

MULTICHANNEL ELECTROENCEPHALOGRAPHY IN HYPOTHERMIA AND CIRCULATORY OCCLUSION

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MOST studies of the electroencephalogram (EEG) during anesthesia and surgery have been confined to a single channel technique recording from a frontal-occipital electrode pair. This has provided interesting information about the response of the brain to these procedures, but has given no clue to changes occurring in segments of the brain. To determine if such changes might escape detection, we have investigated additional combinations of leads during hypothermia and circulatory occlusion.

In a recent report¹ we summarized our observations on the frontal-occipital lead in these circumstances. This communication presents our experience in 36 additional patients in whom multiple EEG leads were observed.

METHOD

Patients undergoing cardiac surgery with hypothermia were studied. Premedication was with opiate, usually morphine 1 mg. per year of age to a maximum of 10 mg., and scopolamine 0.1 mg. for children and 0.2–0.3 mg. in adults. Anesthesia was induced with thiopental, cyclopropane, or a combination of the two. Ether was administered for induction of hypothermia, except that in two cases nitrous oxide was used. Muscle relaxation was obtained with tubocurarine. Pulmonary ventilation was insured throughout by manual compression of the rebreathing bag. Hypothermia to a final temperature of about 30 C. was induced by immersion in ice water, with esophageal and rectal temperatures monitored. At the appropriate time rewarming was accomplished by short-wave diathermy or water-circulated blankets. Nitrous oxide-oxygen (500:500 cc./minute) was usually administered after establishment of hypothermia so

that electrocautery could be used by the surgeons.

Nonpolarizable needle electrodes were inserted in the scalp just after induction of anesthesia, and were placed symmetrically over the motor, premotor, frontal, temporal, parietal, and occipital areas, with midline frontal-occipital electrodes used as well in some patients. The electrode placement corresponded generally to standard positions F7 and 8, T3 and 4, T5 and 6, O1 and 2, F3 and 4, and C3 and 4 for the regular electrodes. With this arrangement, it was possible to record from any pair of electrodes by changing selector switches on the recorder, a Grass model IIID, 4-channel electroencephalograph. Intermittent recordings were made during cooling and rewarming. Continuous records were made during occlusion and for at least 10 minutes after resumption of circulation.

For routine monitoring we used the motor-premotor and occipital-parietal pairs, bilaterally. Only the motor-premotor leads were in place while the patient was in the ice bath. Different combinations were tried from time to time, such as paired leads all on the same side, and a biparietal combination.

Five patients were subjected to hypothermia without circulatory occlusion, and a total of 50 circulatory occlusions was observed in the other 31 patients. The shortest occlusion was less than 30 seconds, and the longest single occlusion 7 minutes. Eight patients had more than one period of occlusion. The patients' ages ranged from 2 to 37 years.

RESULTS

There was a tendency toward lower voltage in the adults, but relative voltages in the same patient were consistent during the period of observation. Age did not appear to influence the response to hypothermia or circulatory occlusion.

The records from 26 patients were adequate for evaluating changes during induction of

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TABLE 1
CHANGES IN THE ELECTROENCEPHALOGRAM AS TEMPERATURE WAS REDUCED

	No Change	Decreased Frequency		Increased Frequency	Increased Voltage	Decreased Voltage
		<25%	>25%			
Number of patients	11	8	4	1	5	3

hypothermia. EEG changes were minimal and similar to the changes recorded from frontal-occipital electrodes (table 1). About half of the records showed some decrease in frequency, usually slight, and in half no change was apparent. A few showed some change in voltage, and one showed increase in frequency. There was no evidence of asymmetry between the hemispheres, and no difference in response of frontal and posterior areas.

Only one patient's temperature was reduced below 29 C.; the one reaching 27.5 C. At 36 C. this patient had 20 cps (cycles per second) 40 microvolt activity, which fell to 15 cps at 31 C. and 10–12 cps at lowest temperatures. Voltage remained essentially unchanged.

In this study of 50 total circulatory occlusions, the response was consistent from patient to patient. After occlusion, there was a lag period in which the EEG did not change. Next came a period of progressive slowing of frequency followed by reduction in amplitude. Then all activity disappeared and the tracing became isoelectric (flat). In one regard these

findings differ from changes seen with fronto-occipital electrodes. The fronto-occipital lead showed consistently a low voltage fast frequency activity just before the tracing became flat. This activity could not be recognized in the other leads. When the fronto-occipital lead was observed, in addition to the other leads, low voltage fast frequency activity was present in it but not seen in those leads recorded simultaneously (fig. 1).

Approximately half the patients showed some asymmetry in the response to circulatory occlusion, while the others showed equal response in different areas of the head. The occipital regions tended to develop slowing before the frontal regions (fig. 2) and also tended to recover earlier (fig. 3). However, the time lag was only a matter of one to three seconds in all instances. Little asymmetry was seen in the appearance of the flat record (fig. 4). Figure 5 is a representative segment of a tracing showing the closeness, but definite lag, in the slowing of frequency seen after circulatory occlusion.

Since these results have shown that various

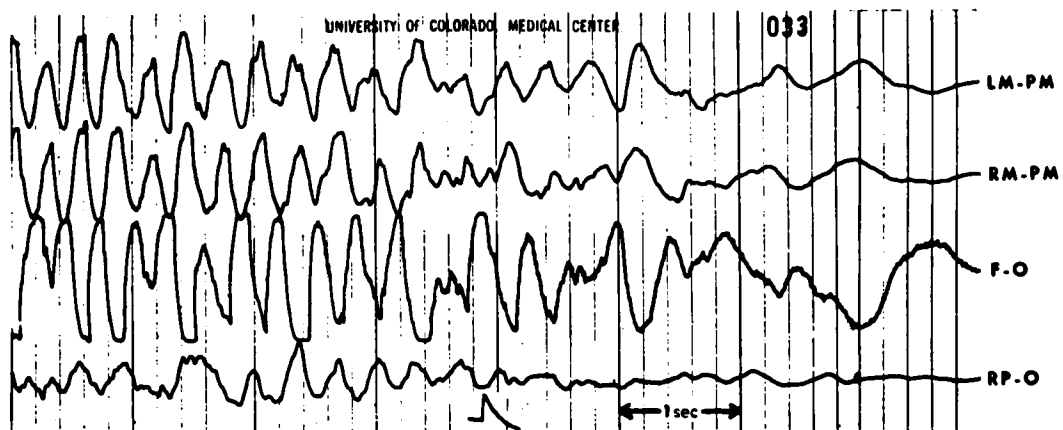


FIG. 1. Segment of the EEG 20 seconds after onset of circulatory occlusion showing low voltage fast ("file") activity only in the frontal-occipital lead. Calibration: 50 microvolts. LM-PM: Left motor-premotor; RM-PM: Right motor-premotor; F-O: Frontal-occipital; RP-O: Right parietal-occipital.

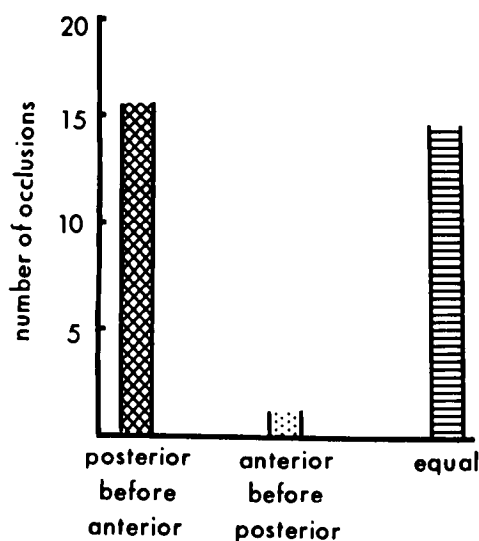


FIG. 2. Response to circulatory occlusion. Asymmetry in time from onset of occlusion to slowing of EEG frequencies in the anterior and posterior areas of the brain.

parts of a patient's brain often require different times to respond with characteristic EEG changes after circulatory occlusion, no average values are reported. However, most of these patients showed slowing in 15 to 25 seconds (range 3–50) and became isoelectric in 25–35 seconds (range 15–60).

After resumption of circulation, 27 patients showed activity reappearing as very low voltage, slow or fast frequency waves which gradually and steadily progressed, in most cases,

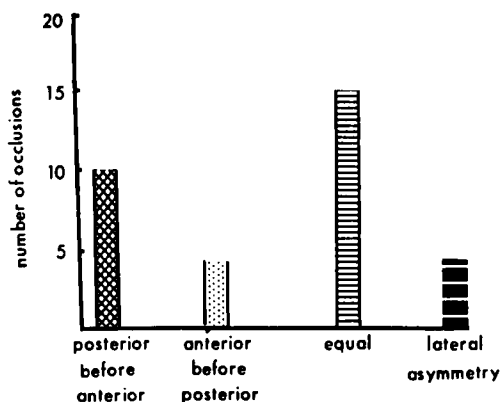


FIG. 3. Response to circulatory occlusion. Asymmetry in time from reinstitution of circulation to first EEG activity, in opposite sides, and anterior and posterior areas of the brain.

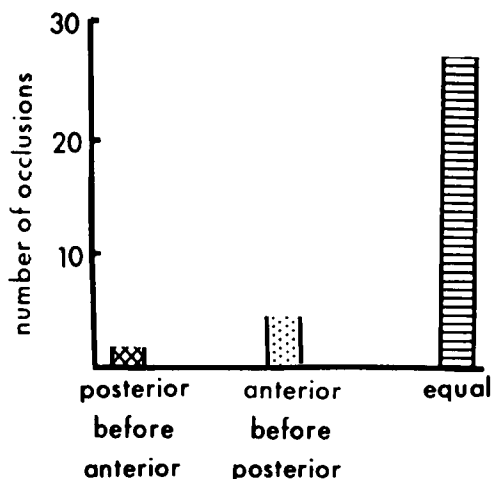


FIG. 4. Response to circulatory occlusion. Asymmetry in time from beginning of occlusion to flat EEG, in the anterior and posterior areas of the brain.

to the pattern seen prior to occlusion. Reappearing activity frequently presented a low voltage fast frequency ("file") pattern, which showed progressively increasing voltage until it reached a preocclusion level. During recovery in 4 patients, EEG activity reappeared as intermittent bursts separated by isoelectric periods. This resembled typical burst-suppression activity. The suppression intervals became shorter until within a minute activity became continuous. There was no evidence that different types of initial activity were prognostically important.

As had been found previously, recovery was slower after longer occlusions, and details of the results in this series parallel our previous figures.

The patient whose circulation was occluded at a temperature lower than 29 C. required 80 minutes after resumption of circulation to return to a preocclusion EEG. This time was prolonged, and did not substantiate our previous findings that the lower temperatures were associated with more rapid recovery. However, the circulation of this patient was occluded twice, 6 minutes and 5½ minutes, and completely normal EEG activity was not obtained between occlusions. Eventual total recovery, however, was uncomplicated.

Two patients in this series had greater than five circulatory occlusions, ranging in length

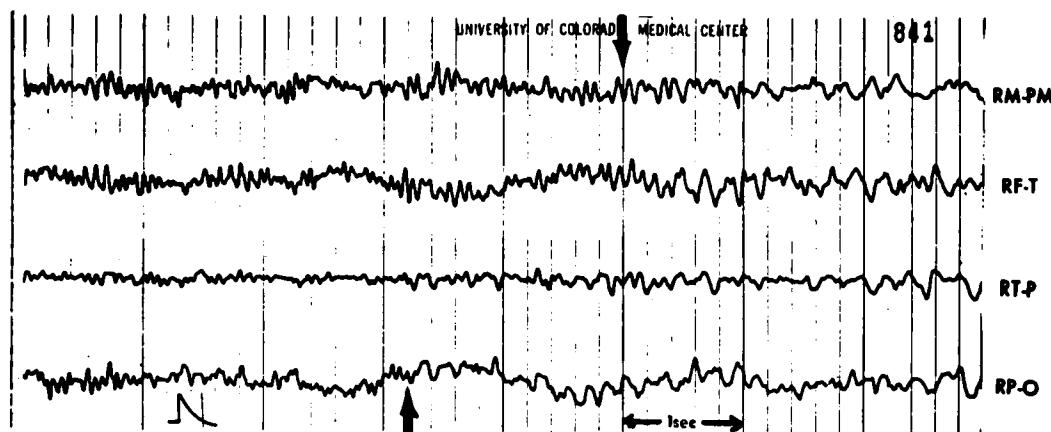


FIG. 5. Leads derived from the right side illustrating posterior slowing before anterior. Arrows indicate first evidence of slowing. Calibration, 50 microvolts. RM-PM: Right motor-premotor; RF-T: Right frontal-temporal; RT-P: Right temporal-parietal; RP-O: Right parietal-occipital.

from 30 seconds to five, and to six, minutes. One of these patients also developed ventricular fibrillation, which added to the time of cerebral ischemia. In this patient, the EEG was flat for 31 minutes, and had not recovered to preocclusion level in 2 hours. The other patient's EEG was flat for 49 minutes and had not recovered to preocclusion activity in 75 minutes when recording was discontinued. Postoperatively both patients had an uneventful recovery.

In addition to the delayed EEG recovery in these two patients, two others required 2 hours or more for the tracing to return to preocclusion condition. One of these 4 patients died. Of the other 27 patients who had circulatory occlusion, one died. Both deaths were upon a circulatory basis, one of pulmonary thrombosis, the other of congestive failure. Therefore, a mortality rate of 1 in 4 occurred in those with prolonged EEG recovery compared with a mortality rate of 1 in 27 of those with EEG recovery within 2 hours. In none of the patients was there clinical evidence of cerebral damage postoperatively.

During rewarming, many records were obscured by muscle and electrical artifact (diathermy was used to rewarm). Most patients required some supplemental anesthetic agent as the temperature rose, so that complete studies of EEG changes could not be obtained. However, the study of 9 fairly complete rec-

ords showed no consistent change after stabilization from the effects of circulatory occlusion. The stabilized pattern was generally maintained through rewarming until the patient was awakened.

In this series 2 patients showed considerable delay in recovery in one hemisphere while the other recovered normally. We have no immediate explanation for this asymmetry. However, our experience in extracorporeal circulation, to be presented in another report, suggests that circulatory embarrassment, particularly venous obstruction, may be paramount in producing this picture.

DISCUSSION

The results of recording 4-channel electroencephalograms during hypothermia and circulatory occlusion have added some additional features to the material available from single channel recording. The two most striking differences seen were the absence of low voltage fast frequency ("file") activity after circulatory occlusion, and the variable response times in different parts of the head.

That the low voltage fast frequency activity was probably still present was indicated by its identification in the frontal-occipital lead when it was simultaneously absent in other leads. One might speculate, therefore, that this peculiar activity represented the summation of extremely small potentials, and could be ob-

served only when inter-electrode distance was great and the number of neurons interposed between the electrodes tremendous. This concept was corroborated by the occasional presence of "file" activity in other leads during recovery from the effects of circulatory occlusion. The frontal-occipital lead has shown that this "file" activity gradually built up in voltage as the brain recovered from ischemia, so that at some point this voltage would be adequate to be recorded from electrodes placed closer together. As this voltage increased, "file" activity became apparent in parietal-occipital and motor-premotor electrodes. Perhaps if equipment were made sensitive enough, and sufficiently free from artifact, one could record many low voltage potentials that are now missed. One might even see activity during the period of circulatory occlusion, which now appears devoid of activity because present rather crude instruments record a "flat" tracing.

In selected cases, there was a definite though short lag in the response of some portions of the brain to circulatory occlusion. There was no consistent pattern for this asymmetry, and we have no evidence at this time that it is clinically significant. However, this front-to-rear difference should come as no surprise, for Schneider *et al.*² have shown time differences in EEG response to nitrous oxide inhalations.

The mortality figures from this series substantiate our previous findings that delayed recovery of the EEG after circulatory occlusion was a poor, but by no means hopeless, prognostic sign. We have no evidence that cerebral injury contributed to mortality, since both of the deaths in this series were precipitated by circulatory aberrations. Moreover there is no evidence that the periods of circulatory occlusion used were harmful to the brain, since all the survivors had apparently normal cerebral function in the postoperative period. Even the patient who had multiple occlusions and no evidence of cerebral electrical activity

for more than 45 minutes recovered without apparent sequelae.

SUMMARY

The results of monitoring multiple leads of the EEG during hypothermia and circulatory occlusion in 36 patients have been presented. Little change was seen as body temperature was lowered to about 30 C., or as temperature was brought back toward normal. During occlusion the EEG progressively became slower, reduced in amplitude, and finally flat. Recovery after resumption of circulation began with low voltage activity which progressively returned to the preocclusion level.

About half of these patients showed a tendency for changes that occurred after circulatory occlusion, and following reinstitution of circulation to appear in one lead (or one portion of the head) before appearing in the others. A few patients showed asymmetry between the two hemispheres during recovery.

A relatively poor prognosis was noted when full EEG recovery was prolonged beyond 2 hours; however, no survivors appeared to suffer cerebral sequelae even though, in two instances, the EEG was flat for more than one-half hour.

Low voltage fast frequency ("file") activity was notably absent in the leads sampling relatively small areas of the brain, while it appeared at the same time in the frontal-occipital lead.

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