OXYGEN AVAILABILITY IN BIOCHEMICAL MODALITIES OF KIDNEY RECIPIENTS

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ABSTRACT

This study was designed to evaluate the oxygen availability of KR in the perioperative period and to detect the impact of correction of MA on haemodynamic, oxygenation and biochemical variables. Tweenty KR were randomized for correction or no correction of MA. Each group consisted of 10 patients. Correction of MA was carried out by 5% $NaHCO_3$. Pulmonary catheter was used for measurments of heamodynamic variables. Haemodynamic variables recorded included HR, MBP, CVP, PCWP, CI; Gasometric variables included pH, $PacO_2$, HCO_3 and BD; measured O_2 variables included PaO_2, PVO_2 and SVO_2; calculated O_2 variables included CaO_2, A-aDPO_2, a-vDO_2, P^{50}, DO_2I, VO_2 I, $O_2ER\%$, and biochemical variables including mixed venous and arterial lactate.

The results of this study showed that there was a significant difference in the acid-base status after correction of acidosis. Otherwise, acidosis correction did not display any significant differences in haemodynamic, oxygenation and biochemical variables. We concluded that KR can maintain adequate tissue oxygenation and correction of MA has no value in improvement of oxygen availability.

INTRODUCTION

Patients with end stage renal failure (ESRF) and subjected to allograft renal transplantation may have limitation in oxygen delivery and availability to the tissues. They have cardiovascular, pulmonary, haematological and biochemical changes that may interfer with tissue oxygenation. Some have uraemic cardiomyopathy, and most are anaemic and suffering from metabolic acidosis (MA). During operation, those kidney recipients (KR) can be exposed to hypothermia due to use of

cold ischaemia during vascular anastmosis. Consequently, KR may suffer from multifactorial limitations of oxygen availability in perioperative period.

O_2 availability to the tissues is defined as the overall ability of the blood to deliver the necessary amount O_2 to the tissues which is suitable for its consumption(1). Adequacy of tissue oxygenation is the net result of O_2 delivery (DO_2) to tissues and O_2 consumption (VO_2) by the tissues. Therefore, tissue oxygenation is a global concept that can't be directly measured and no single measurement or calculation reliably reflects the oxygenation status of vital tissues(2). Monitoring of O_2 variables in clinical settings should include information about O_2 uptake in the lung, O_2 transport in blood, O_2 release to the tissues and O_2 consumption by the tissue.

This study was designed to evaluate the oxygen availability of KR in the pericperative period and to detect the imapct of correction of metabolic acidosis on haemodynamic, oxgynation and biochemical variables. Haemodynamic and O_2 variables will be calculated by themodilution (TD) technique.

PATIENTS AND METHODS

This study included 20 adult patients suffering from end stage renal failure (ESRF) and subjected to allograft renal transplantation. The study protocol was approved by the responsible authorities. Patients gave their written consents. Preoperative assessment included routine haematological and biochemical laboratory investigations, ECG and echocardiography. Kidney recipient (KR) patients were preimedicated with diazepam lOmg (orally) two hours before surgery. Lumber epidural block was performed at L2-3 interspace using 0.8mg. Kg^{-1} bupivacaine 0.5%, 1 mg.kg-1 lidocaine 2% and 30µg.mgkg-1 morphine. Arterial catheter (20G) was fixed in the radial artery for continuous BP monitoring. Pulmonary artery 5 lumen floating catheter was inserted through the right internal jugular vein and connected to Hewllett packard monitor (665). Thermodilution technique was used for measurement for cardiac output and other haemodynamic variables.

Preinduction with midazolam, morphine and atropine was followed by induction by thiopentone-suxamethonium. After endotracheal intubation anaesthesia was main-

tained with No2-02 mixture (FIO_2 = 35%) halothane 0.2-0.4% and tubocurarine. At the end of surgery residual effect of muscle relaxant was reversed by prostigmin and atropine.

Moderate hydration of patients during surgery was achieved by infusion of 60ml. kg^{-1} of normal saline and glucose 5% (2-1) until declamping. After the onset of diuresis, infusion rate was adjusted according to urine volume, central venous pressure (C.V.P.) and pulmonary capillary wedge pressure (PCWP).

Patients were randomly classified into two groups. Group I, Metabolic acidosis was not corrected and no bicarbonate solution was given. The second received sodium bicarbonate solution 5% for correction of their metabolic acidosis.

$NaCO_3$ solution was given in the morning of surgery slowely until the base defict (BD) was less than 5mmol L-1 and/or pH increased to 7.37. Additional calculated dose of bicarbonate might be required intraoperative or in early recovery time to maintain acid-base status near normal.

Monitoring and measurments :

Through the pulmonary artery floating catheter the thermodilution CO mesurment was taken as the avariage of three consecutive readings of temperature.

The following variables were recorded:

1- Haemodynamic variables: included heart rate (HR), mean arterial blood pressure (MBP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), cardiac index (CI).

2-Gasometric variables: included pH, arterial CO_2 tension ($PaCO_2$). serum bicarbonate (HCO_3-) and base deficit (BD), measured oxygen variables [arterial O_2 tension (PaO_2), arterial O_2 saturation (SaO_2), mixed venous O_2 tension (PVO_2) and mixed venous oxygen saturation (SV02)] and calculated O_2 values [arterial O_2 content (CaO_2), Alveoloarterial O_2 tension difference (A-aDpO_2), arteriovenous O_2 content difference (a-vDO_2), P^{50}, O_2 delivery index (DO_2I) O_2 consumption index (VO_2I), O_2 extraction ratio (O_2ER) and core body temperature.

The blood samle (lml) was taken in a heparin coated syringe closed tight-

ly and immediately analysed by blood gas analyser (ABL510, Radiometer, Copenhagen).

3- Biochemical variables: included mixed venous and arterial blood lactate were measured. Each blood sample (2ml) was put in a tube containing sodium fluoride as a preservative and immediately kept in slushed ice and analysed by Enzymatic-photometric method. The normal serum lactate in our laboratory by this technique was 4-16 $mg.dl^{-1}$ (0.3-1.8mEq. L^{-1}).

All haemodynamic, gasometric and biochemical variables were recorded at specific points, namely, before correction of MA, before induction of anaesthesia (after correction), 15 min after induction, before clamping of vessels to start vascular anastmosis, 15 min after declamping of vessels, end of surgery and two hours after surgery, the study end point.

Statistical analysis:

Data were computerized and SPSS statistical programme was used. The data obtained were tested for normality (parametric or nonparametric). Parametric data were analyzed by simple t-test. Nonparametric data were analyzed by Mann-Whitney-U test. data were presented as mean±SD. Difference between two groups was considered significant at $P<0.05$.

RESULTS

The patients demographic data are shown in table (1).

I- Acid-base status:

KR presented to surgery were all acidotic and hypocapnic. However, after correction of MA by 5% sodium bicarbonate, pH returned to normal value and was significantly higher than the acidotic group to the study end point (table 2). $PaCO_2$ was below normal values in the perioperative period indicating that KR were in a state of chronic hypocapnia and $PaCO_2$ did not show a significant difference between the acidotic and acidosis corrected KR during most of the anaesthesia time. However, $PaCO_2$ was significantly higher in the acidosis corrected KR 15 min. postinduction, end of surgery than those measured in the acidotic KR (table 2). HCO_3- was significantly higher in the corrected KR compared with the acidotic ones, (table 2). The maximum dose of 5% $NaHCO_3$ used for correction was 300 mEq. BD was significantly lower in the acidosis corrected group than the aci-

dotic KR. This continued to the study end (table 2).

II- Haemodynamic variables:

HR showed a significant increase in the acidosis-corrected KR compared with the acidotic group. This was cotinued during and after surgery (table 3). MBP was above normal value for all KR subjected to surgery and correction of the acidotic state did not elicit a significant effect (table 3). Measured CVP and PCWP (table 3) did not elicit a noticalbe difference between the two groups. These two variables increased gradually till the end of vascular anastmosis where they declined gradually due to the occurance of brisk diuresis. Cardiac index (CI) was measured by TD, CI was on the high normal value (table 4) and there was no significant difference between acidotic and acidosis corrected KR.

III- Oxygenation variables:

Measured arterial O_2 variables, PaO_2 and SaO_2 did not show stastistically significant difference between the acidotic and acidosis corrected KR, except that PaO_2 was significantly lower in the corrected group after vascular anastmosis and after surgery (table 4). All KR had a within normal preoperative mixed and central venous oxygen saturation (SVO_2) and tension (PVO_2) (table 5). PVO_2 was significantly lower in acidosis-corrected KR before of anaesthesia and after vascular anastmosis while central PO_2 was significantly lower in the corrected KR after surgery (table 5). All KR displayed low values for mixed or central oxygen tension and satuarion during recovery (table 5).

The preoperative total arterial oxygen content (CaO_2) was reducred and A-aDpO_2 was slightly higher than normal (table 6). However, there was no significant difference between the two groups (table 6). All KR have a normal preoperative P^{50} but after correction of MA, P^{50} decreased and become on lower normal value indicating shift of oxygen dissociation curve ODC to the left. However, during anaesthesia all KR showed low P^{50} without any significant difference between the two groups. Following surgery, acidosis-corrected subjects demonstrated a significant lower P^{50} value (table 6). The arterio-venous O_2 content difference (a-vDO_2) showed no significant difference between the acidotic and acidosis-corrected groups during the study

period whether calculated from mixed venous or central venous blood samples (table 6).

DO_2I calculated with TD technique was nearly within normal limits and remained above the critical level. Correction of the acidotic state did not display any significant changes in DO_2I. VO_2I also did not show any significant difference between the acidosis and corrected KR. (table 7) It is observable that V02I decreased in all KR during anaesthesia. After surgery, V02I was on high normal values. O_2 ER was within normal limits in the perioperative period in all patients and showed no significant difference between acidotic and acidosis-corrected KR. However, after surgery, O_2 ER raised to near the critical value (table 7).

IV. Biochemical profile:

Serum lactate-whether measured from arterial, mixed venous or central venous blood samples - was within normal limits preoperatively and all KR had the highest abnormal level of serum lactate in the postoperative period (table 8).

Body temperature changes are shown on (table 9), when a decrease down to 33.5 in postoperative period was recorded.

The results of this study can be summerized and capsulated into the following:

Correction of MA, as carried out in this study, produced:

1- Changes that appeared after the correction and remained during the whole studied period. These include:

a) Increase in HR, b) increase in pH and plasma HCO- and decreased in the degree of BD.

2- Changes that appeared after the completion of surgery. These include:

a) Oxygenation: decreased in PaO_2 and P^{50}, b) Gasometric: increase PaCO

Table (1): Clinical data of kidney recipients (KR). Values are mean (range).

	Acidotic (A)	Acidosis-corrected (c)
Number of patients	10	10
Sex (M/F)	9/1	9/1
Age (yr)	32.0 (24-47)	37.2 (30-52)
Body weight (kg)	62.3 (50-72)	65.4 (49-91)
Height (cm)	169 (157-186)	169.1 (154-175)
Body surface area (m^2)	1.7 (1.49-1.91)	1.8 (1.46-2.13)

Table (2): Perioperative pH, arterial Carbon dioxide tension ($PaCO_2$), serum bicarbonate (HCO_3) and base deficit (BD) of the acidotic (A) and acidosis-corrected (C) KR. (n=20 each). Values are mean (SD).

* Significant difference between A and C, $P<0.05$

	PH		PaCO2 (mmHg)		HCO3 (mmol/l)		BD (mmol)	
	A	C	A	C	A	C	A	C
- Before correction	7.35 (0.06)	7.33 (0.05)	30.1 (3.0)	28.4 (3.2)	16.4 (2.9)	14.9 (2.8)	8.3 (3.6)	9.9 (3.5)
- Preinduction (after correction)	7.35 (0.05)	7.40* (0.03) (n=19)	29.7 (3.0)	29.6 (3.3) (n=19)	16.4 (2.9)	18.1* (2.1) (n=19)	8.4 (3.5)	6.2* (2.1) (n=19)
- 15 min post-induction	7.36 (0.07)	7.40* (0.04)	27.0 (3.2)	29.2* (3.2)	15.7 (2.4)	18.0* (1.5)	9.0 (3.1)	6.4* (1.7)
- Before vascular anastmosis	7.36 (0.08)	7.40* (0.04)	25.7 (3.7)	28.2 (4.1)	14.8 (2.3)	17.6* (1.5)	10.1 (3.1)	6.8* (1.4)
- After vascular anastmosis	7.34 (0.07)	7.38* (0.04)	25.3 (3.4)	27.7 (4.0)	14.0 (2.2)	16.8* (1.6)	11.3 (3.0)	7.9* (1.5)
- End of surgery	7.34 (0.07)	7.93* (0.04)	26.0 (4.2)	29.6* (5.1)	14.3 (2.2)	17.8* (2.6)	11.1 (3.0)	6.7* (2.3)
- 2h post-surgery	7.27 (0.05)	7.37* (0.03)	29.7 (2.8)	31.9* (1.8)	13.9 (1.8)	18.0* (1.8)	12.1 (2.5)	6.9* (1.8)

Table (3): Perioperative heart rate (HR), Mean arterial blood pressure (MBP) and central venous pressure (CVP)and pulmonary wedge pressure (PCWP) of the acidotic (A) and acidosis-corrected (C) KR. (n=20 each). Values are mean (SD).
* Significant difference between A and C, P<0.05

	HR (bpm)		MBP (mmHg)		CVP (mmHg)		PCWP	
	A	C	A	C	A	C	A	C
- Before correction	85 (16)	96 (13) (n=11)	114 (13)	103* (12) (n=11)	2 (3)	2 (2)	9 (3)	9 (4) (n=6)
- Preinduction (after correction)	84 (16)	92 (13)	114 (18)	98* (20)	3 (2)	3 (3)	9 (4)	11 (3)
- 15 min post-induction	80 (14)	89* (12)	98 (14)	91 (16)	6 (3)	5 (3)	12 (5)	13 (4)
- Before vascular anastmosis	72 (16)	79 (16)	111 (17)	114 (23)	8 (2)	8 (3)	14 (6)	15 (5)
- After vascular anastmosis	74 (14)	82* (13)	109 (16)	106 (15)	10 (3)	9 (4)	16 (5)	16 (6)
- End of surgery	69 (13)	80* (12)	107 (13)	109 (16)	6 (30	5 (3)	10 (4)	13 (6)
- 2h post-surgery	79 (18)	90* (12)	106 (11)	106 (11)	2 (2)	2 (2)	8 (2)	9 (3)

Table (4): Perioperative arterial oxygen tension (PaO_2) and saturation (SaO_2), and cardiac index (CI) of the acidotic (A) and acidosis-corrected (C) KR. (n=20 each). Values are mean (SD).

* Significant difference between A and C, $P<0.05$

	PaO_2 (mmHg)		SaO_2 (%)		CI (L/min/m^2)	
	A	C	A	C	A	C
- Before correction	97.4 (12.8)	97.2 (13.5)	97.0 (1.1)	96.8 (1.2)	5.2 (1.1)	5.3 (0.6) (n=6)
- Preinduction (after correction)	97.4 (12.9)	93.2 (13.4) (n=19)	97.1 (0.9)	97.0 (1.2) (n=19)	5.0 (1.3)	5.2 (0.6)
- 15 min post-induction	165.6 (35.4)	141.4 (47.8)	98.5 (0.6)	98.0 (1.4)	4.0 (1.2)	4.4 (0.8)
- Before vascular anastmosis	158.5 (35.7)	136.1 (45.4)	98.4 (0.7)	98.0 (1.5)	3.8 (1.3)	4.5 (0.9)
- After vascular anastmosis	154.1 (36.2)	125.4* (46.4)	98.5 (0.7)	97.9 (1.6)	4.5 (1.0)	4.7 (1.1)
- End of surgery	150.8 (38.1)	125.3 (44.8)	98.4 (0.7)	97.7 (2.2)	3.9 (0.9)	4.0 (0.9)
- 2h post-surgery	92.6 (12.9)	81.2* (15.6)	96.5 (1.20	96.3 (1.5)	3.9 (0.9)	4.0 (0.9)

Table (5): Perioperative mixed and central venous oxygen tension (PVO_2) and saturation (SVO_2) in the acidotic (A) and acidosis-corrected (C) KR. (n=10 each). Values are mean (SD).

* Significant difference between A and C, P<0.05

	(Mixed Venous samples)				(Central Venous samples)			
	PVO_2		SVO_2		PVO_2		SVO_2	
	A	C	A	C	A	C	A	C
- Before correction	39.0 (4.6)	39.7 (3.4) (n=6)	74.6 (7.2)	76.1 (5.9) (n=6)	39.6 (4.0)	39.6 (6.2) (n=7)	75.1 (6.2)	77.5 (4.3) (n=7)
- Preinduction (after correction)	39.4 (4.6)	35.3* (3.6) (n=9)	76.2 (5.2)	73.3 (5.9) (n=9)	38.1 (3.3)	36.7 (2.2)	75.1 (4.0)	75.8 (5.0)
- 15 min post-induction	40.5 (6.6)	39.5 (3.9)	78.7 (5.8)	77.0 (6.3)	45.4 (7.3)	40.9 (6.5)	80.3 (15.7)	80.8 (6.9)
- Before vascular anastmosis	37.5 (7.7)	35.8 (5.4)	75.2 (6.2)	75.6 (6.3)	42.3 (6.1)	39.5 (5.4)	82.0 (6.0)	80.1 (6.8)
- After vascular anastmosis	40.1 (6.0)	33.7* (3.8) (n=9)	79.5 (5.9)	74.0 (6.9)	44.6 (4.7)	40.1 (5.6)	84.4 (4.4)	81.3 (6.0)
- End of surgery	38.3 (6.0)	34.6 (5.9)	77.0 (6.2)	72.0 (7.3)	41.8 (6.2)	37.0 (5.7)	81.5 (5.3)	76.1 (7.5)
- 2h post-surgery	34.9 (3.5)	32.9 (6.4)	68.6 (7.1)	67.7 (7.4)	38.0 (6.1)	32.3* (3.9)	69.4 (11.2)	66.1 (7.4)

Table (6): Perioperative arterial oxygen content (CaO_2), alveolo-arterial oxygen tension difference (A-a DPO_2, P^{50} of the acidotic and artero-venous oxygen content difference (a-VDO_2) of acidotic (A) and acidosis-corrected (C) KR. (n=20 each). Values are mean (SD).
* Significant difference between A and C, P<0.05

	CaO_2 (ml.dl^{-1})		A-a DPO_2(mmHg)		P^{50}(mmHg)		a-V DO_2(ml.dl^{-1})	
	A	C	A	C	A	C	A	C
- Before correction	11.2 (2.8)	11.1 (2.3)	18.5 (12.0)	19.8 (13.1)	27.0 (2.8)	27.7 (23.3)	2.5 (0.7)	2.3 (0.3) (n=6)
- Preinduction (after correction)	11.1 (2.7)	10.3 (2.4) (n=19)	19.7 (12.3)	23.2 (12.9) (n=18)	26.4 (2.7)	24.7* (2.0) (n=19)	2.4 (0.6)	2.3 (0.3)
- 15 min post-induction	10.12 (2.7)	9.3 (2.3)	54.0 (34.8)	68.7 (42.2)	23.8 (2.4)	24.1 (3.4)	2.2 (0.7)	1.9 (0.3)
- Before vascular anastmosis	10.0 (2.4)	9.2 (2.1)	64.1 (34.4)	74.6 (41.6)	23.3 (2.7)	22.7 (2.7)	2.4 (0.7)	2.1 (0.3)
- After vascular anastmosis	10.0 (2.7)	9.8 (2.3)	69.3 (34.9)	91.0 (43.6)	23.2 (2.5)	21.9 (1.5)	2.1 (0.6)	2.3 (0.6)
- End of surgery	10.6 (3.1)	10.2 (2.2)	66.9 (36.5)	88.8 (41.5)	23.4 (3.5)	22.4 (2.0)	2.5 (0.8)	2.6 (0.5)
- 2h post-surgery	10.1 (2.4)	9.7 (2.1)	24.5 (12.4)	32.5 (15.9)	27.4 (3.5)	24.1* (2.2)	3.0 (1.2)	2.6 (0.5)

Table (7): Perioperative oxygen delivery index (DO_2I) oxygen consumption (V2OI) and oxygen extraction ration (O_2ER% of the acidotic (A) and acidosis-corrected (C) KR. (n=10 each). Values are mean (SD).

	DO_2I (ml/min/m^2)		VO_2I (ml/min/m^2)		O_2ER(%)	
	A	C	A	C	A	C
- Before correction	544 (170)	564 (152) (n=6)	124 (30)	121 (8.3) (n=6)	0.24 (0.07)	0.23 (0.07) (n=6)
- Preinduction (after correction)	507 (169)	460 (141)	110 (42)	117 (27)	0.22 (0.05)	0.26 (0.07)
- 15 min post-induction	373 (139)	352 (166)	81 (21)	80 (10)	0.23 (0.05)	0.24 (0.06)
- Before vascular anastmosis	364 (173)	352 (115)	83 (19)	85 (17)	0.26 (0.09)	0.26 (0.07)
- After vascular anastmosis	432 (148)	408 (156)	88 (18)	100 (18)	0.22 (0.05)	0.26 (0.07)
- End of surgery	368 (119)	381 (120)	88 (23)	98 (18)	0.24 (0.06)	0.29 (0.08)
- 2h post-surgery	510 (186)	454 (188)	148 (55)	130 (39)	0.30 (0.08)	0.31 (0.08)

Table (8): Perioperative lactate concentration ($mg.dl^{-1}$) measured in arterial, mixed venous and central venous blood- of the acidotic (A) and acidosis-corrected (C) KR. Values are mean (SD).

	Arterial lactate		Mixd venius lactate		Central venous lactate	
	A	C	A	C	A	C
- Before correction	11.1 (4.5)	15.8 (8.4) (n=10)	11.5 (5.9)	15.1 (8.4) (n=5)	10.9 (5.1)	16.4 (8.2) (n=5)
- Preinduction (after correction)	11.8 (4.9) (n=18)	14.1 (7.5) (n=17)	12,6 (6.2)	13.3 (7.1) (n=9)	11.0 (5.7)	14.5 (8.2)
- 15 min post-induction	13.4 (5.2)	15.6 (7.4) (n=17)	14.2 (6.5)	15.1 (6.8) (n=9)	12.9 (4.6)	15.9 (8.2)
- Before vascular anastmosis	14.2 (4.3)	16.1 (7.3)	14.7 (5.7)	15.2 (6.9)	14.8 (4.3)	17.2 (7.8)
- After vascular anastmosis	16.4 (5.5)	18.7 (8.3)	16.5 (7.0)	18.4 (7.7)	18.2 (5.8)	18.8 (7.4)
- End of surgery	19.1 (7.2)	21.4 (9.3)	18.1 (6.2)	20.8 (6.9)	20.0 (5.8)	22.6 (11.6)
- 2h post-surgery	20.1 (8.4)	23.0 (11.2)	20.9 (6.9)	21.8 (7.5)	20.7 (9.4)	26.0 (16.1)

Table (9): Perioperative central and Core temperature (degree celcius) of the acidotic (A) and acidosis-corrected (C) FK. KR. (n=20 each).

	Central body temperature		Core body temperature	
	A	C	A	C
- Before Correction	35.8	36.3	35.8	36.0
- Preinduction(after correction)	35.6	35.7	35.5	35.3
- 15 min post-induction	34.5	34.7	34.6	34.5
- Before vascular anastmosis	34.0	34.3	34.0	33.9
- After vascular anastmosis	33.6	33.9	33.4	33.3
- End of surgery	33.6	33.9	33.4	33.3
- 2h post-surgery	34.9	34.0	34.5	35.1

DISCUSSION

Tissue oxygenation is a global concept. The adequacy of oxygenation is indicated by the net result of DO_2 and VO_2. In order to get a full picture about tissue oxygenation, it is essential to monitor O_2 variables that indicate O_2 uptake in the lung, O_2 transport to tissues, O_2 release and consumption, in addition to acid-base status. KR have limitations in their O_2 availability. They have haemodynamic, pulmonary, haematological and biochemical changes that may interfer with tissue oxygenation.

Haemodynamics of KR are usually compromised. Hung et al., 1980(3) reported an increase in the preload due to sodium retention, increase in blood volume and increments in venous return by arteriovenous fistula. However, in this study preload of either the right ventricle (CVP) and the left ventricle (PCWP) were within normal limits. This probably because all KR underwent haemodialysis the day before surgery.

Pulmonary functions are affected due to left ventricular dysfunction or increased pulmonary capillary permeability and transudation of fluid(4). Diffusion through the lung may be affected and this is indicated by increased A-aDpO_2 due to severe chronic anaemia(5). This has been proved in these studied KR as they have a slightly high values of $AaDpO_2$. Severe anaemia is charactersitic for all ESRF patients. In this study CaO_2 is similarly reduced by 50%. Various compensatory mechanisms exist to overcome this decrease in O_2 carrying capacity including increase in CO and 2,3 DPG, shifting of ODC to right. This is fairly observed in KR as P^{50} value - which is the mirror of position of ODC - was on the high range of normality.

Metabolic acidosis is a common feature of patients with end stage renal failure and it usually mild and asymptomatic(4). In this study serum HCO_3- was around 15 mmol/ and $PaCO_2$ was around 30mmHg. This state of mild acidosis of prime importance first to protect the patients from hypoclaemic tetany and second to facilitate O_2 release to the tissues by shifting the oxygen dissociation curve to the right. Sears, 1988(6) advised that; keep an adequate tissue oxygenation for this group of patients, the acute fall of CO and increasing the pH should be avoided. In this study half correction of base deficit resulted in significant increase in pH up to 7.4

but HCO_3 to rise the normal range while $PaCO_2$ did not changed transfering the patient from mild acidaemia and hypocapnia into a state of neutralaemia and hypocapnia.

All KR had a normal P^{50} value but as a result of acidosis correction, it decreased significantly compared with acidotic patients. Correction of MA did not elicit significant changes in oxygenation variables except PVO_2 which was significantly lower after correction of acidosis. Also, P^{50} was significantly lower in corrected group indicating shift of ODC to left.

Haemodianamically, correction of MA did not display significant changes during anaesthesia except HR which showed a significant rise in most intraoperative readings. The explanation is - to some extent obscured but it may be the hyperosmolar 5% $NaHCO_3$ that causes volume expansion and increases preload leading to increased HR, but against this is the insignificant difference of CVP, PCWP and CO.

Similarly, correction of acidosis did not significantly change oxygenation during anaesthesia. Most variables did not elicit a significant difference between acidotic and corrected groups in the postoperative period. However, arterial and central venous PO_2 were significantly lower in the corrected group compared with the acidotic one.

KR, whether acidostic or neutralaemic, displayed adequate oxygenation as indicated by normal values of mixed venous oxygen saturation (SVO_2) and serum lactate. It is evident that SVO_2 indicates tissue O_2 reserve[7]. Schmidt et al., 1984[8] reported that SVO_2 value more than 65% represented an adequate tissue oxygen reserve. This denotes that KR are able to maintain a normal tissue O_2 reserve in the periopeartive period whether the patients remain acidotic or acidosis was corrected. This normal tissue O_2 reverse is not affected to some extent by the marked reduction of O_2 transport to tissues. This can be further explained on the basis of concomitant reduction of oxygen consumption by the tissues so that O_2 extraction ratio remains on the normal values and venous blood returning from the tissues has a normal SVO_2. This reduction in oxygen consumption may be attributed to hypothermia that occurs intraoperatively (mean body temperature was down to

33.5°C at end of surgery). Therefore, it seems that this hypothermia is beneficial to meet the reduction of oxygen delivery.

The second evidence of adequate tissue oxygenation in KR was the normal serum lactate value seen in the pre-and intraoperative period. In recovery unit, serum lactate was above normal range in both groups of patients indicating that the tissues may be exposed to oxygen debt in the immediate post-operative period. Another observation recorded in KR in this time was that most of them did not shiver inspite of their low body temperature (around 34.6°C). This phenomenon is difficult to explain.

In conclusion, firstly, tissue oxygenation in KR was preserved in the perioperativee period. This adequate oxygenation can be attributed to the different compensatory mechanisms occuring in ESRF, namely, hyperdynamic circulation increasing tissue perfusion, shift of ODC to right facilitating O_2 release to tissue and hypothermia decreasing VO_2. Secondly, correction of mild metabolic acidosis has no value in improvement of tissue oxygenation.

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