

Cerebral Complication after Open Heart Surgery

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Abstract

(Extra-Corpor. Technol. 19[3] p. 274-280 Fall 1987, 10 ref.) The incidence of brain complication after open heart surgery in Rajvithi Hospital has been around 2-3% each year until 1985. Since January 1986, the Capiox II membrane oxygenator has been on routine use in alternate with the Bentley bubble oxygenator. There has been increasing incidence of cerebral complication since the beginning of the year. Retrospective study is carried on to analyze brain complications after open heart surgery during January to May 1986. During this period there were 96 patients ranging in age from 9 months to 55 years, submitted for open heart surgery. Among these there were 13 patients suffering cerebral complication, namely subarachnoid or subdural hemorrhage and brain infarction, of which 6 patients died. There was no statistical significance of type of oxygenators which would cause cerebral complication. The cerebral complication was related to the weight of patients, degree of acidosis and pO_2 during bypass and immediately after. It is concluded that these cerebral complications could be lessened if control of blood gas and pH is optimum.

Introduction

Cerebral complication is not uncommon after open heart surgery.¹ The causes may be air emboli, microemboli and cerebrovascular accidents. Other factors which may be involved are flow rate, perfusion pressure, temperature and biochemical.^{2,3} The incidence of cerebral complication occurred in membrane oxygenation more often than bubble oxygenation.⁴

Since January 1986 we have used membrane oxygenators in alternate with bubble oxygenators in open

heart surgery. We also found an increasing incidence of cerebral complication in the first 5 months after introduction of the membrane oxygenators (Figure 1). This prompted our concern that it may have had something to do with the new oxygenators.

The present report is our study of cerebral complication after open heart surgery performed in our hospital between January and May 1986 with the aim of trying to identify the factors responsible for the cause of cerebral complication.

Materials and Methods

Between January 1986 and May 1986, there were 95 patients, age ranged from 7 months to 55 years, submitted for open heart surgery. The numbers of each sex were equal. All bypasses were conducted at 22-23°C with ischemic cardiac arrest. Myocardial protection was achieved using modified St. Thomas cardioplegic solution infusing into coronary circulation at the dosage of 10 ml. per Kg every half an hour. Local hypothermia with cold saline and ice sludge was also

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N = 95

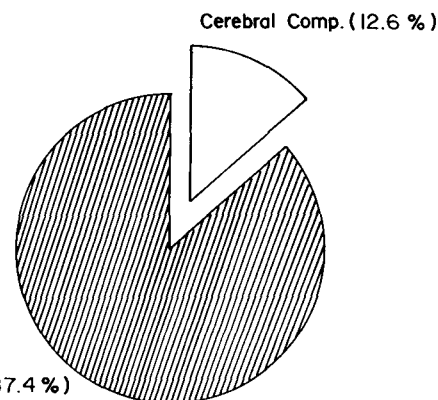


Figure 1

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employed. Two types of oxygenators were used, namely the Bentley Bos type (bubble) and Terumo Capiox II (membrane). The Sarns roller pump was used for all cases. No prebypass filter or arterial filter was used. Before bypass the oxygenator and tubing circuit (silicone tubing) were flushed with 100% CO₂ for 3 minutes. The air was evacuated from the heart before the release of aortic clamp via left atrial or left ventricular vent and also through a stab wound of the proximal aorta. Cerebral complications were recorded if there was any. Analysis was made to determine if factors that were responsible for cerebral complication could be identified. All recorded data were compared between the patients who had cerebral complication and those without the complication using the student's paired t tests.

Results

Twelve patients were classified as having cerebral complications, namely subarachnoid hemorrhage, subdural hemorrhage and brain infarctions, of which 6 patients died (Table 2). Comparison between the complication group and the noncomplication group did not reveal any statistical difference in sex, severity of disease, bypass time, mean arterial or perfusion pressure during bypass, dosage of heparinization, time of circulatory arrest, types of oxygenators (Table 1 and Figure 2), hemoglobin (Figure 3), and Blood pH (Figure 4). No difference was found in pCO₂ obtained before and after perfusion between these two groups (Figure 5). However, there was statistical significance in the difference in pO₂, HCO₃, Sodium and Potassium at specific times during surgery and also there was statistical difference in age, weight, basal surface area and type of heart disease and operation.

Factors Influencing Cerebral Complication

Age. There was statistical significance in age between these two groups at p-value <.05. The mean age in the cerebral complication group was 15.6 years while the mean age in the noncerebral group was 25.1 years.

Weight. Weight was significant at p-value <0.5, having the mean weight of 26.3 Kg. in the cerebral complication group and 36.7 Kg. in the noncerebral group.

Basal surface area. The mean basal surface area of the cerebral complication group was .96 square meter as compared to the 1.23 square meter in the noncerebral group at p-value <0.5.

pO₂. There was significant difference only in pO₂ obtained just prior to the bypass but not to pO₂ obtained during and after bypass. The mean pO₂ was 133.7 mmHg in the cerebral complication group and was 175.2 mmHg in the noncerebral group at p <.05 (Figure 6).

HCO₃. The HCO₃ was low at the beginning of perfusion (mean = 21.7 mEq) in the cerebral complication group comparing to the noncerebral group (mean = 25.1 mEq) at p <.05 (Figure 7).

Prebypass sodium. Sodium was lower in the group of cerebral complications than the non-complication group (125.4 mEq:130.0 mEq) at p <.05.

At two hours after bypass, the mean sodium was 137.3 mEq and 135.1 mEq in the cerebral and noncerebral respectively. Though the level was normal in both groups, it was significant at p <.05 (Figure 8).

Potassium obtained at the end of operation was lower in the cerebral group (mean = 3.0 mEq) than the noncerebral group (mean = 3.7 mEq) at significant level p <.05 (Figure 9).

The mean body temperature during perfusion and in the early hour of intensive care were 19.7°C and 34.9°C in the cerebral complication group comparing

Table 1
Clinical Data of Patients with Factors Not Influencing Cerebral Complication

	with cerebral comp				without cerebral comp				P value
	X	SD	min	max	X	SD	min	max	
Bypass time (min)	105	38.20	44	167	115.78	72.76	16	387	NS
MAP during CPB (mmHg)	30.71	7.34	10	40	33.37	7.13	15	50	NS
Heparin dose (mg)	116.78	84.16	27	263	158.45	78.22	23	428	NS
Circulatory arrest (min)	77	16.97	65	89	64.8	46.37	14	126	NS
Types of Oxygenator	----- NS -----				----- NS -----				
Age (year)	15.61	14.72	7(mo)	49	25.18	15.69	9(mo)	55	<0.05
Weight (Kg)	26.30	17.31	6	50	36.72	14.04	7	64	<0.05
BSA (m ²)	.983	.44	.38	1.55	1.24	.325	.46	1.73	<0.05
Lowest temp during CPB (C°)	19.78	2.45	14	23	21.01	3.87	10	33	<0.05
Lowest temp in ICU (C°)	34.90	.027	34	35	35.07	.529	34.3	37.7	<0.05

Table 2
Twelve Patients Who Had Cerebral Complication

Patient No.	Age yr.	Sex	weight (kg)	Dx	Operation	Bypass time (min.)	Cerebral lesion	Result
1	9 mos	M	6	PS, PFO	Pulmonary valvotomy, annulus enlargement, closure of PFO	44	subdural hematoma	alive
2	29	F	46	AI	Aortic valve replacement	59	muscular weakness	alive
3	1	F	10	Fallot	Corrective surgery	92	subdural hemorrhage	alive
4	9	M	19	Fallot	Corrective surgery	167	subdural hematoma	alive
5	35	F	44	MS,MR,TR	Mitral valve replacement, Tricuspid-valve replacement	145	cerebral embolism	alive
6	4	F	15	Fallot	Corrective surgery	122	subdural hematoma	dead
7	1	M	8	TGA	Senning	124	subarachnoid hemorrhage	dead
8	7 mos	F	6	Cor-Triatriatum	Corrective surgery	115	subdural hematoma	dead
9	14	M	25	Fallot	Corrective surgery	130	convulsion	alive
10	10	F	22	PS	Pulmonary valvotomy Infundibulectomy	63	convulsion	alive
11	49	M	40	MS, clot	Mitral valve replacement, removal clot in LA	134	multiple infarction	dead
12	19	M	50	Ebstein's anomaly	Tricuspid valve replacement, closure of PFO	128	cerebral ischemia	dead

PS = Pulmonary Stenosis
PFO = Patent Foramen Ovale
MR = Mitral Regurgitation
AI = Aortic Insufficiency
MS = Mitral Stenosis

TR = Tricuspid Regurgitation
TGA = Transposition of great artery
PS = Pulmonary stenosis
MS = Mitral stenosis

to the noncerebral group which had the mean temperature of 21.5°C and 35.1°C at the significant level $p < .05$.

There was statistical significance at $p < .05$ for the groups having partial hemodilution or total hemodilution. Using partial hemodilution, there was about 23% of cerebral complication comparing to 6% of incidence when total hemodilution was employed. How-

ever, this might be due to the fact that the group which had partial hemodilution for priming perfusion was a cyanotic group of small children younger than 10 years old. The relation of cerebral complication and type of heart disease is shown in Table 4.

In our unit, any patient who bleeds 2 ml./Kg./hour in the postoperative period is considered to have abnormal bleeding. Among 12 patients who had cer-

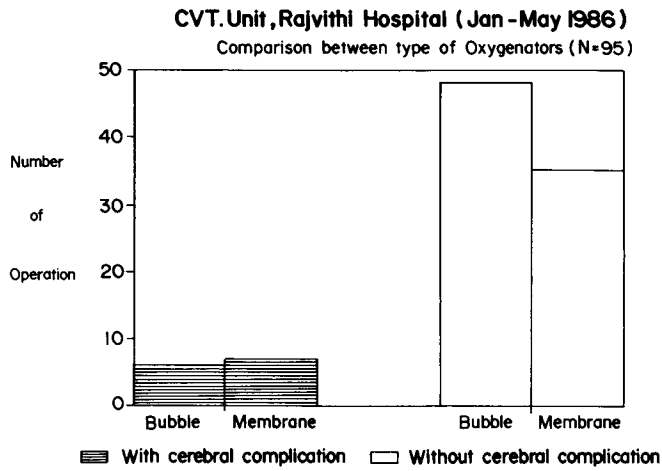


Figure 2

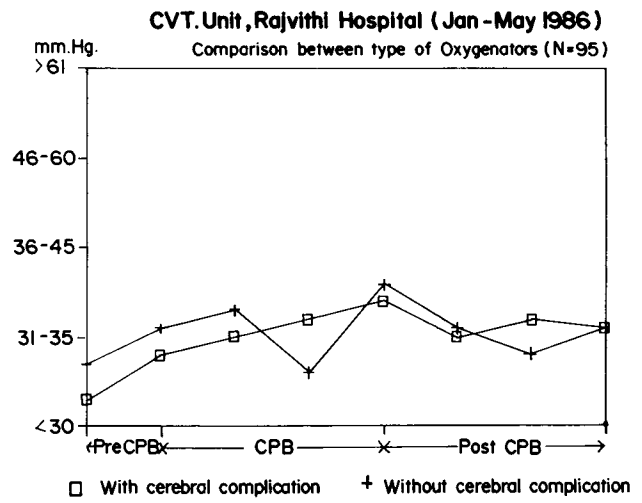


Figure 5

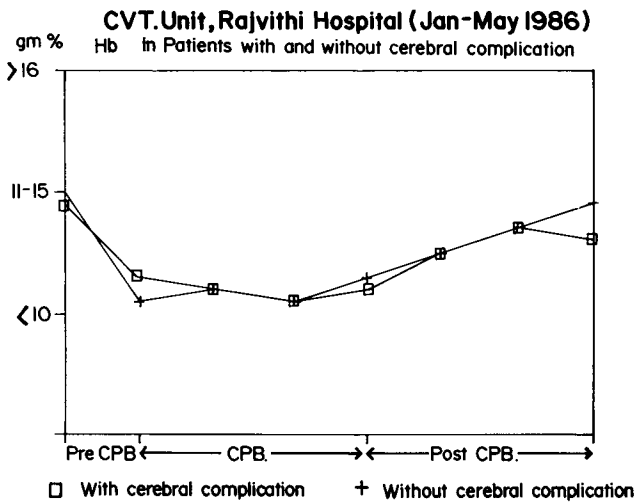


Figure 3

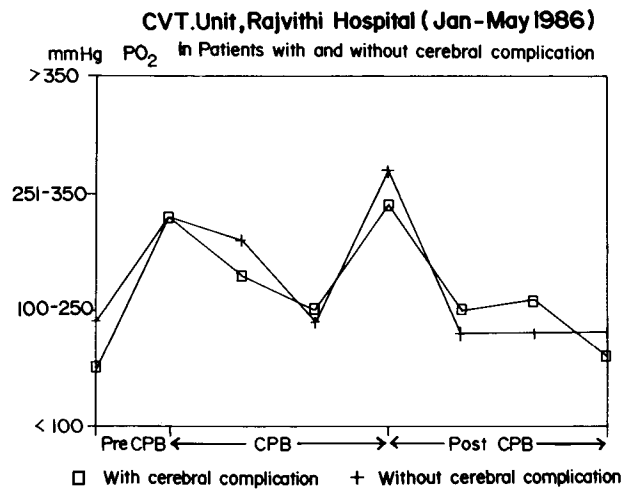


Figure 6

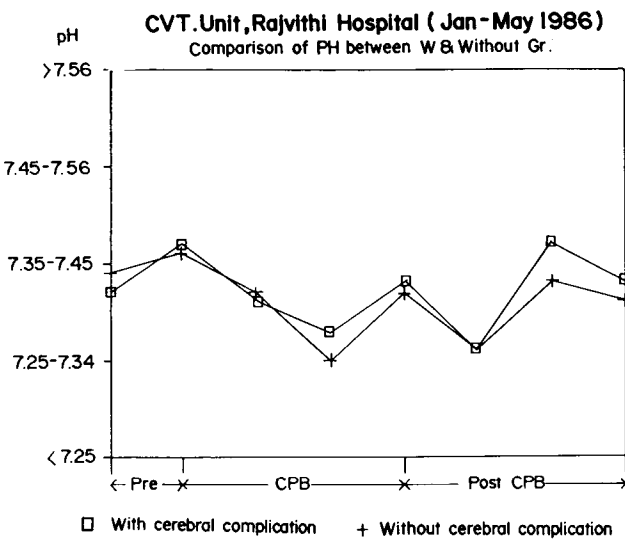


Figure 4

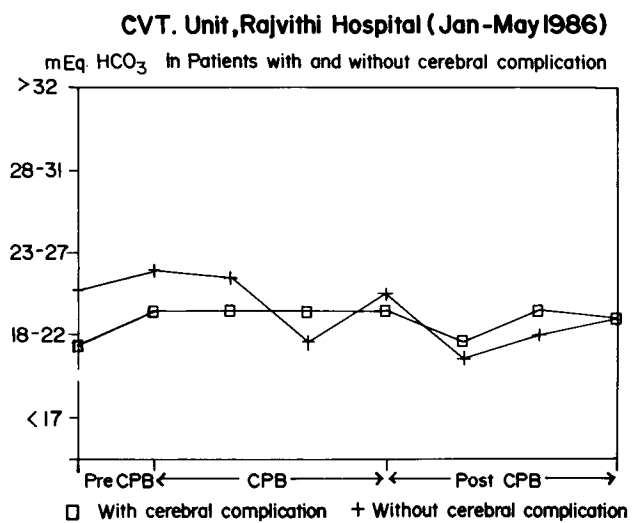


Figure 7

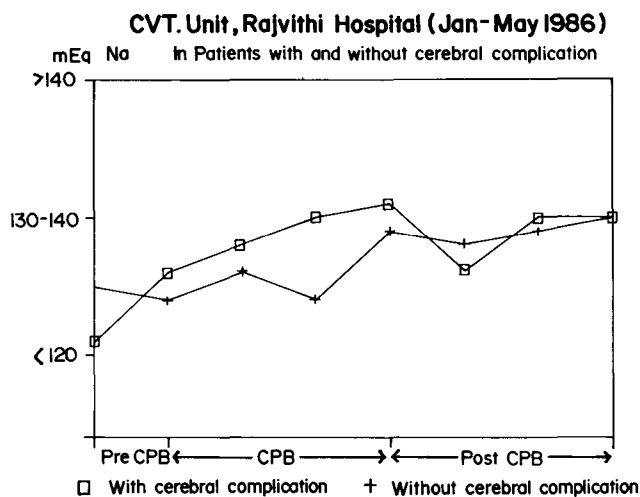


Figure 8

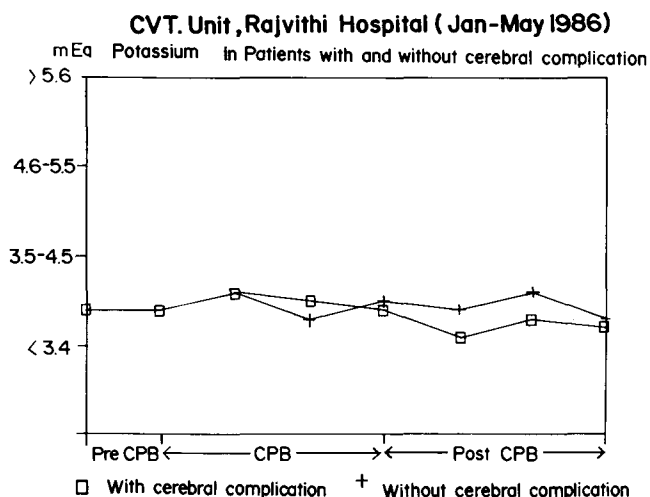


Figure 9

bral complication, there were 8 children of which 6 had abnormal bleeding after operation. However, there were two patients who had abnormal bleeding among adults (Table 3).

Discussion

In this series, the cerebral complication was highest among children younger than 10 years of age who had cyanotic congenital heart disease (Figure 10). This was also noted by others. In our patients there was low pO_2 before bypass, indicating some degree of anoxia which was ordinary in cyanotic heart disease. There was evidence from some literature that in acute stages there was decreased oxygen delivery to the brain due to decreased regional flow. This condition was aggravated by metabolic acidosis that occurred in the beginning of bypass in our patients who developed this complication. We suspected that the sodium which

was significantly low in the prebypass period among the complication group and abnormally rose to a higher level than the no complication group might be the cause of cerebral edema. However, this assumption needs further study. In three patients who had acquired valvular heart disease and had cerebral emboli, we could not rule out the possibility of emboli due to clot or other particulate materials. Age, weight and basal surface area had effects upon the development of cerebral complication in this series. Abnormal bleeding in cyanotic heart disease is well known and intracerebral bleeding due to disturbances of clotting and thrombosis could be the causative factors in our patients.

Our results suggest that cerebral complication occurs more commonly with cyanotic congenital heart disease in small children. Age, weight and basal surface area are factors affecting this complication. Abnormal bleeding plays a significant role as the causative factor in cerebral complications in cyanotic heart disease in children. Decreased cerebral blood flow in the acute anoxic stage causes decreased oxygen delivery to the brain. An abnormal shift of sodium during perfusion could be one of the causes of cerebral edema, though this assumption needs further study. There is no relationship between types of oxygenators used for operation and incidence of cerebral complication. In our patients, blood pH, pCO_2 , Hb, mean arterial pressure and hemodilution are not factors responsible for cerebral complication. Cerebral complication could be

Table 3

Bleeding Tendency among 12 Patients Who Had Cerebral Complication

	Children	Adult	Total
	<10 years		
	N	N	
abnormal bleeding	6	2	8
no abnormal bleeding	2	2	4
Total	8	4	12

Table 4

Relation of Cerebral Complication and Type of Heart Disease

	with cerebral comp		without cerebral comp	
	N	%	N	%
Cyanotic	6	50	11	13
Non-Cyanotic	3	25	20	24
Acquired	3	25	52	63

lessened by improvement of cerebral blood flow. Prevention of anoxia in any period, prevention of sodium imbalance and, correction of abnormal coagulation could prevent cerebral complication.

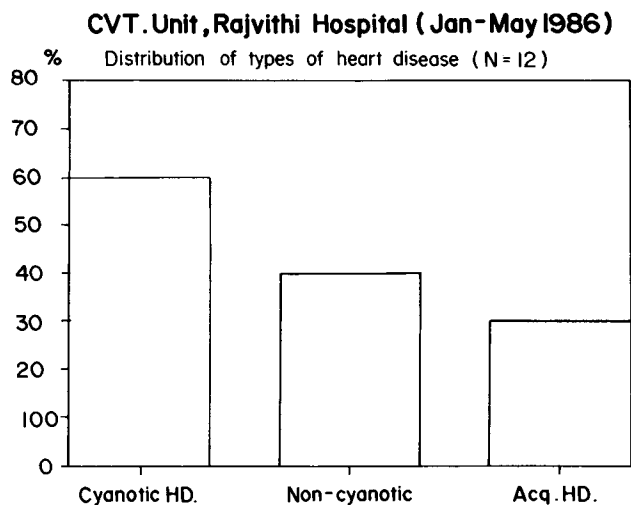


Figure 10

Conclusion

Our results suggest that cerebral complication occurs more commonly among small children with cyanotic congenital heart disease than in other groups. Age, weight and basal surface area are factors affecting this complication. Abnormal bleeding plays a significant role as the causative factor in cerebral complication in cyanotic heart disease in children. Decreased cerebral blood flow in acute anoxic stage causes decreased

oxygen delivery to brain. An abnormal shift of sodium during perfusion could be one of the causes of cerebral edema, though this assumption needs further study. There is no relationship between types of oxygenators used for operation and incidence of cerebral complication. In our patients, blood pH, pCO₂, Hb, mean arterial pressure and hemodilution are not factors responsible for cerebral complication.

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Questions from the Audience

Question: Aaron Hill, Falls Church, VA: An interesting presentation. Regarding the cerebral complication, what was the determining factor on whether or not someone had a cerebral complication following open heart surgery?

Response: They had a cerebral convulsion.

Question: Did you do any pre- or postoperative electroencephalogram?

Response: We have not.

Question: Did you do any testing pre and post for primitive reflexes?

Response: No.

Question: It would appear from your data that it was a significant difference on size. As the patient was smaller in size, the patient also had increased complications. Is that true?

Response: Yes.

Question: You were concerned with sodium. If you're concerned with cerebral edema, did you use Mannitol during these procedures?

Response: Yes.

Question: You didn't feel that it cut back your chances of having cerebral edema? Did you decrease the chances of cerebral edema with the use of Mannitol?

Response: Yes.

Question: Don't you think that might have counteracted the cerebral edema that you thought might be a problem? What I guess I'm asking is: Did you measure the serum oncotic pressure or the osmolality to see if that was a problem?

Response: I'm not sure.

Question: Frank Hurley, Chicago, IL: I'd like to thank you for your presentation. My question pertains to your patients with cyanotic heart disease. Could you explain what your technique was relevant to hemodilution on bypass—especially in regard to patients with cyanotic heart disease? What level of hemodilution do you have in your oxygenator? Your pump prime?

Response: If the hemoglobin is more than 15, we do not use blood.

Question: So it would be fair to say that you maintain your hemoglobin at 15 grams in children?

Response: Yes.

Question: David DeForest, Cincinnati, OH: We found that in children, especially under two years of age, that high levels of sugar, over 200 milligrams, would produce convulsions. And we're very careful in our prime not to include sugar. Also, the anesthesia people are alerted to curtail drips with sugar. So we do that simple test for sugar on a strip when we first go on bypass. And we found that children will convulse. Especially when they are rewarmed, their metabolism does not turn on and you would have to give insulin to curtail this convulsion process. So I might suggest that you run a sugar test. This would cause cerebral edema.