Acta Biomed 2013; 84: 44-52

Original article

Early neurological injury after cardiac surgery: insights from a single centre prospective study

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Abstract. *Background and aim of the work:* The aim of this study was to report the incidence of early neurological complications after heart surgery, to identify preoperative and procedural risk factors for these complications and to assess their influence on postoperative outcome. *Methods:* Data were prospectively collected from 954 procedures: 520 coronary artery bypass grafting (CABG), 233 valve surgery, 100 combined CABG and valve surgery, 88 major aortic surgery, and 13 other procedures. Independent risk factors were analyzed by multivariate stepwise logistic regression model. *Results:* Early cerebral complications occurred in 94 patients (9.85%). Eleven patients (11.7%) had permanent cerebral symptoms and 83 (88.3%) had transient neurological symptoms only. Risk factors for early neurological complications were older age, chronic obstructive pulmonary disease, open cardiac chambers procedures, higher end-cardiopulmonary bypass lactate levels, blood transfusion, and the use of insulin in ICU. *Conclusion:* The results of this study suggest a relationship between metabolic and technical aspects of cardiopulmonary bypass management and early occurrence of neurological injury. Preoperative screening and intraoperative measures that ensure adequate cerebral perfusion, minimize embolization and improve systemic inflammatory response and hemodynamic performance appear mandatory in these patients. Future efforts will be necessary to strongly validate surrogate markers of early neurologic damage to predict neuropsychological dysfunction after cardiac surgery. (www.actabiomedica.it)

Key words: stroke, cardiac surgery, extracorporeal circulation

Introduction

Early injury of the central nervous system following cardiac surgery remains a severe complication despite the advances in cardiopulmonary perfusion, anaesthesiology and surgical techniques. This common adverse event is responsible for high morbidity and mortality as well as longer hospitalization and greater use of health care and financial resources (1-4).

The incidence of such neurological complications varies widely in published studies mainly due to different methodologies employed for its diagnosis or classifications and study design. Thus, the incidence of stroke and neurocognitive dysfunction in the estimated 1.500.000 worldwide cardiac operations performed yearly, reported in the recent literature, ranges up to 1.5% and 83% respectively (5-14).

The objectives of this study were to report the incidence of early neurological complications after heart surgery procedures, to identify preoperative and procedural risk factors for these complications and to assess their influence on postoperative outcome of these patients.

Materials and methods

Study population

From February 2009 to August 2010, we prospectively collected and retrospectively analyzed the data of 1023 adult patients underwent cardiac surgery at Heart Surgery Unit of the University Hospital of Parma. We excluded from the analysis the patients with ongoing neurological complications at time of surgery (2 patients), the patients died in ICU before any clinical evaluation of potential postoperative cerebral injury (8 cases), the patients who underwent off-pump CABG (20 patients), those who underwent major aortic surgery requiring cardiocirculatory arrest (18 patients), and those with incomplete clinical and laboratory data collection, even in absence of postoperative neurological complications (21 patients). Therefore, the study included 954 consecutive patients who underwent heart surgery with the use of extracorporeal circulation (ECC) at our institution.

The study complies with the Declaration of Helsinki, it was given approval by the institutional review committee, and the informed consent was given by the subjects.

Perioperative management

Preoperative epiaortic vessels Doppler ultrasound was performed in all patients who underwent elective surgery. Significant carotid disease was confirmed by CT scan or angiography. Carotid endoarteriectomy was performed in case of 1) symptomatic stenosis greater than 75%, 2) in case of monolateral asymptomatic lesions greater than 90%, 3) bilateral asymptomatic stenosis greater than 75%, and associated with the scheduled cardiac procedure. Transesophageal echography was used in all patients to confirm preoperative diagnosis and to detect the presence of ascending aorta calcifications.

All patients received premedication with morphine sulfate (0.1 mg/kg body weight) and scopolamine (0.01 mg/kg body weight) intramuscularly 60 minutes before transfer to the operating room. Anesthesia was induced with midazolam (0.05 mg/kg), etomidate (0.3 mg/kg) and fentanyl. Neuromuscular block was obtained by cisatracurium besylate intravenously. Anesthesia was maintained with fentanyl, sevoflurane and cisatracurium besylate.

A full intravenous heparin dose was administered (3 mg/kg) in case of standard ECC. Heart surgery was performed using cardiopulmonary bypass (CPB) at 34° C systemic temperature in all patients, 32°C in open chamber procedures, and at 30°C in case of concomitant carotid endoarteriectomy. Normocapnia and hemodilution secondary to priming solutions with crystalloids and cardioplegia with a hematocrit of 20% to 30% were maintained during the ECC. Membrane oxygenators and roller pumps were used. The extracorporeal circulation management was based on an optimal flow rate of 2.4 l/min/m^2 and mean systemic perfusion pressure of 60 mmHg. Perfusion pressure > 70 mmHg during CPB was maintained in all patients with a documented associated cerebrovascular disease. Cold crystalloid cardioplegia or ante-retrograde warm blood continuous cardioplegia were used at the discretion of the operating surgeon. Alpha-stat management was employed for acid-base balance during ECC.

Protamine was administered to reverse the effect of heparin. Anti-Xa dalteparin, 2500 UI or 5000 UI, was administered subcutaneously to all patients until they were mobilized, starting the day of surgery. Acetylsalicylic acid, 250 mg, was given to the patients 6 hours after surgery, and maintained with 100 mg per os, daily, starting on the following day in all the patients who underwent CABG or aortic valve replacement with bioprostheses with sinus rhythm. Warfarin treatment was started, when indicated, on the second postoperative day.

After the operation, the assessment of cardiac output, cardiac index, systemic vascular resistance index and pulmonary vascular resistance index was made by Swan-Ganz catheter insertion. Supraventricular tachyarrhythmias were treated with amiodaron or beta-blockers, and electrical cardioversion was performed in resistant cases.

Definition of the complications

All the preoperative variables included in the EuroSCORE system were defined as reported in the same score system (15).

Cerebral complications were classified as early if the first cerebral symptoms were detected when the patients regained consciousness from anesthesia and the damage was considered to have occurred intraoperatively. The adverse neurologic sequelae were classified into 2 general categories, according to Roach et al (1). Type I included death due to stroke or encephalopathy, nonfatal stroke, transient ischemic attack, or stupor or coma at time of discharge, and Type II included every new deterioration in intellectual function, confusion, agitation, disorientation, memory deficit, or seizure without evidence of focal injury. Cerebral complications were diagnosed based on clinical findings and confirmed by the neurologist with electroencephalogram and CT head scans. In-hospital mortality was considered to be any death occurring within 30 days after surgery.

Statistical analysis

The characteristics of patients were reported using descriptive statistics. All data are expressed as absolute numbers and percentage or means ± standard deviation of the mean and median as appropriate. The Student τ-test with normal distribution data and the Mann-Whitney test with rejected normality distributions were utilized for continuous variables. Categorical variables were compared using the Chi-squared or Fisher Exact tests. The association between pre- intra- or post-operative variables and occurrence of early neurological complications, defined as a dependent binary variable, was first explored in a univariate logistic regression model. Variables with p less than 0.10 were included in a multivariate analysis. The risk for early neurologic complications was assessed with a multivariate stepwise logistic regression model to identify independent predictors with p less than 0.10 required for inclusion. To avoid overfitting and collinearity in assessing the multivariate model, independent variables were tested for intercorrelation and then, when they were present, the most clinically relevant dependent variables were chosen for inclusion in the multivariate analysis. The risk factors analysis included preoperative, intraoperative, and postoperative factors and they are listed in Table 1. A p value of 0.05 or less was considered significant. Statistical analysis was

Table 1. List of the variables included in the statistical analysis

• Preoperative variables
- Age
- Gender
- Body mass index
- Chronic obstructive pulmonary disease requiring pharmacological therapy (COPD)
- Systemic hypertension
- Peripheral arteriopathy
- Previous neurological dysfunction
- Alcohol abuse
- Serum creatinine levels > 2.25 mg/dL
- Renal failure on dialysis
- Diabetes on medication
- Liver dysfunction,
- NYHA functional class
- Left ventricular ejection fraction (LVEF)
- Previous heart surgery
- Active endocarditis
- Recent myocardial infarction
- Unstable angina
- Pulmonary hypertension
- Preoperative IABP

• Intraoperative variables
- Urgency surgery
- Type of surgery
- Open chambers surgical procedures
- Presence of aortic atherosclerosis
- Cardiopulmonary bypass time
- Aortic cross clamping time
- Type of cardioplegia
- Mean arterial pressure <50mmHg during extracorporeal circulation
- Use of IABP
- PaO2/FiO2
- Glicemia
- Temperature
- pH
- Hematocrit at the end of CPB
- Blood lactate levels at the end of CPB

• Postoperative variables
- Low cardiac output syndrome
- Perioperative myocardial infarction
- IABP insertion in ICU
- Temperature on arrival in ICU
- Temperature after 4 hours in ICU
- Mean pH in ICU
- Hyperglicemia
- Mean hematocrit in ICU
- Peak blood lactate in ICU
- PaO2 / FiO2
- Use of insulin in ICU
- Surgical re-exploration
- Transfusion

performed with NCCS statistical software ver. 07.1.2 (2007), Kaysville, Utah.

Results

Clinical and preoperative characteristics of the patients are reported in Table 2. The mean age of the overall population was 68.33 ±10.2 years, and majority were male. The distribution of procedures was as follows: 520 (54.5%) CABG, 233 (24.4%) valve surgery, 100 (10.5%) combined CABG and valve surgery, 88 (9.2%) major aortic surgery (ascending and/or aortic arch surgery), 13 (1.4%) other procedures, including surgery for removal of atrial mixoma, ventricular septal defect correction, ventricular pseudoaneurysm repair, and CABG associated with carotid endoarterectomy (Table 3).

A total of 94 patients (9.85%) had neurological complications: 14 (1.47%) type I and 80 (8.38%) type II complications, respectively. The clinical onset was stroke in 6 patients, coma in 4, agitation and disorientation in 37 patients, stupor and confusion after delayed awakeness in 18 patients, and seizure without evidence of focal injury in 29 patients. Eleven patients (11.7%) had permanent cerebral symptoms and 83 (88.3%) had transient neurological symptoms only.

Patients who suffered neurological complications were significantly older, and more frequently female. Severe dyspnoea, history of chronic obstructive pulmonary disease, chronic renal failure, and dialysis treatment were prevalent among patients who suffered neurological complications. All other preoperative variables were comparable among groups (Table 2).

In Table 3, surgical data for patients with cerebral complications are summarized and compared with data for patients without neurological complications. At univariate analysis, the patients who experienced neurological complications resulted to undergo more frequently open chambers procedures, with significantly prolonged CPB and aortic cross clamping times. In these patients, temperature during CPB was slightly lower, and myocardial protection was obtained more frequently with cold crystalloid cardioplegia. More-

Table 2. Relationship between preoperative variables and incidence of cerebral complications

	Total population	Patients without cerebral complications (N=860)	Patients with cerebral complications (N=94)	*P*
Age (mean,SD,median)	68.33±10.2 (70)	67.85±10.3 (69)	72.64±8.5 (73.5)	*0.0001*
Female (%)	265 (27.78)	223 (25.9)	42 (44.7)	*0.0001*
BMI (mean,SD,median)	28.9±4.1 (26.6)	27.03±4.1 (26.8)	25.64±4.2 (25.3)	*0.002*
COPD (%)	59 (6.18)	47 (5.46)	12 (12.8)	*0.01*
Previous cerebral events (%)	10 (1.05)	7 (0.8)	3 (3.2)	*0.066*
NYHA ≥III (%)	276 (28.93)	236 (27.4)	40 (42.5)	*0.003*
Serum creatinine >2,25mg/dl (%)	27 (2.83)	21 (2.44)	6 (6.38)	*0.041*
Previous Cardiac Surgery (%)	58 (6.08)	48 (5,58)	10 (10.64)	*0.085*
Renal failure on dialysis (%)	9 (0.94)	6 (0.7)	3 (3.19)	*0.05*
Diabetes on medication (%)	256 (26.83)	230 (26.74)	26 (27.66)	*0.946*
Peripheral Vascular Disease (%)	152 (15.93)	136 (15.81)	16 (17.02)	*0.876*
Active Endocarditis (%)	16 (1.68)	13 (1.51)	3 (3.19)	*0.203*
Recent Myocardial Infarction (%)	202 (21.17)	186 (21.63)	16 (17.02)	*0.365*
Unstable Angina (%)	41 (4.29)	37 (4.3)	4 (4.25)	*0.621*
Systemic Hypertension (%)	714 (74.84)	642 (74.65)	72 (76.59)	*0.773*
LVEF% (mean,SD,median)	52.32±10.7 (55)	52.38±10.7 (55)	51.7±11.2 (54.5)	*0.56*
IABP (%)	8 (0.84)	8 (0.93)	0	*0.434*
Pulmonary Hypertension (%)	22 (2.31)	19 (2.21)	3 (3.19)	*0.371*
Liver dysfunction (%)	38 (3.98)	33 (3.84)	5 (5.32)	*0.317*
Alcohol abuse (%)	3 (0.31)	3 (0.35)	0	*0.732*

BMI: Body mass index; COPD: Chronic pulmonary obstructive disease; NYHA: New York Health Association; LVEF: Left vebntricular ejection fraction; IABP: Intra-aortic balloon pump

Table 3. Relationship between operative variables and incidence of cerebral complications

	Total population (N=954)	Patients without cerebral complications (N=860)	Patients with cerebral complications (N=94)	*P*
Type of surgery				
Isolated CABG	520	492	28	*0.0001*
CABG + valve surgery	100	84	16	
Isolated valve surgery	233	203	30	
Major aortic surgery	88	73	15	
Others	13	8	5	
Open chambers procedure (%)	434 (45.49)	368 (42.8)	66 (70.21)	*0.0001*
Aortosclerosis (%)	198 (20.75)	172 (20)	26 (27.66)	*0.564*
CPB time (min) (mean,SD,median)	113.6±45.05 (105)	111.6±41.8 (105)	131.7±65.6 (120)	*0.002*
CPB T°C (mean,SD,median)	31.8±1.2 (32)	31.9±1.1 (32)	31.1±2.4 (32)	*0.006*
Cardioplegia (cristalloid vs hematic)	270/684	228/632	42/52	*0.0003*
Clamping time (min) (mean,SD,median)	74.2±35.2 (65)	72.8±33.6 (64)	86.7±45.9 (80)	*0.007*
Urgent operation (%)	140 (14.67)	122 (14.19)	18 (19.15)	*0.255*
Mean Arterial Pressure <50mmHg (%)	63 (6.6)	56 (6.5)	7 (7.45)	*0.898*
IABP (%)	16 (1.68)	13 (1.51)	3 (3.19)	*0.203*
PaO_2/FiO_2 (mean,SD,median)	331.7±109.9 (327.5)	332.4±108.7 (329)	325±121.4 (321)	*0.486*
Glicemia post CPB (mean,SD,median)	141.1±30.3 (138)	141.2±30.1 (138)	140.7±32.3 (137)	*0.784*
T°C post CPB (mean,SD,median)	36.1±0.5 (36.1)	36.1±0.4 (36.1)	36±0.7 (36)	*0.281*
pH post CPB (mean,SD,median)	7.37±0.04 (7.37)	7.38±0.04 (7.37)	7.36±0.04 (7.36)	*0.045*
Ht post CPB (mean,SD,median)	28.01±3.5 (27.6)	28.1±3.6 (27.6)	27.2±2.9 (27)	*0.023*
End CPB blood lactate level (mean,SD,median)	1.59±1.01 (1.3)	1.52±0.84 (1.3)	2.17 ±1.87 (1.5)	*0.0001*

CPB: cardiopulmonary bypass; IABP: Intra-aortic balloon pump

over, post-CPB acidosis and hemodilution were prevalent in patients with cerebral complications. On the other hand, incidence of aortosclerosis was similar in both groups (Table 3).

Postoperative morbidity was higher in the group of patients who experienced neurological complications, as reported in Table 4.

In hospital mortality was 5.32% (5 of 94) among

Table 4. Relationship between postoperative variables and incidence of cerebral complications

	Patients without cerebral complications (N=860)	Patients with cerebral complications (N=94)	*P*
Transfusion (%)	426 (49.53)	71 (75.53)	*0.0001*
Low cardiac output (%)	106 (12.32)	27 (28.72)	*<0.0001*
Peak blood lactate in ICU (mean,SD,median)	1.61 ±1 (1.3)	2.24 ±1.7(1.5)	*0.0001*
Use of insulin in ICU (%)	338 (39.3)	56 (59.57)	*0.0002*
Reexploration (%)	34 (3.95)	12 (12.76)	*0.0009*
Perioperative Myocardial Infarction (%)	17 (1.98)	4 (4.25)	*0.144*
T °C on arrival in ICU (mean,SD,median)	30.13 ±2.5(30.1)	29.9 ±2.4(30)	*0.378*
T °C after 4 hours in ICU (mean,SD,median)	35.2 ±1.6 (35.4)	35.2 ±1.3 (35.4)	*0.885*
Glicemia (mean,SD,median)	147.9 ±35.2 (141)	147.8 ±35.2 (141)	*0.18*
PaO_2 / FiO_2 (mean,SD,median)	294.58 ±85.2 (296)	278.68 ±82.9 (268)	*0.064*
Ht in ICU (mean,SD,median)	31.9 ±3.4 (31.95)	30.6 ±3.6 (30.3)	*0.0004*
pH in ICU (mean,SD,median)	7.38 ±0.04 (7.39)	7.38 ±0.04 (7.38)	*0.613*
IABP insertion in ICU (%)	17 (1.98)	4 (4.25)	*0.144*

IABP: Intra-aortic balloon pump

Table 5. Stepwise logistic regression analysis of 94 early neurological complications

Variable	Coefficient	Std. Error	Odds Ratio	95% CI	*p*
Age (years)	0.0457	0.014	1.0468	1.0184-1.0759	*0.0011*
COPD (yes/no)	0.8436	0.376	2.3248	1.1125-4.8581	*0.0249*
Open cardiac chambers (yes/no)	-0.6507	0.1366	0.5217	0.3992-0.6818	*<0.0000*
end-CPB blood lactate level (mmol/L)	0.3562	0.097	1.4279	1.1807-1.7268	*0.0002*
Transfusion (yes/no)	0.5881	0.2705	1.8006	1.597-3.0594	*0.0297*
Use of insulin in ICU (yes/no)	0.5957	0.2412	1.8143	1.1308-2.9111	*0.0135*

patients who experienced postoperative neurological complications and 1.98% (17 of 860) in patients without cerebral complications (p=0.05). Three deaths in the former group were related to permanent stroke.

When significant variables obtained by means of univariate analysis were included in a multivariate analysis, 2 preoperative patients-related risk factors, 2 intraoperative and 2 postoperative variables were identified as predictors of cerebral complications (Table 5).

Discussion

The aim of this study was to evaluate the whole population of cardiac surgical patients using multivariate analysis in order to identify risk factors for early neurological complications. Patients who were older, had history of COPD, had undergone open heart chambers procedures, presented higher end-CPB blood lactate levels, received more frequently blood transfusions, or required insulin therapy in ICU were at higher risk for early onset of cerebral complications.

In our experience 94 patients (9.85%) had neurological events: 14 (1.47%) type I and 80 (8.38%) type II complications, according to Roach classification (1). The rate of permanent cerebral symptoms was 1.1% (11 of 954). These rates are similar to those reported in other studies (1, 5-14). The mechanisms behind postoperative cerebral complications in cardiac surgery are multifactorial including embolism of atherosclerotic debris in association with manipulations of the aorta, thromboembolism, gaseous microembolism from the CPB circuit, inadequate perfusion of the brain during CPB, and systemic inflammatory response. The identification of genetic predisposition in cerebral injuries after cardiac surgery has been recently investigated (4). Genetic factors associated with inflammation predicted a three-fold increase in stroke rate over and above other clinical risk factors. The mechanism of this association might be related to an increased prothrombotic activity, which has been shown in studies of coronary artery thrombosis and myocardial infarction. These mechanisms often depend on a common pathway and then they can be associated in the same patient. There is agreement that increased age, diabetes, previous cardiovascular disease, the presence of carotid atherosclerotic disease, and prolonged CPB and aortic cross clamping times are associated with adverse neurologic events (1, 6, 16-18).

Older age by itself is a powerful predictor of higher postoperative neurological dysfunction after cardiac surgery, as recently demonstrated by us and other authors (6, 19-20). The physiological changes of these patients are related to the cerebral atrophy and the diminished cerebrovascular reserve capacity respectively, combined with cerebrovascular atherosclerosis and sub-clinical degenerative brain disease. These factors contribute to the high propensity to develop acute postoperative neurological dysfunction.

Another variable which resulted to be independently associated with neurological complications in our study was COPD. Possible mechanisms behind this relationship include increased haemoglobin levels and carbon dioxide retention with associated changes in cerebral vasoreactivity. Frequently these patients are elderly, and have comorbidities such as atherosclerosis and systemic vascular disease. They presented with an evident pre-thrombotic condition due to the increase in the viscosity of the blood and endothelial dysfunction related to systemic inflammatory disease (21).

The incidence of overall cerebral complications among patients undergoing combined CABG and valve procedures has been reported to be higher compared with CABG surgery or valve replacement alone (5, 22). Also in our study a higher incidence of cerebral complications occurred in patients operated on for isolated heart valve procedure (12.9%), combined CABG and valve surgery (16%) or major aortic procedures (17%). Assessment for atheromatous debris appears then helpful in any cardiac surgery procedure where surgical manipulation of the aorta will occur, including any type of aortic clamping, aortic cannulation, aortic valve incisions, proximal coronary graft anastomoses, and antegrade cardioplegia catheters.

Previous studies have identified CPB time and aortic cross-clamping time as risk factors for neurologic injury (23). Prolonged CPB can be a marker of a complicated procedure or a more severe state of postoperative heart failure. In our study, CPB time and aortic cross-clamping time were significantly longer in the group of patients with neurological complications, but they did not result independent risk factors in the multivariable analysis. An extended aortic cross-clamping time reflected a complicated procedure or advanced atherosclerotic disease. Subsequently, in consideration of the prolonged CPB time and of its associated potential hazards, it can be speculated that extra-corporeal perfusion during CPB may be less adequate for the brain.

Three aspects of CPB management influence the risk of neurological injury during cardiac surgery: the mean arterial pressure (MAP), temperature and acid-base status. The role of MAP during CPB has been discussed by many authors. MAP between 50 and 70 mmHg range are well tolerated by most patients during CPB. Higher MAP may decrease cardiac, renal, and neurologic complications and may be desirable for patients affected by known stenosis of the cerebral vessels (5, 24). Hypoperfusion and embolization may coexist in the pathophysiology of brain ischemia and reduced perfusion limits the ability of the bloodstream to wash out emboli and microemboli and worses the cerebral injury. This issue was also addressed in a randomized trial in which 248 patients undergoing non emergent CPB were assigned to MAP of 80 to 100 mmHg or 50 to 60 mmHg. Higher pressures during CPB was associated with a significant lower rate of neurologic and cardiac complications or death at six months without an increase in bleeding or in transfusion requirements or a difference in cognitive and functional status (25). At our institution perfusion pressure > 70 mmHg during CPB was maintained in all patients with a documented associated cerebrovascular disease, although MAP<50 mmHg did not result a significant risk factor for early neurologic injury at univariate analysis.

Randomized trials comparing the safety of normothermic and hypothermic CPB have producted conflicting results (26-27). In our experience moderate hypothermia in the range of 34°C is commonly employed for closed chamber cardiac operations, 32°C for open chamber procedures, and 30°C for cardiac procedure associated with carotid endoarteriectomy. Our study showed that bypass temperature was significantly lower in the group of patients with neurological complications, but it did not result independent risk factor in the multivariate analysis. This relationship could be probably explained considering that lower CPB temperature is usually maintained in such surgical procedures (isolated heart valve surgery, combined operations or major aortic procedures) that resulted themselves at high risk of early neurological injury. It is well known that hypothermia alters acid-base balance, which in turn affects cerebral blood flow. Cerebral autoregulation is preserved with alpha-stat management, with reported lower rates of postoperative cognitive dysfunction (28). In our experience pH after CPB resulted significantly lower in the group of patients with neurological complications, although it did not reach statistical significance in the multivariate analysis.

Metabolic aspects of CPB management deserve consideration. There is consistency in identifying an intraoperative onset of hyperlactatemia during CPB as a determinant of bad outcomes (29-30). Hyperlactatemia is defined as an arterial lactate concentration > 3 mmol/L. It is a complex condition, due to an impaired tissue oxygenation leading to increased anaerobic metabolism and excessive production of pyruvate, converted to lactate. Several studies have established the use of lactates as a marker of global tissue hypoxia in circulatory shock (29). Hyperlactatemia is strongly

correlated with hyperglicemia during cardiogenic shock and there is evidence about a relationship between peak blood glucose levels and peak blood lactate concentrations during CPB (30). Patients presenting with this condition probably suffered a prolonged inadequate oxygen delivery during CPB, leading to anaerobic energy production. An inadequate oxygen delivery may result from a low cardiac output, a poor arterial oxygen content due to low hematocrit value, increased oxygen demands, and a combination of these factors. In our study, end-CPB higher blood lactate levels and lower hematocrit values, as well as peak blood lactate and the use of insulin in ICU resulted significantly associated to early neurologic complications at univariate analysis. Moreover end-CPB higher blood lactate levels and the use of insulin in ICU confirmed to be risk factors for early cerebral complications also at multivariate stepwise logistic regression analysis. In consideration of the strong correlation between hemodynamic parameters during surgery and cerebral hypoperfusion, a rigorous CPB management appears mandatory.

The limitations of this study should be noted. This is a single centre experience based on a relatively small number of patients, prospectively collected in nearly one year of surgical activity. We have examined only the occurrence of early neurological complications and its relationship with preoperative and procedural risk factors. We have then excluded from the analysis clinical variables as postoperative atrial fibrillation that are reported to be related to late occurrence of cerebral injury. Although we collected the data prospectively, we have used only routine clinical and laboratory parameters and we have no information about the value of specific neurologic markers like protein S 100B in changing the prediction of cerebral injury after cardiac surgery.

Conclusions

Our study proved a strong relationship between metabolic and technical aspects of CPB management and early occurrence of neurological injury. Preoperative screening and intraoperative measures that ensure adequate cerebral perfusion, minimize embolization and improve systemic inflammatory response and hemodynamic performance appear mandatory in these patients. Future efforts will be necessary to strongly validate surrogate markers of early neurologic damage in order to predict and evaluate neuropsychological dysfunction after cardiac surgery.

Acknowledgements

We thank Lois Clegg, English Language Teacher, University of Parma, for her assistance in the revision of the manuscript.

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Accepted: 15th April 2013
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