Electroencephalographic Patterns Related to Hemodynamic Changes at the Onset of Cardio-Pulmonary Bypass Circulation

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SUZUKI, H., KAWAKAMI, Y. and FUJITA, M. Electroencephalographic Patterns Related to Hemodynamic Changes at the Onset of Cardio-Pulmonary Bypass Circulation. Tohoku J. Exp. Med., 1991, 164 (4), 331–337 — Electroencephalographic (EEG) changes in relation to hemodynamic changes at the onset of cardio-pulmonary (C-P) bypass circulation in 31 cases undergoing open heart surgeries were studied. Rapid and extensive changes in systemic circulation, which were followed by abnormal EEGs, appeared within the first five minutes of C-P bypass. Abnormal EEG patterns including disappearance of fast waves, slowing under 6 Hz in frequency with high voltage and flattening were observed in 64% of the cases. The relations between the appearance of these abnormal EEGs and hemodynamic factors such as mean arterial pressure (mAP), central venous pressure (CVP), rate of decrease in mAP and rate of change in CVP, were analyzed in order to elucidate causes of development of the abnormal EEGs. Only the rate of decrease in mAP at the onset of C-P bypass had a high correlation (p < 0.01) with the appearance of abnormal EEGs. The findings suggest that rapid circulatory changes in the first five minutes of C-P bypass may induce disruption of cerebral autoregulation. —— EEG; C-P bypass circulation; systemic circulation; autoregulation; hemodynamic changes

Direct recording of EEGs and cerebral function monitoring (CFM) reveal that EEG patterns are extremely sensitive to changes in the systemic circulation at the onset of cardio-pulmonary (C-P) bypass circulation (Davenport et al. 1959; Brierley 1967; Wright et al. 1972; Branthwaite 1973; Witoszka et al. 1973). Among a number of factors which affect EEG patterns and CFM at the onset of C-P bypass, arterial hypotension is one of the most important. In the conscious state, cerebral blood flow is sustained within a normal range via cerebral autor-
regulation even during periods of severe hypotension. The safe lowest limit of mAP for cerebral autoregulation has been reported previously (Harp and Wollman 1973; Shapiro 1978). The rate of decrease in blood pressure also affects autoregulation of cerebral circulation (Trojaborg and Boysen 1973). In the present study, the degree and rate of change in the systemic circulation and EEG changes were recorded simultaneously during the first five minutes of C-P bypass. The circulatory factors affecting cerebral autoregulation at the onset of C-P bypass were examined in terms of the correlation between these factors and the appearance of abnormal EEG patterns.

METHODS

Studies were performed in a total of 31 patients undergoing open heart surgery, ranging in age from 5 to 20 years. None of the patients had any signs or symptoms of neurological dysfunction in the preoperative period. Their diagnoses were: arterial septal defects [5 cases], ventricular septal defects [13], Tetralogy of Fallot [5], endocardial cushion defects [3], tricuspid valvar stenosis [2], d-TGA [1], pure pulmonary valvar stenosis [1] and mitral valvar insufficiency [1]. In all cases, anesthesia was induced and maintained with halothane (0.5-1.0%), nitrous oxide (50-60%) or air and oxygen. Arterial blood pressure (AP) and central venous pressure (CVP) were continuously monitored through catheters inserted in the radial artery and the external jugular veins, respectively. The mean AP (mAP) and CVP just before C-P bypass circulation and the lowest value of mAP and CVP during the first five minutes of C-P bypass were measured. The rates of change in mAP and CVP were calculated using the following equation: (mAP or CVP before C-P bypass)-(the lowest value of mAP or CVP during first 5 min of C-P bypass)/duration between two points; the first point at which mAP or CVP was measured just before C-P bypass, the second point at which lowest mAP or CVP was measured in the first five minutes of C-P bypass.

A C-P bypass circulation was achieved with a Model-2D (Tow Nak Co., Tokyo) with the Kolobow membrane oxygenator (Sci Med Co., MA, USA). Arterial infusion and venous return flow were measured with an electromagnetic flow meter (Sanei Co., Tokyo). The mean arterial flow rate during C-P bypass was 100-180 ml/kg/min.

EEG patterns recorded during the first five minutes of C-P bypass were compared to that recorded just before C-P bypass initiation. EEGs were recorded from 6 monopolar leads (F3, F4, C3, C4, O1, O2) with a reference electrode at the earlobe. Data were fed into an electroencephalograph recorder (EEG-5109; Nihon-Kohden Co., Tokyo) with a bandpass of 100 Hz-0.3 Hz and stored on magnetic tapes for later analysis. Five second EEGs were digitized and analyzed to obtain the power spectrum (softwear Power Spectrum 48A; Sanei Co.) with a Sanei signal processor 7T08. EEG during first five minutes of C-P bypass were classified into two groups as follows; group I showed no change as compared to that just before C-P bypass, group II had EEG changes including abnormal patterns.

All statistical significance was evaluated using an unpaired t-test.

RESULTS

EEG changes during C-P bypass

Pronounced changes in EEGs (Fig.1) were observed within the first five minutes of C-P bypass circulation in twenty (group II) of 31 cases (Fig. 2). The abnormal EEGs observed in group II were as follows; disappearance of fast waves above 10 Hz in frequency, slowing under 6 Hz with high voltage more than
EEG changes and arterial blood pressure

The preC-P bypass mAP were 82.1 and 77.4 mmHg for groups I and II, respectively. The lowest mAP during the first 5 min of C-P bypass circulation were 38.9 for group I and 36.2 mmHg for group II. There were no significant difference in mAP between the two groups. The mAP decreased, however, faster in group II than in group I. The rate of decrease in mAP was 0.66 mmHg/sec on the average in group II which was statistically significant (p < 0.01) as compared to 0.34 mmHg/sec in group I (Table 1).

EEG changes and central venous pressure

The CVP before and during C-P bypass were identical in the two groups (group I: 15.8 and 14.9 cmH$_2$O; group II: 15.3 and 14.9 cmH$_2$O). The rate of
There was no significant difference between the two groups in perfusion rate, once arterial perfusion flow was kept constant. There was also no significant
difference in the duration from the onset of C-P bypass to the point at which constant perfusion was established (40 sec in groups I, 50 sec in group II). However, the duration significantly \((p < 0.01)\) prolonged to 120 msec in eight cases of group II which showed marked slowing under 3 Hz or flattening.

**Postoperative complications**

Only three of the 31 patients, all in Group II, experienced delayed emergence from general anesthesia. None, however, developed any neurological complications in the postoperative period.

**DISCUSSION**

The remarkable circulatory changes were observed during the first five minutes of C-P bypass in our study. Rapid hypotension and changes in CVP were followed to starting C-P bypass circulation. Balance of flow volume between venous drainage and arterial perfusion was not maintained adequately at the onset of C-P bypass. However, perfusion rate became constant and an adequate C-P bypass circulation was obtained within 5 min after onset of C-P bypass in most cases. Therefore, effects of the initial five-minute circulatory changes on EEG patterns were analyzed.

The mean value of the lowest mAP in the first five minutes of C-P bypass were identical in the two group. The mean value of mAP was, however, lower than the critical limit (Shapiro 1978; Sharbrough et al. 1973). Furthermore, some cases in group I sustained normal EEG patterns even when their mAPs fell below 30 mmHg. Dong et al. (1983) reported that high frequency bands of EEG activities disappeared, only the delta band activities remaining, during trimethaphan induced systemic profound hypotension of 20 mmHg in dogs. Brain cell damage due to profound arterial hypotension was reported at a mAP of 25 mmHg in monkeys (Brierley et al. 1969; Meldrun and Brierley 1969). These observations suggest that a normal EEG pattern can be maintained even under 50 mmHg of mAP under certain conditions. Schneider (1963) suggested that very rapid changes in blood pressure may disrupt autoregulation of cerebral circulation. In our experiment, the high rate of decline of mAP induced abnormal EEG patterns. The results suggest that rapid mAP changes may be the most important cause of abnormal EEG patterns at the onset of C-P bypass. The rate of changes rather than the lowest value of mAP, is likely to correlate with the degree of cerebral autoregulatory dysfunction during C-P bypass.

Six cases of group II showed a slight increase in CVP at the onset of C-P bypass. Paton et al. (1960) reported the six cases of EEG changes due to superior vena caval obstruction at the onset of C-P bypass undergoing open heart surgery. In our cases, no clinical signs of vena caval obstruction were observed. However, rapid and excessive arterial perfusion was detected by a flow meter at the onset of C-P bypass. Rapid and excessive perfusion via the femoral artery has been
shown to augment rCBF and induce abnormal EEGs in anesthetized cats (Suzuki et al. 1979). The rapid change in blood volume also may produce disruption of cerebral autoregulation.

Anesthetic drugs are considered to effect both the EEG and cerebral circulatory autoregulation. In this study, volatile anesthetics were cut off about 10 min before C-P bypass. The blood concentration of anesthetics measured by gas-chromatography were low enough so as not to induce slowing on EEG in the first five minutes of C-P bypass.

Correlations between abnormal EEG patterns during open heart surgery and post-operative neurological complications have been confirmed by Witoszka et al. (1973). As previously reported (Davenport et al. 1959; Wright et al. 1972), EEG changes observed immediately after the onset of the C-P bypass lasted for only a few minutes. These investigators found that the short duration of changes in EEG and CFM did not cause neurological complications (Branthwaite 1973). In our study, although three cases in group II showed delayed emergence from general anesthesia, no clear correlation between this phenomenon and EEG changes could be demonstrated. In addition, no neurological complications occurred during the postoperative period. We previously reported a case with marked postoperative neurological complications which had been preceded by abnormal EEGs (Suzuki et al. 1979). In this case, the abnormal EEG patterns appeared at the onset of C-P bypass and lasted throughout the operation. In contrast, EEG changes of short duration in this study which appeared at the onset of C-P bypass did not have prognostic significance. EEG monitoring during open heart surgery is no wonder to be useful for detection of abnormal EEGs which forecast brain damage following open heart surgeries. Moreover, monitoring of arterial pressure also gives us important informations for brain functions.

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References


