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Title: VENTILATION, THERMAL NOISE, AND ERRORS IN CARDIAC OUTPUTS AFTER CARDIOPULMONARY BYPASS

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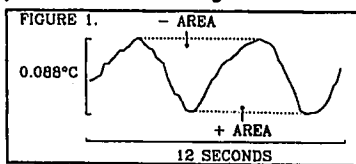
INTRODUCTION: Small variations in pulmonary artery blood temperature (PABT) associated with respiration are a known source of error in thermodilution cardiac output measurements (TDCOs).¹ Since the reported magnitude of potential error is relatively small, many anesthesiologists elect to accept this potential error rather than to stop ventilation. We have observed transient increases in these respiratory variations in PABT (RVPABT) in many patients after cardiopulmonary bypass (CPB), and these increased RVPABTs appeared to cause significant errors in TDCOs. The purpose of this study was to: 1) quantitate the magnitude and time course of RVPABT in patients after CPB; 2) determine if temperature (T) gradients between nasopharyngeal T, rectal/bladder T, and mean PABT could identify patients with increased RVPABT; 3) calculate the potential magnitude of error in TDCOs in patients with increased RVPABTs following CPB.

METHODS: After IRB approval, we studied 13 patients undergoing cardiac surgery. Simultaneous measurements of RVPABT (from strip chart recordings of PABT), nasopharyngeal T, rectal/bladder T, and mean PABT were recorded: #1) within 5 min after discontinuation of CPB; #2) 10 min after #1; #3) 20 min after #1; #3) 30 min after #1. Areas enclosed by both the convex (-AREA) and concave (+AREA) portions of the PABT curve were calculated as shown in the figure. Maximum potential errors in TDCOs were estimated by adding (subtracting) these thermal areas from the theoretical area produced by injection of 10 ml of room temperature injectate into a normothermic patient with a fixed cardiac output of 5 l/min. This method ignores additional errors that could be introduced by: 1) erroneous extrapolation of the terminal portion of the TDCO thermal curve; 2) additive effects from a second respiratory cycle with prolonged TDCO thermal curves.

RESULTS: An example PABT trace at sample time #1 is shown in the figure. The mean \pm SEM ($^{\circ}$ C) of RVPABT for the 4 measurement periods were: #1) $0.037 \pm .005$; #2) $0.025 \pm .003$; #3) $0.020 \pm .003$; #4) $0.012 \pm .002$. There were no consistent correlations between RVPABT magnitude and any of the recorded T gradients. Four patients had RVPABT values over 0.06° C. The average +AREA in these 4 patients at sample time #1 was 0.36° C-sec, decreasing to 0.09° C-sec by time #4; the average -AREA at time #1 was 0.23° C-sec, decreasing to 0.09° C-sec by time #4. These areas at time #1 could cause potential errors in TDCOs as large as 39% (from +15% to -24%, depending on the "zero" reference level and injection timing). This potential error decreases to only 9% (+3% to -6%) at time #4.

DISCUSSION: RVPABT results from differences in blood T in the inferior and superior vena cava, coupled with changes in the proportion of total venous return coming from each cava during the respiratory cycle.² We hypothesize that the transient increase in RVPABT after CPB in some patients results from uneven rewarming of body regions on CPB, leading to an increase in the T difference between blood in the inferior and superior cava. Four of 13 patients exhibited values of RVPABT exceeding the upper range of published values in man (0.05° C).³ These patients could not be consistently identified from the recorded T gradients. Under the conditions specified, potential errors in TDCOs as large as 39% could be anticipated with random injections of thermal indicator not timed to the respiratory cycle. Although using injections timed to the respiratory cycle will reduce both the maximum error amplitude (to -24%) and the variation between TDCO measurements, it will not eliminate the potential for error. As the amplitude of RVPABT decreases over time, the associated error will decrease. Although this results in a more accurate measurement, the resultant "trend" in TDCOs may be misinterpreted. In a hypothetical patient with a fixed cardiac output of 5 l/min, timed-injectate measurements of TDCO could change from an initial value of 3.8 l/min (-24%) to 4.7 l/min (-6%) at 30 minutes with no real change in true cardiac output. We conclude that: 1) patients subjected to CPB may show transient increases in RVPABT; 2) gradients between nasopharyngeal T, rectum/bladder T, and mean PABT are not reliable indicators of the magnitude of RVPABT; and 3) these large RVPABTs may cause significant errors in measured TDCOs.

REFERENCES: (1) Am J Cardio 29:241-246 (2) Surgery 79:469-475 (3) Heart Lung 12:175-176



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Title: VALUE OF TOTAL BODY OXYGEN CONSUMPTION AS A PARAMETER OF GRAFT FUNCTION AFTER LIVER TRANSPLANTATION

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Introduction: In the basal state the liver contributes significantly (about 20%) to total body oxygen consumption (VO_2). After reperfusion of a transplanted liver graft a sudden increase in VO_2 can be observed. Preliminary results (1) showed that a poor or missing increase in VO_2 (< 40% compared to the anhepatic state) indicates primary graft failure. The aim of the present study was to assess the predictive value of VO_2 -increase in the first 60 minutes after reperfusion for early graft function.

Methods: In 36 Patients undergoing orthotopic liver transplantation VO_2 was measured continuously using the Deltatrac^R Metabolic Monitor (Datex Instrumentarium Co., Helsinki, Finland). In all cases FiO_2 was kept constant at 0.5 throughout the procedure. Standard laboratory parameters often fail to indicate liver function in the early postoperative period due to dilution caused by infusion and transfusion. Thus synthesis of clotting factors was used for quantification of liver function. Primary graft function (PGF) was defined by prothrombin time (PT) in % of reference corrected for substitution of fresh frozen plasma (FFP) and surgical bleeding (red blood cells, RBC). A new index (PGF-Index) was created for quantification of PGF in the first 24 h using the formula:

$$\text{PGF-Index} = \text{PT} - 5 \times (\text{FFP-RBC}).$$

A PGF-Index < 0 was defined as primary graft failure.

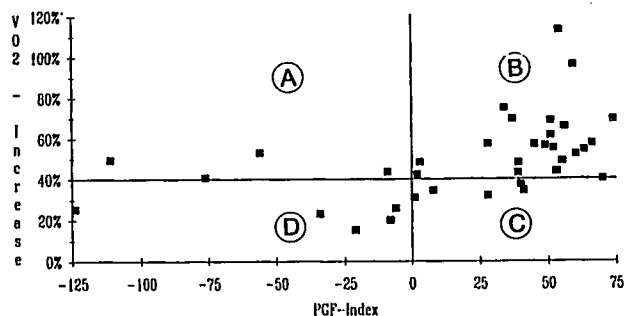
Primary Graft Function and Increase of VO_2 

Fig.1: Increase in VO_2 versus PGF-Index

Results: In 27 patients VO_2 -increase and PGF-index correlated well (Fig.1, quadrant B,D). 4 patients showed a sufficient VO_2 -increase but a poor graft function (A). In 2 cases this bad correlation can be explained by vascular problems. 5 patients had a poor VO_2 -increase despite good graft function (C).

Conclusions:

1. VO_2 can be measured continuously using a noninvasive technique.
2. VO_2 is of some predictive value as an early indicator of PGF after liver transplantation.
3. The excellent correlation seen in our preliminary results could not be confirmed in a larger series.

References: (1) Dieterich H-J, Forst H, Groh J, Denecke H, Peter K: Anesthesiology 73: A488 (1990)