1978

A study of potassium level alterations in twenty cardiac surgical patients subjected to cardiopulmonary bypass

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A STUDY OF POTASSIUM LEVEL ALTERATIONS IN TWENTY CARDIAC SURGICAL PATIENTS SUBJECTED TO CARDIOPULMONARY BYPASS

A Thesis

Presented to

the Graduate Faculty of

the Department of Biological Sciences

University of the Pacific

In Partial Fulfillment

of the Requirements for the Degree

Master of Science

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by

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November 1978

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Dated November 14, 1978

ACKNOWLEDGEMENTS

I would like to thank Dr. Robert Wuerflein for permission to draw samples from his surgical patients. Without his help this investigation could not have been undertaken. Also, I would like to thank Dr. George Bailey for his suggestions and use of laboratory equipment at Good Samaritan Hospital, San Jose, California. The intensive care nurses of Western Heart Associates Medical Group were very patient and understanding of my requests and I extend thanks to them.

The members of my thesis committee deserve a special thanks for their guidance and understanding of my difficulty with appointments and deadlines. Also I would like to thank my parents who have always been supportive of everything I have undertaken, and Kathy for sharing and understanding my periodic frustrations.

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INTRODUCTION

The use of cardiopulmonary bypass in cardiac surgery has introduced a number of physiological problems inherent to the technique. A major concern among these is an observed decrease in serum potassium (K^+) concentration (Barnard et al., 1966). A substantial loss of potassium from the body could have deleterious effects, for it has many important physiological functions. Potassium assists in the transport of oxygen as potassium oxyhemoglobin and facilitates the transport of carbon dioxide as bicarbonate ion in the red blood cell. It has a major role in depolarization and repolarization of nerve fibers as the primary intracellular cation. It decreases protoplasmic viscosity. Potassium is necessary for the utilization of glucose by the cells and for the conversion of glucose to glycogen by the liver. It is also an important ion in the acid-base balance of the body (Burke, 1976).

Since hypokalemia during and after cardiac surgery may cause cardiac arryhthmias, this phenomenon is of clinical importance and has been studied extensively (Mandal et al., 1969). Lockey et al. (1966) suggested that hypokalemia found during and after operations might be the result of potassium depletion secondary to pre-operative diuretic therapy. Another cause of the hypokalemia seen during bypass could be a shift of potassium into the cells (Lampard and Couves, 1970) or hemodilution (Dieter et al., 1970b).

Salt loading with a perfusion prime of Ringer's lactate solution may also contribute to the hypokalemic effect (Dieter et al., 1970a). In addition, overzealous use of mechanical ventilators, including cardiopulmonary devices, resulting in patient alkalosis may reduce serum K^+ by renal excretion (Adrogue et al., 1971).

Although plasma potassium level is decreased by cardiopulmonary bypass (CPB), it is possible to maintain a normal level (3.5-5.5 mEq/l) with supplemental doses of KCl. However, experiments by DeWall and Lillihei (1962) emphasized that a normal serum level of potassium does not preclude total body depletion of potassium. Mandal et al. (1968) biopsied skeletal and myocardial muscle and determined by flame photometric measurements that intracellular, or total body, level of potassium is reduced following CPB; and this reduction may cause cardiac arryhthmias.

Since changes of red blood cell (RBC) potassium level parallels changes in total body potassium (DeWall et al., 1970), RBC K^+ level may be a better indicator of total body potassium than the serum level (Carlmark et al., 1975). Researchers believe the K^+ depletion in serum is a shift of this electrolyte to the intracellular compartment. However, Gay and Ebert (1968) found that K^+ may be a rapidly available source of potassium during the period of bypass. Nuutinen and Holmm (1976) found that urinary K^+ clearance definitely increased as bypass progressed, and an elevated level

existed for a number of hours post-operatively as compared to pre-operative values.

The events of cardiac surgery combined with moderate hypothermic hemodilution perfusion and post-operative respiratory care provide the stimuli for the alteration of K^+ levels in red blood cells, plasma, and urine. The purpose of this study was to measure the deviations from normal potassium ion concentrations in an attempt to understand the physiological processes involved.

MATERIALS AND METHODS

Data obtained from 20 patients undergoing open heart surgery (Appendix) for quadruple coronary artery disease are the basis for this paper. All patients were male, ranging in weight from 165 lbs to 175 lbs, in age from 60 to 65 years, and in height from 5'8" to 5'11".

The following equipment and techniques were employed during surgery which was performed by Dr. Robert Wuerflein. The Bentley disposable bubble oxygenator model Q100 (Bentley Laboratories, Irvine, California) was primed in each case with 1.5 litres of lactated Ringer's solution buffered with sodium bicarbonate. Moderate hypothermic perfusion was employed, body temperature not exceeding 30°C while the aorta was cross clamped, with flow rates ranging from 3.0 to 4.0 litres/minute. Gas to perfusion flow ratio was approximately 2:1, using a mixture of 100% O_2, and 95% O_2 with 5% CO_2. The pH, pO_2, pCO_2, and bicarbonate ion levels were all maintained within accepted limits (7.3-7.5, 100-300, 30-40, 22-26). Arterial mean pressures were maintained between 50 and 60 mm Hg.

Patients did not receive digitalis, insulin, or diuretics prior to surgery or post-operatively in order to prevent electrolyte imbalance or excretion due to these drugs. Neurolept anaesthesia was employed for inducement. Supplemental potassium (30 to 60 mEq/l) was given each

patient during the operation to maintain a normal plasma level (3.5 to 5.5 mEq/l) and as needed post-operatively (Gay et al., 1975; Sliwi et al., 1974). A volume ventilator was used in each case, immediately following bypass, for a period of approximately 12 hours.

Heparinized blood samples and urine samples (collected in 10 cc glass vials) were drawn from each patient for potassium determinations. Samples were taken 12 hrs before bypass, during bypass at 30 minute intervals (maximum of three samplings) and 48 hrs after bypass. Blood samples were centrifuged to separate the plasma and cell fractions and then stored in separate containers. All containers were air-tight to prevent evaporation. Samples were frozen for preservation immediately. Blood cell samples were thawed and frozen 10 separate times to facilitate membrane lysis. All samples were finally thawed and evaluated for potassium ion concentration by flame photometry. Only the supernatant of blood cell samples was used for testing.

In order to evaluate the data obtained a two-way factorial analysis of variance (ANOVA), based on a Model I design, was employed. Student's t-tests were also performed, using pre-bypass data as a control, to determine at what time during surgery and post-operatively variance from control values were significant. Pearson product-moment correlations were also used to determine relational changes in potassium levels of the three fluids examined.

RESULTS

The pre-operative plasma K^+ ranged from 3.8 to 4.8 mEq/l with a mean of 4.2 mEq/l (Table 1). During the operation the following changes occured in plasma K^+: 30 minutes on bypass plasma K^+ increased above pre-operative levels (ranging from 4.5 to 5.5 mEq/l with a mean of 5.0 mEq/l); 60 minutes on bypass plasma K^+ remained essentially the same as the 30 minute levels (ranging from 4.5 to 5.3 mEq/l with a mean of 4.9 mEq/l); 90 minutes on bypass plasma K^+ was still slightly elevated (ranging from 4.7 to 5.4 mEq/l with a mean of 5.0 mEq/l). The post-operative level of plasma K^+ at 48 hrs had decreased from operative levels (ranging from 3.9 to 4.8 mEq/l with a mean of 4.4 mEq/l) (Figure 1).

The pre-operative level of intracellular (RBC) potassium ion ranged from 89.6 to 103.5 mEq/l with a mean of 96.7 mEq/l. During the operation, RBC K^+ concentrations were as follows: 30 minutes on bypass RBC K^+ decreased from pre-operative levels (ranging from 88.2 to 100.5 mEq/l with a mean of 94.8 mEq/l); 60 minutes on bypass, RBC K^+ had again decreased (ranging from 85.3 to 96.5 mEq/l with a mean of 91.7 mEq/l); 90 minutes on bypass, RBC K^+ further decreased (ranging from 83.6 to 92.5 mEq/l with a mean of 88.6 mEq/l). The intracellular K^+ at 48 hrs post-operatively had risen from the operative levels (ranging from 89.1 to 101.2 mEq/l with a mean of 95.6 mEq/l) (Figure 2).

The urinary K^+ level pre-operatively ranged from 22.6 to 41.2 mEq/l with a mean of 33.9 mEq/l. During the operation the urinary K^+ levels were as follows: 30 minutes on bypass the urinary K^+ concentration had increased from pre-operative levels (ranging from 34.1 to 53.5 mEq/l with a mean of 42.3 mEq/l); 60 minutes on bypass the K^+ level again increased (ranging from 42.1 to 64.9 mEq/l with a mean of 53.4 mEq/l); 90 minutes on bypass the urinary K^+ rose further (ranging from 51.2 to 74.2 mEq/l with a mean of 61.3 mEq/l). Following the surgery 48 hrs post-operatively the urinary potassium ion level had fallen from 60 and 90 minute bypass levels (ranging from 33.3 to 54.9 mEq/l with a mean of 42.6 mEq/l) (Figure 3).

Statistically the ANOVA shows that change of the K^+ level in the three fluids examined is not by chance, given the circumstances as described earlier at the .001 significance level (Table 2). Student's t-tests were employed (Table 3) to determine more exactly when K^+ levels were altered significantly from control, pre-operative, values.

Serum K^+ levels are increased significantly 30 minutes, 60 minutes and 90 minutes on bypass, returning to control values 48 hrs post-operatively. RBC K^+ is not significantly elevated until 60 minutes on bypass, remaining so throughout the surgery, and returning to control values 48 hrs post-operatively. Urinary K^+ concentration is elevated significantly 30 minutes on bypass, continuously until the end of

bypass as well as 48 hrs post-operatively.

In order to determine whether any relationship between these three fluids has been temporarily disturbed, with respect to the passage of potassium ion, Pearson product-moment correlation tests were performed (Table 4). Not until 90 minutes on bypass were any definitive changes seen. The exchange of K^+ between intracellular and extracellular fluids is significantly correlated with an r value of .93. The correlation of intracellular and urinary K^+ is 1.0. The correlation of extracellular and urinary K^+ is .89. All correlation values decrease post-operatively returning to non-significant values as determined pre-operatively (Figure 4).

TABLE 1

Data obtained by flame photometric analysis of the potassium level (mEq/l) in serum (S), red blood cells (R), and urine (U) of 20 cardiac surgical patients exposed to cardiopulmonary bypass.

TABLE 1

Pt.	12 Hrs Pre-Bypass			30 Mins on Bypass			60 Mins on Bypass		
	S	R	U	S	R	U	S	R	U
1	4.5	101.9	32.6	5.0	99.5	39.6	4.9	95.1	49.8
2	3.9	99.7	29.4	4.8	98.3	35.4	4.9	94.3	42.4
3	4.6	103.5	41.2	5.1	100.5	52.1	5.3	96.4	61.8
4	4.1	97.9	33.6	4.9	96.4	41.3	4.7	93.7	50.6
5	4.0	98.3	35.5	5.5	96.9	44.2	5.0	94.8	55.4
6	3.9	92.4	25.6	4.7	91.0	34.7	4.6	88.2	43.6
7	4.2	97.3	37.9	5.0	95.2	45.1	5.0	91.6	56.4
8	4.3	96.5	32.4	5.1	96.2	40.2	4.8	92.3	53.7
9	3.8	100.2	29.8	4.5	99.4	36.1	5.1	94.1	49.8
10	4.1	93.8	36.5	4.9	92.2	44.4	4.8	90.3	53.1
11	4.0	98.6	30.3	4.8	96.4	39.3	4.6	91.5	50.5
12	4.8	95.2	41.1	5.5	93.1	52.1	5.3	89.4	64.9
13	4.4	99.1	35.6	5.2	98.5	43.6	5.0	96.5	58.6
14	4.6	96.4	38.4	4.8	94.9	47.8	4.9	91.8	61.0
15	4.2	89.6	29.9	5.0	88.2	36.5	4.9	85.3	45.3
16	3.9	93.4	33.3	4.6	91.6	41.6	4.5	90.2	50.2
17	4.4	98.7	22.6	4.5	90.3	31.4	4.8	87.9	47.4
18	4.0	97.5	34.5	5.3	97.4	42.8	5.1	93.4	52.1
19	4.7	93.2	40.2	5.2	91.7	53.5	5.0	88.7	65.3
20	4.2	90.4	38.1	4.8	88.9	45.2	5.1	87.7	56.8
Mean	4.2	96.7	33.9	5.0	94.8	42.3	4.9	91.7	53.4
Range	3.8-	89.6-	22.6-	4.5-	88.2-	34.1-	4.5-	85.3-	42.1-
	4.8	103.5	41.2	5.5	100.5	53.5	5.3	96.5	64.9
S. D.	0.3	3.5	4.9	0.3	3.7	6.0	0.2	3.1	6.6

TABLE 1 (CONT.)

Pt.	90 Mins on Bypass			12 Hrs Post-Bypass		
	S	R	U	S	R	U
1	5.0	90.2	55.6	4.3	99.4	35.2
2	4.7	89.6	53.7	4.1	98.7	33.3
3	5.0	92.4	54.6	4.7	101.2	43.1
4	4.8	88.9	55.3	4.2	99.5	36.6
5	5.1	91.2	62.9	4.1	98.9	37.4
6	4.8	85.3	51.4	4.3	94.7	34.1
7	5.0	89.7	64.8	4.5	96.3	46.7
8	4.7	90.4	66.3	4.1	98.1	42.3
9	5.2	90.1	58.1	4.6	96.2	43.6
10	5.3	87.5	60.9	4.8	94.3	40.1
11	5.0	88.4	57.3	4.5	96.0	41.8
12	5.2	86.3	72.1	4.4	93.5	52.3
13	4.9	92.5	66.0	4.6	98.9	49.9
14	4.7	90.1	68.4	4.0	97.3	53.4
15	5.2	83.6	51.2	4.3	89.4	33.8
16	5.1	87.5	57.5	3.9	93.8	39.3
17	4.8	85.3	54.2	4.7	91.2	35.6
18	5.4	90.2	63.9	4.2	95.3	47.3
19	5.1	85.9	74.2	4.9	90.5	54.9
20	5.2	86.0	67.8	4.6	89.1	50.5
Mean	5.0	88.6	61.3	4.4	95.6	42.6
Range	4.7-5.4	83.6-92.5	51.2-74.2	3.9-4.8	89.1-101.2	33.3-54.9
S. D.	0.2	2.5	6.8	0.3	3.5	7.0

TABLE 2

Two-way analysis of variance (ANOVA) based on a Model I design with replication. Values calculated from data obtained by flame photometric analysis of the potassium level (mEq/l) in serum, red blood cells and urine of 20 cardiac surgical patients exposed to cardiopulmonary bypass.

TABLE 3

Student's t-test values calculated from data obtained by flame photometric analysis of the potassium level (mEq/l) in serum, red blood cells and urine of 20 cardiac surgical patients exposed to cardiopulmonary bypass. Red blood cell, serum and urine potassium levels during bypass and post-bypass were compared with pre-bypass levels.

TABLE 4

Pearson product-moment correlation values calculated from data obtained by flame photometric analysis of the potassium level (mEq/l) in serum, red blood cells and urine of 20 cardiac surgical patients exposed to cardiopulmonary bypass.

TABLE 2

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Time	4	1607.7	28884.0	1699.1*
Fluids	2	394335.8	401.9	23.6*
Interaction	8	8433.1	197167.9	11598.1*
Error	285	4843.1	1054.4	82.7*
Total	299	409219.7	17.0	

TABLE 3

Time	RBC	Serum	Urine
30 mins on bypass	3.570ns	8.704*	4.824*
60 mins on bypass	4.727*	8.639*	10.390*
90 mins on bypass	8.356*	9.984*	14.663*
48 hrs post-bypass	0.625ns	1.713ns	4.549*

TABLE 4

Time	RBC vs. Serum	RBC vs. Urine	Serum vs. Urine
12 hrs pre-bypass	0.08ns	0.14ns	0.50ns
30 mins on bypass	0.20ns	0.08ns	0.62ns
60 mins on bypass	0.26ns	0.13ns	0.60ns
90 mins on bypass	0.93*	1.00*	0.89*
48 hrs post-bypass	0.32ns	0.19ns	0.30ns

* 99.9% significance level
ns not significant

FIGURE 1

Mean values of serum potassium level of 20 cardiac surgical patients pre-bypass, during bypass, and post-bypass. The vertical bars indicate one standard deviation above and below the line.

FIGURE 1

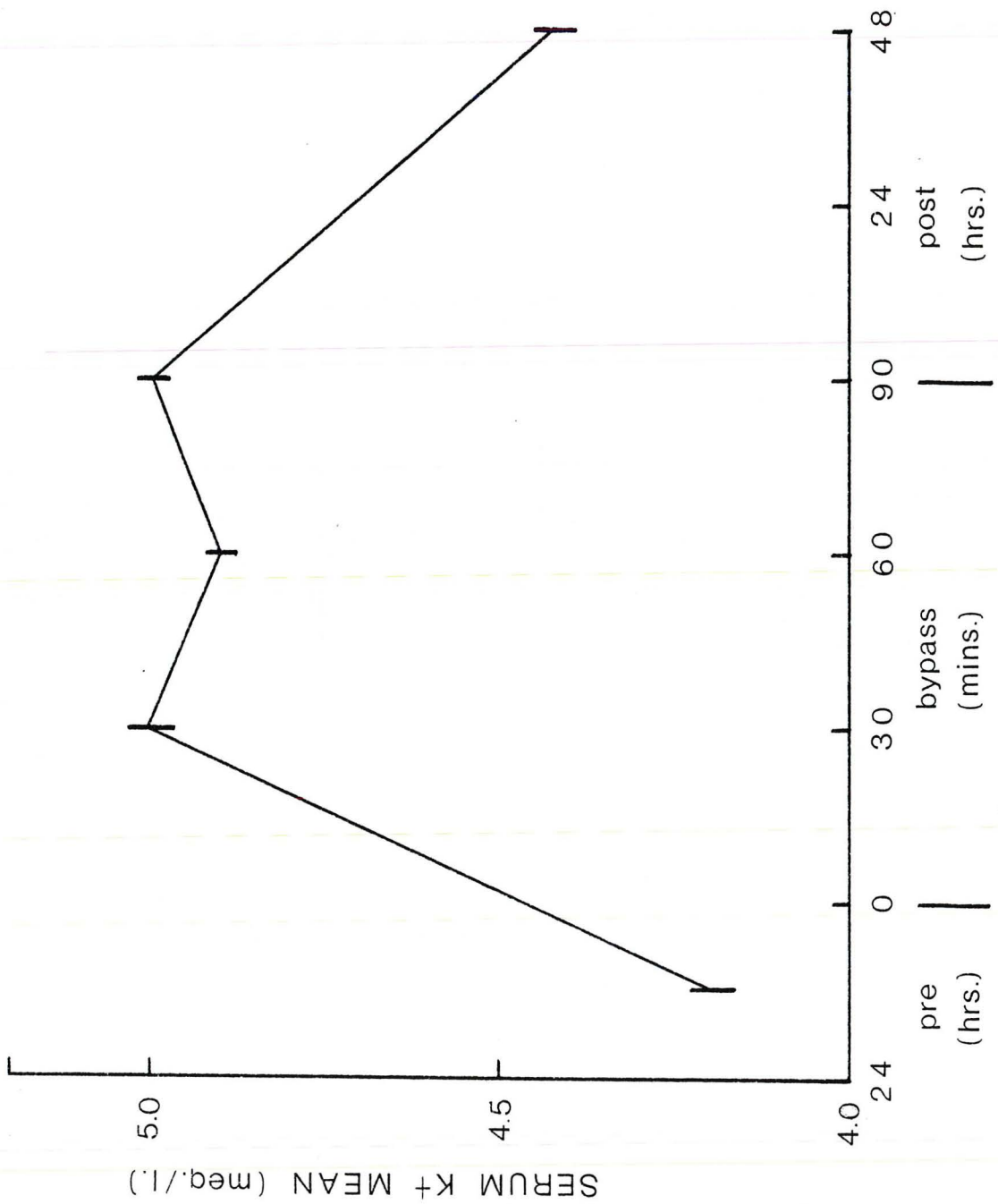

SERUM K+ MEAN (meq./l.)
5.0
4.5
4.0
24
0
30
60
90
24
48
pre (hrs.)
bypass (mins.)
post (hrs.)
TIME

FIGURE 2

Mean values of red blood cell potassium level of 20 cardiac surgical patients pre-bypass, during bypass, and post-bypass. The vertical bars indicate one standard deviation above and below the line.

FIGURE 2

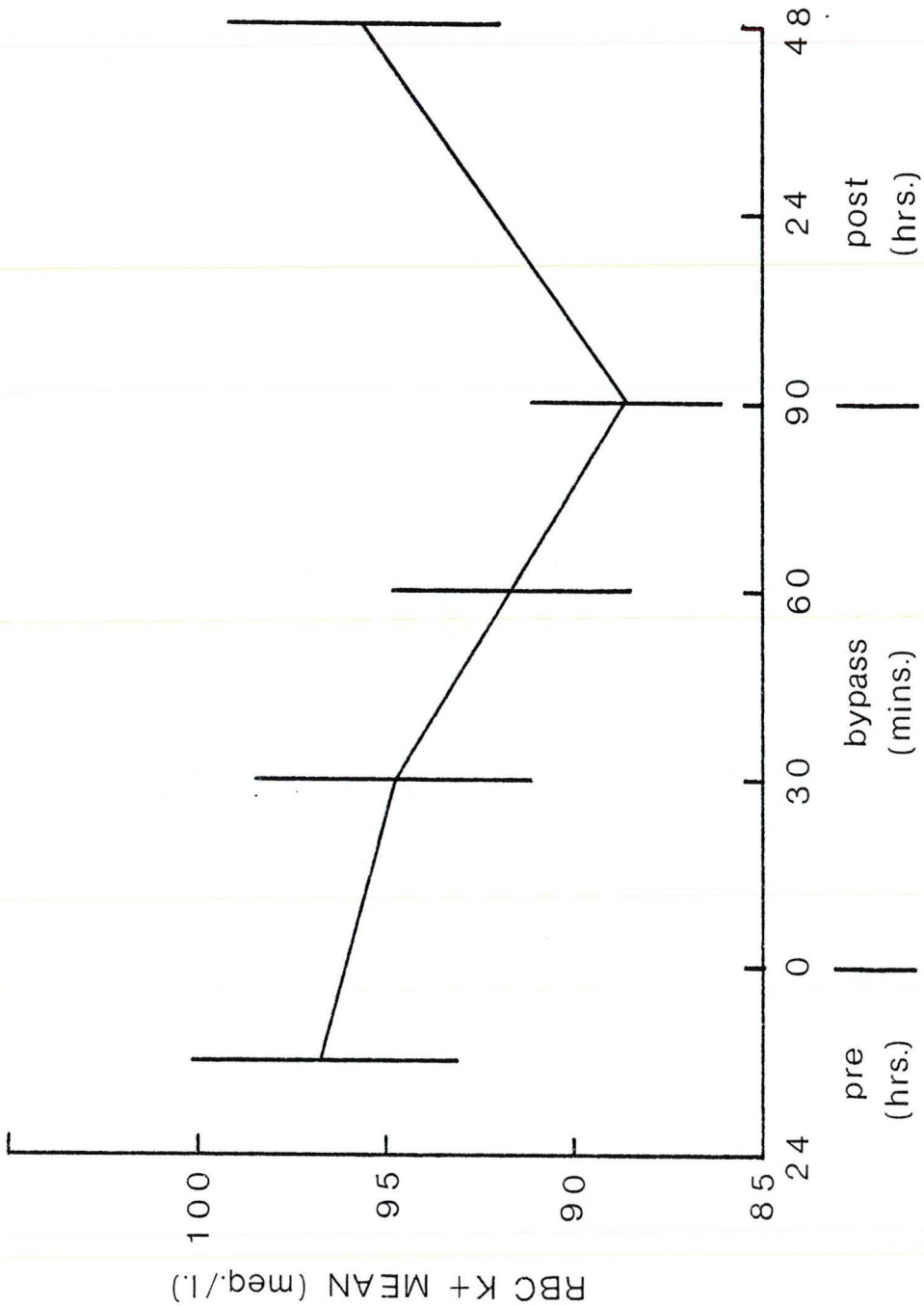

RBC K+ MEAN (meq./l.)
100
95
90
85
24
0
30
60
90
24
48
pre (hrs.)
bypass (mins.)
post (hrs.)
TIME

FIGURE 3

Mean values of urinary potassium level of 20 cardiac surgical patients pre-bypass, during bypass, and post-bypass. The vertical bars indicate one standard deviation above and below the line.

FIGURE 3

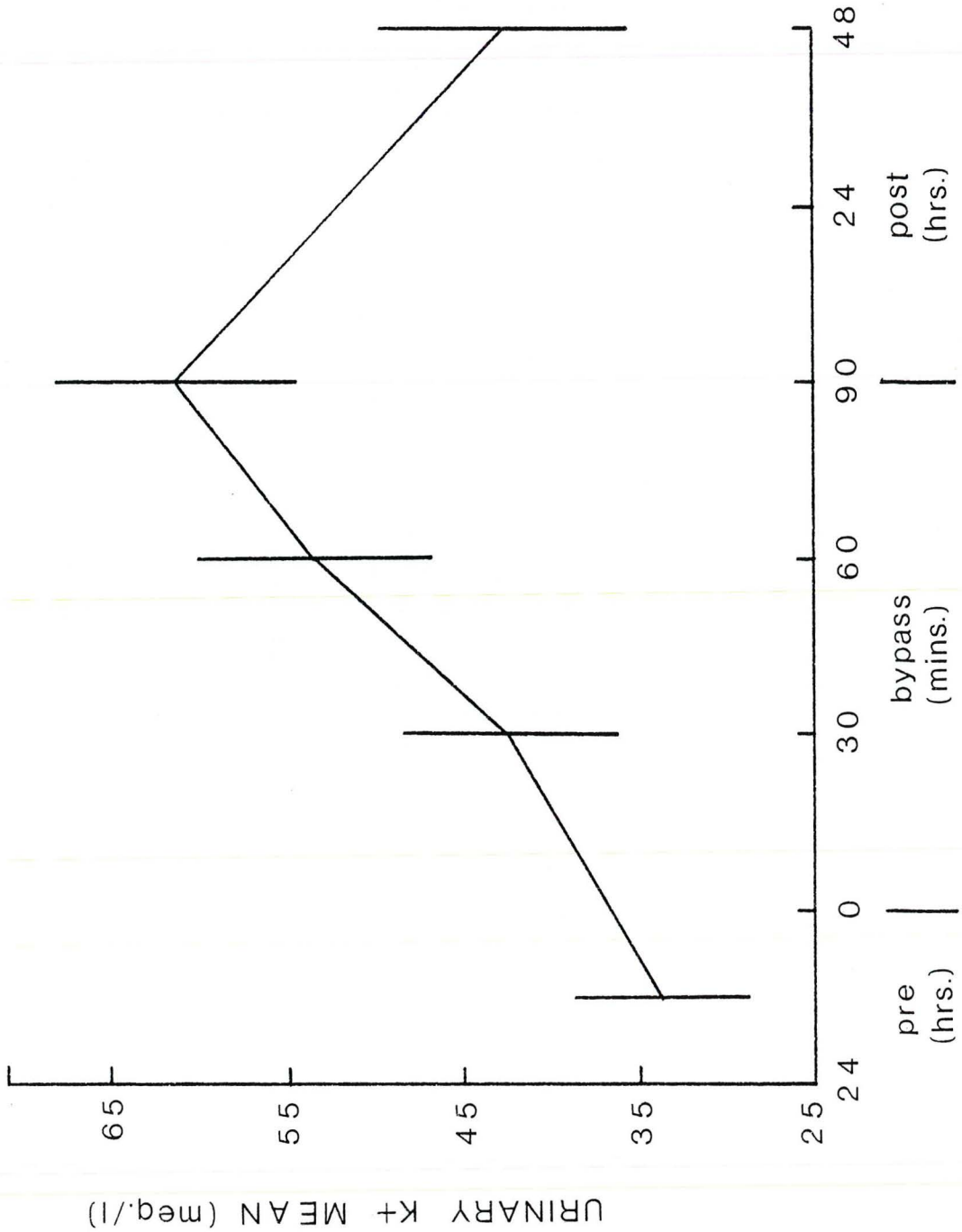

URINARY K+ MEAN (meq./l)
65
55
45
35
25
24
0
30
60
90
24
48
pre (hrs.)
bypass (mins.)
post (hrs.)

FIGURE 4

Pearson product-moment correlation values of potassium level of 20 cardiac surgical patients pre-bypass, during bypass, and post-bypass.

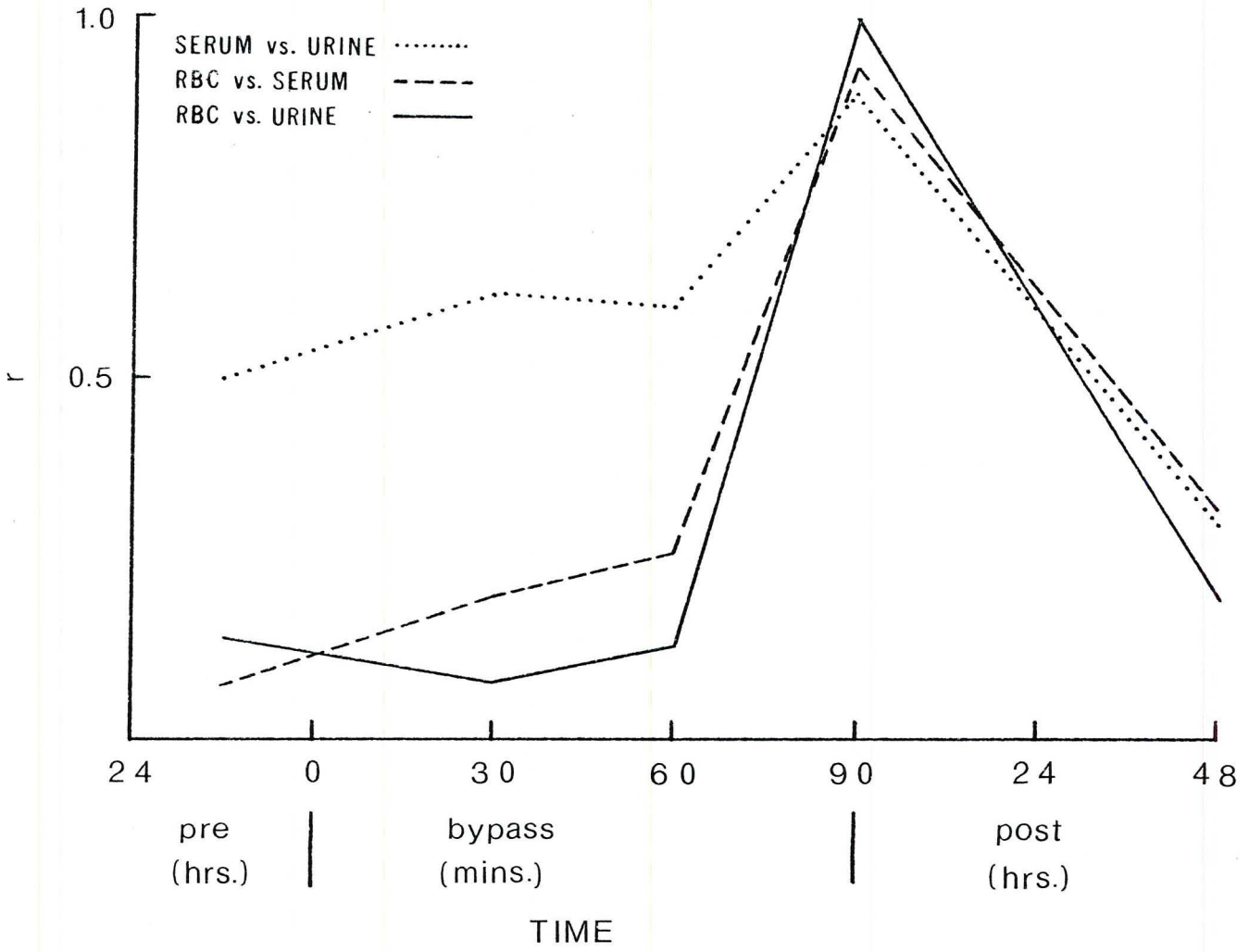

1.0
0.5
r
SERUM vs. URINE
RBC vs. SERUM
RBC vs. URINE
24
0
30
60
90
24
48
pre
(hrs.)
bypass
(mins.)
post
(hrs.)
TIME

FIGURE 4

DISCUSSION

Potassium ion regulation and exchange in the body compartments is affected by several mechanisms including: endocrine function; acid-base balance buffering systems; respiration; renal function, and; metabolic activity. All of these systems work simultaneously and in conjunction with one another, and none exclusively manipulates concentration of any electrolyte.

As with any surgery, insult to the body tissues causes the adenohypophysis to secrete greater quantities of corticotropin, ACTH, which exhibits a humoral effect on the adrenal cortex to permit the production of aldosterone. ACTH does not regulate the rate of aldosterone release, but a minimum level need be present for its secretion. Aldosterone acts directly on the renal tubules to increase the rate of potassium excretion (Guyton, 1976).

The supplemental doses of KCl administered during the surgery cause a brief increase in K^+ concentration in the serum. This elevated level results in an increase in aldosterone secretion by direct action on the zona-glomerulosa of the adrenal gland, and subsequently greater K^+ excretion. Serum K^+ concentration is the major aldosterone secreting mechanism. Not all supplemental potassium seems to be excreted early in the perfusion; rather some may be passed into the intracellular fluid (Lampard and Couves, 1970).

This theory is supported by the significant changes which occur in serum and urine K^+ levels as early as 30 minutes on bypass while RBC K^+ is not altered significantly until 60 minutes on bypass (Table 3).

Alternate mechanisms for aldosterone secretion are also in effect. A reduced cardiac output stimulates the production of aldosterone. Cardiac output is maintained below a normal level during this type of operation because of the reduced nutrient demands of all tissues due to hypothermia and level of anaesthesia. As cardiac output decreases, lower blood flow results in a reduced glomerular pressure in the kidney. A lower glomerular pressure results in a reduced glomerular filtration rate, which in turn causes a decrease in total sodium content in the tubular fluid as it passes the juxtaglomerular apparatus. This low sodium stimulates the release of renin from granules of the juxtaglomerular cells. It is believed that renin combined with converting enzyme causes formation of angiotensin II which subsequently causes a feedback constriction of the renal afferent arterioles. There is an apparent increase in colloid osmotic pressure in the peritubular capillaries as a result and tubular reabsorption of water and salt is markedly increased, with a corresponding increase in potassium secretion. An increase in angiotensin II in the extracellular fluid can cause an immediate but not long lasting effect to increase aldosterone secretion. The effect of

angiotensin II is about 100 times less potent percentage-wise than the direct effect of potassium ion on aldosterone secretion (Ganong, 1977).

Hemodilution and a reduced metabolic rate, due to induced hypothermia, are primarily responsible for extraction of potassium ions from the intracellular fluid (Dieter et al., 1970b). Since diluting the blood with lactated Ringer's solution reduces oxygen carrying capacity due to a lower proportion of RBC per given amount of volume, there is an overabundance of O_2 in the interstitial or extracellular fluid. This situation produces a slight respiratory alkalosis. Contributing to this state is the reduced O_2 consumption of the cell because of the lowered metabolic demand and the lowered blood flow rate (Figure 5). Since there is an increased amount of bicarbonate ion, due to addition of this ion for pH buffering in the oxygenator prime, H^+ is not readily available to compensate the alkalosis. However, the cationic species of potassium ion can be extracted from the intracellular fluid for this purpose (Gay and Ebert, 1968).

Another mechanism by which K^+ is transported from intracellular to extracellular fluid is suggested by Valtin (1973). In order to achieve a sufficient concentration of intracellular K^+, this ion must be actively transported across the cell membrane, expending energy to accomplish the task. In a hypothermic state, the metabolic rate is

lowered and the body is deprived of some energy. Since potassium ion passes readily by simple diffusion from intracellular to extracellular fluid it cannot be maintained in the cell adequately in the lowered energy state. From the combination of biochemical and biological mechanisms discussed here the reasons for a significant increase in correlation values between fluid compartments relevant to the exchange of K^+ are theorized.

Once bypass is terminated the patient is returned to normothermia and metabolic rate is increased. Cardiac output is greatly elevated. Twelve hours post-operatively a mechanical ventilator is no longer needed and respiration is returned to normal. Urine output is still above normal because of sodium retention and an increased circulatory volume from hemodilution (Nuutinen and Hollm, 1976). These conditions may explain the sustained increase in urinary K^+ output as compared to the control while serum and RBC K^+ levels are returned to approximate pre-operative values (Table 1; Table 3). Correlation values are reduced as potassium shifts are supressed (Table 4).

One aspect of the clinical significance of hypokalemia is the occasional cardiac patient with an irritable myocardium (Sliwi et al., 1974). Three patients in this study experienced cardiac arrhythmias attributable to hypokalemia. The conductive tissues of the heart like that of nerve tissue depolarizes in response to displacement of an electrical

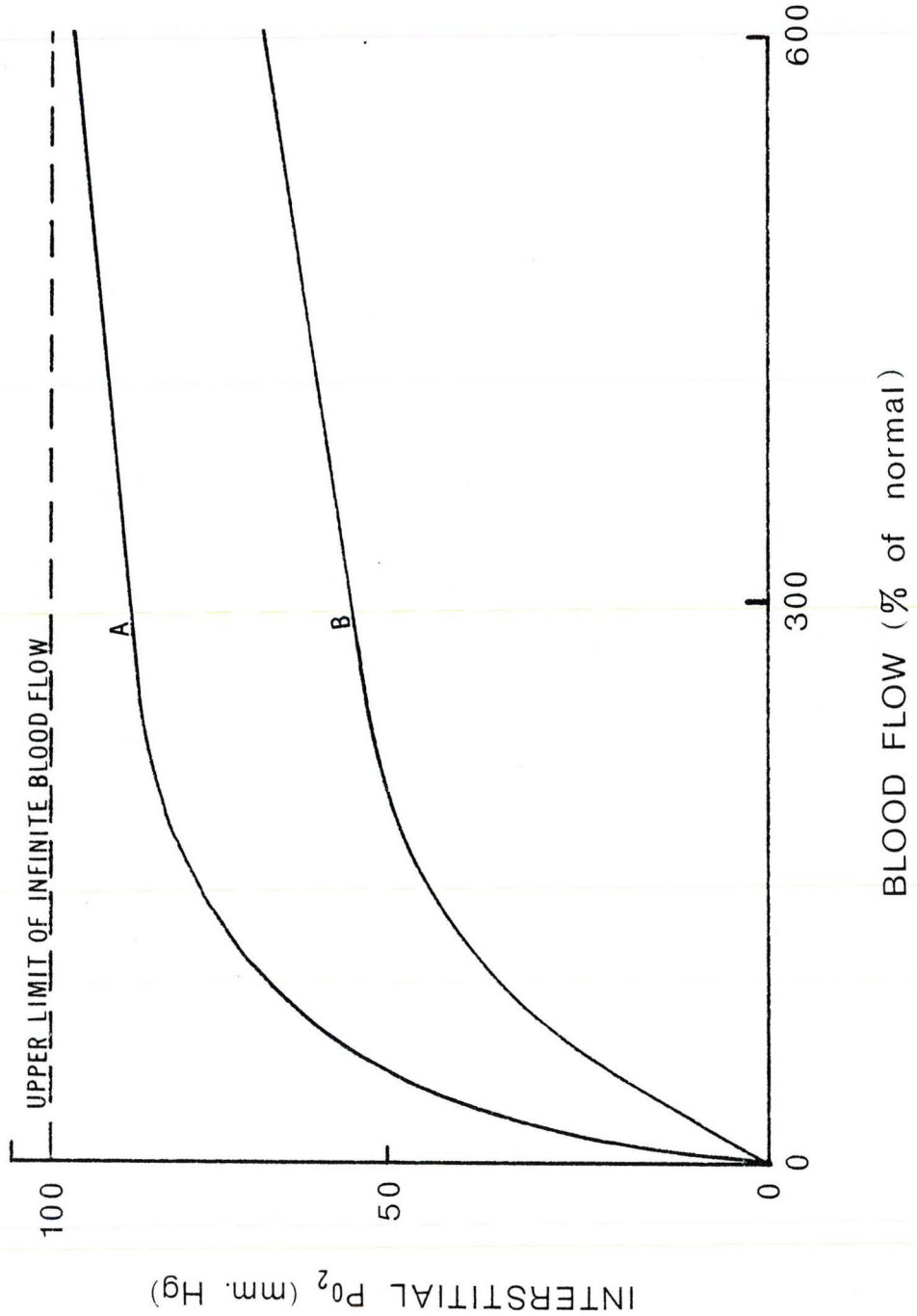
potential. There is a net negative charge on the inner membrane surface of conductive cells and a net positive charge on the outside resulting in a potential difference of -85 millivolts. If potassium is lost from the interior of the cell, increased negativity results. The resting membrane potential is now increased and must decrease by a greater amount to equal the threshold level of -60 millivolts for excitation. This condition causes a slowing of impulses, blockage at some point in the pathway, or total arrest of conduction (Ganong, 1977).

Another clinical consideration, as noted by Gay et al. (1975), is that potassium offers protection to the brain in periods of less than optimal perfusion. This observation is substantiated by the fact that K^+ is needed for utilization of glucose by the cell (Burke, 1976).

FIGURE 5

Effect of blood flow and rate of oxygen consumption on tissue pO_2 at (A) $\frac{1}{4}$ normal O_2 consumption and (B) normal O_2 consumption (Guyton, 1976).

FIGURE 5


UPPER LIMIT OF INFINITE BLOOD FLOW
A
B
100
50
0
0
300
600
INTERSTITIAL P_{O_2} (mm. Hg)
BLOOD FLOW (% of normal)

CONCLUSION

The alterations in K^+ levels were studied in twenty patients undergoing open heart surgery with cardiopulmonary bypass, employing buffered lactated Ringer's solution in the pump oxygenator. Supplemental potassium was administered to prevent endangering any of the patients. Serum K^+ levels were increased during the operation, but RBC K^+ levels were noticeably reduced during the bypass period. Urinary K^+ was increased during bypass and remained elevated 48 hrs following surgery. The stress of surgery with an increased secretion of aldosterone, reduction of cardiac output and metabolic rate, as well as respiratory alkalosis all played a role in the reduction of K^+ in the body fluids.

Since intracellular K^+ is a better indicator of total body potassium than that of serum K^+, possibly incorporation of this value in clinical treatment may prevent complications due to hypokalemic episodes. Also, closer observation and immediate correction of the eventual respiratory alkalosis of these surgical patients may prevent a certain amount of K^+ depletion.

APPENDIX

In order to safely and efficiently perform heart surgery, use of cardiopulmonary bypass is required. As the term implies, circulation and respiration of a patient is performed by an extracorporeal device, one outside of the body.

The patients observed in this study have coronary artery disease, or arteriosclerosis of the arteries supplying the heart. Since these arteries are blocked by plaques, they must be bypassed so that circulation to the heart muscle is restored. The greater saphenous vein is removed from the patient's leg and a portion of it is anastomosed to the aorta and the coronary artery distal to the plaque. This proceedure is repeated for each diseased artery. Since this operation is very tedious and exacting, the movement of the lungs and the heart must be stopped to simplify the process of inserting sutures to join the vessels.

To maintain the patient's circulation and respiration, blood is drawn from the patient through cannulas inserted in the superior and inferior vena cava. This blood is returned to an "oxygenator" then pumped back to the patient through a cannula inserted in the ascending aorta. The blood that is returned to the oxygenator from the patient flows by hydrostatic pressure through a 1/2 inch i.d. polyvinyl chloride tubing. The blood then passes over a perfor-

ated diffusion plate in the oxygenator which allows the passage of blood and gas. The gas is supplied by two source tanks, one containing 100% O_2 and the other containing 95% O_2 with 5% CO_2. As the blood passes over the diffusion plate it is forced through the perforations and consequently forms a bubble. The bubble provides a gas exchange surface in vitro for the red blood cell as does the aveoli of the lungs in vivo. The bubbles pass through columns in the oxygenator and burst eventually as the pressure is increased due to compaction. The gas is then vented to room air and the blood passes to a resevoir. The bursting of the bubbles causes some hemolysis so the RBC artifacts are filtered, and the unvented gas is absorbed by a defoaming agent before the blood passes to the resevoir. The resevoir now contains oxygenated blood, the degree of oxygenation being controlled by the flow of gas from the source tanks. Oxygenated blood is then mechanically pumped, by a roller, through 3/8 inch i.d. poly-vinyl chloride tubing to the patient via the aortic cannula. The patient's cardiac output and mean arterial pressure are controlled by blood flow rate.

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