid advances in monitoring hemodynamic and respiratory functions have increased patient safety; however, the monitoring of cerebral function has not kept pace with these advances.[1]

Since 1975 we have used the cerebral function monitor* in our operating suite in an effort to ascertain first and then to correct those situations that may result in neurologic dysfunction in the postoperative period. The cerebral function monitor is designed to process the cerebral electrical activity and display it in an easily interpretable fashion. The device has some advantages over the conventional electroencephalogram (EEG), including simplicity, cost, size, safety, and reliability. The details of electronics of the cerebral function monitor have been reported.[2-4]

This report is based on 650 patients who had undergone surgery for acquired and congenital

* *The Cerebral Function Monitor 4640, Devices Limited, Johnson & Johnson, Inc. New Brunswick, New Jersey 08903.*

heart diseases and who had been attached to the cerebral function monitor during the entire operative procedure. The purpose of this communication is to analyze the clinically important observations that occur in the cerebral electrical activity recording during normothermic perfusion, deep hypothermia, and during untoward events, e.g., hypotension, technical complications during perfusion, and uncontrolled hemorrhage. The limitations of the cerebral function monitor will also be discussed to avoid overinterpretation of the cerebral electrical activity tracings.

Material and method

The cerebral function monitor recording is provided by the use of three electrodes attached to the scalp. The signal is passed through a filter eliminating high and low frequencies that usually result in interferences and artifacts. It is then amplified, rectified, integrated, compressed, and displayed. The cerebral electrical activity is represented as a thick band on a slow-running paper, where the highest and lowest points correspond with the extremes of cerebral voltage, while the width of the band indicates variability of the signal amplitude. A second channel records electrode impedance and warns of artifacts that might be misinterpreted as a change in cerebral electrical activity.

Six hundred fifty patients have had the cerebral function monitor attached and cerebral electrical activity was recorded during the entire surgical procedure. Periods of hypotension, hypothermia (esophageal temperatures), hypoxia, circulatory arrest, and untoward situations were marked on the tracing.

Two hundred ten patients were operated on under normothermia or slightly hypothermic conditions, esophageal temperature (> 32 C) and 440 were operated on under deep hypothermia (< 25 C) and cardioplegic arrest techniques. Patients undergoing surgery for acquired (coronary and valvular) heart disease in the initial period of the study were operated on with normothermia or mild hypothermia; in the latter part of the study deep hypothermia with cardioplegic arrest was used. The group with congenital heart disease in the early part of the study was operated upon under mild hypothermic conditions (30 to 32 C). Patients with more complex lesions, e.g., large ventricular septal defect, tetralogy of Fallot, complete atrioventricular canal, were operated on under deep hypothermia with cardioplegic arrest in the latter period of this study.

Membrane oxygenators (Travenol) with arterial filters (Pall) and cardiotomy line filters (Pall) were used in most patients. Only emergency procedures were performed with bubble oxygenators.

The pump prime consisted of a nonblood prime, with blood being added if the hematocrit dropped below 20% during perfusion. This protocol differs from that previously reported[4] in that 50% hemodilution was used and no arterial line filters were in place.

All patients were examined after recovery from anesthesia; patients with gross neurologic defects or those who emerged from anesthesia unusually slowly were seen in neurologic consultation. These patients form the nucleus of the report and will be discussed.

Results

In our practice an abnormality in cerebral electrical activity can only be detected by evaluating changes with respect to the base line of the same patient after induction of anesthesia. Factors to be considered in relation to the changes

in cerebral electrical activity are (1) hypothermia, (2) hypotension, and (3) hypoxia. Once the baseline of cerebral electrical activity is established, untoward incidents affecting cerebral electrical activity can be immediately recognized.

The cerebral electrical activity recording obtained can be divided into four groups:

I. Essentially normal throughout the procedure
II. Mild transient abnormalities
III. Severe or prolonged abnormalities or both with return to normal
IV. Severe or prolonged abnormalities or both without return to normal

Of 210 patients operated on under normothermia or slight hypothermia, 198 were in groups I and II. In 161 patients, during the entire operative procedure, the cerebral electrical activity was without changes (*Fig. 1*). Thirty-seven patients had short periods of changes in cerebral electrical activity (depression), mostly during onset of cardiopulmonary bypass. Twelve patients were in group III or IV.

Of 440 patients operated on under deep hypothermic conditions, 380 were in group III and 60 in group IV. Twenty-one of the group IV patients had exceptionally low cerebral electrical activity before surgery secondary to poor hemodynamics. Each of the groups of recordings was obtained and compared to the clinical outcome and neurologic recovery of the patient.

Most patients were in groups I and II and emerged from anesthesia without difficulty and were neurologically intact. However, in each group a number of false negatives were obtained. The neurologic deficit encountered in these patients was focal rather than diffuse and except in one case had completely resolved before the patient's discharge. The limitation of this method is obvious since the recording is an integrated signal rather than discrete regional signals. Patients with small localized particulate or air emboli will often escape detection by this method.

The mild, transient abnormalities, group II, were most often seen at the onset of cardiopulmonary bypass when nonpulsatile flow and mild hypotension

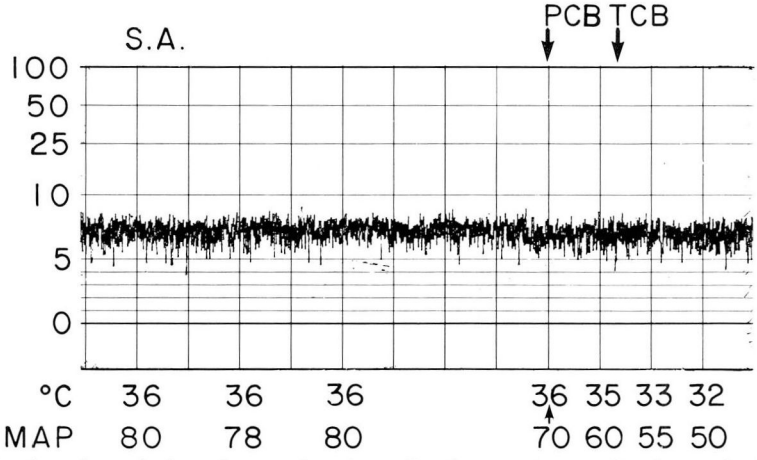

Fig. 1. Induction of anesthesia and normothermic cardiopulmonary bypass. No changes in the cerebral electrical activity (CEA) throughout the procedure. The mean arterial blood pressure (MAP) was between 80 to 50 torr. SA = start of the anesthesia, PCB = partial cardiopulmonary bypass, TCB = total cardiopulmonary bypass.

were frequently experienced. Often short periods of hypotension at normothermia resulted in transient depression of electrical activity but did not correlate with postoperative neurologic dysfunction.

Group III (*Fig. 2*) recordings were universally obtained in patients undergoing procedures in which hypothermia was used (< 25 C). In these cases the cerebral activity falls continuously as the temperature decreases, reaching the zero cerebral electrical activity level at temperatures below 20 C (*Fig. 2*). As the patient is rewarmed, the cerebral function monitor tracing demonstrates reactivation and return to baseline activity. During the period of deep hypothermia, hypotension, low flow, or even circulatory arrest appears to be well tolerated.

Patients whose tracings return to baseline after rewarming have recovered neurologic function that is similar to those in groups I and II. Again, small focal lesions escape detection by this method.

Tracings of the type found in group IV were the most diagnostic and unfortunately demonstrated the worst prognosis. Unlike those in group III, the cerebral electrical activity does not return to baseline. The setting is usually one of sudden unexpected change. One patient who underwent mitral valvuloplasty was off cardiopulmonary bypass with the arterial cannula still in place in the ascending aorta (*Fig. 3*). A sudden deterioration in the cerebral function monitor tracings was noted without change in the hemodynamic parameters. At this point a large bolus of air was noted in the base of the aortic cannula. Maneuvers were performed to lessen the devastating effects of the air embolus. After several minutes the cerebral function monitor tracings returned to a more normal (but not baseline) level. The patient awoke slowly and had a left hemiplegia, which cleared over 2 days. This type of tracing has been almost universally associated with severe neurologic dysfunction after surgery, unfortunately often without recovery.

Discussion

Inadequate cerebral perfusion is a well-recognized hazard of open heart

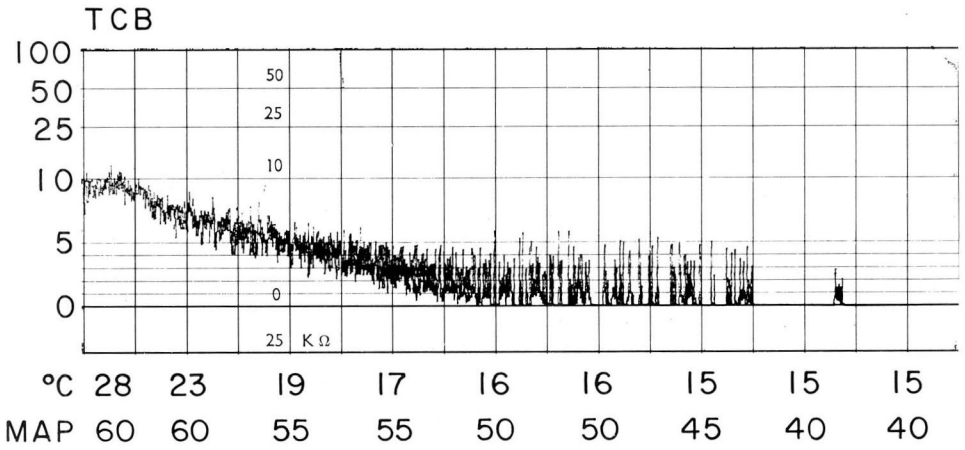

Fig. 2. Recording of cerebral electrical activity during deep hypothermia (total cardiopulmonary bypass [TCB]). MAP = mean arterial pressure.

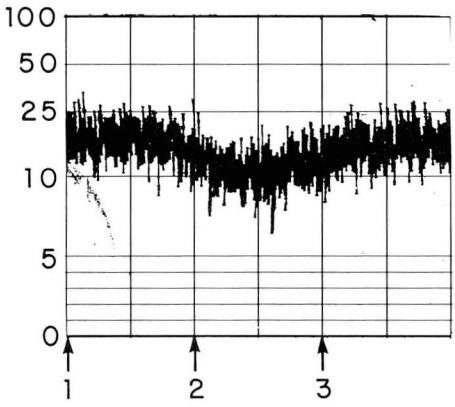

Fig. 3. Patient after mitral valvuloplasty. 1. Mean arterial pressure, 65; partial cardiopulmonary bypass. 2. The large bolus of air was noted in the base of the aortic cannula.

surgery with cardiopulmonary bypass. Routine intraoperative assessment of cerebral integrity is not yet widely established.[2,5] Garman,[6] in a survey of 26 institutions, showed that recording of cerebral electrical activity was performed only in five centers. Reasons for this can be found in the cost and complexity of standard EEG equipment and the logistics of introducing these into the operating room. Prior[2] described requirements for a cerebral monitoring system that could be used intraoperatively. They were (1) simplicity, (2) reasonable cost, (3) reliability, (4) direct continuous information about cerebral electrical activity, (5) quantifications of output of reading available for long periods, and (6) noninvasive automatic operation. In our experience the cerebral function monitor has proved to meet these requirements.

In instances of deep hypothermia and circulatory arrest, return of cerebral electrical activity before generalized rewarming has begun is undesirable. It is believed that return of cerebral electrical activity signals the return of metabolic activity in a truly unfavorable milieu.

Evaluation of cerebral function monitor tracings obtained in various clinical settings has enabled us to make some predictions and more importantly some therapeutic interventions to reverse the damaging neurologic effects. Patients who demonstrate sudden drops in cerebral electrical activity without any evidence of hemodynamic deterioration and whose tracings do not return to baseline within 1 to 2 minutes are now treated in the operating room as if a massive cerebral insult has occurred. Steroids in pharmacologic doses are given and a trial of high-dose barbiturate infusion as proposed by Safar[7] is also started in an effort to reduce neurologic dysfunction. In case of air embolism accidents, treatment in a hyperbaric chamber may be considered.

In patients whose cerebral circulation may be compromised preoperatively,[8,9] e.g., patients with carotid bruits or previous cerebrovascular accident, levels of mean arterial pressure that would otherwise be acceptable may be found to result in decreased cerebral electrical activity. If the cerebral function monitor tracings decrease unexpectedly, vasopressor agents are used to maintain high pressures and reverse the downward trend. Further study is necessary to evaluate the salutory effect of these interventions.

The cerebral function monitor has also aided in cases in which cardiac performance has been marginal. The decision to use cardiotonic drugs, intraaortic balloon pumping or reinstitute cardiopulmonary bypass is complex. Before cardiac output measurements can be obtained or urine production significantly decreases, the cerebral function monitor tracing may show a definite adverse trend. Measures may then be taken sooner to reverse the perfusion deficit. The cerebral function monitor does not replace the usual parameters

for determining adequate function but becomes another reliable, immediately available scale that can be of assistance to the operating team.

In deep hypothermia and circulatory arrest, return of cerebral electrical activity before generalized rewarming has begun is undesirable. It is believed that return of cerebral electrical activity signals the return of metabolic activity in a truly unfavorable milieu. When the cerebral function monitor demonstrates the return of cerebral electrical activity, the limits of safe circulatory arrest may have been reached. The surgeon may then elect to restart cardiopulmonary bypass, providing some flow to the cerebral circulation as a measure of protection. It is hoped that this will assist in preventing neurologic dysfunction that is evidenced years later in the child's development.

As experience widens with the cerebral function monitor, confidence is also gained in its use. Patients who require resuscitation in the operating room or intensive care unit can be evaluated with the cerebral function monitor to determine the efficacy of the resuscitative measures. Decisions to continue support or institute complex heart assist devices will not be made easier but may be based on more objective data.

Summary

The cerebral function monitor is a simple, reliable method of evaluating cerebral electrical activity in the operating room. Use in 650 patients who underwent operations for acquired and congenital heart disease has provided information helpful in detection, prevention, and early treatment of neurologic dysfunction. The subgroup of patients whose tracings develop severe depression of cerebral electrical activity without prompt return to baseline are those in whom intervention must be undertaken rapidly to prevent or at least reduce neurologic complications. Such disastrous occurrences can only be prevented by rapid recognition of precipitating events. We believe that although the cerebral function monitor is limited in that it provides no information as to degree or location of injury, it does provide the knowledge that there is something amiss with cerebral perfusion requiring immediate attention.

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