서 울 의 대 잡 지 제 17권 제 3 호
The Seoul Journal of Medicine
Vol. 17, No. 3, September, 1976

Clinical and Metabolic Effects of Cardiopulmonary Bypass*

Chong Whan Kim, M.D.

Department of Thoracic Surgery, College of Medicine,

Seoul National University

INTRODUCTION

Intracardiac anatomic correction of the cardiac malformations and other pathology utilizing temporary extracorporeal cardiopulmonary support is as yet the inherently complicated modality of surgical treatment, and it accompanies more or less untoward alterations of function in a patient. And, much attention and refinements have been contributed to the study of cardiopulmonary support with total body perfusion. Blood trauma and protein denaturation, hemodynamic and metabolic alterations, and damage on the specific organs are the complications of major importance. Correlations have been made with length of perfusion, hypothermia, perfusion rates and the types of priming fluid in the oxygenator. And many other factors were studied and the great efforts were put into finding means of conducting in such a way not to cause damage which should not have occurred to begin with, that is, the physiological perfusion.

We have studied these effects of extracorporeal circulation where sigmamotor pump and Rygg-Kyvsgaard oxygenator had been used (Kim et al., 1969; Lee et al., 1973; Kim et al., 1974). Since August, 1974, we have been running the 5-headed roller pump (American Optical Co., deLuxe model), and fifty-three patients underwent open heart surgery during the whole year of 1975. This report studied our whole procedures with cardiopulmonary bypass to review the effects of extracorporeal circulation to the patients by collecting routine laboratory examinations and by looking through the clinical results. All the more, one of the main purposes of this study is to try to establish our own baselines in conducting extracorporeal circulation.

CLINICAL MATERIALS AND METHODS

The consecutive fifty-three patients underwent open heart surgery (OHS) at Seoul National University Hospital in the year of 1975. The patients were classified into four groups for the study: Group I, 9 patients with atrial septal defect (ASD); Group II, 11 patients with ventricular septal defect (VSD); Group III, 19 patients with cyanotic heart disease (CHD), consisted of 17 patients with tetralogy of Fallot (TOF) and 2 patients with double outlet right ventricle (DORV); and Group IV, 14 patients

* Presented at 8th annual meeting of Korean Thoracic and Cardiovascular Surgical Society on May 22, 1976.

Supported partly by the Grant No. 73-300-14 from the China Medical Board of New York, Inc.

Supported partly by the Clinical Research Fund from Seoul National University Hospital.

<Received for publication on August 20, 1976>

with acquired valvular heart disease (AHD). Their ages, sexes, body weights and surface areas were shown in Table 1.

The circuit for extracorporeal circulation (ECC) was established with aortic cannulation for the return of the arterialized blood into patients and venous drainage through the two tubes into the superior and inferior venae cavae after midline sternotomy. In 40 cases the Bentley oxygenator was used, and the Harvey or Polystan bubblers were utilized for the remaining patients. The compositions of prime and the conduct of cardiopulmonary bypass (CPB) were summarized in Table 2. Generally, we prime the oxygenator with relatively low volume, but enough to fulfill the recirculation. According to the hemodilution principle, the heparinized fresh ACD blood (Foote et al., 1961) was diluted with Hartmann and 15 percent mannitol solutions. Potassium (one mEq./Kg. of body weight) and sodium bicarboate (one mEq./Kg. of body weight plus 12mEq./400 ml. of blood prime) were added into the prime. Epsilon aminocaproic acid (125 mg./Kg. of body weight) was administered in all patients by intravenous route before starting CPB, and dexamethasone (one mg./Kg. of body weight) in cases where the CPB might be prolonged. Anticoagulation was done with 3 mg. of heparin/Kg. of body weight.

Blood flow rates were regulated to keep ones within the ranges from 2 to 2.5 L./Min./M^2. of body surface area, and oxygen flows passed into the oxygenator were adjusted in the ranges between 1.5 and 3 times of blood flow rates. No carbon dioxide gas was mixed. Throughout the procedure, mean arterial pressure, central venous pressure and electrocardiogram were continuously monitored. Body temperature was measured through the electrodes placed in both the esophagus and the rectum. And, urinary output was collected through the Foley catheter. Mean arterial pressure was tried to keep higher than the level of 70 mm. Hg, and central venous pressure to maintain the level high enough for the adequate venous return into the oxygenator. And, mild to moderate degrees of hypothermia were employed with the cases where the prolonged CPB had been anticipated. The length of CPB time ranged from the shortest of 21 minutes with the repair of ASD to the longest of 236 minutes with the operations in Groups III and IV.

All defects of ASD were repaired primarily except a case where the partial anomalous pulmonary venous return had been associated and the ASD had been closed in a fashion to drain the pulmonary venous blood into the left atrium with a Dacron patch. Of the patients in Group II, 4 had Type I defect, 5 had Type II and 2 had Type III. Seven patients revealed mild to moderate elevations of systolic pulmonary arterial pressure below 70 mm. Hg in the preoperative catheterisation studies, while the remaining 4 were normal in pressure. A patient had a ruptured coronary sinus of Valsalva into the right ventricle through his VSD. Four defects were closed primarily and 7 with Teflon patches. All 17 patients with TOF had intracardiac total correction and 11 hearts received right ventricular outflow tract gusset patches. For the 2 patients with DORV, the interventricular correction was performed using intraventricular prosthetic conduits. Six patients of Group IV were in NYHA Class III and 8 were in Class IV. Nine patients had mitral valve replacement where one had tricuspid annuloplasty and another had repairs of the associated ASD and VSD; 2 patients had aortic valve replacement where one had open mitral commissurotomy; one patient had both mitral and aortic valve replacement and tricuspid

annuloplasty; 2 patients had open mitral commissurotomy only.

The laboratory examinations were performed periodically with appropriate intervals. Routine hematological studies included hemoglobin, hematocrit, white blood cell and platelet counts. Serum electrolytes were measured for sodium, potassium and chloride. And, among the heavy metals, total calcium and magnesium were also measured with Hitachi 203 atomic absorption spectrophotometer. Blood pH and gases were studied for the arterial blood samples using Instrumentation Laboratories model 113 or 213 blood gas analyzer. Serum enzymes were measured for glutamic oxaloacetic transaminase (SGOT), glutamic pyruvic transaminase (SGPT) and lactic dehydrogenase (SLDH). Postoperative fluid and blood balances were also studied along with the urine specific gravity and urinary excretion of electrolytes. The clinical results were evaluated for complications and mortality.

Table 1. Age, sex and body size.

Group	I	II	III	IV	Total
Number of Patients	9	11	19	14	53
Male: Female	5 : 4	5 : 6	11 : 8	10 : 4	31 : 22
Age(yrs.)	15.44±9.35	11.00±3.31	11.16±6.21	31.79±11.93	17.30
Range	4−35	3−26	3−31	18−49	3−49
Body Weight(kg.)	32.39±14.97	23.50±18.46	26.22±10.27	49.87±10.49	34.14
Range	11−58	10−66	12.5−52.5	27−64.8	10−66
Body Surface Area(M^2)	1.109±0.374	0.991±0.420	0.977±0.250	1.495±0.187	1.142
Range	0.52−1.64	0.51−1.78	0.60−1.56	1.04−1.75	0.51−1.78

Mean±SD.

Table 2. Cardiopulmonary bypass.

Group	I	II	III	IV
Priming, Total(ml.)	1370.0±248.3	1376.4±396.3	1449.4±249.3	1836.4±204.0
Whole Blood(ml.)	800.0± 0.0	836.4±205.7	866.7±149.1	1017.9±196.0
Hartmann Solution(ml.)	333.3± 99.4	300.0±153.7	363.9±105.2	478.6±108.1
15% Mannitol(ml.)	138.6± 56.7	133.6± 46.2	105.6± 28.3	192.9± 17.5
Potassium chloride(mEp.)	26.6± 5.5	23.6± 7.2	23.7± 6.0	35.2± 6.3
Sodium bicarbonate(mEq.)	54.7± 16.1	54.5± 14.8	56.3± 12.1	78.0± 9.9
Blood Flow Rate(L/Min/M^2)	1.98±0.28∼ 2.54±0.20	1.84±0.28∼ 2.67±0.21	1.66±0.22∼ 2.44±0.23	1.62±0.27∼ 2.46±0.17
Oxygen Flow Rate (Times of Blood Flow Rate, L/Min/M^2)	2.17±0.42∼ 2.61±0.34	2.24±0.16∼ 3.03±0.90	2.12±0.31∼ 2.62±0.54	2.10±0.35∼ 2.49±0.50
Lowest Temperature(°C)				
Esophagus	31.23±2.91	25.50±1.57	24.78±2.68	27.17±1.80
Rectum	32.99±2.12	28.80±1.44	28.64±2.01	29.30±1.30
Mean Arterial Pressure (mm Hg.)	63.9±8.4∼ 82.2±14.2	46.8±12.3∼ 85.5±19.8	49.4±13.6∼ 80.4±11.5	55.7±10.8∼ 96.4±14.1
Central venous Pressure (cm H_2O)	10.2±6.1∼ 19.3±6.1	6.5±5.0∼ 15.5±4.6	7.3±4.2∼ 17.9±4.9	8.7±5.1∼ 20.3±4.0
Total Duration of Bypass(Min.)	37.1±17.3	71.5±26.1	121.4±47.3	148.5±48.9
Range	21−74	24−110	63−236	52−236

Mean±SD.

RESULTS

Hemoglobin and Hematocrit

The levels of hemoglobin and hematocrit during anesthesia and before starting CPB were much the like of the control levels on admission. As soon as CPB was started, these values revealed abrupt and highly significant decreases ($P<0.001$ vs. admission control) reaching to the levels of about 25 percent of hematocrit from hemodilution, but the mean hematocrit value was 32.59 percent in Group III because of their polycythemia. These low levels lasted unchanged during the whole period of CPB. Although the values revealed some increases at 3 hours off bypass and thereafter, the mean hemoglobin and hematocrit were yet low and many patients had mild degrees of anemia even at the time of dismissal from hospital.

Table 3. Hemoglobin and Hematocrit.

Group	Hemoglobin (Gm. %)			
	I	II	III	IV
Admission	12.80±1.78	11.67±1.27	17.56±3.62	13.3 ±2.15
Anesthesia	12.23±1.81	10.75±1.61	16.09±3.21	12.57±2.64
Bypass				
30 Min.	8.50±1.25*	7.58±1.16*	10.77±2.35*	9.06±1.16*
60 Min.		8.33±1.19*	10.76±2.32*	8.90±0.94*
90 Min.			10.63±3.16*	9.57±1.51*
120 Min.				9.45±1.35*
Off Bypass				
3 Hrs.	10.62±1.56'	9.57±1.06*	10.80±2.01*	10.44±1.61*
1 Day	10.04±1.28*	9.43±0.76*	11.32±2.13*	10.66±1.16*
2 Days				10.18±1.07*
5-7 Days			11.38±1.11*	10.38±0.73*
Discharge	10.20±0.95*	10.38±0.87'	11.10±1.50*	10.17±1.16*

Group	Hematocrit(%)			
	I	II	III	IV
Admission	38.33±4.78	34.73±3.44	54.28±13.07	40.79±6.17
Anesthesia	37.44±5.34	33.30±4.31	49.76±11.39	39.21±7.86
On Bypass				
30 Min.	27.13±3.37*	23.25±2.63*	32.59±7.96*	27.90±3.33*
60 Min.		25.57±3.25*	32.50±7.05*	26.80±1.94*
90 Min.			32.43±9.04*	29.43±3.96*
120 Min.				28.67±4.31*
Off Bypass				
3 Hrs.	33.56±4.40'	28.60±3.98*	33.27±5.74*	31.92±5.02*
1 Day	31.44±3.69*	29.55±2.23*	35.00±6.31*	32.83±4.37*
2 Days				31.73±3.67*
5-7 Days			35.08±3.40*	32.10±1.81*
Discharge	31.88±2.32*	33.00±2.66	34.69±4.97*	31.09±4.03*

Mean±SD: *$P<0.001$, '$P<0.01$ vs. Admission.

Additional primings were necessary in many patients to keep the adequate blood levels during CPB. Whole blood was added in an average amount of 780 ml. to 10 cases of Group IV, 697 ml. to 9 of Group III, 342 ml. to 6 of Group II, and 150 ml. to a single case of Group I, respectively. The amount of Hartmann solution added into the oxygenators averaged from 388 to 455 ml. in 21 patients of Groups II, III and IV. The hemoglobin and hematocrit values of the fresh ACD blood from blood bank varied with wide ranges: hemoglobin from 5.4 to 13.5 gm./dl. (average of 10.2 gm./dl.) and hematocrit from 20 to 48 percent (average of 34.4 percent) with the measurements for 27 individual units of blood. And, with the samples from 21 oxygenators after recirculation and before starting CPB, hemoglobin levels varied between 1.9 and 8.3 gm./dl. (average of 4.7 gm./dl.) and hematocrit levels between 6 and 20.5 percent (average of 12.4 percent).

Plasma Hemoglobin

The mean levels of plasma hemoglobin measured during anesthesia and before CPB ranged between 26 and 73 mg./dl. Increases of mean plasma hemoglobin were noted during CPB, especialy with the prolongation of bypass and in Group III ($P<0.01$ vs. anesthesia control). However, the values seen in each patient varied so widely that no significance could be elicited statistically. After completion of CPB and thereafter, plasma hemoglobin was cleared off rapidly from blood to return to or below prebypass levels.

White Blood Cell and Platelet Counts

There were no significant changes in number of white blood cells during the period of CPB from control counts at admission, although some increases were noted when CPB has been prolonged. However, the mean white blood cell counts revealed highly significant increases ($P<0.001$ vs. admission cotrol) from as early as patients were off CPB and at 3 hours off CPB. And, these patterns of leukocytosis lasted for a day or two in the early postoperative periods. On the other hand, the changes in platelet counts were characteristic. They dropped to 48 percent or less of the admission control in mean values at 30 minutes on CPB, and it was lowest, 21 percent of control in Group III at 90 minutes on bypass. Until postoperative

Table 4. Plasma hemoglobin (mg.%).

Group	I	II	III	IV
Admission	42.17±26.20	37.57±17.09	75.79±52.77	64.38±33.70
Anesthesia	32.63±16.10	26.68±17.32	50.32±37.05	73.32±56.22
On Bypass				
30 Min.	50.51±43.65	45.60±28.97	59.92±33.46	65.04±46.82
60 Min.		44.90±12.13	88.71±62.97	59.37±57.27
90 Min.		49.20±10.08*	89.65±33.46*	
120 Min.			168.00±88.69*	90.41±59.48
Off Bypass				
3 Hrs.	52.76±41.18	24.62±12.79	99.45±60.60	81.56±60.22
1 Day	19.06± 7.24	28.49±22.59	33.01±24.64	55.88±30.60
7 Days				39.66±18.78
Discharge	22.00±15.00	38.08±24.86	29.80± 3.80	35.55± 9.46

*$P<0.01$ vs. Anesthesia; Mean±SD.

day 2 they remained low in the ranges between 52 and 76 percent of the control. The platelet counts increased upto 159 percent of the control sometime after postoperative days 5 and 7.

Serum Electrolytes

During anesthesia and before commencing CPB, the levels of serum sodium were generally lower than the values of the admission controls, and the decreases were significant ($P<0.01$ vs. admission control) in Groups Ⅱ, Ⅲ and Ⅳ. These low levels lasted unchanged throughout the CPB. In the early postoperative days, these levels showed upward trend until discharge from hospital when they returned to normal or the mean admission control values. Serum potassium levels were also low during anesthesia,

Table 5. White blood cell and platelet counts.

Group	White Blood Cells(/mm³)			
	Ⅰ	Ⅱ	Ⅲ	Ⅳ
Admission	7622±2484	6573±1913	9544±3903	8821±3840
Anesthesia	10763±4124	9389±2728'	14340±3129*	10992±3703
On Bypass				
30 Min.	7286±2407	6600±1776	7960±3579	6420±2189
60 Min.		6560±1443	10027±4155	8625±3360
90 Min.		6833±1579	9650±3675	8160±3854
120 Min.			12750±6457	10500±4616
Off Bypass				
3 Hrs.	20333±4494*	17830±6178*	22169±6426*	19183±6803*
1 Day	13175±4596'	13255±4227*	15773±5604*	14492±3419*
2 Days	10360±2838	13725±2314*		12473±3499'
5-7 Days			8830±1961	12220±3694'
10-14 Days				12720±1975'
Discharge	6500±2143	6891±2636	7369±2560	7173±2541

Group	Platelets (X100/mm³)			
	Ⅰ	Ⅱ	Ⅲ	Ⅳ
Admission	2077±396	2225±702	2491±1432	2077±585
Anesthesia	2227±478	1857±449	2400±1051	2040±595
On Bypass				
30 Min.	988±357*	876±247*	1027±297*	940±431*
60 Min.		660±126*	675±228*	987±223*
90 Min.		480±241*	513±197*	1103±610
120 Min.			600±260*	972±460*
Off Bypass				
3 Hrs	1580±465	1417±408'	1300±818'	1380±364'
1 Day	1500±482	1471±408'	1278±366*	1070±354*
2 Days				1210±513'
5-7 Days	3300±1123		2033±8818	1687±1027
10-14 Days				3276±1366
Discharge	3130±1101	2568±1069	2382±739	2048±880

Mean±SD; *$P<0.001$, '$P<0.01$ vs. Admission.

Table 6. Serum electrolytes (sodium, potassium and chloride; mEq/L.).

Group	Sodium			
	I	II	III	IV
Admission	134.2±4.6	134.9±3.3	132.9±3.6	132.1±3.0
Anesthesia	128.0±7.6	126.8±4.8*	126.4±8.1′	122.8±4.9*
On Bypass				
30 Min.	125.4±3.5*	120.4±9.5*	126.2±8.0*	121.4±9.6*
60 Min.		124.8±6.8′	126.5±5.5*	120.7±7.1*
90 Min.		121.3±8.7′	121.3±9.2*	
120 Min.			126.7±5.0	125.3±5.9*
Off Bypass				
3 IIrs.	129.9±2.3	130.3±3.7*	128.1±7.2	126.8±6.0′
1 Day	127.0±4.1*	128.0±6.3*	129.1±5.4	129.1±4.6
5-7 Days		127.5±4.4*	129.8±3.7	128.7±4.8
Discharge	132.0±5.0	131.8±3.2′	134.3±2.6	130.7±4.1
	Chloride			
	98.9±3.7	99.5±2.7	97.8±2.9	97.3±2.0
	94.8±4.8	94.6±4.7′	94.1±5.6	90.8±4.7*
	93.0±2.4*	89.3±7.1*	93.4±5.0*	92.0±5.6′
		93.0±3.2*	94.1±3.3′	90.1±5.2′
		91.0±4.2*	90.4±5.5*	
			95.0±2.2	92.4±4.5*
	96.9±2.3	96.1±2.9′	94.8±3.7′	94.3±4.2
	95.0±3.1	95.5±4.7	96.6±94.3	96.6±3.4
		96.8±2.9	96.4±2.5	95.5±3.4
	97.0±4.7	96.9±2.6	98.7±2.9	96.8±2.7

Group	Potassium			
	I	II	III	IV
Admission	4.11±0.24	4.25±0.30	4.37±0.56	4.21±0.52
Anesthesia	3.46±0.67′	3.40±0.48*	3.91±0.71	4.03±0.52
On Bypass				
30 Min.	3.70±0.86′	3.40±0.48*	4.18±0.91	3.64±0.77
60 Min.		3.78±0.75	3.47±0.68*	3.47±0.55′
90 Min.		3.20±0.79	3.40±0.67*	
120 Min.			3.90±0.51	3.41±0.74*
Off Bypass				
3 Hrs.	3.61±0.83*	3.70±0.52′	3.68±0.55*	3.58±0.80
1 Day	4.43±0.59	3.99±0.72	4.27±0.48	4.71±0.62
5-7 Days		4.05±0.21	4.10±0.33	3.98±0.46
Discharge	4.25±0.54	4.21±0.43	4.25±0.24	4.36±0.42

Mean±SD; *P<0.001, 'P<0.01 vs. Admission.

and the mean values of potassium were less than the level of 4 mEq./L. during CPB and at 3 hours off bypass in all groups. However, these were corrected with exogenous supplement

of potassium chloride on the values at day 1 and thereafter. The changes of serum chloride levels were similar to the ones seen with serum sodium, being decreased during anesthesia and CPB and showing upward trend in the early postoperative days. Exogenous potassium was added to the oxygenator in 6 patients during CPB, and the average amounts ranged between 12 and 27 mEq. The values of sodium and potassium measured for the 15 samples from oxygenator after recirculation and before CPB were 153 mEq./L. of the average and higher than the levels of 10 mEq./L., respectively.

Heavy metals were measured for total calcium and magnesium. The mean values of total calcium were in the normal range on admission, and they were significantly lowered ($P<0.01$) during anesthesia in Groups Ⅰ and Ⅳ where the admission controls were lower than the levels in other groups. During CPB, these levels increased in all groups, especially at 30 minutes on CPB. However, these increases were not always significant in all groups. With the samples at 3 hours after CPB, the mean

Table 7. Serum electrolytes (total calcium and magnesium; mEq/L.).

Group	Total Calcium			
	Ⅰ	Ⅱ	Ⅲ	Ⅳ
Admission	5.49±0.59	5.15±0.25	4.84±0.35	4.64±0.42
Anesthesia	5.03±0.74	4.95±0.48	4.41±0.56′	4.17±0.39*
On Bypass				
30 Min.	6.05±0.42′	5.76±1.12	5.20±0.94	4.74±0.46
60 Min.			4.43±0.31*	4.88±0.56
90 Min.		5.42±0.70		
120 Min.				4.91±0.63
Off Bypass				
3 Hrs.	5.18±0.39	4.89±0.38	4.82±0.78	4.57±0.43
1 Day	4.73±0.72′	4.64±0.31*	4.35±0.46*	4.74±0.21
5-7 Days			4.35±0.54	4.64±0.39
Discharge	5.16±0.23	4.56±0.39*	4.93±0.38	5.13±0.34′

Group	Magnesium			
	Ⅰ	Ⅱ	Ⅲ	Ⅳ
Admission	1.211±0.188	1.439±0.109	1.415±0.250	1.397±0.153
Anesthesia	1.334±0.174	1.291±0.169	1.521±0.192	1.381±0.064
On Bypass				
30 Min.	1.244±0.099	1.344±0.143	1.295±0.225	1.240±0.120′
60 Min.			1.439±0.183	1.263±0.159
90 Min.		1.203±0.127*		
120 Min.				1.194±0.329
Off Bypass				
3 Hrs.	1.446±0.178′	1.366±0.183	1.372±0.228	1.234±0.333
1 Day	1.510±0.230*	1.415±0.238	1.344±0.362	1.289±0.124
5-7 Days			1.443±0.178	1.503±0.183
Discharge	1.483±0.130*	1.583±0.327	1.310±0.094	

Mean±SD; *$P<0.01$, ′$P<0.05$ vs. Admission.

levels of total calcium decreased below the control values. And, at day 1, the decreases were significant ($P<0.01$) in all groups except in Group Ⅳ. The calcium levels in the oxygenators varied from 5.4 to 13.5 mEq./L. with the average of 8.88 mEq./L. from 6 oxygenators before CPB and after recirculation. The mean levels of serum magnesium during CPB were lower than the values of the mean admission control in all groups at the most parts of time. Although there were upward tendency in the postoperative days, the variations in individual cases were so wide that these changes were not significant statistically. The samples from 5 oxygenators after recirculation revealed the average of 0.684 mEq./L. of magnesium with the range from 0.5 to 1.1 mEq./L.

Serum Enzymes

Of the transaminases, SGOT showed characteristic changes. The mean levels at admission were in the normal range in all but Group Ⅳ where the mean value was a little higher with wide variations. There were highly significant rises ($P<0.001$ vs. admission control) of SGOT at day 1. The mean value was elevated also in Group Ⅳ, though it was not significant statistically because of wide variations among patients. Although the mean SGOT levels decreased at days 5 and 7 and thereafter, they were still higher than the normal range.

On the other hand, the changes of SGPT were slow and mild as compared with the ones of SGOT. There were significant rises ($P<0.01$ vs. admission control) of the mean levels of SGPT in Groups Ⅲ and Ⅳ at day 1, but no significant rises in Groups Ⅰ and Ⅱ. The mean SGPT remained high, though not sign-

Table 8. Serum enzymes(glutamic oxalacetic transaminase, glutamic pyruvic transaminase and lactic dehydrogenase; units/ml.).

Group	Serum Glutamic Oxalacetic Transaminase			
	Ⅰ	Ⅱ	Ⅲ	Ⅳ
Admission	32.2±11.5	27.9± 9.9	32.4± 9.4	57.4±58.0
Day 1	153.7±55.7*	161.3±75.3*	183.0±87.8*	170.7±39.9
Day 5-7	53.0±24.0	50.6±14.3*	57.8±20.9*	71.0±19.1
Discharge	42.9±18.2	49.8±19.1	50.0±53.6	44.6±16.9
	Serum Glutamic Pyruvic Transaminase			
	22.8± 9.7	19.3± 6.6	22.2±15.5	32.6±26.3
	39.5±21.0	34.5±22.9	46.4±18.7*	59.5±26.3′
	41.5± 1.5*	42.7±30.1	41.5±27.2	54.3±31.6
	26.9±11.5	39.0±34.3	33.8±24.4	23.0± 7.1
Group	Serum Lactic Dehydrogenase			
	Ⅰ	Ⅱ	Ⅲ	Ⅳ
Admission	388.6±308.6	565.5±182.9	743.1±376.3	697.3±340.8
Day 1	1238.6±437.9*	1306.1±498.4*	1559.5±349.0*	1662.2±407.6*
Day 5-7	1140.0± 0.0	1061.6±405.8′	1185.0±406.6′	1556.7±379.8*
Discharge	784.3±305.1	875.0±245.0	814.3±233.7	1386.3±307.6*

Mean±SD; *$P<0.001$, ′$P<0.01$ vs. Admission.

ificant, until days 5 and 7 before returning normal at discharge. For the convenience of calculation, the values reported to be higher than 197 units with SGOT and 98 units with SGPT were taken as high as those levels.

The changes of SLDH were also characteristic, being elevated sharply and uniformly in all groups at day 1 after surgery ($P<0.001$ vs. admission control). The mean levels remained elevated high above 1,000 units even at days 5 and 7 ($P<0.01$). With Group Ⅳ where most patients had prosthetic valve replacement, the mean SLDH level was 1386±307.6 units even at dismissal from hospital, though the mean levels in other groups were still a little high.

Blood Gases

Arterial blood pH was normal during anesthesia and before starting CPB except in Group Ⅲ where the mean pH indicated mild acidosis. However, the general features were the alkalosis during CPB, and the changes in pH toward alkalosis were highly significant ($P<0.001$ vs. anesthesia control) in Groups Ⅲ and Ⅳ throughout the CPB. Upon conclusion of CPB, pH remained within normal range until it revealed mild alkalosis in the morning of day 1. The partial pressure of carbon dioxide in arterial blood revealed a little low levels during anesthesia, and they were further significantly decreased ($P<0.01$ vs. anesthesia control) by CPB. Mean pCO_2 levels, sometimes, decreased as low as the one below 20 mm. Hg. After CPB, it increased to show mild degree of hypocapnia in the next morning of surgery.

The metabolic component was shown by the values of base excess or deficit. These levels were in the normal range after anesthesia and before CPB, but the patients in Group Ⅲ had moderate degree of base deficit. The changes of metabolic components varied without significant decreases in mean values in all groups at any time. And, it was the tendency toward being corrected from the anesthesia control in Group Ⅲ, especially at 90 minutes on CPB ($P<0.01$). Most of the patients with good hemodynamics showed mild degree of base excess in the next morning of operation, being significant in Groups Ⅲ and Ⅳ ($P<0.001$). However, the exogenous sodium bicarbonate administration was necessary in 43 patients during CPB and in the early postoperative hours: the average amounted 42 mEq. in 5 patients of Group Ⅰ, 47 mEq. in 10 of Group Ⅱ, 81 mEq. in 17 of Group Ⅲ, and as high as 156 mEq. in 11 of Group Ⅳ, respectively.

The mean levels of partial pressure of oxygen in arterial blood were satisfactorily high, above 150 mm. Hg, except 54.4±23.6 mm. Hg in Group Ⅲ during anesthesia. But, these increased mostly above 300 mm. Hg on CPB and they were high enough for the good oxygenation of blood after surgery while patients were on respirator.

Fluid and Blood Balances

Postoperative fluid was 5 percent dextrose in water. Fluid administration was limited to 500 ml./Day/M^2. on the day of operation, and it was increased gradually on the first postoperative day and thereafter with a little more allowance of oral fluid in Groups Ⅰ and Ⅲ. Accordingly, urine amount increased by degrees, and, generally, increased urinary output was the tendency toward mild degree of diuresis from day 2 or 3. This tendency was reflected in the urine specific gravity shown in Table 11, where ranges of specific gravity were narrowed and specific gravity themselves decreased day by day until diuresis ensued.

Blood loss through chest tubes ranged from 188 to 553 ml. of mean drainage until the next morning of surgery. Thereafter, the loss was

Table 9. Blood gas studies (pH; pCO_2, mmHg.;base excess, mEq/L.; pO_2, mmHg.).

Group	pH				pCO_2			
	I	II	III	IV	I	II	III	IV
Anesthesia	7.443±.062	7.430±.081	7.318±.115	7.409±.087	32.53± 5.46	29.33±6.93	36.32±11.40	32.88±7.76
On Bypass								
15 Min.	7.466±.096	7.471±.070			25.33± 5.48′	25.06±1.09′		
30 Min.	7.482±.081	7.473±.086	7.462±.083*	7.533±.092*	24.75± 3.75	21.15±4.27*	24.05± 7.69*	22.68±6.74*
60 Min.		7.520±.101	7.511±.096*	7.556±.112*		19.36±5.12*	21.28± 5.55*	23.33±4.59*
90 Min.		7.528±.063	7.543±.132*			17.63±6.44′	22.28± 7.51*	
120 Min.			7.589±.137*	7.560±.085*			18.26± 5.25*	23.16±7.78′
Off Bypass								
15 Min.			7.412±.052′	7.456±.119			29.87± 8.27	27.90±7.30
30 Min.	7.417±.092	7.323±.096	7.416±.067′		32.69±11.78	41.20±8.81′	31.98± 7.29	
1-2 Hrs.		7.358±.047	7.381±.108	7.398±.051		39.29±9.77	32.48± 7.08	30.50±6.02
3 Hrs.	7.413±.073	7.433±.099	7.369±.108	7.416±.065	31.93± 1.85	30.97±9.93	35.82±14.51	29.70±7.49
1 Day	7.489±.080		7.546±.067*	7.544±.025*	30.22± 6.60		32.73±10.43	31.89±5.29
	Base Excess				PO_2			
Anesthesia	−0.30±3.17	−3.04±4.69	−7.31±5.33	−3.85±4.32	194.0± 64.4	189.9± 50.9	54.4± 23.6	176.1± 62.1
On Bypass								
15 Min.	−1.05±4.18	−3.06±3.28						
30 Min.	−3.50±1.50	−6.11±6.80	−5.06±4.40	−2.53±4.06	164.5± 68.5	337.0± 71.7	368.0±131.8	323.3± 90.1
60 Min.		−4.61±5.82	−4.18±4.24	−1.00±5.72		321.6±161.7	293.2±184.3	319.0± 86.7
90 Min.		−7.83±4.19	−2.38±3.01′			205.0± 75.0	341.2±205.4	
120 Min.			−2.14±5.52	−1.04±3.22			346.8±136.1	157.8± 67.4
Off Bypass								
15 Min.			−3.69±3.92	−3.68±4.95			307.0±174.1	450.7± 21.0
30 Min.	−1.96±3.73	−3.97±4.22	−4.49±2.45					
1-2 Hrs.		−3.19±3.15	−5.12±4.36	−4.84±4.18		205.4± 57.9	322.2±100.4	323.7±146.1
3 Hrs.	−2.98±4.34	−1.61±6.25	−4.67±3.80	−3.53±5.82	267.8±135.5	264.7± 73.0	293.8±122.4	245.5± 77.4
1 Day	+3.20±4.52		+5.00±7.07*	+5.07±2.58*	189.8± 87.9		273.3± 34.6	157.7± 74.9

Mean±SD; $P<0.001$, $'P<0.01$ vs. Anesthesia.

Table 10. Fluid and blood balances (ml/M²/Day).

Group	Fluid Intake				Urine Output			
	I	II	III	IV	I	II	III	IV
Day #0	515±233	426±138	549±106	501±148	579±199	687±212	450±135	441±112
Day #1	1106±338	852±202	1040±740	837±223	789±383	669±205	740±237	574±136
Day #2	1481±416	1511±563	1450±536	1000±363	1048±202	770±388	834±257	696±270
Day #3	1355±267	1496±382	1355±552	1282±312	1214±279	1181±590	975±259	683±211
	Blood Transfusion				Chest Tube Drainage			
Day #0	652±474	607±343	906±615	1301±488	435±372	188±76	395±293	553±244
Day #1	230±180	166±150	135±171	236±302	142± 66	103±70	140± 92	175±119
Day #2	314±526	65± 87	0± 0	68±139	88± 57	43±44	67± 74	120± 93
Day #3			79±131	191±162			62± 56	116±106

Mean±SD.

Table 11. Urine specific gravity.

Group	I		II			
Day #0	1.018±.004	—	1.030±.004	1.020±.005	—	1.034±.007
Day #1	1.015±.006	—	1.025±.009	1.017±.008	—	1.028±.005
Day #2	1.010±.003	—	1.017±.005	1.011±.006	—	1.016±.006
Day #3						
Group	III			IV		
Day #0	1.018±.006	—	1.033±.007	1.019±.004	—	1.032±.007
Day #1	1.019±.007	—	1.032±.005	1.018±.007	—	1.029±.006
Day #2	1.015±.005	—	1.025±.007	1.015±.006	—	1.026±.007
Day #3	1.011±.004	—	1.016±.005	1.014±.004	—	1.025±.002

Mean±SD-Mean±SD is the range of specific gravity.

minimal. The largest blood loss amounted to 1172ml. in a patient with ASD on the day of operation. Blood replacement was a little positive side to keep the good hemodynamics in the early postoperative hours, and it ranged from 607 to 1301 ml. of the mean amounts of transfusion in the groups. Blood replacement was minimal in a small number of patients at day 1 and thereafter.

Urine Electrolytes

Urinary excretions of sodium, potassium and chloride were measured for many patients postoperatively including the urine collected on the table. With the urine collected in operating room, the mean amount of sodium excretion varied from 14.0 to 32.6 mEq., while potassium excretion ranged from 13.3 to 20.8 mEq. and chloride excretion from 7.9 to 16.7 mEq. However, the mean values of potassium excretion exceeded the one of sodium excretion in urine collected in the next morning of surgery and thereafter. The highest level of sodium excretion ranged between 11.5 and 69.5 mEq. of the mean values, while the one of potassium excretion varied from 24.0 to 81.8 mEq. in the same amount of urine measured in the postoperative day. The values of mean chloride excretion were mostly lower than the ones of

Table 12. Urine electrolytes (mEq. in the amount of urine collected).

Group	Ⅰ			
	Na	K	Cl	Urine (ml.)
OR	32.6±28.2	19.1±18.1	16.3±6.0	500±185
Day #0	41.7±28.1	50.0±27.4	25.8±15.2	600±211
Day #1	54.6±32.0	58.9±20.1	29.0±14.9	986±405
Day #2	22.7± 6.1	38.7± 8.4	23.3± 6.2	1433±419
		Ⅱ		
	15.9±13.5	13.3±13.1	7.9± 7.4	396±311
	69.5±47.2	62.0±25.7	26.8±12.5	813±497
	35.0± 9.7	47.5±12.5	21.7±14.8	667±236
	15.0±10.2	24.0± 8.0	13.0± 5.0	500±100
Group	Ⅲ			
	Na	K	Cl	Urine (ml.)
OR	28.5±30.3	17.4±11.7	16.7±16.8	390±168
Day #0	23.8±12.3	33.7±10.6	16.8± 9.4	419±133
Day #1	26.0±11.1	39.0± 9.8	15.4± 9.7	557±176
Day #2	11.5±10.0	26.8±14.2	12.3±11.0	538±204
		Ⅳ		
	14.0± 6.7	20.8± 7.5	12.3± 8.1	459±106
	34.3±29.4	40.8±21.9	27.4±18.0	669±229
	46.1±43.2	81.8±18.7	31.0±30.3	865±309
	57.4±25.8	64.2±27.0	30.8±22.6	1060±332

Mean±SD.

cations, ranging between 12.3 and 31.0 mEq. postoperatively.

Clinical Results

The clinical results were seen over complications and hospital mortality. Among the postoperative complications, wound infection was the most common one, and this was experienced by 24 patients or 45 percent of all cases. The infections, however, were fortunately superficial, being limited to the involvement of soft tissues with no cases of bony infection. And, the infections were managed readily without reoperation. The next common complication was the disturbance of cardiac rhythms and conductions. Complete right bundle branch block was noted in 11 patients of Groups Ⅱ and Ⅲ, where the infundibular resection or the repair of VSD were the major part of operation, on the electrocardiograms from immediate postoperative course. Another 2 patients of Group Ⅱ had postoperative right bundle branch block for only several days, and the bundle branch block pattern disappeared while they were in hospital. Temporary atrioventricular dissociation or nodal rhythm were experienced by 5 patients. A 23-year-old man had another exploratory thoracotomy to see the minimal intrapericardial bleeding 10 days after repair of ASD. Another patient of 4-year-old boy who had been ill with severe malnutrition had the clinical findings of low output after surgery, and he had to be kept in postanesthetic recovery room for 16 days. His

Table 13. Complications.

Group	I	II	III	IV	Total
Wound Infection	4	7	9	4	24
Complete RBBB		4	9		11
Temporary A-V Dissociation		1	2	1	4
Temporary RBBB		2			2
Temporary Nodal Rhythm			1		1
Intrapericardial Bleeding	1				1
Low Output	1				1
Heart Failure		1			1
Bacterial Endocarditis				1	1
Alopecia	1				1

Table 14. Hospital mortality.

Group	I	II	III	IV	Total
Hospital Mortality (%)	1 (11.2)		5 (26.3)	4 (28.6)	10 (18.9)
Cause of Death:					
Ventricular Arrhythmia	1			3	4
Complete A-V Block			2		2
Low Output			2		2
Acute Aortic Insufficiency			1		1
Coronary Embolism				1	1

preoperative pulmonary arterial pressure was high, 75 mm. Hg systolic, and his ASD was so big that the operative findings had a description of almost a single atrium. Postoperative alopecia was also seen in this case. One of the twin brother, both of whom had VSD, revealed the clinical findings of congestive heart failure from a week after operation, but this was managed easily to be dismissed from hospital in a good condition at day 20 after surgery. The patient who had mitral valve replacement along with the concomitant repairs of the associated VSD and ASD had fever at his home on 44th postoperative day, and blood culture revealed positive growth of Alkaligenes fecalis on readmission.

There were 10 deaths, or 18.9 percent mortality rate, while patients were in hospital. A patient of Gorup I who had closure of ASD followed by exploratory right ventriculotomy died from sudden occurrence of ventricular fibrillation in the next morning of operation when resuscitative measures failed. Three patients of Group IV died also from ventricular arrhythmias: one developed intractable ventricular fibrillation after aortic valve replacement for his aortic insufficiency and closure of aortotomy when he could not come off CPB; another patient who had prosthetic valve replacement for his tightly stenotic mitral valve and removal of huge left atrial thrombus occupying almost whole left atrial lumen had suddden ventricular tachycardia followed by ventricular fibrillation, and he died on table after closure of sternotomy wound; the third patient was in coma postoperatively and died from ventricular tachycardia developed suddenly in the evening next day of mitral valve replacement of her calcified valve.

A patient of 18-year-old boy who had both mitral and aortic valve replacements along with tricuspid annuloplasty had been doing well postoperatively until he died from sudden occurrence of ventricular fibrillation in ward at postoperative day 18. Two patients with DORV died after interventricular correction. One could not come off CPB because of low output; and another had more complexed anomalies, DORV, large VSD, combined pulmonic stenosis, bilateral superior venae cavae, and dextrocardia with situs inversus and she died from bradycardia followed by cardiac standstill in postanesthetic recovery room on the day of surgery. Two patients of Group III developed complete atrioventricular block after surgery and they were supported with intravenous isuprel administration until they died at day 1 and day 3 respectively. Another patient of TOF died manifesting the clinical features of acute aortic insufficiency at the night of operation, when the injury to the aortic root was suspected at the time of overzealous pulmonary valvulotomy.

DISCUSSION

It is hard to study why the ECC with a heart lung machine shoud be inherently harmful or damaging to the biological system. The function of the heart is to pump blood and the one of the lung is to oxygenate blood and remove carbon dioxide. And, when patients undergo OHS these functions are taken over with mechanical systems. Therefore, the conduct of CPB should aim at pumping blood as well as the heart does and at oxygenating blood and removing carbon dioxide as efficiently as the lung does. When the machine perfusion is so much like physiological perfusion there would be no after effects requiring special treatment. And, if a patient is worse after CPB, then, it must be because the hemodynamics, blood flow rates or respiration were inadequate to the needs or because the ECC were injurious to the patient by damaging blood or by adding some toxic substance. As a matter of course, surgical correction and operative details are the utmost importance. The risk of complication associated with CPB is reportedly remarkably low, perhaps one percent for perfusions lasting three hours or less, being related to the equipment, the perfusate and technical execution of the procedure (Litwak, 1971).

Generally, an intraoperative fall in hematocrit is the consequence of perfusate dilution, and this is well tolerated despite the reduced oxygen carrying capacity of the blood provided that flow rates, perfusion pressure and gas exchange are properly maintained. One of the characteristics seen in this report is the trend of decreased hemoglobin and hematocrit which lasted to the time of dismissal from hospital. In the study of body fluid shifts after CPB, Cohn et al. (1971) reported an over-all decline of 25 percent in red cell mass at postoperative day 8 where total hemodilution was used and followed by auto-transfusion of centrifuged red cells to restore the hematocrit to preoperative levels immediately after operation. And, Yashar et al.(1971) studied the blood changes after OHS and reported that the gradual increase in the blood volume approaching the preoperative level within 8 to 10 days was mainly due to an increase in plasma volume, as evidenced by a progressive fall in hematocrit from 40 to 33 percent. They suggested that increased plasma volume following OHS may be caused by redistribution of body water between the extracellular and intracellular fluid, and observed an increase in red cell mass at the expense of plasma volume at the end of 3 months. Mig-

ration of fluid from the intracellular space into the extracellular space during the week following operation was pointed to be main reason of blood and fluid limitations in the early postoperative days. The mean hematocrit levels in our patients ranged between 31 and 35 percent at discharge.

Blood cell trauma, as measured by red cell hemolysis has long been a popular guide. Although significant increase of plasma hemoglobin was seen in Groups II and III when CPB had been prolonged, the lethal trauma causing hemolysis was not always evident on mean values of plasma hemoglobin. The more important sublethal trauma to red blood cell will cause delayed hemolysis and this is reported to be parallel to a rise of plasma LDH released from cells(Bernstein et al., 1967). This was evidenced by increased mean SLDH in all groups for a week after CPB, and, moreover, the increased mean value lasting long even till the time of discharge from hospital in Group IV suggests some hemolysis in patients who had prosthetic valve replacement. And, this delayed hemolysis will explain at least partly the trend of postoperative anemia. There is, in the postperfusion, a tendency for the levels of white blood cell count to exceed the normal (Galletti, 1965). In general, levels of all types of white blood cell tend to fall in a perfusion and rise as the perfusion is prolonged (Peirce, 1969).

The fate of platelets during and after CPB has been thoroughly investigated by many authors. In human subjects, most authors found a reduction of the platelet count to values from 30 to 50 percent of the preoperative count (Schmidt et al., 1961; McKenzie et al., 1969; McKenna et al., 1975); in our patients this values ranged from 43 to 47 percent of anesthesia control at 30 minutes of CPB, from 28 to 48 percent at 60 minutes of CPB, and from 54 to 76 percent at 3 hours after CPB. Normal levels of platelet count are regained only after 6 to 7 days. And, this tendency was also seen in this study, showing increases of platelet count in the mean values between 115 and 161 percent of prebypass level at one week after CPB or sometime thereafter. The mechanism responsible for this thrombocytopenia during ECC and postoperative platelet rebound is not completely understood, but correlations have been made with the type of oxygenator used and the duration of CPB (Hill et al., 1972; Bartlett et al., 1972). Several mechanisms have been suggested for the low platelet count including loss in the extracorporeal circuit (Salzman, 1963), extracorporeal injury (Ashmore et al., 1968), and splenic and/or hepatic sequestration (Mielke et al., 1973), and many other important factors were presented as the mechanism of disapperance of platelets. Whatever the main reason for the decrease in circulating platelets may be, pretreatment of the patient with drugs inhibiting platelet aggregation, such as dipyridamole, will result in an attenuation of platelet drop and diminution of circulating microemboli (Mielke et al., 1973; Nuutinen & Mononen, 1975).

Low serum sodium on the electrolyte pattern before starting CPB in this report is chiefly because most of patients were on low salt diet after admission. Hyponatremia during CPB results from hemodilution and it persists afterward (Tarhan & Moffitt, 1971). However, the average of sodium level from 6 oxygenators in our cases was 153 mEq./L. with the ranges from 131 to 164 mEq./L. And, there were no significant changes during CPB when the sodium levels were compared with the ones at mean anesthesia control, although the highly significant decreases were seen when compared with the mean admission control at 30 minutes.

of CPB. Early changes of serum sodium are dependent upon the sodium level in the prime. Pacifico et al. (1970) reported an absolute increase in the total body sodium and a significant decrease in exchangeable potassium immediately following CPB. An actual increase in the body sodium and loss of body potassium occurs during the first 4 days after CPB, and decrease in sodium excretion averaging 50 percent was demonstrated, while increases of 200 percent in urinary potassium, often 75 to 100 mEq./L., occurred in all patients (Cohn et al., 1971). Pacifico et al. (1970) suggested that either neurohumoral factors secondary to operation or else cellular injury with potassium release and secondary excretion of potassium were the possible cause of the marked reduction in body potassium and kaliuresis. The mean potassium levels in this report were low at anesthesia and during CPB, despite exogenous addition of potassium into the prime. And, serum levels of potassium returned to normal range at day 1 for the first time after liberal administration of supplemental potassium in the early postoperative hours. Breckenridge et al.(1972) reported the postoperative negative potassium balances lasting for 3 to 4 days after open intracardiac operations and the cumulative deficit in potassium from 120 to 260 mEq. in first 3 postoperative days. The cumulative average of mean potassium excretion in urine for postoperative 3 days were from 100 to 187 mEq. of mean values with our patients, while the one of mean urinary excretion of sodium ranged from 61 to 138 mEq. The importance of potassium to improve vascular tone with increases in both arterial and venous pressures and the mechanism of restoration of normal blood pH following correction of hypokalemia were also studied during CPB (Vasko et al., 1973).

A critical period in open repair of heart lesions is the point at which the heart resumes function and the pump is turned off. The ability of the heart muscle to maintain an adequate output at the end of CPB is in part dependent upon the serum level of ionized calcium. It was reported that the level of serum ionized calcium was inversely related to the concentrations of serum citrate before CPB and, during CPB with a hemodiluted prime, the serum ionic calcium remained stable at the prebypass level in spite of a decrease in serum total calcium and proteins (Das et al., 1971). However, the hypercalcemia resulted by CPB lasting through the day of operation with the prime of diluted ACD blood where extra amounts of calcium were added (Tarhan & Moffitt, 1971). The increases of calcium levels, though not uniformly significant, at 30 minutes of CPB in our patients can be explained by the hypercalcemic primes. The level of ionized calcium was not studied in this report. The necessity of direct measurement of ionic calcium was reported, and it was pointed that the marked distortion of plasma proteins and pH, the addition of large amounts of citrate, and the differences between various calcium salts indicated that it was probably not possible to predict ionic calcium with assurance (White et al., 1976). The changes in the level of magnesium during CPB are decreases in serum levels due to hemodilution from low magnesium prime (Romero et al., 1973); and the magnesium levels in the oxygenator prime were also low in our patients.

Many tissues contain the enzyme glutamic oxalacetic transaminase, but the highest concentrations are found in the myocardium and in the liver. Tissue injury often raises the serum levels of this enzyme, presumably by releasing intracellular GOT as a consequence of necrosis or altered permeability of cell membranes. And the changes in SGPT levels

are reportedly more specific index of hepatocellular injury than that in SGOT levels. It needs further study to indicate the myocardial injury was responsible to the elevated SGO T in our patients. The characteristics of 24-hour uncomplicated ECC in animal included increase in both heart and liver enzymes (Bartlett et al., 1972). The mean SLDH levels seen in our patients revealed marked increases in all groups at day 1 and these lasted for 5 to 7 days. The increased SLDH level in Group IV persisted even at the time of discharge. The LDH activity is a reliable indicator of red cell hemolysis (Mayhre & Rasmussen, 1970; Mayhre et al., 1970). The early rise of SLDH may be interpreted by the tissue injury and by early hemolysis from CPB itself. However, the persisting high level of SLDH in Group IV must be from the hemolytic condition in patents who have a prosthetic valve in their heart. Chronic hemolysis is a recognized complication of prosthetic heart valves (Donnely et al., 1973; Eyster et al., 1971). Ahmad et al. (1976) studied the chronic hemolysis by red cell survival and other parameters including SLDH, and they correlated elevation of SLDH with hemolysis. Valve function was assessed by clinical examination and only patients with good valve function were included in their study.

Respiratory alkalosis is produced during CPB and occurs postoperatively (Moffitt et al., 196 9), and acid-base balance during ECC remains a major concern. Various concentrations of carbon dioxide added to the gaseous inflow of the oxygenator have also been suggested to prevent hypocapnia and its accompanying lactic acidosis and hypokalemia (Carson et. al., 1964; Andersen et al., 1963). This was also stressed by us (Kim et al., 1974), but, unfortunately, the medical grade of gases containing exact amount of carbon dioxide are not readily available with us presently. Hallowell et al. (1967) reported that the arterial pCO_2 and pH could be controlled predictably by varying oxygen flow rates in relation to perfusion through the disc oxygenator without adding carbon dioxide, and that metabolic acidosis was not an inevitable concomitant of CPB of less than 4 hour in length without the need of alkanizing agents. However, they mentioned about the fact that bubble oxygenators required large volumes of oxygen to oxygenate the blood and the degree of gas exchange was not readily predictable. Although the metabolic component of blood gas studies was generally acceptable in our patients, many needed varying amounts of sodium bicarbonate during and after CPB.

Several authors have already domonstrated changes in the normal blood-clotting mechanism after OHS in which ECC was used (Gans & Krivit, 1962; Bentall & Alwork, 1968). However, these changes are not usually of such a degree as to cause excessive bleeding (Bentall & Alwork, 1968). Although, there are centers where they routinely perform sequestration of the patient's own blood during perfusion and autotransfusion after perfusion to diminish the bleeding tendency that is partly associated with the destructive action of the pump oxygenator on circulating coagulation factors (Hardesty et al., 1968; Garcia et al., 1973). The mean blood losses through the chest tubes varied among groups of our patients, and no case underwent re-exploration for excessive bleeding. More meticulous intraoperative hemostasis and prebypass administrations of epsilon aminocaproic acid may have something to do with postoperative bleeding. The reported bleeding tendency in cyanotic patients(Gomes & McGoon, 1970) was not evident with our patients.

The infection of sternotomy wound was experienced in about half of our patients. Though the involvements were only soft tissues and no bony infection was noted, these infections were one of the major reason for the patient's prolonged stays in hospital after surgery; mean hospital stays of 25.8±11.7 days in Group I, 22.6±6.9 days in Group II, 26.9±7.6 days in Group III, and 27.6±10.4 days in Group IV, making almost no differences among groups for the length of hospital stays. Disturbances in cardiac rhythm or conduction mechanism were the next common complications. Ventricular arrhythmias and complete surgical heart blocks were the fatal complications. Two patients with DORV died from low output condition and a patient with ASD who had pulmonary arterial hypertension recovered from a postoperative low output. However, 14 patients had supports with isuprel sometime during postoperative course for the better hemodynamics or slow cardiac rhythm, although clnical findings of low output were not definite.

CONCLUSIONS

The clinical and metabolic effects of cardiopulmonary bypass were stduied with fifty-three patients who underwent open heart surgery by evaluating serial laboratory examinations and clinical results. Mild anemia was a finding after extracorporeal circulations along with the thrombocytopenia followed by a platelet rebound. Hypokalemia was a characteristic of cardiopulmonary bypass being evidenced by low serum potassium levels during and after extracorporeal circulation and by the increased urinary excretion of potassium which exceeded the one of sodium in the early postoperative days. The changes of total calcium and magnesium during cardiopulmonary bypass were dependent upon their levels in the prime of oxygenator. Of serum enzymes the significant increases of SGOT and SLDH after cardiopulmonary bypass suggested various tissue injuries including thoracotomy, cardiotomy, sublethal damage to red blood cells and hemolysis from the presence of prosthetic cardiac valves. The changes of SGPT were mild and delayed. Marked respiratory alkalosis accompanied with mild degrees of metabolic acidosis were the characteristic pattern during and early after cardiopulmonary bypass.

The most common complications were the infection of sternotomy wounds. Although these were superficial in nature, they prolonged the patient's hospital stays. Disturbances of cardiac rhythms or conduction mechanisms were the next commonly seen complications and they were fatal when ventricular arrhythmias were intractable and surgical block was not treated with pacemaker implantation. There were 10 deaths, or 18.9 percent, while patients were in hospital. The hospital mortality rates among groups were 11.2 percent (1 of 9) after repairs of atrial septal septal defect, none after closure of ventricular septal defect, 26.3 percent (5 of 19) after total correction of tetralogy of Fallot and interventricular correction of double outlet right ventricle, and 28.6 percent (4 of 14) after valve replacement.

》國文抄錄《

體外循環의 臨床 및 代謝效果

서울大學校 醫科大學 胸部外科學敎室

金 鍾 煥

先天性 및 後天性 心臟疾患에 對하여 心肺器을 使用한 開心術을 받은 53名의 患者를 對象으로 各種 檢査室成績과 臨床結果를 綜合하여 體外循環이 人體에 미치는 臨床的 및 代謝上 效果를 檢討하였다. 體外循環

後 輕度의 貧血像이 退院時까지 繼續되며 血小板은 急激한 術中減少와 後續되는 術後增加를 보였다. 血淸電解質中 카륨의 低下가 顯著하고 尿中排泄의 增加를 보였다. 血淸內 칼슘總量과 마그네슘은 主로 酸化器充塡液中의 含量에 따라 變動하였다. 各種 血淸酵素中 SGOT 및 SLDH의 顯著한 上昇을 보였으며, 開胸, 開心, 赤血球의 損傷等을 包含하는 組織損傷과 人工辨膜의 存在에 따른 溶血現象等에 起因하는 것으로 看做되었다. 體外循環中 및 術後早期에는 中等度의 呼吸性鹽基症과 輕度의 代謝性酸症이 特徵的 所見이었다.

術後合併症으로 開胸創의 感染이 가장 흔하였으며 胸骨侵襲은 없었으나 患者의 在院期間을 延長하는 主因의 하나였다. 心臟律動이나 傳導機構의 障碍가 다음으로, 治療에 抵抗하는 心室性不整脈 또는 人工心搏裝置없이 加療한 外科的心부룩은 致命的이었다. 入院中 10名이 死亡하여 在院死亡率은 18.9%였고, 各疾患群에 따른 死亡率은 心房中隔缺損症에서 11.2%, 心室中隔缺損症에서 없고, 靑色群에서 26.3%, 및 後天性辨膜疾患群에서 28.6%였다.

REFERENCES

Ahmad, R., S.M. Manohitharajah, P.B. Deverall & D.A. Watson: *Chronic hemolysis following mitral valve replacement. A comparative study of the Björk-Shiley, composite-seat Starr-Edwards, and frame-mounted aortic homograft valves. J. Thorac. Cardiovasc. Surg., 71:212, 1976.*

Andersen, M.N., M. Mendelow & G. William-Olsson: *Relationship of respiratory alkalosis to metabolic acidosis during extracorporeal circulation. Surgery, 53:730, 1963.*

Ashmore, P.G., V. Svitek & P. Ambrose: *The incidence and effects of particulate aggregation and microembolism in pump-oxygenator systems. J. Thorac. Cardiovasc. Surg., 55:691, 1968.*

Bartlett, R.H., N.E. Burns, S.W. Fong, A.B. Gazzaniga, B.M. Achauer & J. Fraile: *Prolonged partial venoarterial bypass; Physiologic, biochemical, and hematologic responses. Surg. Forum, 23:178, 1972.*

Bentall, H. H. & S.P. Alwork: *Fibrinolysis and bleedig in open heart surgery. Lancet, 1:4, 1968.*

Bernsterin, E.G., P.L. Blackshear, Jr. & K.H. Keller: *Factors influencing erythrocyte destruction in artificial organs. Amer. J. Surg., 114:126, 1967*

Breckenridge, I.M., P.B. Deverall, J.W. Kirklin & S.B. Digerness: *Potassium intake and balance afer open intracardiac operations. J. Thorac. Cardiovasc. Surg., 63:305, 1972.*

Carson, S.A.A., L.E. Morris, K.W. Edwards, T.W. Jones, G.A. Logan, L.R. Sauvage & G.L. Thomas: *Acid-base management for open-heart surgery. Circulation, 29:456, 1964.*

Cohn, L.H., W.W. Angell & N.E. Shumway: *Body fluid shifts after cardiopulmonary bypass: I. Effects of congestive heart failure and hemodilution. J. Thorac. Cardiovasc. Surg., 62:423, 1971.*

Das, J.B., A.J. Eraklis, J.G. Adams, Jr. & R.E. Gross: *Changes in serum ionic calcium during cardiopulmonary bypass with hemodilution. J. Thorac. Cardiovasc. Surg., 62:449, 1971.*

Donnelly, R.J., A.V. Rahman, S.M. Manohitharajah, P.B. Deverall & D.A. Watson: *Chronic hemolysis following mitral valve replacement. A comparison of the frame-mounted aortic homograft and the composite-seat StarrEdwards Prosthesis. Circulation, 48:823, 1973.*

Eyster, E., J. Rothchild & O. Machajliw: *Chronic intravascular hemolysis after aortic valve replacement. Circulation, 44:657, 1971.*

Foote, A.V., M. Trede & J.V. Maloney, Jr.: *An experimental and clinical study of the use of acid-citrate-dextrose (ACD) blood for extracorporeal circulation. J. Thorac. Cardiovasc. Surg., 42:93, 1961.*

Galletti, P.M.: *Laboratory experience with 24 hour partial heart-lung bypass. J. Surg. Res., 5:97, 1965.*

Gans, H. & W. Krivit; *Problems in hemostasis during open-heart surgery. IV. On the changes in the blood clotting mechanism during cardiopulmonary bypass procedures. Ann. Surg., 155:353, 1962.*

Garcia, J.B., B.C. Pakrashi, D.A. Mary, R.K. Tandon, & M.I. Ionescu: *Postoperative blood loss after extracorporeal circulation for heart valve surgery. J. Thorac. Cardiovasc. Surg., 65:487, 1973.*

Gomes, M.M.R. & D.C. McGoon,: *Bleeding patterns after open-heart surgery. J. Thorac. Cardiovasc. Surg., 60:87, 1970.*

Hallowell, P., W.G. Austen & M.B. Laver,: *Influence*

of oxygen flow rate on arterial oxygenation and acid-base balance during cardiopulmonary bypass with use of a disc oxygenator. Circulation, 35 & 36 *(Suppl.):I-199, 1967.*

Hardesty, R.L., W.L. Bayer, & H.T. Bahnson,: *A technique for the use of autologous fresh blood during open-heart surgery. J. Thorac. Cardiovasc. Surg., 56:683, 1968.*

Hill, J.D., M.R. de Leval, C.H. Mielke, Jr. et al.: *Clinical prolonged extracorporeal circulation for respiratory insufficiency: Hematological effects. Trans. Am. Soc. Artif. Intern. Organs, 18:546, 1972.*

Kay, E.B., C. Noguiera, D. Mendelsohn, Jr. & H.A. Zimmerman: *Corrective surgery for tetralogy of Fallot: Evaluation of results. Circulation, 24:1342, 1961.*

Kim, C.W., C.W. Choi, J.S. Kim, K.H. Sohn, K.P. Suh & Y.K. Lee: *Acid-base balance in open heart surgery utilizing ACD blood hemodilution. Seoul J. Med., 10:199, 1969.*

Kim, C.W., S.M. Chang, Y.J. Song, J.R. Rho, K.P. Suh & Y.K. Lee: *Metabolic effects of extracorporeal circulation. Korean Central J. Med., 26:561, 1974.*

Lee, Y.K., K.P. Suh, C.W. Kim, Y.K. Park, J.R. Rho, Y.J. Song, S.M. Chang, C.H. Kim, H. Chae & C.C. Park: *Open mitral commissurotomy:A report of 21 cases. Korean J. Thorac. Cardiovasc. Surg., 6:1, 1973.*

Litwak, R.S.: *Extracorporeal circulation. Calif. Med., 115:79, 1971.*

Mayhre, E. & K. Rasmussen: *Serum lactic dehydrogenase activity and intravascular hemoglobin. Lancet, 1:355, 1970.*

Mayhre, E., K. Rasmussen & A. Andersen,: *Serum lactic dehydrogenase activity in patients with prosthetic heart valves: A parameter of intravascular hemoglobin. Am. Heart J., 80:463, 1970.*

McKenna, R., F. Bachmann, B. Whittaker, J.R. Gilson & M. Weinberg, Jr.: *The hemostatic mechanism after open-heart surgery: II. Frequency of abnormal platelet functions during and after extracorporeal circulation. J. Thorac. Cardiovasc. Surg., 70:298, 1975.*

McKenzie, R.N., D.P. Dhall, K.E. Arfors, S. Nordlund & N.A. Matheson: *Blood platelet behaviour during and after open heart surgery. Br. Med. J., 2:795, 1969.*

Mielke, C.H., Jr., M. de Leval, J.D. Hill, M.F. Macur & F. Gerbode: *Drug influence on platelet loss during extracorporeal circulation. J. Thorac. Cardiovasc. Surg., 66:845, 1973.*

Moffitt, E.A., J.W. Rosevar, C.H. Townsend & D.C. McGoon: *Myocardial metablism in patients having aortic-valve replacement. Anesthesiology, 31:310, 1969.*

Nuutinen, L.S. & P. Mononen,: *Dipyridamole and thrombocyte count in open-heart surgery. J. Thorac. Cardiovasc. Surg. 70:707, 1975.*

Pacifico, A.D., S.B. Digerness & J.W. Kirklin: *Acute alterations of body composition after open intracardiac operations. Circulation, 41:331, 1970.*

Peirce, E.C., II: *Extracorporeal circulation for open-heart surgery. Thomas, Springfield, 1969.*

Romero, G., J.L. Castillo-Olivares, F. O'Connor, J. Guardiola & D.F. Aymerich: *The importance of calcium and magnesium ions in serum and cerebrospinal fluid during cardiopulmonary bypass. J. Thorac. Cardiovasc. Surg., 66:668, 1973.*

Salzman, E.W.: *Blood platelets and extracorporeal circulation. Transfusion, 3:274, 1963.*

Schmidt, P., J. Peden, G. Brecher & A. Barnovsky: *Thrombocytopenia and bleeding tendency after extracorporeal circulation. N. Eng. J. Med., 265:1181, 1961.*

Tarhan, S. & E.A. Moffitt: *Anesthesia and supportive care during and after cardiac surgery. Ann. Thorac. Surg., 11:64, 1971.*

Vasko, K.A., R.A. DeWall & A.M. Riley: *Hypokalemia. Physiological abnormalities during cardiopulmonary bypass. Ann. Thorac. Surg., 15:347, 1973.*

White, R.D., R.S. Goldsmith, R. Rodriguez, E.A. Moffitt & J.R. Pluth: *Plasma ionic calcium levels following injection of chloride, gluconate, and gluceptate salts of calcim, J. Thorac. Cardiovasc. Surg., 71:609, 1976.*

Yashar, J.J., G.L. Hallman, R.D. Leachman, & D.A. Cooley: *Cardiac output and blood volume changes after open-heart surgery. J. Thorac. Cardiovasc. Surg., 61:724, 1971.*