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VENO-ARTERIAL GRADIENTS FOR PCO2 AND TITLE:

pH REFLECT TISSUE HYPOXIA DURINĞ

HEMORRHAGIC SHOCK IN DOGS

AUTHORS: Vanderlinden P, Gilbart E, Pâques P,

Bakker J, Vincent JL

AFFILIATION: Dept of Anesthesiology and Intensive

Care, Erasme University Hospital,

Brussels, Belgium

When oxygen delivery (DO $_2$) is reduced below a critical value (DO $_2$ crit), the tissues oxygen uptake (VO $_2$) becomes DO $_2$ dependent. This reduction in DO $_2$ below DO $_2$ crit is associated with the development of tissue hypoxia, as reflected by an increase in blood lactate levels (Lac). Increases in the venoarterial gradients (VA) for PCO₂ (VAPCO₂) and pH (VApH) can also be observed in low flow states. The present study tested the hypothesis that DO₂crit obtained from repeated measurements of VO₂, Lac, VAPCO $_2$ and VApH are in fact similar. We used an anesthetized dog model in which ${\rm DO}_2$ is reduced by progressive hemorrhage.

MATERIAL & METHODS

The study included 13 dogs (weight $28.6 \pm 2.5 \text{ kgs}$). Anesthesia was induced with thiopental (20 mg kg⁻¹) and maintained with isoflurane (1 MAC = 1.4 % endtidal). After endotracheal intubation, the dog was mechanically ventilated with air. After splenectomy, ${\rm DO}_2$ was reduced by successive withdrawal of 100 ml of blood every 15 min. ${\rm VO}_2$ was determined from the expired gas analysis and ${\rm DO}_2$ from the product of thermodilution cardiac output (CO) and the arterial O₂ content. Measurements of CO, arterial and mixed venous blood gases, Lac and expired gas concentrations were performed before every blood withdrawal.

In each dog, the DO2crit was determined from a dual regression analysis using the least sum of squares technique.

RESULTS

The DO₂crit obtained from VO₂, Lac, VAPCO₂ and VAPH were 9.2 \pm 1.4, 8.8 \pm 1.1, 9.0 \pm 1.2, and 8.9 \pm 1.1 ml.kg⁻¹.min⁻¹, respectively. The DO₂crit obtained from VO₂ correlated well with those obtained from Lac (r = .89), VAPCO₂ (r = .81) and VAPH (r = .75). The VO₂, Lac, VAPCO₂ and VAPH at DO₂crit were 5.4 \pm 0.9 ml.min⁻¹.kg⁻¹, 3.1 \pm 1.3 mEq.1, -10.9 \pm 3.5 mmHz and 0.05 \pm 0.02 ll. respectively. mmHg and 0.05 ± 0.02 U, respectively.

CONCLUSIONS

In this hemorrhagic shock model, the onset of tissue hypoxia associated with the profound reduction in DO2 is reflected by abrupt increases not only in but also in VAPCO₂ and VAPH. Accordingly, these parameters, easily obtained from arterial and mixed venous blood gas sampling, could represent valuable indicators of cellular hypoxia.

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VENTILATION-PERFUSION INDEX CAN ACCURATELY REFLECT VENOUS ADMIXTURE IN CRITICALLY ILL TITLE:

PATIENTS

AUTHORS:

AFFILIATION:

G.B. RUSSELL,M.D., J.M. GRAYBEAL, CRTT, M.T. SNIDER, M.D., PH.D. Division of Respiratory & Intensive Care, Department of Anesthesia, Penn State University College of Medicine, Hershey, PA 17033

INTRODUCTION: The clinical course of the hypoxemic state associated with the Adult Respiratory Distress Syndrome (ARDS) can be monitored with serial measurements of venous admixture (Qs/Qt). The determination of Qs/Qt uses simultaneous arterial and mixed venous blood gas measurements and is calculated with the equation, Qs/Qt = (CcO2 - CaO2 / CcO2 - CvO2), utilizing oxygen contents of pulmonary capillary, systemic arterial, and mixed venous blood, respectively. The ventilation-perfusion index (VQI = 100 - SaO2 / 100 - SvO2), utilizing oxygen saturations of arterial and mixed venous blood, respectively, has been advocated as a simplified, immediate and less expensive method of monitoring Qs/Qt. Previous clinical comparisons of VQI and Qs/Qt have been made when Qs/Qt has been ≤ 45%. We hypothesized that VQI would also reliably assess Qs/Qt particularly in the range between 45 - 100% as seen in patients critically ill with ARDS.

METHODS: With approval of the institutional Clinical Investigations Committee, 12 adult patients (5 male, aged 25 ± 7 yrs,) were studied and 370 comparisons of calculated Qs/Qt and VQI values made. All patients had severe ARDS; 11 required either extracorporeal membrane oxygenation or experimental use of intravenous oxygenation for respiratory support. Blood gases were sampled as clinically indicated and measurements made utilizing both an IL - 1323 Blood Gas Analyzer and an IL - 282 Hemoximeter. Qs/Qt was calculated by the clinical laboratory computer using the classic equation. Comparisons were made over a wide range of Qs/Qt (8 to 100%), FiO2 (0.21 - 1.0), PEEP (5 - 30 cm H2O), SaO2 (65 -99%), SvO2 (35 - 92%), hemoglobin (6.6 - 14.9 gm%), and PaCO2 (23 -74 mmHg). VQI and Qs/Qt correlation was determined by linear regression. A p value < 0.05 was considered significant. Bias and standard deviation of the differences (SDD) were also calculated.

RESULTS: The frequency distribution of calculated Qs/Qt was: <15% (18), 15-30% (81), 30-45% (103), 45-60% (44), 60-75% (33), and >75% (91). Qs/Qt was above 60% in 124 of 370 comparisons. There was a strong correlation (r = 0.973, p < 0.001, slope = 1.072, y intercept = -8.4) over the range of VQI and Qs/Qt values measured. Overall, calculated bias was 4.7 (± 7.6 SDD)%. Maximum bias was 8.7 (± 7.0)% for comparisons in the 30 - 45% Qs/Qt range (figure). Changes in VQI correctly predicted changes in Qs/Qt in 88% of measurements. The incorrectly predicted changes were evenly distributed; 21 false predictions of deterioration and 22 false predictions of improvement.

CONCLUSIONS: In patients with severe ARDS, there was a high correlation between VQI and Qs/Qt over a wide range of Qs/Qt values. Maximal bias was found in the clinically significant 30-45% Qs/Qt range. The direction of Qs/Qt changes was correctly predicted in 88% of comparisons. VQI can provide rapid, relatively inexpensive and, if the correlation extends to saturation measurements utilizing pulse oximetry and fiberoptic SvO2 monitoring, continuous assessment over a wide Qs/Qt range. Clinical use of VQI should be made with the understanding that variations in correlation and predictability of Qs/Qt changes do occur.

FIGURE The agreement between VQI and Qs/Qt is demonstrated across the range of Qs/Qt measured in patients critically ill with ARDS.

