Anesthesiology
73:632–636, 1990

Anterior Fontanel Pressure and Visual Evoked Potentials in Neonates and Infants Undergoing Profound Hypothermic Circulatory Arrest

Frederick A. Burrows, M.D., F.R.C.P.C., Simon C. Hillier, M.B., Ch.B., F.F.A.R.C.S.,†*
M. Elizabeth McLeod, M.D., F.R.C.P.C. Karey S. Iron, B.A.,‡ Margot J. Taylor, Ph.D.§*

To determine the effects of cardiopulmonary bypass with profound hypothermic circulatory arrest (PHCA) on anterior fontanel pressure (AFP) and visual evoked potentials (VEPs), 21 neonates and infants undergoing cardiopulmonary bypass (CPB) with PHCA for surgical correction of congenital heart defects were studied. Mean (± SD) minimum nasopharyngeal, esophageal, and rectal temperatures of 16.4 ± 2.2, 11.2 ± 2.7, and 17.7 ± 1.9° C, respectively, were achieved for a mean duration of PHCA of 51.6 ± 18.7 min. AFP increased significantly above pre-CPB values for the first 21.7 ± 8.1 min of rewarming. The duration of this increase in AFP was related logarithmically and directly to the product of the nasopharyngeal temperature (NPT) at the end of PHCA and the duration of PHCA ($r^2 = 0.82$, $P < 0.0001$). Nineteen of these patients had simultaneous monitoring of VEPs. The latency of both the N70 and P100 components of the VEPs increased as temperature decreased. The cerebral perfusion pressure was linearly and inversely related to the AFP ($r^2 = 0.72$, $P < 0.01$). The VEPs disappeared at a nasopharyngeal temperature (NPT) of 18.9 ± 2.8° C and reappeared after 21.9 ± 8.8 min post-PHCA at an NPT of 32.8 ± 1.4° C. There was no significant difference between duration of increased AFP (20.9 ± 8.1 min) and the duration of absence of VEPs during the post-PHCA period. The duration of increased AFP correlated linearly and directly with the duration of absence of VEPs ($r^2 = 0.84$, $P < 0.005$). These data demonstrate that transient neurophysiologic dysfunction occurs after PHCA. This dysfunction is related to the duration of elevation of the AFP and cannot be explained solely by a temperature effect. (Key words: Anesthesia: pediatric. Monitoring: anterior fontanel pressure; visual evoked potentials. Surgery, Cardiac: congenital heart disease; cardiopulmonary bypass; circulatory arrest.)

ANTERIOR FONTANEL PRESSURE (AFP), an accurate estimate of intracranial pressure (ICP),[1,2] decreases during cardiopulmonary bypass (CPB) with profound hypothermic circulatory arrest (PHCA) and then rebounds above pre-CPB levels during reperfusion and rewarming.[3,4] These changes in AFP with reperfusion are associated with a decrease in cerebral perfusion pressure (CPP).[3,4] A delayed return to normal of cerebral blood flow (CBF) velocity (an index of cerebral perfusion)[5] has been noted to occur during the same time period.[6] The relationship between neurophysiologic function and changes in AFP and CPP during this rewarming period has not been investigated.

Visual evoked potentials (VEPs) have been used as an objective measure of neurophysiologic function in the visual pathway during PHCA in neonates and infants.[7,8] The visual cortex is sensitive to small changes in cerebral perfusion since it lies in the watershed area of the posterior and middle cerebral arteries. The sensitivity of this organ to inadequate perfusion has been supported clinically by reports of visual disturbances after cardiac surgery.[9,10] Predictable changes in both the latency and amplitude of VEPs with changes in temperature have been demonstrated during CPB and PHCA.[7] In addition, the resolution of VEP changes during rewarming has been shown to correlate with the duration and temperature of the PHCA period.[8]

The current study, performed in neonates and infants with congenital heart defects undergoing corrective cardiac surgery, evaluated the changes in AFP during rewarming CPB after PHCA and investigated possible correlations to the duration and temperature of the PHCA period. Simultaneous measurements of the VEPs were made to determine the relationship between changes in VEPs and AFP.

Materials and Methods

With the approval of the Human Subject Review Committee, 21 patients (11 male, 10 female) less than 9 months of age and admitted to The Hospital for Sick Children for surgical repair of congenital cardiac defects requiring CPB and PHCA, were studied.

Patients received intramuscular atropine 20 $\mu g \cdot kg^{-1}$ 30–60 min preoperatively. Anesthesia was induced and maintained with intravenous fentanyl 50–100 $\mu g \cdot kg^{-1}$. Neuromuscular blockade was achieved with pancuronium 0.15 $mg \cdot kg^{-1}$ given intravenously. Supplemental doses of fentanyl and pancuronium were administered as necessary. Nasotracheal intubation was performed, and intermittent positive pressure ventilation was commenced with an air/oxygen mixture (FI_{O_2} of 0.7–1.0). An arterial catheter was inserted for measurement of systemic arterial pressure and intermittent blood sampling. A central ve-

* Assistant Professor of Anaesthesia.
† Fellow in Anaesthesia.
‡ Research Assistant.
§ Assistant Professor of Paediatrics.

Received from the Department of Anaesthesia, the Research Institute and the Department of Paediatrics (Division of Neurology), The Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada. Accepted for publication May 10, 1990.

Address reprint requests to Dr. Burrows: Department of Anaesthesia, The Hospital for Sick Children, 555 University Avenue, Toronto, Ontario, Canada M5G 1X8.

nous catheter was inserted percutaneously into the superior vena cava *via* the external or internal jugular vein for measurement of the central venous pressure (CVP). No cerebral vasoactive agents were administered.

After induction of anesthesia, hair over the anterior fontanel was shaved and tincture of benzoin was applied to the scalp. A calibrated Ladd transducer was placed over the fontanel and secured with a 3-cm by 3-cm piece of adhesive foam (3M Canada Inc., North York, Ontario). Excessive application pressure was avoided and a positive response to jugular compression was documented in each instance. AFP was continuously recorded on a single-channel recorder.

After anticoagulation with heparin 300 IU $\cdot$ kg^{-1}, nonpulsatile CPB was established with a standard roller pump (Cobe Canada Ltd., Scarborough, Ontario) and a 0.8- or 1.5-m^2 SciMed® membrane oxygenator (SciMed Life Systems, Inc., Minneapolis, MN) or a 0.8- or 1.6-m^2 Capiox® hollow fiber membrane oxygenator (Terumo, Tokyo, Japan). The CPB circuit was primed with blood and Plasmalyte (Travenol, Mississauga, Ontario) solution plus mannitol 1 g $\cdot$ kg^{-1} to maintain a hematocrit of 30% during CPB. CPB flows were maintained between 2.4 and 3.2 l $\cdot$ m^{-2} $\cdot$ min^{-1}. Cardioplegia consisted of modified Roe's solution,[11] with 20 mEq $\cdot$ l^{-1} of sodium bicarbonate, with an initial dose of 300 ml $\cdot$ m^{-2} body surface area, and 150 ml $\cdot$ m^{-2} given at approximately 30-min intervals. Patients were cooled at a rate of 1–3° C $\cdot$ min^{-1}. PHCA was used with mean (± SD) minimum nasopharyngeal, esophageal, and rectal temperatures of 16.4 ± 2.2, 11.2 ± 2.7, and 17.7 ± 1.9° C, respectively, before induction of circulatory stasis and venous exsanguination.

Alpha stat acid–base management was used[12] and aimed at maintaining Pa_{CO_2} between 32–37 mmHg and *p*H between 7.35–7.40. Arterial blood gas determinations were performed with a Nova® Stat Profile 4 blood gas analyzer (Nova Biomedical, Waltham, Massachusetts). During CPB arterial acid–base status was continuously monitored using a CDI 300® monitoring system (Cardiovascular Devices, Inc., Irvine, CA).

Throughout the study, AFP, mean arterial pressure (MAP), and CVP were measured continuously. An increase or decrease in AFP was considered to be present when the value differed from the mean pre-CPB value by greater than 1 mmHg. CPP was calculated, every 5 min, as the difference between the MAP and the AFP or CVP, whichever was greater. During each of the periods—prebypass, cooling bypass, circulatory arrest, and warming bypass—nasopharyngeal, esophageal, and rectal temperatures were measured and recorded every 5 min, and arterial blood gas, hematocrit, and serum ionized calcium analysis was performed and recorded every 15 min. The duration of each phase of CPB and PHCA was recorded as was the time for AFP to return to the pre-CPB bypass level during rewarming.

In 19 patients (9 male, 10 female) VEPs were simultaneously measured. In 2 patients VEP studies could not be completed due to intraoperative failure of the equipment. VEPs were in response to binocular stimulation with light-emitting diode goggles secured over the infant's eyes after anesthetic induction. Both eyelids were taped with translucent tape. The VEPs were recorded with a single active surface electrode to Oz referenced to Fz; a grounding electrode was placed on the infant's forehead. A 500- or 1000-ms sweep was used, and the gain was 10 K, and filters were 1–100 Hz. Impedances were less than 2000 Ω. Each average contained 64 artifact-free trials collected at a rate of 0.9 flashes $\cdot$ s^{-1}. The amplitude and latency of the two main components of the VEP, a negative wave occurring around 100 ms (N70) and a positive wave occurring at 155 ms (P100), were measured (the latencies were due to the age of the infants tested). Baseline VEPs were established after administration of the anesthetic and before the start of surgery. VEPs were recorded repeatedly prior to CPB; during the cooling process until a response could no longer be obtained; during the rewarming of the patient to normothermia; and then after CPB. We defined the reappearance of the VEPs during rewarming CPB as the first recording in which an identifiable and reproducible P100 wave was present.

The observers of AFP measurements (SH, NM, FB) and of the VEP measurements (KI, MT) were blinded until analysis of the data.

All patients underwent preoperative and postoperative clinical neurologic evaluation. Neurologic status was assessed by clinical neurologic examination of mental and developmental states, cranial nerves, motor and sensory systems, and cerebellar function.

Data are presented as mean (± SD). Time-dependent data were analyzed with repeated-measures analysis of variance and the Tukey test for multiple comparisons. Individual comparisons were made with the paired Student's t test. The strength of the relationship between variables were determined with least-squares regression analysis and reported as the coefficient of determination (r^2). The coefficient of determination was used because it expresses the proportion of total variation that was accounted for by the fitted regression line.[13] The power of the regression analysis was determined using standard techniques[13,14] ($\alpha = 0.05$). The coefficients of correlation (r) of the linear relationships were compared to r of the nonlinear relationships with the use of Fisher's transformation.[13] Statistical significance was accepted as $P < 0.05$.

Results

The demographic data of the 21 patients studied is presented in table 1. The mean (± SD) age and weight of the infants were 3.94 ± 2.72 months and 5.28 ± 1.89

TABLE 1. Demographic Data

Patient	Age (months)	Weight (kg)	Diagnosis	Operation
1*	6.25	5.13	AVSD	Complete repair
2*	5	4.65	TGA	Arterial switch
3	5.25	5.77	TGA	Atrial repair
4	6.5	6.61	TGA	Atrial repair
5	8	7.89	VSD	VSD closure
6	4.75	6.60	TGA	Arterial repair
7	0.25	3.71	TGA	Arterial repair
8	8	8.15	TGA	Atrial repair
9	0.25	1.28	IAA/VSD	Complete repair
10	1	3.3	AVSD	Complete repair
11	6	6.42	VSD	VSD closure
12	5.5	5.58	AVSD	Complete repair
13	5	8.07	TGA	Atrial repair
14	5	6.87	TGA	Arterial switch
15	0.25	2.93	TGA	Arterial switch
16	3.5	6.06	TGA	Atrial repair
17	6.25	6.48	VSD	VSD closure
18	4.5	5.12	AVSD	Complete repair
19	0.5	3.36	TGA	Arterial switch
20	0.25	3.25	AVSD	Complete repair
21	0.75	3.54	VSD	VSD closure
Mean ± SD	3.94 ± 2.72	5.28 ± 1.89		

AVSD = atrioventricular septal defect. TGA = transposition of the great arteries. VSD = ventricular septal defect. IAA = interrupted aortic arch.

* Only AFP monitored.

kg, respectively. The mean (± SD) duration of circulatory arrest was 51.6 ± 18.7 min.

MAP was significantly less during the cooling and rewarming phases of CPB compared to the prebypass period (table 2). AFP increased only during the rewarming period. These changes resulted in a significant reduction in CPP during CPB. During the initiation of rewarming bypass when the AFP was maximal, the CPP value was significantly lower than prebypass and cooling bypass values. During rewarming CPP correlated linearly and inversely with the magnitude of elevation of AFP above cooling bypass values ($r^2 = 0.72$, $P < 0.01$). There was no significant change in Pa_{CO_2} during these periods.

TABLE 2. Summary of Data for VEP Group

	CPB		
Pressure (mmHg)	Pre-CPB†	Cooling‡	Warming¶
Mean arterial	53.1 ± 9.1	37.4* ± 10.3	36.2* ± 9.8
Anterior fontanel	8.1 ± 4.0	7.9 ± 4.6	18.6** ± 5.9
Cerebral perfusion	42.5 ± 10.4	26.1* ± 2.2	17.7** ± 6.2
Pa_{CO_2}	34.9 ± 5.5	34.2 ± 5.1	32.2 ± 4.2

Data are mean values (±SD) of the 19 patients monitored with both VEPs and AFP.

† Values obtained immediately prior to CPB during hemodynamic stability at 32.4 ± 3.2° C (NPT).

‡ Values obtained on stable CPB immediately prior to PHCA at 16.4 ± 2.2° C (NPT).

¶ Values obtained on initiation of warming CPB (time of maximum AFP elevation) at 19.3 ± 2.4° C (NPT).

* $P < 0.05$ compared to pre-CPB values.

** $P < 0.05$ compared to pre- and cooling CPB values.

CPB = cardiopulmonary bypass.

The time (mean ± SD) for the AFP to return to baseline values during rewarming after PHCA was 21.7 ± 8.1 min. Although the duration of the increase in AFP post-PHCA correlated weakly with the duration of PHCA ($r^2 = 0.22$, $P < 0.05$) and with NPT at the end of PHCA ($r^2 = 0.13$, $P < 0.05$), it did increase logarithmically and directly with the product of these two variables (T × C) ($r^2 = 0.82$, $P < 0.0001$) (fig. 1). T × C correlated weakly with peak AFP elevation ($r^2 = 0.07$, $P < 0.05$).

Mean VEP latencies for N70 and P100 prior to cooling were 104 ± 13 and 159 ± 27 ms, respectively. The latency of VEPs increased as temperature decreased. The VEPs disappeared at a mean (± SD) NPT of 18.9 ± 2.8° C. They remained absent throughout the period of profound hypothermia and circulatory arrest. During reperfusion and rewarming, the VEPs reappeared after an average of 21.9 ± 8.8 min at a mean NPT of 32.8 ± 1.4° C, with the N70 and P100 at latencies of 150 ± 24.8 and 219 ± 29 ms, respectively. The VEP components decreased in latency during normothermia, and usually stabilized by the end of surgery (an average of 31.4 ± 16.3 min after their reappearance), with mean latencies of 105.5 ± 15 (N70) and 170 ± 34 ms (P100). These values were not significantly different from the precooling latencies.

The latencies of both N70 and P100 correlated significantly with the NPT during cooling (N70: $r^2 = 0.72$, $P < 0.0001$; P100: $r^2 = 0.67$, $P < 0.0001$), whereas they correlated significantly less closely with NPT during rewarming (N70: $r^2 = 0.11$, $P < 0.05$; P100: $r^2 = 0.34$, $P < 0.001$).

In the 19 patients who had both AFP and VEPs mea-

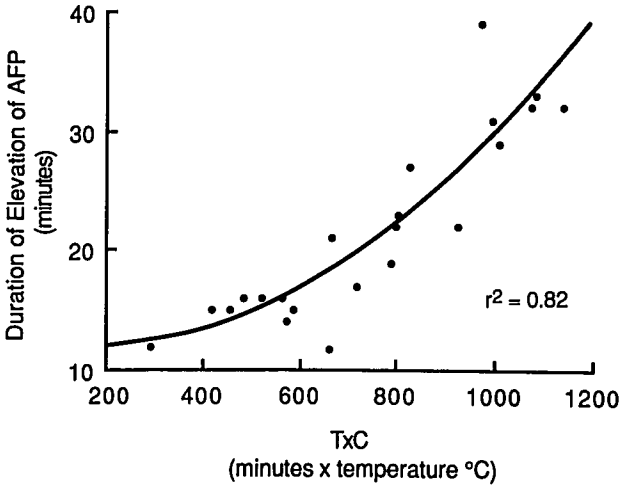

FIG. 1. The duration of the increase in anterior fontanel pressure (AFP) post-PHCA above baseline values as a logarithmic function of the product of the duration of PHCA and temperature during circulatory arrest (T × C).

sured (tables 1 and 2), the mean times for AFP to return to their pre-CPB baseline values (20.9 ± 8.1 min) and for VEPs to return to their pre-CPB values (21.9 ± 8.8 min) after PHCA were not statistically different. The duration of the increase in AFP upon reperfusion correlated with the time taken for the VEPs to reappear ($r^2 = 0.84$, $P < 0.005$) (fig. 2).

All patients survived their operations. Twenty of the 21 infants showed no evidence of neurologic changes from their preoperative assessments. One patient (patient 12) developed transient cortical blindness, left hemiparesis, and choreoathetosis postoperatively. At follow-up 4 months postoperatively the patient's neurologic examination had returned to normal. In this patient both AFP and VEPs had been measured. This infant experienced a long period of PHCA (65 min) and an NPT of 16.7° C at the end of PHCA, producing a high T × C (1086). Of the patients studied, this patient demonstrated the greatest increase in the N70 and P100 components of the VEP postoperatively.

Discussion

The etiology of the increase in AFP post-PHCA is not clear. Previous investigators attributed the increase in AFP to an increase in cerebral blood volume (CBV) possibly due to cerebral vasodilation secondary to the accumulation of metabolic byproducts during the period of PHCA[3,4] as cerebral metabolism continues during hypothermic arrest.[15,¶] Although this is consistent with our finding of a logarithmic correlation between the duration of AFP elevation and T × C, an index of the severity of the ischemic insult (fig. 1), it is unlikely that the increase in AFP is due solely to increased CBV for two reasons: 1) CPP was relatively low during this period, and so by itself would tend to limit CBF and CBV; and 2) recent studies indicate that CBFV does not return to pre-PHCA values during and after rewarming.[6] Increased CBV due to venous obstruction also is an unlikely explanation since the CVP before and after PHCA was not significantly different; however, cerebral venodilation could increase CBV without an increase in CBF or in CVP.

In this study of neonates and infants undergoing CPB with PHCA, the measurements of AFP (to estimate ICP) allowed us to calculate the CPP during CPB. During reperfusion and rewarming we found a marked decrease in CPP that was temporally related to the delay in the reappearance of VEPs. This decrease in CPP has been reported previously[3,4] but has never been correlated to the delay in return of the VEPs.

In contrast to previous work using VEPs during CPB in infants,[16] we have found VEPs to be an easily applicable and objective modality to evaluate the possible neurophysiologic side effects of various cardiopulmonary support techniques used in the repair of congenital heart lesions.[7,8] Hypothermia causes consistent and quantifiable changes in the VEP through the effects of cooling on the central nervous system, *e.g.*, a decrease in the amplitude and an increase in the duration of the action potential, and a decrease in nerve conduction velocity, axonal resting potential, and presynaptic transmitter release. We found that the VEPs disappeared just below 20° C, the temperature at which synaptic transmission fails.[17] However, the reverse pattern was not seen on rewarming; *i.e.*, VEPs did not appear at 20° C with prolonged latencies and then

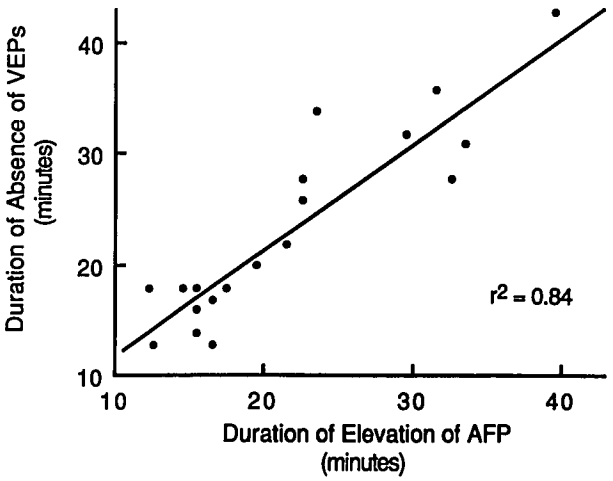

FIG. 2. The delay in return of the visual evoked potentials (VEPs) as a function of the duration of increased anterior fontanel pressure (AFP) above pre-cardiopulmonary bypass (CPB) values on the initiation of CPB and rewarming after PHCA.

¶ Hickey PR, Anderson NP. Deep hypothermic circulatory arrest: A review of pathophysiology and clinical experience as a basis for anesthetic management. J Cardiothorac Anesth 1:135–55, 1987

decrease in latency with increasing temperature. Rather, VEPs were absent until an NPT of 32.8 ± 1.4° C was reached, with improvement in latencies thereafter. This could be due to uneven recovery of brain temperature or to metabolic effects. The VEPs had not returned to normal by the end of CPB as demonstrated by the latency changes in both the N70 and P100 components of the VEP after rewarming. Previous studies have demonstrated similar findings with decreased cerebral electroencephalographic activity after PHCA during and after rewarming.[18]

The principal finding of this study was the linear relationship between the duration of absence of VEPs and the duration of increase in AFP μpon rewarming (fig. 2). This relationship suggests that PHCA induces a neurophysiologic abnormality that delays the return of VEP, and that this abnormality occurs while the AFP is increased. It is unclear whether the delay in recovery of VEPs is caused by the same abnormality that increases AFP or whether it is secondary to the increase in AFP, *i.e.*, decreased CPP.

CBF velocity is decreased during rewarming post-PHCA compared to the same temperature during cooling prior to PHCA.[6] Initially, the decrease in CBFV may be due to a decrease in CPP secondary to an elevated ICP; however, the duration of decreased CBFV and CBF[6,19] is greater than the duration of the increase of AFP and reduction of CPP, and extends into the post-CPB period.[19] Similarly, there is a delay in return to normal of neurophysiologic activity (N70, P100) after PHCA. Although it is possible that the degree of hypothermia itself might induce the same neurophysiologic dysfunction upon rewarming, neither increase in AFP nor lag in return of VEPs during rewarming after continuous low flow bypass at similar temperatures (NPT < 20° C) has been noted.[7,**] In total, these observations suggest a generalized disturbance of cerebral electrophysiology, metabolism, and blood flow after PHCA.

In summary, we have demonstrated transient neurophysiologic dysfunction, not explained solely by a temperature effect, upon reperfusion and rewarming after PHCA. The duration of impairment corresponds to the duration of increased ICP and decreased CPP. The duration of the increased ICP in turn is related logarithmically to the magnitude of the ischemic insult as indicated by the product T × C. In order to improve our understanding of this observation, further studies closely examining cerebral hemodynamics and metabolism during this early rewarming period of CPB are necessary.

** Personal observation.

References

1. Hill A, Volpe JJ: Measurement of intracranial pressure using the Ladd intracranial pressure monitor. J Pediatr 98:974–6, 1981
2. Vidyasagar D, Raju TNK: A simple non-invasive technique of measuring intracranial pressure in the newborn. Pediatrics 59: 957–61, 1977
3. Stow PJ, Burrows FA, McLeod ME, Coles JG: The effect of cardiopulmonary bypass and profound hypothermic circulatory arrest on anterior fontanelle pressure in infants. Can J Anaesth 34:450–4, 1987
4. Friesen RH, Thieme R: Changes in fontanel pressure during cardiopulmonary bypass and hypothermic circulatory arrest in infants. Anesth Analg 66:94–6, 1987
5. Lundar T, Lindberg H, Lindegaard K-F, Tjønneland S, Rian R, Bø G, Nornes H: Cerebral perfusion during major cardiac surgery in children. Pediatr Cardiol 8:161–65, 1987
6. Hillier SC, Burrows FA, Bissonnette B, Lerman J. Cerebral hemodynamics following profound hypothermic circulatory arrest in infants (abstract). ANESTHESIOLOGY 71:A1038, 1989
7. Keenan NK, Taylor NH, Coles JG, Prieur BJ, Burrows FA: The use of visual evoked potentials in assessing cardiopulmonary bypass and circulatory arrest. Electroencephalogr Clin Neurophysiol 68:241–6, 1987
8. Reilly EL, Kondo C, Brunberg JA, Doty DB: Visual evoked potentials during hypothermia and prolonged circulatory arrest. Electroencephalogr Clin Neurophysiol 45:100–6, 1978
9. Myendorf R: Psychopathoophthalmology, gnostic disorders and psychosis in cardiac surgery. Arch Psychiatr Nervenkr 232:119–25, 1982
10. Smith JL, Cross SA: Occipital lobe infarction after open heart surgery. J Clin Neuro Ophthalmol 3:23–30, 1983
11. Roe BB, Hutchinson JC, Fishman NH, Ullyot DJ, Smith DL: Myocardial protection with cold, ischemic, potassium-induced cardioplegia. J Thorac Cardiovasc Surg 73:366–70, 1977
12. Swan H: The importance of acid-base management for cardiac and cerebral preservation during open heart operations. Surg Gynecol Obstet 58:391–414, 1984
13. Zar JH: Biostatistical Analysis. Second edition. Englewood Cliffs, Prentice-Hall, Inc., 1984, pp 185–190, 268–272, 306–317, 312–318
14. Cohen J: Technical appendix: Computational procedures, Statistical Power Analysis for the Behavioral Sciences, revised edition. Toronto, Academic Press Inc., 1977, p 458
15. Kurth CD, Steven JM, Nicolson SC, Norwood WI, Chance B: Changes in brain oxygenation during cardiopulmonary bypass (CPB) and hypothermic circulatory arrest (CA) in neonates (abstract). ANESTHESIOLOGY 71:A1035, 1989
16. Markland ON, Warren CH, Moorthy SS, Stoelting RK, King RB: Monitoring of multimodality evoked potentials during open heart surgery under hypothermia. Electroencephalogr Clin Neurophysiol 59:432–40, 1984
17. Lougheed WM: The central nervous system in hypothermia. Br Med Bull 17:61–9, 1961
18. Weiss M, Weiss J, Cotton J, Nicolas F, Binet JP: A study of the electroencephalogram during surgery with deep hypothermia and circulatory arrest in infants. J Thorac Cardiovasc Surg 70: 316–29, 1975
19. Greeley WJ, Ungerleider RM, Smith LR, Reves JG: The effects of deep hypothermic cardiopulmonary bypass and total circulatory arrest on cerebral blood flow in infants and children. J Thorac Cardiovasc Surg 97:737–45, 1989