Cerebral functional changes following cardiac surgery: Neuropsychological and EEG assessment

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Abstract

Objective: Some form of organic and functional cerebral deficit may occur in up to one third of patients following cardiopulmonary bypass surgery. This study was designed to assess cerebral functional deficit in cardiac surgical patients.

Methods: Neuropsychological and quantitative electroencephalographic (EEG) changes were assessed in 62 first time coronary artery bypass graft surgery patients before surgery and within 1 week and 2 months after surgery. Patients underwent surgery with a standard Hammersmith Hospital anaesthesia and hypothermic cardiopulmonary bypass (28°C), using either bubble (Harvey 1700, n = 28) or membrane (Cobe CML, n = 34) oxygenators with arterial line filters (Pall 40 μm). Neuropsychological performance was assessed using a well established battery of ten tests. Four EEG relative power frequency bands; δ (1–3.5 Hz), θ (4–7.5 Hz), α (8–11.5 Hz), and β (12–23 Hz), were determined using Fast Fourier Transformation (FFT).

Results: Neuropsychological and EEG deficits were found in 48% of patients 1 week after surgery and in 34% 2 months after surgery. Post-operative deficits were not associated with duration of perfusion, type of oxygenator used in surgery or patient age. Neuropsychological and EEG deficits were associated 2 months after surgery, but not 1 week after surgery. Post-operative EEG deficit was associated with pre-operative deficit.

Conclusions: Cerebral functional deficit was found following CABG surgery using quantitative EEG and neuropsychological assessments. Patients who had neuropsychological deficit were also more likely to show EEG deficit. EEG deficit before and after surgery suggests vulnerability of patients with already compromised cerebral function to the effects of CPB procedure. © 1998 Elsevier Science B.V.

Keywords: Quantitative EEG; Brain damage; Neuropsychological deficit; Cardiac surgery; CABG; (Human)

1. Introduction

Cardiopulmonary bypass (CPB) has been associated with cerebral morbidity in cardiac surgery patients. The aetiology of subtle cerebral deficit includes cerebral ischaemia and resulting hypoxia which are believed to be common pathophysiological consequences of damage mechanisms, such as microemboli [1–3] and systemic hypotension [4,5]. Post-operative neurological and neuropsychological (NP) deficits have been demonstrated 1 week and 2 months [6–8], 1 year [9] and longer after CPB surgery [10]. Incidence of sub-clinical cognitive deficit was reported to range between 20 and 80% [11]. The severity and duration of functional deficit was not found in other forms of major thoracic surgery [6,7].

EEG has been used mainly intra-operatively as a means of monitoring and detecting changes in cerebral function which may result in post-operative cerebral abnormalities [3,12–14]. An increase in slow frequency power in the region of 2–3 S.D. has been related to
post-operative neurological deficit following CPB surgery [3,13,15].

Pre-operative and post-operative EEG, together with neurological and neuropsychological assessment have been used to investigate post-operative functional deficit in CPB patients [15]. This study showed that the presence of early post-operative neurological signs predicts EEG abnormalities years after surgery. EEG abnormalities represented an increase in slow frequency power (1–3.5 Hz), presence of sharp waves and δ-wave disturbances. Zeinthofer et al. (1988) reported a slight increase in δ, α and β frequency power in CPB patients after surgery [16]. Mazzoni et al. (1993) found increased δ and θ power in over one third of CABG and valve replacement patients [17].

We have previously studied CABG patients using quantitative EEG (QEEG) together with magnetic resonance imaging (MRI) and neuropsychological assessment together with long latency evoked potentials P300 [8]. These studies found a structural and functional deficit in up to one third of patients after surgery. The objective of the present study was to investigate cerebral functional deficit after CABG surgery using neuropsychological assessment and QEEG and to identify patients who had most deficit. Also, to investigate any association between neuropsychological and QEEG deficits.

2. Methods and materials

A total of 62 patients between 45 and 75 years old, undergoing elective, first time CABG surgery were recruited at random from the cardiac surgical waiting list. The patients cerebrovascular condition was not investigated before surgery. All of the patients were receiving aspirin, anticoagulants and β-blockers for the control of angina. The effect of these drugs on cerebral function is not considered to be significant and was not investigated further in this study. A total of 20 age-matched healthy volunteers had a single EEG recording. This group was introduced because we considered that no reliable age-matched control data exists for 21 channel QEEG. These 20 formed the EEG control group. Only English-speaking patients who were willing to attend as outpatients pre- and post-operatively were approached. Patients with a history of neurological and psychiatric illness were excluded. The study had ethical committee approval from the Royal Postgraduate Medical School and all patients gave written informed consent.

Anaesthesia and surgery, in brief, premedication included lorazepam (1–2 mg), papaveretum (10–20 mg), hyoscine (0.2–0.4 mg) and droperidol (2.5 mg). Anaesthesia was induced with thiopentone (0–3 mg/kg and fentanyl (45 μg/kg). Muscle relaxation was achieved with pancuronium (0.1 mg/kg) and the lungs were ventilated to maintain an end-tidal arterial PCO₂ of 4–5 kPa. Anaesthesia was maintained with 50% nitrous oxide in oxygen. In most cases, an additional dose of fentanyl was given to patients before the onset of perfusion and at the start of rewarming.

Hypothermic bypass (28°C) was established between the right atrium and the ascending aorta at a flow of 2.4 l/min. Mean radial arterial pressure was maintained between 50 and 70 mmHg. At the time of the study (between 1990 and 1993), both bubble and membrane oxygenators were in use in our unit. Patients were non-selectively allocated either a bubble (Harvey 1700, Bard Cardiopulmonary, Santa Ana, CA) or membrane oxygenator (Cobe CML flat sheet oxygenator, Cobe Laboratories, Lakewood, CO) with an additional inline filter (Pall 40 mm). The oxygenators were primed with Hartmann’s solution. Arterial blood gases were maintained using an α-stat protocol. The internal mammary artery and saphenous vein grafts were used for myocardial re-vascularisation.

2.1. Neuropsychological method

The neuropsychological method and rationale have been described before and are shown in the Table 1 [8,14,18]. NP tests of memory (verbal and non-verbal), attention and concentration, perceptual speed, psychomotor performance and manual dexterity were administered before, within 1 week and 2 months after surgery. Post-operative deficit on any test represented a decrease in performance of at least 1 S.D. from the patients pre-operative record. NP deficit after surgery was calculated as a failure on at least two out of ten tests. The criteria used to determine deficit after surgery have been described before [7,9].

<table>
<thead>
<tr>
<th>Neuropsychological test battery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rey auditory verbal learning test (immediate, recent, and delayed, Rey 1–7)</td>
</tr>
<tr>
<td>Two computerized non-verbal memory tests (NVM levels 1, 2)*</td>
</tr>
<tr>
<td>Two trail making tests A and B (TMA, TMB)</td>
</tr>
<tr>
<td>Letter cancellation test (LCT)</td>
</tr>
<tr>
<td>Purdue pegboard (PGR, L, B)</td>
</tr>
<tr>
<td>Two choice reaction time tests* (CRT)</td>
</tr>
<tr>
<td>A Symbol-Digit replacement test* (SDR)</td>
</tr>
<tr>
<td>Weschler block design test (BD)</td>
</tr>
<tr>
<td>IQ tests (from Weschler Adult Intelligence Scale)</td>
</tr>
<tr>
<td>Vocabulary</td>
</tr>
<tr>
<td>Picture completion test</td>
</tr>
<tr>
<td>Mood assessment</td>
</tr>
<tr>
<td>Spielberger state/trait anxiety questionnaire (STAI 1, 2)</td>
</tr>
<tr>
<td>Beck depression questionnaire*</td>
</tr>
</tbody>
</table>

Computerized tests are marked with asterisks *.
2.2. EEG recording and processing

The EEG method has been described before [8,14]. EEG was recorded using a 21-channel topographic brain mapper (Brain Atlas iii, Bio-logic Systems, Mundelein, IL) using an electrode cap. EEG recording was carried out in a quiet research laboratory with the subject seated in a comfortable chair. Due to the clinical constraints, a limited time was available for research studies. For this reason a single recording condition, eyes closed, was chosen. To ensure that subjects were alert during recording and to standardize the procedure, a simple mental task was used. Subjects counted backward from 100 by 5s to 0. This could have caused change in EEG rhythm. However, this effect was not further investigated since the aim of this study was to assess quantitative EEG change over time. The electrode placement was in accordance with the International 10–20 System, with a nose reference, impedance was kept <5 kOhm, the band-pass filter setting was between 1 and 30 Hz. EEG was sampled at the rate of 128 Hz over a period of ∼60 s and the data were analysed off-line. At least 40 s of artifact free EEG were selected visually for further analysis. EEG data were summarised, averaged and expressed in terms of relative power (% total power). The frequency content was determined by Fast Fourier Transformation (FFT). Relative power was determined for the four frequency bands: δ (1–3.5 Hz), θ (4–7.5 Hz), α (8–11.5 Hz) and β (12–23 Hz). An increase in slow frequency power in a region of 1–7.5 Hz (δ and θ), and reduced power in the fast frequency power in the region of 8–23 Hz (α and β) have been associated with cerebral damage [19]. EEG data were normalised by comparing each pre-operative and post-operative sample to the mean of all control data.

Pre-operative deficit EEG was compared to the control data to detect any difference between patient and non-patient groups. Post-operative EEG was compared to pre-operative EEG. Considering that a sub-clinical neuronal dysfunction may be expected in patients, incidence of EEG deficit was determined taking into account intensity and area of change. EEG deficit before surgery represented a slow frequency power (1–7.5 Hz) greater than mean total of control data by at least 1 S.D. over at least five of the 21 channels. Post-operative deficit represented increase in slow frequency power from pre-operative EEG. To assess any relationship between NP and EEG deficit, a number of NP tests failed (out of 10) was then correlated with the number of channels showing deficit, i.e. increase in slow frequency power, for each patient.

2.3. Statistical analysis

The main aim in this study was to identify the patients who showed most deficit after surgery and to assess any relationship between NP and EEG changes. Analysis of variance was used to determine significant change within each EEG frequency band and between samples. Correlation analysis was used to assess the relationship between NP and EEG changes. Statistical significance was taken to be \( P < 0.05 \).

3. Results

There were 55 male and 7 female patients in the study (mean age 60.3 years, range 30–75, S.D. 8.7). All patients had an uneventful peri-operative course, showed normal recovery during ICU treatment and no new neurological abnormality detected by a routine post-operative clinical neurological examination. The mean bypass time was 85 min (range 45–143, S.D. 21.9). Bubble oxygenators were used in 28 operations and membrane oxygenators in 34 operations.

3.1. Neuropsychological findings

Table 2 shows NP tests performance which changed significantly from pre-operative level, i.e. improved or showed deficit, 1 week and 2 months after surgery.

One week after surgery, most deficit was found in tests of attention (LCT) and psychomotor performance using right, left and both hands simultaneously (PEGR, PEGL, PEGB), while performance on the tests of non-verbal memory (NVM) improved.

Two months after surgery, no significant deficit was found. Tests of divided attention (TMG), verbal (R1) and non-verbal memory (NVM) showed an improvement from pre-operative level.

Mood scores, anxiety (STAI1) and depression (BECK) showed no significant change 1 week after surgery and an improvement 2 months after surgery.

Before surgery, older patients took longer to perform on tests of divided attention (TMB, CRT), while patients who had higher trait anxiety (STAI2) performed better on psychomotor tests (PEGR, PEGB, BL). Patients who had higher scores of depression were slower on reaction-time test (CRT). Patients who had higher verbal and non-verbal IQ scores performed better on most tests (Spearman’s rank correlation coefficient \( P < 0.05 \)).

A total of 1 week and 2 months after surgery, patients who had higher verbal and non-verbal IQ scores performed better on tests of attention (LCT, TMA, TMB), test of visual-spatial ability (BD), motor ability (PEGR), memory (NVM) and reaction-time (CRT) \( P < 0.05 \). Duration of perfusion was not asso-
Table 2

Neuropsychological tests which changed significantly 1 week and 2 months after surgery

<table>
<thead>
<tr>
<th>Test</th>
<th>Before surgery</th>
<th>1 Week</th>
<th>Change</th>
<th>2 Months</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (S.D.)</td>
<td>Mean (S.D.)</td>
<td></td>
<td>Mean (S.D.)</td>
<td></td>
</tr>
<tr>
<td>LCT</td>
<td>147.6 (38)</td>
<td>166.6 (47)**</td>
<td>Deficit</td>
<td>152 (37)</td>
<td></td>
</tr>
<tr>
<td>TMB</td>
<td>106.7 (46)</td>
<td>109.9 (49)</td>
<td></td>
<td>92.3 (37)**</td>
<td>Improvement</td>
</tr>
<tr>
<td>PEGR</td>
<td>13.3 (1.9)</td>
<td>12.7 (2)*</td>
<td>Deficit</td>
<td>13.5 (1.8)</td>
<td></td>
</tr>
<tr>
<td>PEGL</td>
<td>13 (1.7)</td>
<td>12.4 (1.7)*</td>
<td>Deficit</td>
<td>13 (1.8)</td>
<td></td>
</tr>
<tr>
<td>PEGB</td>
<td>10.9 (1.9)</td>
<td>10.1 (2)*</td>
<td>Deficit</td>
<td>10.7 (2)</td>
<td></td>
</tr>
<tr>
<td>R1</td>
<td>5.4 (2.3)</td>
<td>5.7 (1.4)</td>
<td></td>
<td>6.2 (1.5)**</td>
<td>Improvement</td>
</tr>
<tr>
<td>NVM1</td>
<td>12.2 (2)</td>
<td>12.5 (2.1)</td>
<td></td>
<td>12.8 (1.3)*</td>
<td>Improvement</td>
</tr>
<tr>
<td>NVM2</td>
<td>12.4 (2.1)</td>
<td>13.1 (2)**</td>
<td>Improvement</td>
<td>13.1 (1.4)</td>
<td>Improvement</td>
</tr>
<tr>
<td>STAI1</td>
<td>30.4 (8.3)</td>
<td>29.6 (10)</td>
<td></td>
<td>26.9 (9.4)*</td>
<td>Improvement</td>
</tr>
<tr>
<td>BECK</td>
<td>7 (3.9)</td>
<td>5.4 (3.9)</td>
<td></td>
<td>4.8 (3.8)**</td>
<td>Improvement</td>
</tr>
</tbody>
</table>

LCT, letter cancellation time; TMB, trail making B; PGR, Purdue pegboard right; PGL, Purdue pegboard left; PGB, Purdue pegboard both hands; NVM1, non-verbal memory test (level 1); NVM2, non-verbal memory test (level 2); Rey 1, immediate verbal memory; STAI 1, Spielberger state anxiety questionnaire; BECK, Beck depression questionnaire.

The table showed mean test scores (range, S.D.), *P < 0.05, **P < 0.01, and direction of change for each test.

Associated with performance on NP tests 1 week and 2 months after surgery (Spearman’s rank correlation coefficient > 0.05).

3.2. Incidence of neuropsychological deficit

One week after surgery, 30 patients (48%), and at 2 months, 21 patients (34%) had significant cognitive deficit defined as failure on at least two of ten NP tests.

3.3. EEG before surgery

EEG data were normally distributed (P > 0.05 Kolmogorov–Smirnov test of normality). EEG absolute power (μV²), was used in the assessment of any differences between patients before surgery and controls. Means were calculated for each frequency range over all 21 channels. The data were analysed using ANOVA for unrelated data. Fig. 1 shows means for the four frequencies and level of significance (*P < 0.05).

Before surgery, patients had more δ power than the controls and less θ and α power. There was no difference in β power between groups.

3.4. EEG after surgery

Mean relative power for each frequency range over all 21 channels was calculated for patients before and after surgery. The difference before and after surgery was analysed using ANOVA for related data.

Fig. 2 shows mean EEG relative power for all frequencies before, 1 week and 2 months after surgery. The level of significance (*P < 0.05, **P < 0.01) represents the difference between pre-operative and post-operative values. One week after surgery, most decrease was found in mean slow power (δ, θ). α Power had also decreased, while β showed a significant increase from pre-operative level. At 2 months, all frequencies showed a significant decrease in power with the two fast frequencies, α and β, showing most decrease.

3.5. Incidence of EEG deficit

Before surgery, 27 patients (43%) had EEG deficit compared to the control group, seen as a significant increase in slow frequency power (1–7 Hz) over five or more recording channels. This deficit was not associated with patient age (rs = 1.2, P = 0.219). 30 patients (48%) had EEG deficit 1 week after surgery, and 21 patients (34%) had EEG deficit 2 months after surgery.

Patients who had significant EEG deficit before surgery were more likely to show deficit 2 months after surgery (rs = 2.4, P = 0.017), but not 1 week after surgery (rs = 1.5, P = 0.211). Patient age was not associated with EEG deficit 1 week (rs = 1.3, P > 0.05) or 2 months after surgery (rs = 1.4, P > 0.05). Also, bypass time was not associated with EEG deficit 1 week (rs =
Fig. 2. Mean (S.E.) EEG relative power in four frequencies for patients before, 1 week and 2 months after surgery. Significant difference between samples is indicated with an asterisk (* \( P < 0.05 \), ** \( P < 0.01 \)).

0.15 \( P > 0.05 \)) or 2 months after surgery (\( \rho = 0.10 \) \( P > 0.05 \)).

3.6. EEG deficit and oxygenators used in surgery

Two months after surgery, seven out of 28 patients (25%) operated on using bubble oxygenators and 14 out of 34 patients (41%) operated on using membrane oxygenators had EEG deficit. A similar proportion of bubble and membrane oxygenators was found in the no-deficit group. Table 3 shows the number (%) of patients who had EEG deficit and who were operated on using either a bubble or membrane oxygenator. To assess whether EEG deficit found 2 months after surgery can be associated with the type of oxygenator used in surgery, patients were split into EEG ‘Deficit’ and ‘No-deficit’ groups. The association between proportions was not significant (\( \chi^2 \) Yates corrected 0.199, \( P > 0.05 \)).

3.7. The relationship between neuropsychological and EEG deficit after surgery

For each patient, the number of NP tests failed after surgery was correlated with the number of EEG channels which had a significant increase in slow power. Table 4 shows the association between NP tests failed and the number of EEG channels which had a significant deficit 1 week and 2 months after surgery. Patients who failed the greater number of NP tests, also had greater EEG deficit 2 months (\( P < 0.05 \)), but not 1 week, after surgery.
4. Discussion

The main finding from this study was a significant cerebral functional deficit 1 week and 2 months after CABG surgery. This was seen as a deficit in cognitive performance and quantitative EEG.

A similar level of cognitive deficit was reported previously using similar [6] or the same [7,8,18] methods. One week after surgery, most deficit was found in the area of attention, concentration and psychomotor performance, the ability to perform fine motor tasks with speed and accuracy. As a group, 2 months after surgery, patients showed an overall improvement in most NP tests compared to their pre-operative performance. Using criteria for deficit, we identified a significant deficit in cognitive performance in half of the patients 1 week after surgery, and in one third 2 months after surgery. The incidence of cognitive deficit found in this study supports a number of previous reports [6–9,18].

Before surgery patients had more EEG deficit compared to the age-matched controls. This was shown as significantly more $\delta$ power and less $\theta$ and $\alpha$ power in the patient than in the control group. EEG changes found in the patient group before surgery may indicate presence of cerebrovascular changes in cardiac patients. A similar level of deficit in EEG has been reported previously [8,10,15]. Also, we have previously reported sub-clinical cerebral structural deficit in 80% of cardiac patients before surgery, investigated using MRI methods [18].

In this study, EEG showed reduced power in all frequencies 1 week after surgery, with the two slow frequencies, $\delta$ and $\theta$, showing most reduction. At 2 months, most reduction was found in the two fast frequencies, $\alpha$ and $\beta$. The two slow frequencies, $\delta$ and $\theta$, although still reduced, showed a return to pre-operative levels. Decrease in EEG power after surgery may indicate a disturbed global and regional CBF (rCBF) and cerebral metabolism, as a consequence of CPB procedure. The relationship between EEG, CBF and cerebral metabolism is now well established. A significant increase in $\delta$ and decrease in $\alpha$ relative power has been associated with a decrease in the mean hemispheric oxygen metabolism (CMRO$_2$) and CBF in carotid surgery [19] and in CPB surgery [3,20]. In CABG patients, regional CBF and performance on NP tests before and after surgery, have previously shown a high and consistent correlation [7]. A diffuse reduction in CBF was reported in some patients 1 year after CPB surgery and was taken to represent diffuse neuronal damage [3]. Even when perfusion pressure is kept within ‘safe’ limits (between 50–70 mmHg) during surgery, regional differences in cerebral perfusion may occur [3]. It has been proposed that a compromised regional cerebrovascular bed may cause disruption in local haemodynamics and autoregulation independent of the mean arterial blood pressure, total perfusion volume and systemic temperature. Even when CPB parameters remain constant, rCBF might occasionally fall below the critical volume for a given region [9]. This may be detected as a regional EEG slowing generally found in CPB patients.

One of the aims of this study was to identify patients who had most EEG deficit after surgery. Using set criteria, EEG deficit was found in half of the patients 1 week before surgery, and in one third 2 months after surgery. Similar levels of EEG deficit have been reported before, although that different method of measurement and criteria for assessment were used. Sotaniemi [10,15], using a clinical method of assessment, found immediate post-operative deficit in 97% of patients who showed clinical signs and in 67% of patients who did not show clinical signs of cerebral damage. EEG deficit persisted 1 year after surgery in 29% of patients who did not show clinical signs and in 67% of patients who showed clinical signs of cerebral damage. EEG deficit persisted 1 year after surgery in 29% and 20%, respectively. Diffuse $\delta$ activity was one of the main features of deficit found in 78% of patients 10 days after surgery. Sotaniemi related this to the possible effect of intra-operative cerebral hypoxia. Two other studies of adult CPB patients of varied aetiology found increased slow $\alpha$ and $\beta$ power 10 days after surgery [16], and a significant increase in slow frequency and decrease in $\alpha$ frequency power in 37% of patients 1 week after surgery [17].

In this study, no significant effect of patient age or duration of perfusion on post-operative EEG deficit was found. Some of the earlier studies found association between patient age and duration of perfusion with post-operative neurological outcome [5]. It is generally agreed that older patients, who had longer bypass, are more likely to show post-operative abnormalities. In recent years, bypass time has been much reduced and reported effect of age is inconclusive [4]. It is possible that the combination of patients age and longer bypass time may have an effect on post-operative cerebral outcome.
In the present study, the association between the type of oxygenator used in surgery and post-operative EEG deficit was not statistically significant. These findings do not support previous reports which found greater cerebral deficit when bubble oxygenators were used in surgery [1]. If anything, marginally more patients, operated on using membrane rather than bubble oxygenators, had EEG deficit 2 months after surgery. Bubble oxygenators are now used less frequently in CPB surgery. Studies which used retinal angiography [3] and doppler [21] have shown a markedly higher level of microemboli in the systemic circulation when bubble oxygenators are used. The use of membrane oxygenators has been associated with reducing neurological and neuropsychological deficit in cardiac patients [7]. In the present study, a relatively small number of patients in each oxygenator group may be responsible for this finding.

Pugsley et al. used transcranial doppler throughout surgery in an attempt to assess microembolic events in patients with or without an arterial line filter [22]. He reported an association between frequency of microemboli and NP deficit. It is generally accepted that a diffuse neuronal cell injury cannot be related only to the number and size of microemboli. Research is turning towards changes in cell physiology in relation to a combined effect of different intra-operative factors. Moreover, most authors agree that microemboli are one of the contributing factors related to ischaemic cerebral injury, rather than the main cause. It is conceivable that metabolic changes associated with a partial microvasculature occlusion, caused by smaller gaseous microemboli generated by a membrane oxygenator, may be more damaging than a complete occlusion caused by larger gaseous microemboli generated by a bubble oxygenator. However, we are not aware of any reported human studies in this area.

The relationship between EEG and cognitive deficit 2 months after surgery is of particular interest. It shows the association between physiological and cognitive measures of cerebral dysfunction in these patients. The fact that a stronger correlation between the two measures was found 2 months, but not 1 week, after surgery indicates the importance of assessing cerebral deficit as a consequence of CPB procedure, after patients have recovered from post-operative trauma. The effect of anaesthesia and post-surgical trauma on cerebral function may last as long as 1 week after surgery [11].

In conclusion, this study has shown concordance between EEG and cognitive measures in assessing cerebral functional deficits after CABG surgery. Post-operative cerebral functional deficit could not be directly related to patient age, type of oxygenator or duration of perfusion. These findings may indicate vulnerability of some patients to CPB procedure and the need for particular intra-operative care of these patients. QEEG has shown to be a sensitive method in detecting slight cerebral functional changes and may be useful in identifying patients suspected of cerebrovascular problems before surgery. However, computerised paperless QEEG equipment is expensive and not accessible to some cardiac units. The interpretation of QEEG is complex and may require particular expertise. For these reasons, suitability of QEEG in routine screening of CPB patients is at present doubtful.

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