Assessment of cognitive functions after coronary artery bypass grafting and its relation to cerebral blood flow

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Abstract

Background: It is widely assumed that decline in cognition after coronary artery bypass grafting (CABG) is related to use of the cardiopulmonary bypass pump. Because most studies have not included comparable control groups, it remains unclear whether postoperative cognitive changes are specific to cardiopulmonary bypass, general aspects of surgery, or vascular pathologies of the aging brain. Methodology: A prospective study was done on 30 patients undergoing CABG and 18 heart healthy controls. Patients were evaluated at baseline (preoperatively), 1 month and 3 months after the operation by Wechsler intelligence scale, Wechsler memory scale and transcranial Doppler. Results: the patient group showed cognitive decline at 1 month followed by return to near baseline values at 3 months. This decline was related to decreased cerebral blood flow during the operation. Conclusion: Neurocognitive dysfunction is a frequently occurring complication of coronary artery bypass grafting. The etiologic contribution of cardiopulmonary bypass to this complication will remain unclear until a randomized trial that directly compares off-pump and on-pump bypass surgery is carried out.

Introduction

Coronary artery bypass grafting (CABG) with the use of cardiopulmonary bypass (CPB) is associated with significant cerebral morbidity. The two main clinical manifestations of brain injury after CABG are stroke and cognitive decline (Roach et al. 1996). Cognitive sequelae after coronary artery bypass grafting (CABG) have been the focus of research interest for several decades. As a review study by Mahanna et al. in 1996 has shown, the reported incidence of morbidity varies; incidence of inhospital postoperative neuropsychological impairment as high as 79% has been cited. The discrepancy in results is very likely multifactorial and can be explained by heterogeneous definitions of cognitive decline and differences in neuropsychological test instruments, practice effects, testing intervals, surgical technique, and others. The assessment of cognition before and after cardiac operation is now used extensively as a measure of surgical outcome, after reports a decade ago that suggested "intellectual dysfunction" could affect as many as 80% of patients (Shaw et al. 1986, Newman et al. 1987). The incidence of cognitive decline after cardiac operation has been subsequently used to compare outcomes in different centers, to assess the efficacy of neuroprotective drugs, and to improve perfusion techniques and surgical management (Patel et al. 1996, Murkin 1993).

The precise pathophysiologic features of cognitive impairment are not certain but have been attributed to the microembolic, inflammatory, and nonphysiologic perfusion factors associated with cardiopulmonary bypass (CPB) (Benedict 1994). Cognitive impairment also occurs after noncardiac operations but is reported to be more frequent and severe after cardiac

operation with CPB. Although cognitive impairment after noncardiac operation is explained by patient-related factors (eg, advanced age, ill health), impairment that occurs after cardiac operation is invariably attributed to CPB (Smith 1988, Hammeke and Hastings 1988, Vingerhoets et al. 1997). The feasibility of the performance of certain coronary grafts without CPB has recently been established. It has been postulated, but not proved, that avoidance of CPB may reduce the postoperative morbidity associated with extracorporeal circulation. Specifically, it is suggested that coronary artery bypass grafting (CABG) without CPB should minimize postoperative cognitive impairment (Treasure et al. 1989).

At a consensus meeting in 1994, several guidelines for the assessment of neuropsychologic deficits after CPB were established. It was recommended that the neurologic and neuropsychologic state be assessed before the operation to provide accurate baseline information. A second important recommendation was that the analysis should be based on the individual change in performance from baseline to a particular time after the operation. In general, practice effects cause the overall group performance to improve after the operation. Accordingly, when the overall postoperative mean is compared with the preoperative mean, the decline of some individuals is overshadowed by the improvement of others. Also, it was agreed that a late assessment (ideally after 3 months) should be included in the study design, because the patients' performances are unstable in the immediate postoperative period (Murkin et al. 1995).

It remains unclear whether changes in cognitive performance after open-heart surgery are specifically related to the use of cardiopulmonary bypass (CPB), to more general aspects of surgery such as type and duration of anesthesia, and type and duration of surgery or to underlying pathologies related to the aging brain such as cerebrovascular disease. To answer these questions we studied the effect of extracorporeal circulation on the cognitive functions of the brain in CABG surgery.

Methods

A case control study was carried out using data collected from 30 patients undergoing first-time CABG by a single surgeon and 18 heart healthy controls. All data were collected prospectively at the time of operation, 1 month, and 3 months after the operation. Cases were defined as patients who underwent the CABG and controls were defined as all other voluntary participants.

A written informed consent was obtained from all participants who took part in the study. Patients with previous neurological or psychiatric illness were excluded by taking detailed and full neuropsychiatric history and carrying on the general health questionnaire. The control group was screened for the following medical history variables: diabetes mellitus, kidney disease, hypertension, coronary artery disease (including myocardial infarction, angina, high cholesterol), stroke/TIA, peripheral vascular disease, and medications used to treat the above conditions. The controls were selected to be comparable with the patient group in terms of age and sex.

The preoperative assessment included all of the usual clinical and investigational measures and a detailed general level of risk was expressed to the patients. Full neurological assessment with special

emphasis on information on previous neurological events was done including a detailed neurological examination according to the National Institutes of Health Stroke Scale (NIHSS) (Brott et al. 1989) was carried on the day before the surgery.

The baseline neuropsychological assessment was carried out on the day before the operation and consisted of Wechsler Intelligence Scale (WAIS) and Wechsler Memory Scale (WMS). These are accepted tests of global cognitive function assessment. The tests were presented in a fixed order according to conventional practice and the scores were arranged so that higher scores indicated better neuropsychologic performance. In addition because cognitive functions may be affected by mood, each patient's current mental health was also assessed by means of the Hamilton Depression Scale (HDS).

The patients were also assessed neurologically, in addition to the history and neurological examination, by using Transcranial Doppler ultrasound (TCD) to measure the changes in mean flow velocity (MFV) of the cerebral vessels. We used the TCD MDX- TCD-7 software (version 7.3) apparatus multi-channel Doppler operating at 2 MHz. Insonation of the middle cerebral artery (MCA) on both sides through transtemporal window. MCA blood flow velocity was recorded bilaterally with TCD. Transducers were immobilized throughout the operations with a head ring. Measures of cerebral perfusion were recorded manually for each side at 15-minutes intervals during the operation for each patient. And the readings were summed and their mean was calculated for the 3 different phases (before anesthesia, during anesthesia and during on-pump period).

General anesthesia was induced in all patients of the study by Midazolam 2-5 mg, Fentanyl 150-250 mcg and propofol 30-50 and maintained by Sevofluarane inhalational anesthetic. Cistracrium was used as skeletal muscle relaxant in all patients given at top-up dose every 45 minutes in a dose of 0.3 mg/kg.

Standard surgical technique was used in all patients. Normothermic cardiopulmonary bypass (CPB) was instituted and maintained using ascending aortic cannulation and two-stage venous cannulation of the right atrium after palpation of the ascending aorta to avoid the calcified segments. Myocardial protection was achieved with intermittent antegrade hyperkalemic warm blood cardioplegia.

During cardiopulmonary bypass, pump flow was maintained between 2.0 and 2.5 L/minute per square meter and mean arterial pressure between 50 and 60 mm Hg. Norepinephrine was used as needed to maintain the mean arterial pressure at the above mentioned values.

Both neuropsychological assessments and TCD were repeated after one month and 3 months postoperatively to assess if there is cognitive decline and affection of cerebral blood flow after on-pump CABG which is the main aim of our study.

Statistical analysis

Statistical analysis was carried out with SPSS 10.0 software. Continuous variables are presented as mean and standard deviation. Comparisons between patients and controls were evaluated by t test. Statistical significance was associated with a p value of less than 0.05.

Results

In this study we investigated 30 patients undergoing CABG surgery, 18 (60%) males and 12 (40%) females with mean age of 54.1 ± 10.8. Of these, 50% (n=15) had diabetes mellitus, 60% (n=18) had hypertension and 30% (n=9) were smokers.

The control group was 18 individuals, 9 (50%) males and 9 (50%) females with mean age of 53.3 ± 8.2. 50% (n=9) had diabetes mellitus, 66.7% (n=12) had hypertension and 33.3% (n=6) were smokers.

Results of TCD mean flow velocity (MFV), Wechsler intelligence scale and Wechsler memory scale preoperative and 1 month and 3 months postoperative consecutively are shown in tables 1, 2 and 3.

When comparing MCA MFV before anesthesia, during anesthesia and during on-pump period, we found that during anesthesia the MCA MFV decreased significantly on both sides then it was increased again significantly during the on-pump time (Table 4).

Direct relation between cognitive functions and the lt. MCA mean flow velocity was found during anesthesia but not during on-pump period. This relation was not found with rt. MCA (Table 5). Figure 1 shows sample of TCD of MCA before anesthesia, during anesthesia and during on-pump period.

Table 1: Results of MCA mean flow velocity (MFV), Wechsler intelligence scale and Wechsler memory scale preoperatively.

	Control	Patients	p value
Number	18	30	
Age	53.3 ± 8.2	54.1 ± 10.8	0.797
Rt. MCA MFV	71 ± 10.3	74.5 ± 19.3	0.418
Lt. MCA MFV	74.5 ± 5.4	69.4 ± 15.2	0.105
Verbal IQ	99.8 ± 5	100.9 ± 17.9	0.761
Performance IQ	99.5 ± 2.8	96.7 ± 15.1	0.331
Total IQ	100.5 ± 3.9	96.5 ± 16.7	0.219
PINF	5.66 ± 0.4	5 ± 1	0.004*
Orientation	5 ± 0	4.7 ± 0.4	0.001*
Memory control	6 ± 0	3.3 ± 2.2	< 0.001*
Immediate recall	11.5 ± 0.5	6.2 ± 2.9	< 0.001*
Memory span	10.5 ± 0.5	7.6 ± 1.9	< 0.001*
Visual reproduction	9.5 ± 0.5	3.4 ± 2.8	< 0.001*
Paired associated learning	11.3 ± 2.5	7.8 ± 4.9	0.002*
Total memory score	113 ± 3.7	78 ± 17.8	< 0.001*
Total HDRS	3.5 ± 0.9	11.5 ± 7.5	< 0.001*

HDRS: Hamilton Depression Rating Scale, IQ: Intelligent Quotient, Lt.: Left, Rt.: Right, MCA: Middle cerebral artery, MFV: Mean flow velocity, PINF: Personal and current information. * $p < 0.05$-- significant

Table 2: Results of MCA mean flow velocity (MFV), Wechsler intelligence scale and Wechsler memory scale 1 month postoperative.

	Control	Patients	p value
Rt. MCA MFV	71.3 ± 9.1	77.5 ± 18.8	0.136
Lt. MCA MFV	73 ± 7.3	71.9 ± 18.1	0.776
Verbal IQ	103 ± 4.5	95.4 ± 19.7	0.2
Performance IQ	99 ± 3	92.7 ± 15.3	0.037*
Total IQ	100.5 ± 4	91.6 ± 17.6	0.012*
PINF	5.66 ± 0.4	5.2 ± 0.6	0.008*
Orientation	5 ± 0	4.4 ± 0.8	< 0.001*
Memory control	6 ± 0	3.2 ± 1.6	< 0.001*
Immediate recall	11.6 ± 0.4	7.2 ± 2.4	< 0.001*
Memory span	10.3 ± 0.48	7.3 ± 1.87	< 0.001*
Visual reproduction	9.33 ± 0.4	2.5 ± 2.3	< 0.001*
Paired associated learning	11.5 ± 1.6	5.4 ± 3.3	< 0.001*
Total memory score	114.1 ± 2.8	73.9 ± 13.5	< 0.001*
Total HDRS	3.1 ± 0.7	14.8 ± 7.4	< 0.001*

HDRS: Hamilton Depression Rating Scale, IQ: Intelligent Quotient, Lt.: Left, Rt.: Right, MCA: Middle cerebral artery, MFV: Mean flow velocity, PINF: Personal and current information. * p < 0.05-- significant

Table 3: Results of MCA mean flow velocity (MFV), Wechsler intelligence scale and Wechsler memory scale 3 months postoperative.

	Control	Patients	p value
Rt. MCA MFV	71.3 ± 9.5	81.1 ± 16.7	0.013*
Lt. MCA MFV	72.8 ± 8	71.9 ± 14.5	0.776
Verbal IQ	100.8 ± 4.2	101.6 ± 18.25	0.827
Performance IQ	100.3 ± 2.8	97.2 ± 15	0.278
Total IQ	101.1 ± 3.9	97.1 ± 16.9	0.217
PINF	5.5 ± 0.5	5.3 ± 0.6	0.246
Orientation	4.8 ± 0.3	4.9 ± 0.3	0.5
Memory control	5.8 ± 0.38	4.2 ± 1.7	< 0.001*
Immediate recall	11.1 ± 0.3	6.9 ± 2.3	< 0.001*
Memory span	10.6 ± 0.4	7.9 ± 1.6	< 0.001*
Visual reproduction	9.6 ± 0.4	4 ± 2.4	< 0.001*
Paired associated learning	12.3 ± 2.1	8.3 ± 4.5	< 0.001*
Total memory score	114.5 ± 2.5	80.2 ± 16.7	< 0.001*
Total HDRS	3 ± 0.5	6.9 ± 2.2	< 0.001*

HDRS: Hamilton Depression Rating Scale, IQ: Intelligent Quotient, Lt.: Left, Rt.: Right, MCA: Middle cerebral artery, MFV: Mean flow velocity, PINF: Personal and current information. * p < 0.05-- significant

Table 4: changes in MCA MFV before, during anesthesia and during on-pump period

	Difference	p value
Rt. MCA MFV before and during anesthesia	35.3 ± 17.2	< 0.001*
Rt. MCA MFV during anesthesia and on-pump period	-14.1 ± 19.6	< 0.001*
Rt. MCA MFV before anesthesia and during on-pump period	21.2 ± 26.7	< 0.001*
Lt. MCA MFV before and during anesthesia	33.1 ± 12.5	< 0.001*
Lt. MCA MFV during anesthesia and on-pump period	-24.7 ± 15.4	< 0.001*
Lt. MCA MFV before anesthesia and during on-pump period	8.4 ± 24.4	0. 070

Lt.: Left, Rt.: Right, MCA: Middle cerebral artery, MFV: Mean flow velocity. * p < 0.05-- significant

Table 5: Relation between cognitive functions and the MCA mean flow velocity

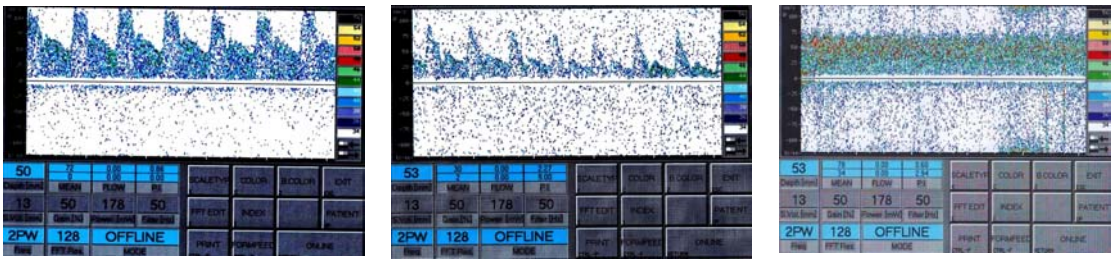
	RT. MCA MFV during anesthesia	LT. MCA MFV during anesthesia	RT. MCA MFV on-Pump	LT. MCA MFV on-Pump
Verbal IQ after 1 month	0.874	0.003*	0.674	0.978
Performance IQ after 1 month	0.264	0.031*	0.775	0.923
Total IQ after 1 month	0.837	0.004*	0.746	0.938
Personal and current information after 1 month	0.757	0.423	<0.001*	0.064
Orientation after 1 month	0.005*	0.185	0.061	0.446
Mental control after 1 month	0.694	<0.001*	0.196	0.471
Immediate recall after 1 month	0.657	<0.001*	0.701	0.396
Memory span for digits after 1 month	0.219	0.028*	0.372	0.057
Visual reproduction after 1 month	0.592	0.543	0.252	0.851
Paired associated learning after 1 month	0.005*	0.309	0.100	0.190
Total Wechsler memory IQ after 1 month	0.777	0.011*	0.885	0.679
Total HDRS after 1 month	0.075	0.058	0.098	0.465
Verbal IQ after 3 months	0.954	0.001*	0.516	0.693

Table (5) continue:

	RT. MCA MFV during anesthesia	LT. MCA MFV during anesthesia	RT. MCA MFV on-Pump	LT. MCA MFV on-Pump
Performance IQ after 3 months	0.600	0.010*	0.974	0.471
Total IQ after 3 months	0.870	0.001*	0.654	0.960
Personal and current information after 3 months	0.511	0.130	0.051	0.712
Orientation after 3 months	0.048*	0.670	0.415	0.048*
Mental control after 3 months	0.211	<0.001*	0.015*	0.007*
Immediate recall after 3 months	0.420	0.002*	0.411	0.942
Memory span for digits after 3 months	0.477	0.002*	0.669	0.289
Visual reproduction after 3 months	0.421	0.078	0.465	0.732
Paired associated learning after 3 months	0.677	0.005*	0.413	0.770
Total Wechsler memory IQ after 3 months	0.295	<0.001*	0.904	0.784
Total HDRS after 3 months	0.959	0.012*	0.209	0.052

HDRS: Hamilton Depression Rating Scale, IQ: Intelligent Quotient, Lt.: Left, Rt.: Right, MCA: Middle cerebral artery, MFV: Mean flow velocity. * p < 0.05-- significant

Figure 1: TCD for MCA before anesthesia, during anesthesia and during on-pump period



Discussion

No fewer than six pathophysiologic effects of cardiopulmonary bypass have been established in the world literature. These are (1) activation of the complement cascade with increased capillary permeability (Singh et al. 1980), (2) metabolic acidosis, (3) severe interstitial fluid accumulation, (4) elevated systemic vascular resistance, (5) arteriovenous shunting, and (6) impaired brain oxygenation (Runge et al. 1992). The National Institutes of Health has recognized that there are important problems with cardiopulmonary bypass and emphasizes the prevalence of neurologic damage produced by conventional cardiopulmonary bypass, particularly in patients who are extremely young or older than 60 years of age (Kirklin et al. 1983). Cognitive memory loss is now an acknowledged complication of the procedure, particularly in patients older than 60 years. There is no longer a question of whether this occurs, only questions of its frequency and what can be done about it (Runge et al. 1992).

Cognitive performances after cardiac operations may be influenced by several important factors. First, scores may be affected by the cardiac operation itself. Our results provide typical examples of this postoperative cognitive change. Second, the effects of practice may influence results, with group performances often improving when psychometric tests are administered repeatedly (Feinstein et al. 1994). Practice effects can be minimized by the use of alternative forms and long follow-up intervals or controlled by a comparison group that undergoes the same assessments at the same time points and this we applied in our study.

Most of the studies reporting late cognitive decline after CABG did not include a control group. It is therefore difficult to determine if the cognitive changes are caused by cardio-pulmonary bypass with general anesthesia years earlier or caused by normal aging, development of Alzheimer's disease or other causes. The choice of appropriate control for CABG has been controversial. The presence of diabetes, hypertension and other cardiovascular risk factors has increased in the candidates for CABG. Therefore ideal controls should also include patients with similar frequency of risk factors for cerebrovascular disease. Neither duration nor the severity of these risk factors can be easily quantified and how these risk factors translate into cerebro-vascular disease is also unknown (Vibha et al. 2006).

Our results showed that the patient group performed lower at baseline than did the healthy controls in the cognitive domains of immediate recall, memory control, memory digit span and paired associate learning. This type of cognitive profile is comparable to that seen in prospective studies of patients with MRI-defined subcortical cerebrovascular disease (Dixon and Raz 2000). Other large-scale epidemiologic studies of community-dwelling individuals indicate that the presence of risk factors of cardiovascular disease, such as hypertension and diabetes, is associated with decline in cognitive performance over time even in the absence of cardiac surgical intervention. Thus, our conclusion is that CABG patients, like similar patients, with long-standing coronary artery disease, have some degree of cognitive dysfunction secondary to cerberovascular disease before surgery (McKhann et al. 2005).

Another possibility is that the surgical group has a falsely baseline because of adverse testing conditions associated with the proximity to surgery, such as anxiety and depression. This possibility may explain the significant difference between the surgical group and healthy control on the total Hamilton Scale score for depression, which we found in our study. However, this would not explain why only specific cognitive domains, such as visual reproduction and memory control in contrast to other domains as performance IQ, are lower preoperatively (McKhann et al. 2005).

The change between baseline, one month and three months in the 2 groups is of some interest. In our study, there was a cognitive decline after one month postoperatively, this could be explained by the previous psychopathological factors of CABG stated before, especially that all of our patients underwent on-pump procedure. This was supported by most of the studies as that done by Van Dijk et al. (2002) who showed that there were significant differences in the incidence of decline between patients having conventional on-pump versus off-pump surgery at 3 months.

Cognitive dysfunction has been reported to persist for several months or even years after CABG (Taggart et al. 1999). In our study the cognitive performances of the patient group declined 1 month after the operation then improved 3 months postoperatively, nearly it returns to base line. Thus, we did not find selective effects of CABG on longitudinal cognitive performance. In general, these findings are in keeping with data from other studies by Vingerhoets et al. (1996) and Toner et al. (1998) that report significant improvements in cognitive function late after cardiac operations and the clinical impression that cognitive impairment is now uncommon late after cardiac operations.

The interpretation of these findings is that patients in the current study have known cardiovascular disease, and thus it is likely that at least some of these patients also have underlying cerberovascular disease, although we exclude neurological deficit by clinical examination. Those patients may have deficits that appear on imaging studies. This is supported by study of 421 patients scheduled for CABG, in which MRI was obtained preoperatively, 126 (30%) had small brain infarctions and 83 (20%) had multiple, larger infarctions. In addition, they found those with infarctions were more likely to have lower cognitive performance before surgery (Goto et al. (2001).

The discrepancy between decline in test performance and functional decline is also expressed by the methodological difficulties of defining a cognitive deficit. Mahanna and colleagues (1996) demonstrated the enormous influence of the definition of cognitive deficit that is chosen by applying five different definitions on the same patient sample. The single-case analysis technique, recommended in the consensus statements (Murkin et al. 1995, Murkin et al. 1997), uses the patient as his or her own control and defines a cognitive deficit as a 20% decrease in at least 20% of the tests. This method also has some drawbacks. In the first place, reducing the continuous test scores to a dichotomous outcome measure (presence of a 20% decrease or not) is a "costly" way of data handling that reduces statistical power and may have made several randomized studies fail to reach statistically significant results. The 20% decrease rule is as arbitrary as the 1

standard deviation decrease rule. The problem may be overcome by refraining from "dichotomizing" data and just calculating how much the patient's performance deviates from the expected (baseline or control group) performance (Van Dijk et al 2000). Our study design involves control group, so, we did not need to rely on arbitrary criteria for determining change of cognition. This is a strength that makes our study different.

We used TCD to measure blood flow velocity bilaterally in the middle cerebral arteries (MCAs) because it is the only continuous means of measuring changes in cerebral hemodynamics noninvasively and has become an essential part of neurologic monitoring (Edmonds et al. 1996). A close relationship between changes in cerebral blood flow and changes in blood flow velocity as measured by TCD has been demonstrated in patients with symptoms suggesting cerebrovascular disease and in patients during cardiac surgery (Van der Linden et al. 1991, Dahl et al. 1992, Trivedi et al 1997). In our study we found that the MCA mean flow velocity decreased significantly during anesthesia on both sides then it was increased again significantly during the on-pump time. Although the changes were on both sides, we found that the cognitive decline was related more to the decreased lt. MCA mean flow velocity during anesthesia.

There is no longer a question of whether cognitive decline after CABG occurs, but the actual cause of it may still be debatable (Vibha et al. 2006). The reason for this uncertainty about the cause comes from data from earlier studies that suggested that CBP was not the sole cause of cerebral dysfunction after cardiac operations (Smith 1988, Hammeke and Hastings 1988, Vingerhoets et al. 1997). Collectively, these studies question the established dogma that CPB is responsible for cognitive impairment after cardiac operations, at least in patients undergoing closed operations and with at least moderate ventricular function. If, however, CPB is not specifically responsible for cognitive impairment at early and late follow-up, what alternative intraoperative components common to both operations could be involved? Several candidates present themselves that include hypotension, general surgical injury, and anesthesia.

Although severe intraoperative hypotension may cause cognitive dysfunction, most prospective studies have failed to confirm this when mean arterial pressure is maintained above 50 mm Hg. (Ellis et al. 1980, Sotaniemi et al. 1981, Savageau et al. 1982, Arom et al., 1989 Slogoff et al. 1990), a level at which cerebral autoregulation normally occurs. In our study the mean blood pressure was maintained at 50 to 60 mm Hg.

The contribution of the general effects of surgical injury warrants consideration, given previous findings that patients undergoing major noncardiac operation also showed cognitive dysfunction (Smith 1988, Hammeke and Hastings 1988, Vingerhoets et al. 1997). These studies cannot, however, distinguish the effects of surgical injury from those of concomitant anesthesia.

General anesthesia per se can result in postoperative cognitive impairment in patients undergoing non-cardiac surgery. In a study on patient aged 40-60 year, 19% were found to have cognitive decline 7 days after surgery compared to only 4% in matched controls (Johnson et al. 2002). By 3 months after surgery, cognitive decline was minimal and not different from control.

In the largest trial to date that investigated the effects of general and epidural anesthesia in orthopedic patients, 5% of all patients showed clinically significant deterioration on a large battery of neuropsychologic tests at 6 months (Williams-Russo et al. 1995). Furthermore, 27% of all patients showed a clinically important deterioration in verbal memory at 6 months.

Although it is generally recognized that anesthesia can produce short-term cognitive dysfunction (Karhunen and John 1982, Herbert et al. 1987), the long-term effects remain open to question. Furthermore, when evaluating the effects of anesthesia, most studies typically consider only the effects of a single agent. Few, if any, consider the potential for the cumulative and/or interactive effects of different agents commonly used (Halsey 1995). As emphasized by Klafta and colleagues in 1995, the typical anesthetic regimen for cardiac operations produces "deeper anaesthesia than is normally required for non-cardiac operation." Taggart et al. (1999) speculate that this cognitive dysfunction might result from anesthetic dependent cerebral ischemia or disturbances of cerebral autoregulation.

This raises an important question about whether the cognitive impairment is due to the anesthetic drugs used or due to the decrease in cerebral perfusion as noticed in our study during anesthesia, or both. Taggart et al. (1999) suggest that the particular anesthetic regimen in association with nonspecific effects of the general operation may be responsible for producing the common pattern of cognitive dysfunction after cardiac operations. To resolve this issue would require submitting age-matched patients to the same anesthetic regimen but without operation, not an ethical proposition.

So, could CABG per se be innocent from the postulation that it is the cause of cognitive dysfunction? These questions need to be addressed in further studies to answer them.

Limitations

A potential weakness of our study was that the neuropsychologic tests were performed by one observer who was not blinded to the groups. In addition to the fact that many of the neuropsychologic tests are objective and quantifiable assessments of cognitive performance and not easily influenced by the examiner, our postulated a priori bias was that the control group would perform better than the patient group. Second, the TCD done was not directed for emboli detection which is an incriminated factor in cognitive affection. Nevertheless, it helped to alarm the anesthetist for the cerebral blood flow during anesthesia.

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تقويم الخلل المعرفي بعد عمليات توصيل الشرايين التاجية و علاقته بمعدل الارتواء الدماغي

يعتقد دائما أن الخلل المعرفي للمرضى الذين أجريت لهم عمليات توصيل الشرايين التاجية مرتبط باستخدام جهاز القلب و الرئة الصناعي. نظرا لأن معظم الدراسات السابقة التي أجريت لم تتضمن استخدام عينات ضابطة، لذا سيظل من غير الواضح ما إذا كان هذا الخلل ناتج عن استخدام جهاز القلب و الرئة الصناعي أم هناك أسباب أخرى مثل أسباب تتعلق بالجراحة أو تغيرات مرضية بشرايين المخ ناتجة عن التقدم بالعمر. اشتملت الدراسة على ٣٠ مريض أجريت لهم عمليات توصيل الشرايين التاجية و ١٥ شخصا أصحاء كعينة ضابطة. تم تقويم النواحي المعرفية لجميع الحالات قبل بدء الدراسة و بعد العملية بشهر و ثلاثة أشهر. كما تم تقويم معدل الارتواء الدماغي باستخدام جهاز الموجات الصوتية عبر الدماغ. أظهرت الدراسة وجود تدهور بالنواحي المعرفية بعد شهر من إجراء الجراحة ثم تحسن تلك النواحي إلى المستوى السابق قبل إجراء الجراحة عند ثلاثة أشهر، كما لوحظ ارتباط هذا التدهور بهبوط معدل الارتواء الدماغي أثناء الجراحة.تعد مساهمة جهاز القلب و الرئة الصناعي في تدهور النواحي المعرفية بعد عمليات توصيل الشرايين التاجية غير مؤكد حتى الآن و نحتاج إلى دراسات عديدة لبيان جميع الأسباب.