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Off-Pump Coronary Bypass Surgery Adversely affects Alveolar Gas Exchange

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A B S T R A C T

While the introduction of off-pump myocardial revascularization (OPCAB) has initially shown promise in reducing respiratory complications inherent to conventional coronary surgery, it has failed to eradicate them. Our study focused on quantifying the lactate release from the lungs and the dysfunction at the level of the alveolar-capillary membrane precipitated by OPCAB at different time points after the insult. Furthermore, we aimed to determine the impact of pulmonary lactate production on systemic lactic acid concentrations. The study was conducted in a prospective observational fashion. Forty consecutive patients undergoing OPCAB were analyzed. The mean patient age was 60±10 years. The mean EUROScore was 3.8±2.9. The alveolar-arterial O_2 gradient increased from 19 [range 9 to 30] to 26 [range 20 to 34] kPa ($P<0.001$) and remained elevated up to 6 hours after surgery. It rapidly declined again by 18 hours postoperatively. The observed increase in the pulmonary lactate release (PLR) from a baseline value of 0.022 [range –0.074 to 0.066] to 0.089 [range 0.016 to 0.209] mmol/min/m^2 at six hours postoperatively did not reach statistical significance ($P=0.105$). The systemic arterial lactate (L_S) concentration increased from 0.94 [range 0.78 to 1.06] to 1.39 [range 0.97 to 2.81] mmol/L ($P<0.001$). The venoarterial pCO_2 difference showed no significant change in comparison to baseline values. The mortality in the studied group was 2.5% (1/40). The pulmonary lactate production showed a statistically significant correlation with the systemic lactate concentration ($R=0.46$; $P=0.003$). Pulmonary injury following off pump myocardial revascularization was evidenced by a prompt increase in the alveolar-arterial oxygen gradient. The alveolar-arterial O_2 gradient correlated with the duration of mechanical ventilation.

***Key words**: off pump coronary artery bypass, lung injury, lactate, alveolar-arterial oxygen gradient*

Introduction

The principal idea behind off pump myocardial revascularization is the avoidance of cardiopulmonary bypass (CPB), which is known to invoke a complex systemic inflammatory response with clinical sequelae on multiple organ systems. The critical event leading to the activation of this defense response is exposure of blood to a non-endothelial surface during CPB. Various cellular elements, including neutrophils, monocytes, macrophages, endothelial cells and platelets are activated, as well as the complement and contact cascades with the resultant release of multiple anaphylatoxins and the terminal cytotoxic complex C5b-9[1,2]. The overwhelming thrombogenic stimulus must be counteracted by systemic anticoagulation, which contributes to the already multifaceted coagulation disorder. The negative aspects of CPB are further enhanced by its embolic potential, which includes both gaseous and particulate emboli.

Respiratory dysfunction after cardiac surgery employing CPB remains a common problem. The presentation varies from subclinical gas exchange impairment to the frequently fatal adult respiratory distress syndrome[3]. The systemic inflammatory response has been hypothesized to be of paramount importance in the development of this condition. Reduction in the plasma oncotic pressure that occurs as the aftermath of hemodilution, coupled with left atrial pressure elevation during the process of weaning from CPB contribute to the accumulation of fluid in the lung interstitium which further exacerbates pulmonary dysfunction[4,5]. It is difficult to ascertain whether

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exclusion of the pulmonary functional circulation additionally enhances lung injury. Lung perfusion strategies during CPB have been employed but have not gained wider acceptance[6–8]. The pulmonary injury can be quantified by the increase in the alveolar-arterial oxygen gradient, increase in pulmonary vascular resistance or changes in the pulmonary shunt[1]. The incidence of adult respiratory distress syndrome following cardiac surgery is below 2% [3,9,10].

Off-pump coronary artery bypass surgery (OPCAB) holds promise of attenuating these effects, but is not without shortcomings itself. It provokes a hypercoagulable state similar to those observed after other major surgical procedures. Furthermore, the dislocation of the heart from its normal position, which is the prerequisite for successful revascularization of the circumflex territory, may induce a period of temporary hypoperfusion. Similarly, myocardial perfusion may be compromised during the occlusion of the target coronary artery while the creation of the bypass anastomosis is taking place. Serum lactate concentrations have been validated as markers of systemic hypoperfusion in a variety of clinical scenarios[11–19]. Should the availability of oxygen become the limiting factor for meeting the demand for energy production, the metabolism will shift toward its anaerobic route. The conversion of pyruvate to acetyl coenzyme A will abate, and lactate will accumulate through the action of lactate dehydrogenase. The venoarterial difference in the partial pressure of carbon dioxide has also been used for documenting hypoperfusion[20,21].

The purpose of our study was to assess the effect of off pump surgical myocardial revascularization on alveolar gas exchange. Additionally, our report, to the best of our knowledge, is the first study to chronologically evaluate pulmonary lactate production in the setting of OPCAB surgery.

Patients and Methods

Patient selection

Following the approval of the institutional ethics committees we enrolled 40 patients into this study, which was conducted in a prospective observational manner. Patients with native coronary artery disease requiring primary surgical myocardial revascularization which was deemed feasible as an off pump procedure were enlisted for the study. Informed consent was obtained from all patients. Exclusion criteria were concomitant valvular pathology requiring surgery, preoperatively documented debilitating pulmonary disease, reoperative surgical myocardial revascularization as well as coronary artery disease secondary to allograft vasculopathy. Furthermore, patients in whom it was found intraoperatively that the myocardial revascularization required the use of cardiopulmonary bypass due to either their deteriorating hemodynamic performance or anatomic unsuitability for off--pump coronary artery bypass surgery were also excluded from the study.

Perioperative management

All patients received diazepam and morphine 30 minutes prior to induction of anesthesia. Endotracheal tube, urinary catheter, as well as radial artery and pulmonary artery catheters were inserted. The induction and maintenance of anesthesia was conducted with midazolam, fentanyl and pancuronium bromide. This was coupled with sevoflurane inhalation. The surgical revascularization was performed on a beating heart, using the Octopus stabilizer (Medtronic, Minneapolis, USA). Intracoronary shunts were employed during the suturing of the coronary artery bypass grafts. Inotropic support was initiated in order to maintain a cardiac index greater than 2.2 L/min/m^2. The inotropic agent of choice was dobutamine. Norepinephrine was used if dobutamine was responsible for hypotension due to excessive systemic vasodilatation. Epinephrine was used if the hemodynamic performance remained inadequate with the previously mentioned catecholamines. The indications for an intraaortic balloon pump were requirement for circulatory support when inotropes alone were insufficient, as well as ongoing myocardial ischemia.

Measurement and calculations

Arterial blood gas samples from the radial artery and mixed venous blood samples from the distal port of the pulmonary artery catheter were collected and analyzed. Every effort was made to draw the mentioned blood samples simultaneously. Thermodilution cardiac indices were obtained in triplicate and then averaged. The first sampling (T1) of radial and pulmonary arterial blood was performed upon induction of anesthesia and prior to the surgical incision. These values constituted the control samples against which all subsequent samples were compared. The second sample (T2) was taken intraoperatively, after completing the myocardial revascularization. The third (T3) and fourth (T4) samples were drawn in the intensive care unit, 6 and 18 hours after surgery, respectively. Thermodilution cardiac indices were obtained at all of the above mentioned stages of blood sampling. The raw data was then processed to obtain the transpulmonary lactate release and the pulmonary lactate release from the following formulas:

$$\text{TPLG} = \text{Lactate}_{\text{RA}} - \text{Lactate}_{\text{PA}} - \\ - ((\text{Hb}_{\text{RA}} - \text{Hb}_{\text{PA}})/\text{Hb}_{\text{RA}}) \times \text{Lactate}_{\text{RA}}$$

$\text{PLR} = \text{TPLG} \times \text{cardiac index}$

TPLG – transpulmonary lactate gradient

PLR – pulmonary lactate release

Hb_{PA} – pulmonary artery hemoglobin

Hb_{RA} – radial artery hemoglobin

$\text{Lactate}_{\text{PA}}$ – lactate concentration in the pulmonary artery

$\text{Lactate}_{\text{RA}}$ – lactate concentration in the radial artery

The effects of hemodilution were taken into account in order to obtain a hemoglobin adjusted pulmonary lactate release as was suggested by Kellum et al[22]. The A-a oxygen gradient was calculated by subtracting the partial pressure of oxygen in arterial blood from the calcu-

lated alveolar oxygen tension[23]. The venoarterial pCO_2 difference was obtained by subtracting the partial pressure of CO_2 in radial arterial blood from the partial CO_2 pressure measured in the pulmonary artery.

Statistical analysis

The data are presented as mean values ± standard deviation or medians followed by the interquartile range for variables with a distribution that is not normal. Longitudinal comparisons between samples of the same subject were performed using the ANOVA test. Correlations were analyzed using the nonparametric Spearman R correlation methodology. Analyses between different groups of patients were performed using the Mann-Whitney U test. A $P<0.05$ was considered to be of statistical significance. The data were processed using the Statistica software package (StatSoft Inc., Tulsa, USA).

Results

Perioperative summary

Forty patients undergoing off pump coronary artery bypass grafting (OPCAB) were enrolled into our study (mean age 60±10 years, EuroSCORE 3.8±2.9, ejection fraction 50±12). The pertinent patient demographic profile is presented in Table 1. The spectrum of comorbidities seen in our patient population is typical for the contemporary cardiac surgical practice.

The perioperative summary is presented in Table 2. Inotropic support was used in 33% (13/40) of patients. In one patient the hemodynamic status required additional mechanical cardiac support, which was achieved with the insertion of an intra-aortic balloon pump (IABP). One patient had an IABP inserted preoperatively for a failed percutaneous coronary intervention accompanied by ongoing ischemia. Two of the three patients that required renal replacement therapy had preoperatively documented chronic renal failure. The renal function recovered in both of these patients. One patient died due to multiple organ failure that followed an episode of ventricular fibrillation on postoperative day 4. The median values of the pulmonary lactate release increased from a baseline value (T1) of 0.022 [range –0.074 to 0.066] to 0.089 [range 0.016 to 0.209] mmol/min/m^2, observed at 6 hours postoperatively ($P=0.105$). The transpulmonary lactate gradient increased from 0.011[range –0.030 to 0.037] to 0.035 [range 0.007 to 0.085] mmol/L ($P=0.160$). The A-a O_2 gradient increased from a baseline median value of 19 [range 9 to 30] to a maximum value of 26 [range 20 to 34] kPa,

TABLE 1
PREOPERATIVE PATIENT CHARACTERISTICS PRESENTED AS MEAN VALUES ± STANDARD DEVIATION, OR AS PERCENTAGES

Age	60±10
Gender (n/%)	
Male	37 (93)
Female	3 (8)
EUROScore	3.8±2.9
Ejection fraction	50±12
Hyperlipidemia (n/%)	30 (75)
Hypertension (n/%)	33 (83)
Diabetes mellitus (n/%)	15 (38)
Smoking history (n/%)	18 (45)
Preop. MI (n/%)	25 (63)
Left main stenosis (n/%)	8 (20)
Three vessel disease (n/%)	26 (65)
Mild-moderate MR (n/%)	8 (20)

Preop. MI – preoperative myocardial infarction, MR – mitral regurgitation

TABLE 2
PERIOPERATIVE PATIENT DATA PRESENTED AS MEAN VALUES ± STANDARD DEVIATION, OR AS PERCENTAGES

Number of grafts	2.1±1
Inotropic support (n/%)	13 (33)
ICU (days)	2±1
Mechanical ventilation (hours)	15±21
Complications (n/%)	
Stroke	2 (5)
Periop. myocardial ischemia	4 (10)
Reexploration for bleeding	2 (5)
Acute renal failure	3 (7.5)
Sternal wound infection	2 (5)
IABP requirement	2 (5)
Death (n/%)	1 (2.5)

ICU – length of stay in the intensive care unit, IABP – intra-aortic balloon pump

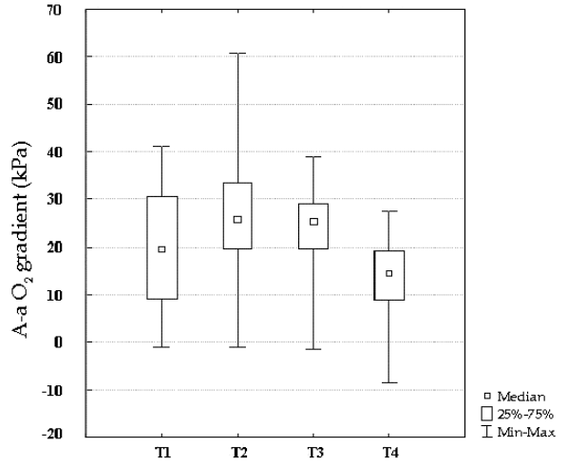

Fig 1. Box and Whisker plot of the alveolar-arterial oxygen gradient (T1 – prior to surgery; T2 – after completion of surgery; T3 – 6 hours after surgery; T4 – 18 hours after surgery).

TABLE 3
LONGITUDINAL EVOLUTION OF THE STUDIED VARIABLES. DATA ARE SHOWN AS MEDIAN WITH QUARTILES

Variables	T1	T2	T3	T4
Pulmonary lactate release (mmol/min/m^2)	0.022 [–0.074/0.066]	0.070 [0.003/0.119]	0.089 [0.016/0.209]	0.003 [–0.114/0.150]
Transpulmonary lactate gradient (mmol/L)	0.011 [–0.030/0.037]	0.032 [0.001/0.049]	0.035 [0.007/0.085]	0.001 [–0.042/0.075]
Alveolar-arterial oxygen gradient (kPa)	19 [9/30]	26 [20/34]	25 [20/29]	14 [9/19]
Systemic lactate (mmol/L)	0.94 [0.78/1.06]	1.08 [0.89/1.23]	1.39 [0.97/2.81]	1.27 [1.01/1.63]
Veno-arterial pCO_2 difference (kPa)	0.8 [0.6/0.9]	0.7 [0.6/0.8]	0.7 [0.6/1]	0.8 [0.6/0.9]
Pulmonary vascular resistance (dyne·sec/cm^5)	89 [41/134]	92 [46/133]	102 [45/123]	89 [55/123]

T1 – preoperative value, T2 – value after completion of surgery, T3 – value 6 hrs postoperatively, T4 – Value 18 hrs postoperatively

immediately upon completion of the procedure ($P<$ 0.001). The changes in the alveolar-arterial oxygen gradient are depicted in Figure 1. Higher A-a O_2 gradients were documented in the subgroup of patients requiring mechanical ventilation for more than 18 hours when compared to those weaned earlier from the ventilator (33 kPa [range 25 to 35] vs. 24 kPa [range 18 to 31], $P=$ 0.039). The total arterial lactate measured in the radial artery increased from a baseline median value of 0.94 [range 0.78 to 1.06] mmol/L to a maximum median value of 1.39 [range 0.97 to 2.81] mmol/L at 6 hours postoperatively ($P<0.001$). The serum lactate concentration did not reach preoperative values by the end of the study protocol. We found no significant change in the dynamics of the pulmonary vascular resistance in our study ($P=$ 0.927). Similarly, the values of the venoarterial pCO_2 gradient (V-A dpCO2) remained constant throughout the study ($P=0.617$).

The evolution of the pulmonary lactate flux, A-a O_2 gradient, total arterial lactate, venoarterial pCO_2 difference and pulmonary vascular resistance from the values obtained prior to surgery to those found at 18 hours postoperatively are presented in more detail in Table 3.

Correlations

A correlation between the pulmonary lactate production and the total serum concentration of lactate was demonstrated at two time points. It was evident immediately after completion of the revascularization procedure, as well as six hours thereafter ($R=0.46$; $P=0.003$). The peak value of the alveolar-arterial oxygen gradient correlated with the duration of mechanical ventilation ($R=0.44$; $P=0.004$). A prolonged elevation of the serum lactate concentration was related to the length of stay in the intensive care unit, and was indicative of a more complex postoperative course. The correlation coefficients (R) for relationships between the studied variables and clinical parameters are presented in Table 4, as are their relevant P values.

TABLE 4
SUMMARY OF THE STATISTICALLY SIGNIFICANT CORRELATIONS USING THE NON-PARAMETRIC SPEARMAN ANALYSIS. A POSITIVE CORRELATION COEFFICIENT INDICATES THAT THE SECOND VARIABLE INCREASES WHEN THE FIRST VARIABLE INCREASES

Correlation	Spearman R	P
PLR^{T2} & $L_S{}^{T2}$	0.35	0.026
PLR^{T3} & $L_S{}^{T3}$	0.46	0.003
A-a O_2 G^{T1} & M. vent	0.40	0.01
A-a O_2 G^{T2} & M. vent	0.44	0.004
$L_S{}^{T4}$ & ICU	0.33	0.039

PLR – pulmonary lactate release, L_S – systemic arterial lactate, A-a O_2 G – alveolar-arterial oxygen gradient, M. vent – duration of mechanical ventilation, ICU – length of stay in the intensive care unit

Discussion

Prolonged exposure of heparinized blood to a non-endothelial surface during cardiopulmonary bypass is the decisive incident leading to the evolution of the systemic inflammatory response[2]. This defense response includes activation of neutrophils, monocytes, platelets and endothelial cells[2,24,25]. This is further complemented by the release of various proinflammatory cytokines, and activation of the complement, coagulation and fibrinolytic cascades.

The initial enthusiasm concerning the reduction of pulmonary complications following off-pump cardiac surgery in comparison to the conventional technique employing CPB has been largely dispelled[2,26]. One of the possible explanations is that respiratory complications following cardiac surgery are not solely the aftermath of post-perfusion lung injury, but stem also from inherent pulmonary disease, pulmonary edema due to a reduction in cardiac function reserve as well as phrenic nerve palsy[1,9].

Our study assessed the affect of off pump surgical myocardial revascularization on alveolar gas exchange. This was complemented by a chronological evaluation of pulmonary lactate release and its relationship with systemic lactate concentrations in the same homogenous patient population.

The present study recorded pulmonary injury after OPCAB at the level of the alveolar-capillary membrane. We were able to show a consistent and statistically important increase in the alveolar-arterial oxygen gradient after the procedure when compared to the control values

obtained prior to surgery. A significant relationship between the increased peak alveolar-arterial oxygen gradient and the duration of mechanical ventilatory support was found. The duration of mechanical ventilation in our study exceeded the expected values for isolated coronary artery surgery. This was more a reflection of an absence of fast track protocols than it was of prolonged respiratory insufficiency. Only one patient was ventilated for more than 48 hours. The accentuation of the A-a O_2 gradient following off-pump coronary artery bypass surgery was of temporary duration, and the values reached preoperative ones eighteen hours after surgery. We have documented a discrete positive transpulmonary lactate gradient even at baseline, i.e. prior to any demonstrable surgical insult. The pulmonary lactate release was calculated by subtracting the lactate concentration in the afferent pulmonary circulation from that measured in the efferent circulation, and then multiplied by the indexed pulmonary blood flow. The pulmonary lactate release in our patient population increased after exposure to surgical trauma, and reached its peak value six hours after the procedure. While the absolute values of pulmonary lactic acid production quadrupled in the observed period, this did not reach statistical significance. We believe that the production of lactate from the lungs is a dynamic occurrence, and responds to the pulmonary insult caused by the surgical procedure. The values of systemic serum lactate concentrations showed a statistically significant increase from baseline to the maximal values observed at six hours after surgery. The dynamics of these changes paralleled the release of lactic acid from the lungs. We were able to demonstrate a significant correlation between the pulmonary lactate production and the total serum lactate concentration, indicating that the lungs were, at least in part, responsible for the augmentation of the systemic lactic acid content. We found no significant change in the pulmonary vascular resistance to further corroborate a pulmonary lesion, nor were the dynamics of the venoarterial pCO_2 gradient indicative of systemic hypoperfusion.

Conclusion

Our study demonstrated a significant impact of off--pump coronary artery bypass surgery on the alveolar--capillary membrane, as evidenced by a prompt increase in the alveolar-arterial oxygen gradient. The A-a O_2 gradient at the completion of the surgical procedure was found to be an early predictor of prolonged mechanical ventilation, and may identify patients at risk for this complication. While the increases in the transpulmonary lactate gradient and systemic lactate concentration remained below the threshold of clinical significance, they were indicative of a mild degree of impairment of oxygen utilization during OPCAB.

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OFF-PUMP KIRURŠKA REVASKULARIZACIJA MIOKARDA NEGATIVNO UTJEČE NA IZMJENU PLINOVA U PLUĆIMA

S A Ž E T A K

Hipoteza da će uvođenje kirurške revaskularizacije miokarda bez upotrebe izvantjelesnog krvotoka (OPCAB, od engl. Off pump coronary artery bypass) dramatično smanjiti incidenciju respiratornih komplikacija u odnosu na klasični zahvat nije našla svoju potvrdu u praksi. U ovoj studiji evaluirali smo utjecaj kirurške traume koja prati OPCAB na alveolo-kapilarnu membranu te kvantificirali količinu laktata koju pluća otpuštaju u cirkulaciju kao odgovor na istu. Nadalje, analizirana je korelacija između laktata otpuštenog iz pluća te ukupnog sistemskog laktata. Studija je dizajnirana kao prospektivno opservacijsko istraživanje. Analizirano je 40 konzekutivnih bolesnika kod kojih je učinjena revaskularizacija miokarda bez upotrebe izvantjelesnog krvotoka. Prosječna dob ispitanika bila je 60±10 godina. Prosječni EUROScore bio je 3,8±2,9. Alveolo-arterijski gradijent parcijalnog tlaka kisika (A-a gradijent O_2) porastao je sa 19 [raspon 9 do 30] na 26 [raspon 20 do 34] kPa ($P<0,001$). Povišene vrijednosti A-a gradijenta O_2 bile su registrirane do 6 sati iza zahvata, nakon čega je postojao nagli trend normalizacije vrijednosti. Iako je zabilježen porast u otpuštanju laktata iz pluća kao odgovor na kiruršku traumu sa 0,022 [raspon –0,074 do 0,066] na 0,089 [raspon 0,016 do 0,209] mmol/min/m^2, isti nije dosegao statističku značajnost ($P=0,105$). Sistemska koncentracija laktata je porasla sa 0,94 [raspon 0,78 do 1,06] na 1,39 [raspon 0,97 do 2,81] mmol/L ($P<0,001$). Venoarterijska razlika u parcijalnom tlaku ugljičnog dioksida nije ukazala na značajniju varijabilnost u promatranom razdoblju. Mortalitet u ovoj kohorti bolesnika bio je 2,5% (1/40). Pokazali smo korelaciju između otpuštanja laktata iz pluća i ukupne sistemske koncentracije laktata (R=0,46; $P=0,003$). Plućna ozljeda nakon OPCAB kirurgije u našoj je studiji demonstrirana naglim porastom alveolo-arterijskog gradijenta parcijalnog tlaka kisika. Klinička implikacija navedenog porasta A-a gradijenta O_2 bila je u njegovoj korelaciji sa dužinom trajanja mehaničke ventilacije.