

# BASE DEFICIT IN IMMEDIATE POSTOPERATIVE PERIOD OF OPEN HEART SURGERY AND OUTCOME OF PATIENTS

H. Teimori<sup>1</sup>, F. Sabzi<sup>2</sup>, V. Hassani<sup>3</sup>, S. Nadri<sup>1</sup> and H. Mahmoudvand<sup>4\*</sup>

1) Department of Anesthesiology, Lorestan University of Medical Sciences, Khoram-Abad, Iran

2) Department of Anesthesiology, Imam Ali Hospital, Kermanshah University of Medical Sciences, Kermanshah, Iran

3) Department of Anesthesiology, Iran University of Medical Sciences, Tehran, Iran

4) Lorestan University of Medical Sciences, Khoram-Abad, Iran

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**Abstract-** Base deficit is a non-respiratory indicator of acid base status. Aim of this study is to assess relationship between the base deficit value in immediate post operative period of CABG and valvular heart disease with cardiopulmonary and in hospital outcome of patient. A total of 136 consecutive with CABG and valvular heart disease scheduled in study. 20 variables were determined during the pre-intra- and postoperative period. Statistical univariate analysis was performed differentiating patients whose initial base deficit after weaning from cardiopulmonary bypass was  $-8$  meq and these whose base deficit was equal or more than  $-8$  meq. Secondly a logistic regression model was performed on the variables shown to have a statistically significant difference in univariate analysis with determination of the odd ratio. 3 variables had a statistically significant difference in univariate analysis and 2 of them high lighted by the linear logistic model. The value of base deficit measured during the immediate postoperative open-heart surgery is correlated with volume of fresh frozen plasma and blood transfusion after open heart surgery and using of intra aortic balloon pump after surgery.

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## INTRODUCTION

The base deficit is a non-respiratory indicator of acid-base statuses that evaluates the severity of shocked at the cellular level. A drop in the total number of base or buffers such as bicarbonate, hemoglobin and plasma protein reflect between tissue oxygen supply and demand.

Base deficit results from cellular metabolism of pyruvate under anaerobic condition (1). Therefore base deficit related to total oxygen debt and the magnitude of tissue hypo perfusion (2, 3).

Several studies have suggested that base deficit has prognostic value in patients with circulatory shock (4-6). A base deficit from 0 to  $-2$  is considered normal mildly in adequate cellular and tissue per fusion is reflected in a base deficit between  $-3$  and  $-5$ , a moderate base deficit is between  $-6$  and  $-9$  and a severe base deficit is  $-10$  or greater. Advanced age or alcohol use falsely increases the reading (1). The purpose of this study is to test the hypothesis that an elevated base deficit in open-heart surgery patients is indicative of impaired systemic oxygen utilization and portends poor outcome.

## MATERIALS AND METHODS

This study was a retrospective analysis of a prospectively-collected database. The study population included all patients (136 consecutive)

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**\* Corresponding Author:**

H. Mahmoudvand, Lorestan University of Medical Sciences, Khoram-Abad, Iran

Tel: +98 916 6616195

Fax: +98 916 6616195

E-mail: hormoz\_mahmoudvand@yahoo.com

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admitted to cardiac intensive care after open heart surgery. We obtained informed consent from all patients.

The patients with alcohol abuse and more than 75 year old were excluded from study. The base deficit obtained from the first blood gas after weaning from cardiopulmonary bypass and the patients were divided in to those who maintained a persistently high base deficit ( $\geq 8$  mmol/l) (group A) and those who achieved base deficit ( $< 8$  mmol/l) (group B). Variable compared with *t* test (continuous variable) and X<sup>2</sup> as univariate analysis and a linear regression model was performed on the variable shown to have a statistically significant difference in univariate analysis with determination of the odd ratio. ROC curve was obtained for determination of sensitivity and specificity of this test. Factors considered for univariate analysis include age, sex, body surface area.

All patients were treated with the same CPB technique. After systemic heparinization the aorta and vena cava were cannulate and CPB initiated using either roller or centrifugal pump, membrane oxygenators were used and circuit was primed with 2 l of a crystalloid solution (Ringer lactate). Systemic temperature was decreased to 28 °C during perfusion. Flow rate were maintained at 2.2 l.min.m<sup>2</sup> at 37 °C and 2 l.min.m<sup>2</sup> at 28 C. mean systemic arterial pressures was continuously monitored and maintained between 60 and 70 mmHg. Arterial blood samples were drawn after weaning from CPB and stored immediately on ice to prevent lactate production blood gas was measured on a ABX commercial analyzer (Nuva USA). For the purpose of the study base deficit levels after CPB with a pump prime solution containing lactate as in ringers lactate were expected to be normally between 0- -2 mmd/l. according to one previous study (1).

Hypertension and diabetes were diagnosed if the patient had a history of the condition. Congestive heart failure was defined as present or previously documented episode of pulmonary congestion in a patient with left ventricular dysfunction. Left ventricular function was evaluated by echocardiography.

Myocardial infarction documented by new Q wave on the electrocardiogram or CPK MB concentration higher than 100 IU/L/ 24 to 48 hours after the operation. Low cardiac output syndrome defined by the use of or inotropic drugs for 24 hours or more or the use of an intra aortic balloon pump. Pulmonary dysfunction as mechanical ventilation for 48 hours or more or acute respiratory failure necessitating reintubation and renal failure as creatinine more than 1.5 mg/L and post operative mortality was defined as death occurring during hospitalization for operation.

## RESULTS

Patients in the study (n=136) had a mean age of 54 ± 10/67 years. Preoperative and intraoperative characteristics of patients are shown in table 1.

There were 88 men and 47 woman. Coronary artery bypass operation was performed in 80 of the study population and valvular operation in 56. Among the patients included in the present study, 39 patient had base deficit levels less than -8 and 91 had a level of  $\geq -8$  meq/l. Patients with a base deficit level of -8 meq or more were older and were more of them woman. The prevalence of left ventricular ejection fraction less than 30% and coronary artery disease was not significantly higher in patients with base deficit of  $\geq -8$ . No difference were found according to the presence of hypertension or diabetes with base deficit levels of -8 or higher than in patients with base deficit levels less than -8 (*P* value  $> 0.05$ ). According to ROC curve analysis the best threshold value of base deficit for predicting the occurrence of postoperative complication (Table 2) had sensitivity and specificity of 0/50.

A logistic regression model was performed on the variables shown to have a statistically significant difference in univariate analysis with determination of odd ratio, 14 variables had a statistically significant difference in univariate analysis and three of them high lighted by linear regression model that one of them was base deficit value in the 1<sup>st</sup> hour post operatively (Table 3).

**Table 1.** Preoperative and intraoperative characteristics of 136 patients undergoing cardiac operation according to base deficit levels after cardiopulmonary bypass

Variable	$\geq -8$ meq/L	$< -8$ meq/L	P
Age	55.3 $\pm$ 12.4	54.1 $\pm$ 10.58	NS
Men	61.1%	65.8%	NS
Woman	38.9%	34.2%	NS
Diabetes	29.4%	25.4%	NS
Preoperative Hemoglobin	13.6 $\pm$ 1.2	13.8 $\pm$ 1.2	NS
CHF	2%	6.5%	0.04
Hypertension	35%	27%	NS
Redo operation	1.6%	7.1%	NS
Emergency operation	0	4.5%	NS
CPB time	90 $\pm$ 17	83 $\pm$ 21	0.05
Clamp time	46 $\pm$ 8	43 $\pm$ 13	NS
Lowest HB (gr/l)	11.3 $\pm$ 1.3	11.8 $\pm$ 1.7	NS
Maximum CPB flow	3996 $\pm$ 1338	4267 $\pm$ 985	NS
Minimum CPB flow	3263 $\pm$ 845	3877 $\pm$ 3514	NS

Abbreviation: NS, not significant; CHF, congestive heart failure; CPB, cardiopulmonary bypass; HB, hemoglobin.

**Table 2.** Postoperative characteristics of 136 patients undergoing cardiac operation according to base deficit levels after cardiopulmonary bypass

Variable	$\geq -8$ meq/L	$< -8$ meq/L	P value
Neurological complication	6.36%	3.64%	NS
Myocardial infarction	9.0%	1.0%	0.01
Mortality (hospital)	6.67%	3.33%	NS
Homodynamic stability	7.19%	2.81%	0.004
Pulmonary complication	7.27%	2.73%	NS
IABP	4.44%	5.56%	0.013
Gastro intestinal complication	6.92%	3.08%	NS
Inotropic using	5.71%	4.29%	0.001
Renal failure	1.00%	0 %	NS
Infection	6.67%	3.33%	NS

Abbreviation: NS, not significant.

## DISCUSSION

Outcome after cardiac operation is determined by preoperative characteristics of the patient in addition to intraoperative factors such as surgical technique, myocardial protection, and CPB.

Several risk factor models have been developed to predict outcome after cardiac operation but very few consider intra operative events (5-16).

Such intra operative factors may identify the postoperative course. Monitoring of such factors during the surgical procedure may lead to early therapeutic intervention that might improve outcome. Cardiopulmonary bypass is widely used to maintain systemic perfusion and oxygenation during coronary artery bypass and other open heart surgery. Tissue hypoperfusion with lactic acidosis during CPB may occur despite normal gas concentration (10). Therefore high blood lactate level during CPB may reflect as base deficit and may be used as a marker of inadequate tissue oxygen delivery.

The goal of this study was to evaluate the association between base deficit after CPB and postoperative morbidity and mortality in an adult cardiac surgical population. Under anaerobic condition, oxidative phosphorylation is not possible and ATP is produced from pyruvate metabolized to lactate that reflected as base deficit.

In this situation, base deficit is a sensible marker of the magnitude of anaerobic metabolism and tissue oxygen deficit (1). Several studies have showed a strong positive correlation between base deficit and the risk of morbidity and mortality in clinical situation such as circulatory shock, extra corporal support and in children after operation for complex congenital heart disease (1-4, 17, 18).

As early as 1964, Broder and Weil (2) documented that only 11% of patients with excess levels of base deficit survived circulatory shock. Later these authors showed that as base deficit increased from -2 to -8 mmol/ L the probability of survival from shock decreased from 9% to 10% (3). These finding were confirmed more recently by other groups (1, 4).

More over serial base deficit determination was also reported to be useful to assess response of patients in shock to therapy as a decrease in base

**Table 3.** Summary of linear regression of base deficit

Variable	Unstandardized Coefficients		Standardized Coefficients	<i>t</i>	<i>P</i> value
	B	Std. Error	Beta		
Preoperative. HB	7.49	4.28	0.23	1.75	0.088
Renal. Complication	9.09	4.58	0.204	1.98	0.054
ICU stay	-1.92	0.85	-0.84	-2.24	0.031
Minimum hemoglobin during CPB	-1.39	0.49	-2.07	-2.83	0.007

Abbreviations: HB, hemoglobin; ICU, intensive care unit; CPB, cardiopulmonary bypass.

deficit exceeding -6 mmol/L (19). In high risk general surgical patients a positive correlation between the estimated intraoperative oxygen deficit and post operative base deficit was observed (20).

It was concluded that base deficit determination maybe used to assess the degree of accumulated oxygen deficit and in titrating therapy to support postoperative physiologic compensation. Tissue perfusion and oxygenation during CPB is achieved by adjusting flow rate, temperature, gas flow, and hemoglobin to maintain oxygen delivery. Monitoring of the balance between oxygen supply and demand usually consists of serial arterial blood gas determination.

In 1958, Clowes and colleagues (6) reported the occurrence of metabolic acidosis with base deficit determination during CPB. In some patients early experimental evidence showed the critical value of high perfusion flow rate and hypothermia in preventing lactic acidosis and base deficit during CPB (6, 8).

Several contributing factors have been linked to regional tissue hypo perfusion and increased base deficit during CPB, recently splanchnic hypo perfusion during CPB, related to the production of endogenous vasoactive mediators and a decrease in arterial oxygen content during bypass was proposed as an important event in the generation of lactate and increased base deficit during an apparently adequate oxygen supply. Extreme hemodilution, hypothermia, low flow CPB, and excessive neurohormonal activation have also been linked to increased base deficit (10).

An abrupt increase in lactate concentration may result at the institution of CPB when priming solutions containing lactate are used. Several studies comparing, different priming solutions have shown however that base deficit are lower than -1 mmol/L

evens when lactate is present in the priming fluid (9, 14, 15).

An impaired hepatic clearance of lactate during CPB related to a hypothermia- induced defect in pyruvate metabolism has also been suggested. In the immediate postoperative period, a hyper metabolic response characterized by increased oxygen consumption and carbon deficit production has been shown to occur at CPB (21-23).

Base deficit measurement usually indicates the need for volume resuscitation in the acutely injured patient. However the implication of a severe base deficit in the ICU patient etiologies for perfusion impairment (24).

In one study, (25), the base deficit has been specifically examined in the pediatric trauma population. Base deficit less than -4 meq/L and the presence of a closed head injury were predictors of mortality. For this group an admission base deficit of -8 meq/L corresponded to a probability of mortality. Significant correlation were found between base deficit and emergency department systolic blood pressure, injury severity score and deficit value in immediate postoperative period of coronary surgery for CPB and length of stay in the ICU (26). A logistic regression model was performed on the variables shown to have a statistically significant difference in univariate analysis with determination of odd ratio, 14 variables had a statistically significant difference in univariate analysis and three of them high lighted by linear regression model that one of them was base deficit value in the 1<sup>st</sup> hour post operatively (Table 4).

Kincaid and colleagues (25) showed that in patient, the admission value of the arterial has deficit stratifies injury severity, predicts complication and is correlated with arterial lactate concentration in theory, elevated base deficit and lactate

concentration after shock and hypo perfusion such as CPB are related to oxygen transport imbalance at the cellular level. Subgroup analysis revealed that patients with a persistently high base deficit had higher rates of multiple organ failure compared with patients who achieved a low base deficit (25). Patients with a persistently high base deficit had lower oxygen consumption and a lower oxygen utilization coefficient and higher lactate level compared with patients with a low base deficit. They conclude that in the patients, a high arterial base deficit is associated with altered oxygen utilization and an increased risk of multiple organ failure and mortality. Serial monitoring of base deficit may be useful in assessing the adequacy of oxygen transport and resuscitation. The above response maybe related to intraoperative oxygen deficit and may represent a high risk period for decompensation. In the present study, patients undergoing cardiac operation with base deficit of -8 or higher after CPB were older, more likely to be female, and were at higher risk according to clinical study.

They also had a lower ejection fraction, lower preoperative and preoperative hemoglobin concentration and were more likely to have preoperative or complex operation. There were a weak but significant correlation between CPB time and base deficit more than -8 meq, suggesting that higher base deficit are more likely to occur with long CPB duration but are dependent of other preoperative factor. Postoperative mortality was significantly higher in patients with high base deficit after CPB.

Moreover postoperative complication and myocardial infarction were not significantly were frequent in these patients. This group of patients also needed greater hospital care. Finally a peaked base deficit of -8 meq/L or higher after CPB was identify as a strong independent prediction of mortality and morbidity by logistic regression analysis and suggests that occult tissue hypo perfusion occurred during CPB. However further studies are needed to evaluate the association between blood gas base deficit and occult oxygen deficit occurring during CPB.

Identification of high base deficit during CPB should prompt further evaluation of all potential

factors that may modify oxygen delivery and consumption. Several limitation are inherent to the present study, first, the population studied was heterogeneous including all the patients undergoing cardiac operation with CPB during 3 month. And the proposed threshold value for base deficit levels differed from the value for base deficit chosen by others authors for different clinical situation.

In conclusion, our data suggest that a base defecation level of -8 meq/L or higher after CPB is associated with an in creased risk of preoperative mortality and morbidity. Further studies are needed to determine if serial blood gas during CPB and intervention based on base deficit valves improve survival in cardiac surgical patients.

### Conflict of interests

The authors declare that they have no competing interests.

## REFERENCES

1. Mizock BA, Falk JL. Lactic acidosis in critical illness. *Crit Care Med.* 1992 Jan; 20(1):80-93.
2. Broder G, Weil MH. Excess lactate: an index of reversibility of shock in human patients. *Science.* 1964 Mar 27; 143: 1457-1459.
3. Weil MH, Afifi AA. Experimental and clinical studies on lactate and pyruvate as indicators of the severity of acute circulatory failure (shock). *Circulation.* 1970 Jun; 41(6):989-1001.
4. Vitek V, Cowley RA. Blood lactate in the prognosis of various forms of shock. *Ann Surg.* 1971 Feb; 173(2):308-313.
5. Higgins TL, Estafanous FG, Loop FD, Beck GJ, Lee JC, Starr NJ, Knaus WA, Cosgrove DM 3rd. ICU admission score for predicting morbidity and mortality risk after coronary artery bypass grafting. *Ann Thorac Surg.* 1997 Oct; 64(4):1050-1058.
6. Clowes GH Jr, Neville WE, Sabga G, Shibota Y. The relationship of oxygen consumption, perfusion rate, and temperature to the acidosis associated with cardiopulmonary circulatory bypass. *Surgery.* 1958 Jul; 44(1):220-239.

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7. Ballinger WF 2nd, Vollenweider H, Pierucci L Jr, Templeton JY 3rd. Anaerobic metabolism and metabolic acidosis during cardiopulmonary bypass. *Ann Surg.* 1961 Apr; 153:499-506.
8. Ballinger WF, Vollenweider H, Pierucci L, Templeton JY. The accumulation and removal of excess lactate in arterial blood during hypothermia and biventricular bypass. *Surgery.* 1962; 5: 738-745.
9. Alston RP, Singh M, McLaren AD. Systemic oxygen uptake during hypothermic cardiopulmonary bypass. *J Thorac Cardiovasc Surg.* 1989;98:757-768.
10. Fiaccadori E, Vezzani A, Coffrini E, Guariglia A, Ronda N, Tortorella G, Vitali P, Pincolini S, Beghi C, Fesani F, et al. Cell metabolism in patients undergoing major valvular heart surgery: relationship with intra and postoperative hemodynamics, oxygen transport, and oxygen utilization patterns. *Crit Care Med.* 1989 Dec; 17(12):1286-1292.
11. Landow L, Phillips DA, Heard SO, Prevost D, Vandersalm TJ, Fink MP. Gastric tonometry and venous oximetry in cardiac surgery patients. *Crit Care Med.* 1991 Oct; 19(10):1226-1233.
12. Landow L. Splanchnic lactate production in cardiac surgery patients. *Crit Care Med.* 1993 Feb; 21(2 Suppl):S84-91.
13. Raper RF, Cameron G, Walker D, Bowey CJ. Type B lactic acidosis following cardiopulmonary bypass. *Crit Care Med.* 1997 Jan; 25(1):46-51.
14. McKnight CK, Elliott MJ, Pearson DT, Holden MP, Alberti KG. The effects of four different crystalloid bypass pump-priming fluids upon the metabolic response to cardiac operation. *J Thorac Cardiovasc Surg.* 1985 Jul; 90(1):97-111.
15. Himpe D, Van Cauwelaert P, Neels H, Stinkens D, Van den Fonteyne F, Theunissen W, Muylaert P, Hermans C, Goossens G, Moeskops J, et al. Priming solutions for cardiopulmonary bypass: comparison of three colloids. *J Cardiothorac Vasc Anesth.* 1991 Oct; 5(5):457-466.
16. Higgins TL, Estafanous FG, Loop FD, Beck GJ, Blum JM, Paranandi L. Stratification of morbidity and mortality outcome by preoperative risk factors in coronary artery bypass patients. A clinical severity score. *JAMA.* 1992 May 6; 267(17):2344-2348.
17. Grayck EN, Meliones JN, Kern FH, Hansell DR, Ungerleider RM, Greeley WJ. Elevated serum lactate correlates with intracranial hemorrhage in neonates treated with extracorporeal life support. *Pediatrics.* 1995 Nov; 96(5 Pt 1):914-917.
18. Cheifetz IM, Kern FH, Schulman SR, Greeley WJ, Ungerleider RM, Meliones JN. Serum lactates correlate with mortality after operations for complex congenital heart disease. *Ann Thorac Surg.* 1997 Sep;64(3):735-738.
19. Vincent JL, Dufaye P, Berre J, Leeman M, Degaute JP, Kahn RJ. Serial lactate determinations during circulatory shock. *Crit Care Med.* 1983 Jun; 11(6):449-451.
20. Waxman K, Nolan LS, Shoemaker WC. Sequential perioperative lactate determination. Physiological and clinical implications. *Crit Care Med.* 1982 Feb; 10(2):96-99.
21. Chiara O, Giomarelli PP, Biagioli B, Rosi R, Gattinoni L. Hypermetabolic response after hypothermic cardiopulmonary bypass. *Crit Care Med.* 1987 Nov; 15(11):995-1000.
22. Ariza M, Gothard JW, Macnaughton P, Hooper J, Morgan CJ, Evans TW. Blood lactate and mixed venous-arterial PCO<sub>2</sub> gradient as indices of poor peripheral perfusion following cardiopulmonary bypass surgery. *Intensive Care Med.* 1991; 17(6):320-324.
23. Cremer J, Martin M, Redl H, Bahrami S, Abraham C, Graeter T, Haverich A, Schlag G, Borst HG. Systemic inflammatory response syndrome after cardiac operations. *Ann Thorac Surg.* 1996 Jun; 61(6):1714-1720.
24. Himpe D. Anion gap, lactate, and acid-base status after cardiac surgery. *Crit Care Med.* 1993 May; 21(5):807-808.
25. Kincaid EH, Chang MC, Letton RW, Chen JG, Meredith JW. Admission base deficit in pediatric trauma: a study using the National Trauma Data Bank. *J Trauma.* 2001 Aug; 51(2):332-335.
26. Hugot P, Sicsic JC, Schaffuser A, Sellin M, Corbineau H, Chaperon J, Ecoffey C. Base deficit in immediate postoperative period of coronary surgery with cardiopulmonary bypass and length of stay in intensive care unit. *Intensive Care Med.* 2003 Feb; 29(2):257-261.