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Cerebral ischaemia during cardiac surgery in children detected by combined monitoring of BIS and near-infrared spectroscopy

M. Hayashida*, N. Kin, T. Tomioka, R. Orii, H. Sekiyama, H. Usui, M. Chinzei and K. Hanaoka

Department of Anesthesiology, Graduate School of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan

**Corresponding author: Surgical Center Research Hospital, Institute of Medical Science, University of Tokyo, 4-6-1 Shiroganedai, Minato-ku, Tokyo 108-8639, Japan. E-mail: hayashida-todai@umin.ac.jp*

Background. Children frequently suffer transient cerebral ischaemia during cardiac surgery. We measured cerebral ischaemia in children during cardiac surgery by combining two methods of monitoring.

Methods. We studied 65 children aged between 5 months and 17 yr having surgery to correct non-cyanotic heart disease using hypothermic cardiopulmonary bypass (CPB). During surgery, we measured the Bispectral Index (BIS) and regional cerebral haemoglobin oxygen saturation (Sr_{O_2}) with near-infrared spectroscopy (NIRS). Cerebral ischaemia was diagnosed if both Sr_{O_2} and BIS decreased abruptly when acute hypotension occurred. In each patient, the relationship between Sr_{O_2} and arterial blood pressure (AP) was indicated by a plot of mean Sr_{O_2} against simultaneous mean AP.

Results. We noted 72 episodes of cerebral ischaemia in 38 patients. Sixty-three ischaemic events were during CPB. Cerebral ischaemia was less frequent in older patients. Cerebral ischaemia was more common and more frequent in children under 4 yr old. Haematocrit during CPB was lower and Sr_{O_2} was more dependent on AP in children under 4 yr.

Conclusions. Children less than 4 yr of age are more likely to have cerebral ischaemia caused by hypotension during cardiac surgery. Ineffective cerebral autoregulation and haemodilution during CPB may be responsible.

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During cardiac surgery, haemodynamic changes such as acute hypotension and cardiac arrest can cause cerebral ischaemia.[1–4] When cerebral blood flow (CBF) decreases by more than half, electroencephalographic evidence of cerebral ischaemia appears as acute slowing of the raw electroencephalogram (EEG) frequency.[5] Acute slowing of the EEG occurs within seconds of hypotension, occlusion of the carotid artery and cardiac arrest, as indicated by a variety of processed encephalographic methods.[1–4 6 7] The Bispectral Index (BIS) also allows detection of cerebral ischaemia,[8–11] particularly if it is used along with near-infrared spectroscopy (NIRS) to indicate cerebral tissue oxygenation.[11] When acute slowing of the EEG develops because of cerebral ischaemia, an abrupt and profound reduction in BIS is seen, although the depth of anaesthesia does not change.[8–11]

By using both BIS and NIRS measurements in children having cardiac surgery, we found that transient cerebral ischaemia caused by acute hypotension was frequent, especially in younger children.[11] We measured the incidence of cerebral ischaemia using a combination of BIS and NIRS monitoring and hoped to identify factors that could affect the incidence of cerebral ischaemia in children having cardiac surgery.

Methods

With approval of the review board and informed consent of a parent, we studied 65 children aged 5 months to 17 yr about to have surgery to correct non-complex non-cyanotic heart disease using hypothermic cardiopulmonary bypass (CPB). Thirty-one had ventricular septal defect (VSD), 20 had atrial septal defect (ASD), six had both ASD and VSD, six had stenosis of the right ventricular outflow tract, pulmonary valve or pulmonary conduit, one had mitral valve regurgitation, and one had aortic valve regurgitation. We did not study infants under 4 months of age because they usually have more severe heart disease and more complex surgery, so comparison with older children is difficult. We also noted that infants under 4 months have consistently low BIS values (usually between 30 and 50) that do not relate to anaesthetic doses, suggesting that BIS monitoring is inappropriate (data presented in poster form at the 7th America–Japan Anesthesia Congress in Kofu, Japan, October 2002).

Anaesthesia was maintained with sevoflurane in oxygen-enriched air and intermittent doses of fentanyl and vecuronium. We routinely monitored five-lead electrocardiogram, invasive arterial blood pressure (AP), central venous pressure (CVP), oximetry (Sp_{O_2}), end-tidal carbon dioxide tension (E'_{CO_2}) and oesophageal and rectal temperatures. In addition, we monitored BIS throughout surgery using a processed EEG monitor (A-1050 version 3.4; Aspect Medical Systems, Natick, MA, USA) and four electrodes (Aspect Zipprep) placed on bilateral temporal areas (At1 and At2), with Fpz as the reference and Fp1 as the ground. At the same time we measured regional cerebral haemoglobin oxygen saturation (Sr_{O_2}) to assess cerebral oxygen delivery,[12 13] using a dual-detector three-wavelength NIRS oximeter (PSA-3N; Biomedical Science, Kanazawa, Japan)[14] with its emitter–detector placed laterally to the EEG electrode in the mid-forehead Fpz position. Analogue signals of AP, CVP, Sp_{O_2}, E'_{CO_2} and Sr_{O_2} were sampled at 100 Hz using PowerLab and Chart for Windows (version 4; AD Instruments, Colorado Springs, CO, USA) and the data were recorded with an off-line computer. Digital values of BIS and other processed EEG variables were taken every 5 s and recorded off-line with Microsoft Hyperterminal for Windows.

The CPB circuit was primed with blood-containing (n = 18) or bloodless (n = 47) priming solution (Table 1). After the circulation had been stabilized on CPB, midazolam, fentanyl and vecuronium were given via the CPB circuit and a low concentration of sevoflurane was also given. When CPB was prolonged, the same drugs were added every 1.5–2 h. We induced mild or moderate hypothermia, aiming for a rectal temperature of 28–30°C for correction of VSD or an incompetent left heart valve or 30–32°C for correction of ASD or a right-sided stenotic lesion. Blood gases and haematocrit were measured every 20 min during CPB. After surgical repair, we gave dobutamine, dopamine and nitroglycerine if necessary during and after weaning from CPB. Sevoflurane was used for maintenance of anaesthesia after CPB.

Cerebral ischaemia was diagnosed when simultaneous abrupt reductions in Sr_{O_2} and in BIS (by >10 units) were noted with acute hypotension and without changes in anaesthesia (Fig. 1).[11] We noted the number or frequency of cerebral ischaemic events that occurred intraoperatively in each patient. The initial, mean and minimum haematocrit during CPB were noted. The minimum (instantaneous) AP during CPB was determined and the mean AP during the whole period of CPB was computed from the logged AP data. The relationship between Sr_{O_2} and AP was assessed in each patient by plotting the mean Sr_{O_2} measured every minute during surgery (MSr_{O_2}) against the simultaneous mean AP (MAP) and examining the correlation between these variables by simple regression analysis. The presence or absence of a statistically significant correlation between MAP and MSr_{O_2} was taken to show that Sr_{O_2} changes were AP-dependent or AP-independent (Figs 1–3). The slope of the regression line for the MSr_{O_2}–MAP plot [slope ($\Delta Sr_{O_2}/\Delta AP$)] was also determined to estimate how Sr_{O_2} was affected by AP. The relationship of Sr_{O_2} to AP and a value of slope ($\Delta Sr_{O_2}/\Delta AP$) was assessed for each patient for paired data sets obtained not only during the whole period of surgery, including CPB (Fig. 3A and C), but also during the prebypass period alone (Fig. 3B and D). The AP-dependency of Sr_{O_2} during the prebypass period was assessed to exclude any influence of haemodilution on Sr_{O_2}, which may decrease cerebral oxygen delivery and thus Sr_{O_2}, by a decrease in the oxygen content of arterial blood (Ca_{O_2}), irrespective of a decrease in CBF, so that the MSr_{O_2}–MAP relationship could be a very close approximation to the CBF–MAP relationship.

Patients were divided into five age groups: Group 1 (0.4–1.0 yr, n = 16), Group 2 (1.0–2.0 yr, n = 15), Group 3 (2.0–4.0 yr, n = 13), Group 4 (4.0–8.0 yr, n = 10) and Group 5 (over 8.0 yr, n = 11). Parametric data are summarized as mean (SD) and groups were compared using ANOVA followed by Fisher's protected least significant difference test. Frequency variables were compared using the χ^2 test. Multiple and simple regression analyses were used to identify factors that may influence the frequency of cerebral ischaemia. A value of $P < 0.05$ was considered statistically significant.

Results

The patients were aged 3.9 (4.2) yr (mean, SD). Their details are listed in Table 1. Trends of AP, Sr_{O_2} and BIS in a 5-month-old and an 11-yr-old child undergoing patch closure of VSD are shown in Figs 1 and 2 respectively. In the younger child, transient cerebral ischaemic events were suggested by simultaneous abrupt transient reductions in Sr_{O_2} and BIS on three occasions, associated with acute

Table 1 Group comparisons for the number and percentage of patients with cerebral ischaemia, frequency of cerebral ischaemia per patient, initial, mean and minimum haematocrit (Hct) during CPB, mean blood pressure during the whole period of CPB, minimum instantaneous blood pressure during CPB, number and percentage of patients with AP dependency of Sr_{O_2} and slope ($\Delta Sr_{O_2}/\Delta AP$) during surgery and during the prebypass period. *$P < 0.05$ *vs* Group 6; †$P < 0.05$ *vs* Group 5; ‡$P < 0.05$ *vs* Group 4; §$P < 0.05$ *vs* Group O. Results of group comparisons by ANOVA or the χ^2 test are shown as (NA) (not applied), NS (not significant) or S (significantly different between and among groups). Slope ($\Delta Sr_{O_2}/\Delta AP$) is the slope for the MSr_{O_2} *vs* MAP relationship

Variable	All patients	Group 1	Group 2	Group 3	Group 4	Group 5	Group Y	Group O	ANOVA or χ^2 test
Age	0.4–17.0	0.4–1 yr	1–2 yr	2–4 yr	4–8 yr	>8 yr	0.4–4 yr	⩾4 yr	
No. of patients	65	16	15	13	10	11	44	21	
Age (yr)	3.9 (4.2)	0.6 (0.2)	1.4 (0.3)	2.7 (0.6)	5.6 (1.3)	12.0 (2.6)	1.5 (0.9)	8.9 (3.9)	(NA)
Body height (cm)	92 (29)	65 (4)	76 (6)	87 (8)	108 (10)	143 (16)	75 (11)	127 (22)	(NA)
Body weight (kg)	14.3 (10.8)	5.9 (0.9)	9.2 (2.4)	11.7 (2.8)	17.2 (3.9)	34.2 (10.6)	8.7 (3.2)	26.1 (11.8)	(NA)
Boy/girl	36/29	11/5	7/8	8/5	4/6	6/5	23/16	10/11	NS
ASD/VSD/ASD + VSD/others	20/31/6/8	2/9/4/1	4/10/0/1	6/3/2/2	3/6/0/1	5/3/0/3	11/20/5/3	8/23/0/3	NS
Duration of surgery (min)	185 (52)	174 (39)	193 (65)	179 (57)	182 (42)	197 (54)	182 (54)	190 (48)	NS
Duration of CPB (min)	79 (34)	79 (21)	87 (47)	74 (40)	75 (25)	76 (33)	81 (37)	76 (29)	NS
Blood prime/bloodless prime	18/47	11/5	6/9	1/12	0/10	0/11	13/26	0/21	(NA)
Initial Hct (%) during CPB	22.1 (3.1)	21.3 (2.9)*	20.7 (2.9)*	20.6 (2.9)*	21.3 (2.7)*	24.0 (2.8)	20.9 (2.8)§	22.7 (3.0)	S
Mean Hct (%) during CPB	20.5 (2.7)	22.6 (3.0)*	20.9 (2.9)*	20.9 (2.9)*	21.2 (2.3)*	25.0 (2.1)	21.5 (3.0)§	23.2 (2.9)	S
Minimum Hct (%) during CPB	21.7 (3.3)	20.6 (2.6)*	19.3 (2.1)*	20.0 (2.8)*	20.1 (2.7)*	23.3 (1.8)	20.0 (2.5)§	21.7 (2.8)	S
Mean AP (mm Hg) during CPB	46.5 (7.8)	46.6 (8.2)	42.4 (5.7)†*	46.0 (5.4)	49.5 (10.5)	50.2 (7.5)	44.9 (6.8)§	49.9 (8.8)	S
Minimum AP (mm Hg) during CPB	24.1 (6.2)	22.2 (4.7)*	21.3 (4.5)*	23.9 (6.8)*	24.3 (5.4)*	30.7 (6.1)	22.4 (5.3)§	27.6 (6.6)	S
Minimum oesophageal temperature (°C)	29.1 (2.0)	28.3 (1.9)	29.2 (1.6)	29.8 (1.8)	28.9 (1.9)	29.9 (2.5)	29.0 (1.8)	29.4 (2.4)	NS
Minimum rectal temperature (°C)	30.6 (2.7)	29.4 (1.6)	30.3 (2.3)	30.9 (1.9)	29.9 (1.7)	30.9 (2.3)	30.0 (2.1)	30.4 (2.1)	NS
No. of patients with/without AP-dependent Sr_{O_2} (% with AP-dependent Sr_{O_2})									
During surgery	50/15 (77%)	14/2 (88%)*	13/2 (87%)*	12/1(92%)*	6/4 (60%)	5/6 (46%)	35/4 (90%)§	11/10 (52%)	S
During the prebypass period	43/22 (66%)	13/3 (81%)*	13/2 (87%)†*	10/3 (77%)*	5/5 (50%)	2/9 (18%)	32/7(82%)§	7/14 (33%)	S
Slope ($\Delta Sr_{O_2}/\Delta AP$) (%/mm Hg)									
During surgery	0.133 (0.118)	0.171 (0.124)†*	0.186 (0.114)†*	0.171 (0.119)†*	0.059 (0.063)	0.026 (0.031)	0.176 (0.117)§	0.042 (0.051)	S
During the prebypass period	0.114 (0.120)	0.174 (0.123)†*	0.158 (0.096)†*	0.149 (0.128)†*	0.036 (0.060)	–0.005 (0.040)	0.161 (0.114)§	0.015 (0.053)	S
No. of patients with/without cerebral ischaemia (% with cerebral ischaemia)	38/27 (59%)	13/3 (81%)†*	12/3 (80%)†*	8/5 (62%)*	3/7 (30%)	2/9 (18%)	29/10 (74%)§	5/16 (24%)	S
Frequency of cerebral ischaemia per patient (times)	1.11 (1.20)	1.56 (1.32)†*	1.53 (1.19)†*	1.23 (1.17)*	0.60 (1.08)	0.18 (0.41)	1.46 (1.21)§	0.38 (0.81)	S

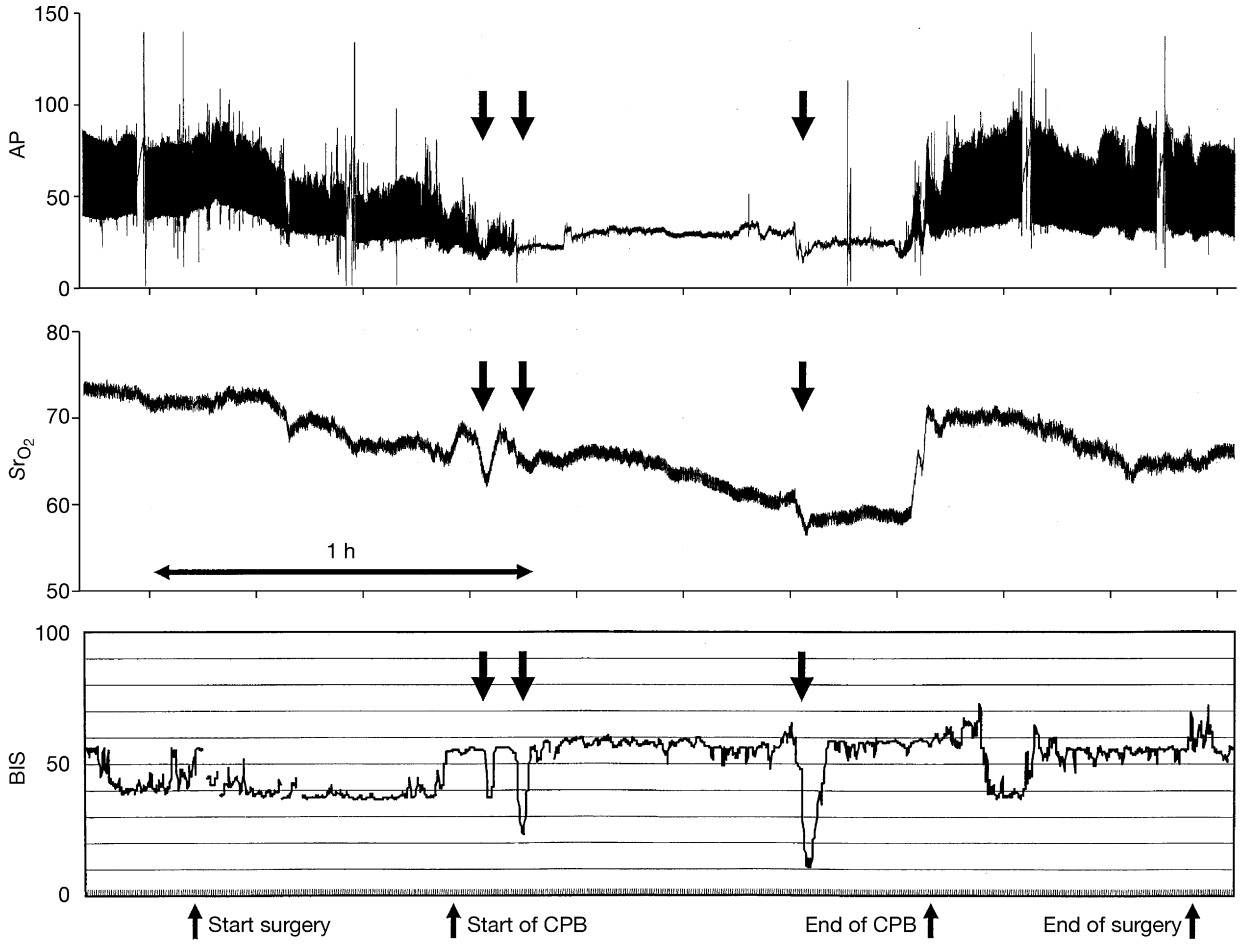

Fig 1 Relationships of arterial blood pressure (AP), regional cerebral haemoglobin oxygen saturation (Sr_{O_2}) and Bispectral Index (BIS) in a 5-month-old boy undergoing patch closure of ventricular septal defect. Three episodes of simultaneous transient reductions in Sr_{O_2} and BIS suggest transient cerebral ischaemia during the initial hypotension at the onset of CBP and during the reduced pump flow rate on aortic clamp release (arrows). Note that Sr_{O_2} continued to change in parallel with changing AP, not only during the whole period of surgery including CPB, but also during the prebypass period, suggesting that cerebral blood flow depended on AP.

hypotension during CBP (Fig. 1). Such events were not noted in the older child (Fig. 2). The MSr_{O_2}–MAP plots indicated that MSr_{O_2} was related to MAP in the younger child both during surgery (Fig. 3A) and during the prebypass period (Fig. 3B). In the older child there was no relationship between MAP and MSr_{O_2} either during surgery (Fig. 3C) or during the prebypass period (Fig. 3D).

In 65 patients, 72 episodes of cerebral ischaemia were detected, in which BIS decreased abruptly from 65.1 (12.1) to 38.8 (15.5) and returned abruptly to 65.5 (12.8) within several min and Sr_{O_2} decreased from 67.5 (6.1) to 60.8 (9.8)% and returned to 67.9 (7.7)%. BIS never reached 0, indicating isoelectric silence[15] during these ischaemic events. In all the ischaemic periods except three, BIS reductions lasted <3 min. Twenty-seven patients did not suffer any cerebral ischaemia, while 38 experienced cerebral ischaemia one to four times during surgery. Nine ischaemic events occurred before CPB and 63 during CPB. Of the events during CPB, 34 developed during initial hypotension after the start of CPB, while 29 were noted during transient reductions in the pump flow rate and thus in perfusion pressure on unclamping of the aorta, made to prevent atrial discharge (Fig. 1).

Group comparisons are summarized in Table 1. There were no differences between the groups in types of heart disease, duration of surgery, duration of CPB, and minimum oesophageal and rectal temperatures. More patients experienced cerebral ischaemia, and more often, in Groups 1, 2 and 3 than in Group 5. Haematocrit values during CPB were less in Groups 1, 2, 3 and 4 than in Group 5. The minimum AP during CPB was lower in Groups 1, 2, 3 and 4 than in Group 5 and the mean AP during CPB was lower in Group 2 than in Groups 4 and 5.

More patients showed a significant relationship between MAP and MSr_{O_2} in Groups 1, 2 and 3 than in Group 5, and slope ($\Delta Sr_{O_2}/\Delta AP$) was greater in Groups 1, 2 and 3 than in

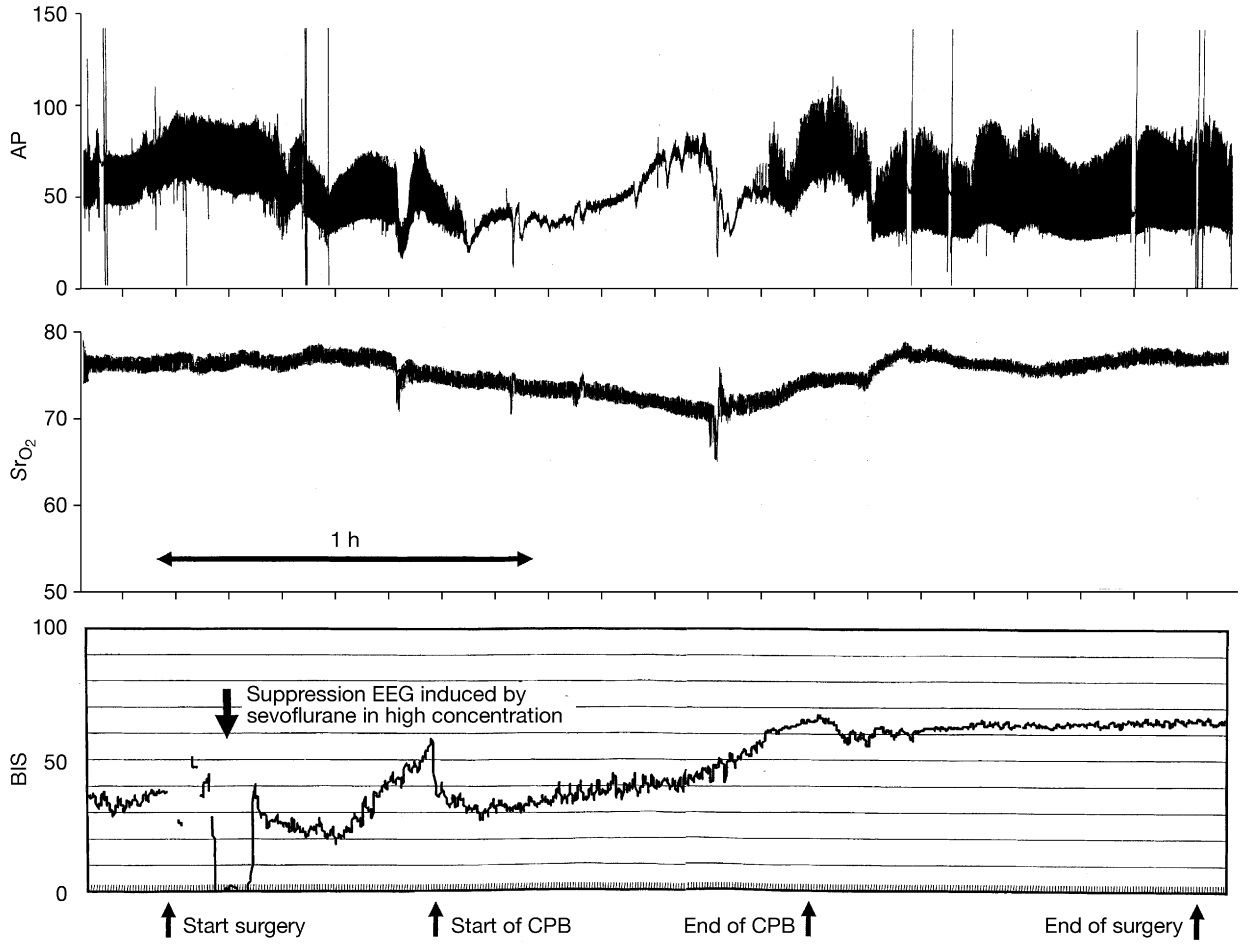

Fig 2 Arterial blood pressure (AP), regional cerebral haemoglobin oxygen saturation (Sr_{O_2}) and Bispectral Index (BIS) in an 11-yr-old boy undergoing patch closure of ventricular septal defect. Simultaneous abrupt reductions in Sr_{O_2} and BIS indicative of cerebral ischaemia did not occur, although a marked reduction in BIS, reflecting EEG suppression caused by high-concentration sevoflurane, occurred at the start of surgery. A decrease in BIS caused by anaesthesia can be differentiated from that caused by ischaemia if Sr_{O_2} does not decrease. Note that Sr_{O_2} was not related to changes in AP, although extremely transient reductions in Sr_{O_2} are seen, suggesting prompt recovery of cerebral blood flow during acute hypotension.

Groups 4 and 5, both during surgery and during the prebypass period. Cerebral ischaemic events and slope ($\Delta Sr_{O_2}/\Delta AP$) during the prebypass period became less with increasing age, particularly when age was over 4 yr (Table 1).

By simple regression analysis, the frequency of cerebral ischaemia in each patient correlated inversely with age In particular, age younger than 4 yr was associated with a higher frequency of ischaemia (Fig. 4). Multiple regression analysis was performed on the dependent variable of frequency of cerebral ischaemia using independent variables that could affect this: the minimum and mean AP, the initial, mean and minimum haematocrit, and slope ($\Delta Sr_{O_2}/\Delta AP$) during surgery and during the prebypass period. The mean haematocrit, the minimum AP and slope ($\Delta Sr_{O_2}/\Delta AP$) during the prebypass period gave the highest regression coefficient ($r = 0.623$, $P < 0.0001$). The frequency of cerebral ischaemia correlated significantly with slope ($\Delta Sr_{O_2}/\Delta AP$) during the prebypass period (standard regression coefficient $r = 0.353$, $P < 0.01$) and with the mean haematocrit ($r = –0.295$, $P < 0.01$), but not with the minimum AP ($r = –0.196$, not significant).

Simple regression analyses showed that the frequency of cerebral ischaemia correlated positively with slope ($\Delta Sr_{O_2}/\Delta AP$) during the prebypass period ($r = 0.527$, $P < 0.0001$), which in turn correlated inversely with age ($r = –0.535$, $P < 0.0001$). In particular, age less than 4 yr was associated with higher values of slope($\Delta Sr_{O_2}/\Delta AP$) (Fig. 5). The frequency of cerebral ischaemia correlated negatively with the mean haematocrit ($r = 0.469$, $P < 0.0001$), which in turn correlated positively with age ($r = 0.361$, $P = 0.01$). The frequency of cerebral ischaemia did not correlate with the minimum oesophageal or rectal temperature.

When patients were divided into groups <4 yr old (Group Y) and ⩾4 yr old (Group O), cerebral ischaemia was more common and more frequent, haematocrit values during CPB

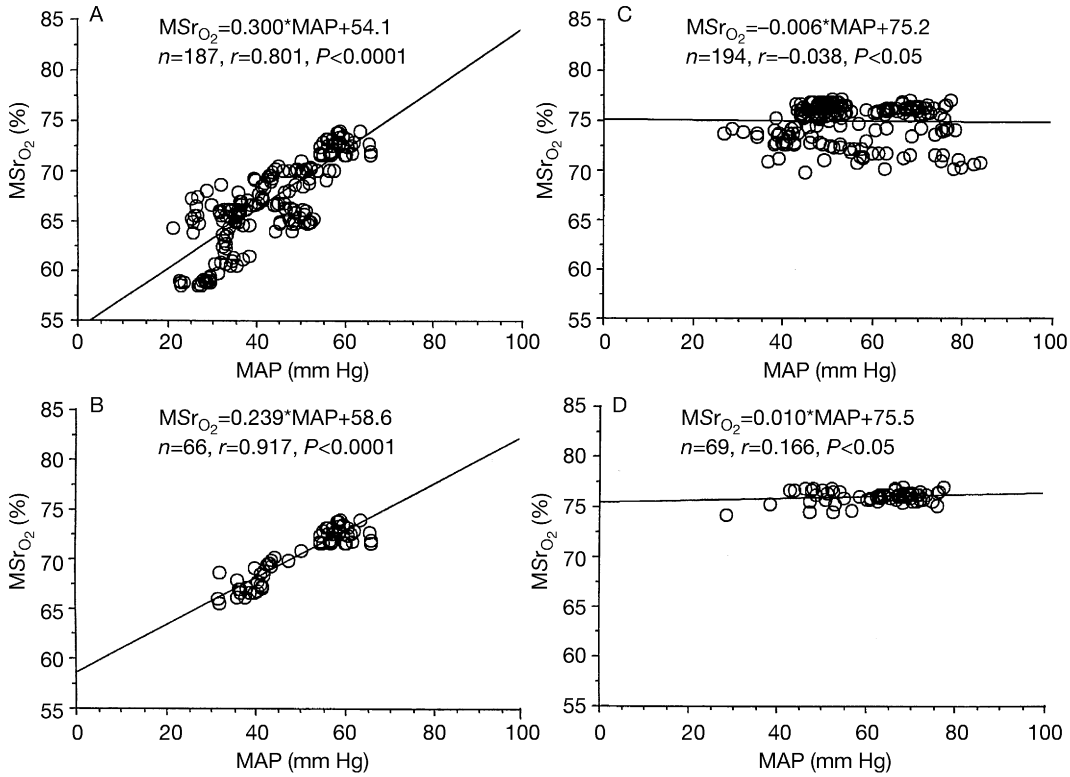

Fig 3 Relationships of mean arterial blood pressure (MAP) and mean regional cerebral haemoglobin oxygen saturation (MSr_{O_2}) in a 5-month-old child (A and B) and in an 11-yr-old child (C and D) undergoing patch closure of ventricular septal defect. In the 5-month-old child, whose monitor trends were shown in Fig. 1, MSr_{O_2} correlated positively with MAP not only during surgery (A) but also during the prebypass period (B), suggesting that cerebral blood flow (CBF) was dependent on arterial blood pressure. The values of slope ($\Delta Sr_{O_2}/\Delta AP$) during surgery and the prebypass period were 0.300 and 0.239 %/mm Hg respectively. In the 11-yr-old child, whose monitor trends were shown in Fig. 2, MSr_{O_2} did not correlate with MAP during surgery (C) or before bypass (D), suggesting that CBF was independent of AP. The slopes were only –0.006 and 0.010. Slope ($\Delta Sr_{O_2}/\Delta AP$) is the slope of the regression line for the MSr_{O_2}–MAP plot.

were lower and AP-dependency of Sr_{O_2} was more common and more evident in Group Y (Table 1).

We did not detect any gross neurological deficits after surgery, such as newly developed mental retardation, convulsions or motor palsy.

Discussion

A decrease in Sr_{O_2} associated with hypotension suggests a decrease in cerebral oxygen delivery.[12–14] An abrupt decrease in BIS reflects abrupt slowing of the EEG,[11] which is an early indication of cerebral ischaemia.[5] With the combination of BIS and NIRS, transient cerebral ischaemic events were frequent during paediatric cardiac surgery, especially in younger children.

Cerebral ischaemia developed most commonly during CPB. Both haematocrit and AP during CPB were less in children younger than 4 yr, in whom ischaemic events were frequent. On multiple regression analysis, haematocrit but not AP significantly affected the frequency of cerebral ischaemia. More haemodilution, but not lower blood pressure, during CPB contributed to more cerebral ischaemia in the younger children, presumably through greater

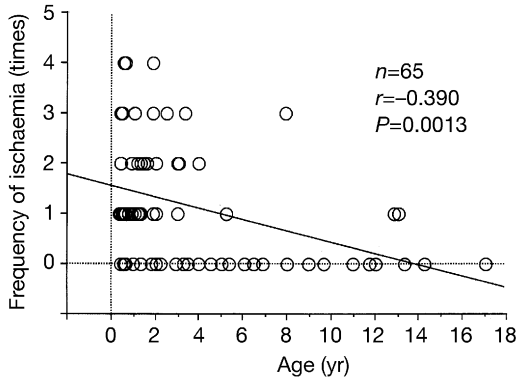

Fig 4 Relationship between age and the frequency of cerebral ischaemia in 65 patients.

decreases in Ca_{O_2} and thus in cerebral oxygen delivery, irrespective of a reduction in CBF.

Our previous report suggested that cerebral autoregulation may be less effective in younger children, because Sr_{O_2}, a correlate of cerebral oxygen delivery,[12–14] tends to change in parallel with changing blood pressure throughout surgery.[11] Cerebral oxygenation in children <4 yr of age

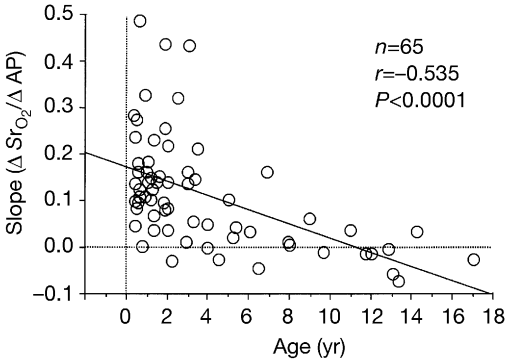

Fig 5 Relationship between age and slope ($\Delta Sr_{O_2}/\Delta AP$) before bypass in 65 patients. Slope ($\Delta Sr_{O_2}/\Delta AP$) is the slope of the regression line for the MSr_{O_2}–MAP plot.

was more dependent on AP, not only during surgery but also during the prebypass period. Because Sr_{O_2} should be related to CBF especially before bypass (i.e. in the absence of acute haemodilution), these results suggest that CBF is more dependent on AP and thus cerebral autoregulation is poor in children <4 yr old. Simple and multiple regression analyses showed that the more frequent cerebral ischaemia in younger children is due primarily to immature autoregulation, indicated by greater slope ($\Delta Sr_{O_2}/\Delta AP$), and secondarily to more marked haemodilution during CPB.

Information about the development of cerebral autoregulation in children is limited.[16] Some studies show no autoregulation in critically ill preterm infants[17–19] or limited autoregulation in stable preterm infants.[20] Younger children, compared with older children, undergoing coarctation repair have lower CBF velocity after aortic clamp release, with less compensation for acute hypotension.[21] Compared with adults, even healthy adolescents have a slightly delayed return of CBF velocity in response to transient hypotension.[22] Our present study supports these suggestions that cerebral autoregulation is immature during infancy and childhood. Our study also suggests that cerebral autoregulation may develop rapidly during early childhood to become nearly complete by the age of 4 yr. In contrast, one recent study found no differences in cerebral autoregulation in infants, children and adults.[23] In that study, blood pressure was gradually changed with a slow infusion of phenylephrine,[23] whereas in other studies blood pressure was altered more quickly, causing greater transient changes in CBF velocity.[20–22]

With an abrupt reduction in blood pressure, CBF will decrease for a brief period (1–2 min) before autoregulation restores CBF.[24] Immature autoregulation may take more time to restore CBF, so that a decrease in CBF long and severe enough to depress EEG activities may occur more easily in young children. Even in such children, however, decreases in BIS lasted for no longer than 3 min in most ischaemic events, suggesting that cerebral autoregulation restored CBF and EEG during hypotension, albeit slowly.[11] In addition, BIS never reached 0, indicating a total loss of EEG activity and suggesting that the ischaemia we observed was incomplete and that cerebral oxygen delivery was present but reduced, being sufficient to sustain some EEG activities.[5] Presumably, by these mechanisms neurological damage did not occur, despite frequent cerebral ischaemia. In this sense, transient ischaemia during paediatric cardiac surgery may not be of grave clinical significance. However, our observation was made only in a small number of subjects, and no other systematic studies have been carried out on transient cerebral ischaemia during paediatric surgery. The clinical significance of transient ischaemia should be assessed in further studies using detailed perioperative neurological examinations. Non-invasive monitoring with BIS and NIRS appears to be useful, especially in children <4 yr old (but >4 months old), as cerebral ischaemic events are frequent and are sometimes prolonged (e.g. >3 min in three of our patients), and prolonged ischaemia can cause irreversible EEG changes during cardiac surgery.[1 2 4]

The NIRS oximeter cannot indicate the saturation level at which cerebral ischaemia will occur.[12 13] By combining NIRS and BIS, we can determine if a reduction in CBF, indicated by a decrease in Sr_{O_2}, is sufficient to cause cerebral dysfunction. Conversely, we can determine that a decrease in BIS is caused by decreased CBF and not by hypothermia or deepened anaesthesia (Fig. 2) if there is a concurrent reduction in Sr_{O_2}. Thus the BIS–NIRS combination can be a convenient ischaemic/anaesthetic monitor.

In conclusion, children less than 4 yr of age are more likely to have transient cerebral ischaemia caused by acute hypotension during cardiac surgery mainly due to ineffective cerebral autoregulation.

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