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Real-time continuous estimation of gas exchange by dual oximetry

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Abstract. We designed a ventilation-perfusion index (VQI) to estimate venous admixture (Q_{sp}/Q_t) in a real-time fashion by simultaneous pulse and pulmonary artery oximetry in 17 patients with acute respiratory failure. Changes in Q_{sp}/Q_t were produced by altering the level of continuous positive airway pressure. VQI correlated well with Q_{sp}/Q_t ($r = 0.78$). This contrasts with the poor correlation found between Q_{sp}/Q_t and the commonly used oxygen tension based indices such as PaO_2/FIO_2 ($r = -0.51$), PaO_2/PAO_2 ($r = -0.47$), and PAO_2-PaO_2 ($r = 0.23$). The use of dual oximetry to derive a VQI appears to be a reliable and accurate method for real-time assessment of pulmonary gas exchange in patients with acute respiratory failure.

Key words: Oxygenation – Intrapulmonary shunt – Oximetry

Calculation of venous admixture (Q_{sp}/Q_t) currently is the best method available to accurately assess pulmonary gas exchange in critically ill patients. However, the complexity of the calculation, the necessity for frequent sampling of arterial and mixed venous blood, and the time delay associated with blood gas analysis have led to the development of a number of bedside estimates of Q_{sp}/Q_t such as arterial blood oxygen tension to inspired oxygen concentration ratio (PFI), arterial to alveolar oxygen tension ratio (AAI), and alveolar-arterial oxygen tension difference (AAD) [1]. The accuracy of these indices as estimates of Q_{sp}/Q_t, however, is reduced by the nonlinear relationship between oxygen tension and oxygen content, and by the fact that they do not account for changes in mixed venous oxygenation [2–4]. Therefore, we derived a new estimate of Q_{sp}/Q_t, the ventilation-perfusion index (VQI), by simplifying the intrapulmonary shunt equation:

$$Q_{sp}/Q_t = 100 \cdot \frac{(1.32 \cdot Sc'O_2 \cdot Hgb + 0.0031 \cdot PAO_2) - (1.32 \cdot SaO_2 \cdot Hgb + 0.0031 \cdot PaO_2)}{(1.32 \cdot Sc'O_2 \cdot Hgb + 0.0031 \cdot PAO_2) - (1.32 \cdot S\bar{v}O_2 \cdot Hgb + 0.0031 \cdot P\bar{v}O_2)}$$

(A = alveolar, a = arterial, $\bar{v}$ = mixed venous, and c′ = pulmonary end-capillary blood, and Hgb = blood hemoglobin concentration). If oxygen dissolved in arterial, and mixed venous blood is discounted:

$$Q_{sp}/Q_t \approx 100 \cdot \frac{(1.32 \cdot Sc'O_2 \cdot Hgb + 0.0031 \cdot PAO_2) - (1.32 \cdot S\bar{v}O_2 \cdot Hgb)}{(1.32 \cdot Sc'O_2 \cdot Hgb + 0.0031 \cdot PAO_2) - (1.32 \cdot SaO_2 \cdot Hgb)}$$

Assuming that pulmonary end-capillary blood is fully saturated with oxygen, i.e. $Sc'O_2 \approx 1.0$, an estimate of Q_{sp}/Q_t may be calculated using arterial and mixed venous oxygen saturations:

$$Q_{sp}Q_t \approx 100 \cdot \frac{1.32 \cdot Hgb \cdot (1 - SaO_2) + 0.0031 \cdot PAO_2}{1.32 \cdot Hgb \cdot (1 - S\bar{v}O_2) + 0.0031 \cdot PAO_2} = VQI$$

VQI can be expected to be related to Q_{sp}/Q_t in a linear fashion when SaO_2 is less than 100%. Moreover, it will account for changes in peripheral circulation, since $S\bar{v}O_2$ is included in the calculation.

Simultaneous pulse and pulmonary artery oximetry (dual oximetry) provides real-time values for arterial and mixed venous blood oxygen saturations. These values can be fed into a microcomputer which, when updated for changes in inspired oxygen concentration (F_IO_2) and hemoglobin concentration, allows

continuous, real-time monitoring of VQI. The purpose of this investigation was to determine the reliability of real-time VQI as an estimate of Q_{sp}/Q_t in patients with acute respiratory failure.

Patients and methods

The study protocol was approved by the Human Subjects Review Committee of the Ohio State University. Informed consent was obtained from the families of 17 patients who were treated in the Surgical Intensive Care Unit. All patients had acute lung injury that responded to change in continuous positive airway pressure (CPAP) with a change in Q_{sp}/Q_t. The patients were hemodynamically stable, they were not bleeding actively, and were not receiving blood transfusions or rapid fluid infusions at the time of the study. Variables reflecting the cardiopulmonary function of the patients at the time of data collection are summarized in Table 1.

All patient's tracheas had been intubated before the study and they were receiving positive airway pressure with either a ventilator or a venturi powered CPAP apparatus, as part of their treatment. Previously placed indwelling arterial catheters allowed sampling of blood and measurement of systemic blood pressure. A pulmonary artery catheter equipped with a fiberoptic cable (American Edwards Laboratories, Santa Ana, CA, USA) was inserted using standard technique and connected to an oximetric mixed venous saturation monitor (Sat-1, American Edwards Laboratories) which also was used for thermodilution cardiac output measurements. The saturation monitor was calibrated prior to catheter insertion using in-vivo calibration method suggested by the manufacturer. A pulse oximeter finger probe (Nellcor Inc., Hayward, CA, USA) was attached to an appropriate finger.

After instrumentation, CPAP was changed in steps of 2.5 cmH_2O to cover a range of ± 7.5 cmH_2O from the previously selected level. Inspired oxygen concentration initially was adjusted to produce arterial blood oxygen saturation between 94–96% and was not changed during data collection. After at least 5 min of equilibration at each level of CPAP, vascular pressures, cardiac output, heart rate, respiratory rate, pulmonary artery temperature, and oximeter readings were obtained. Immediately after recording the oximetric saturations, arterial and mixed venous blood were sampled. Progressive change of CPAP in a given direction was discontinued if any of the following conditions was met: SaO_2 less than 90%, increase in $PaCO_2$ by 15%, spontaneous respiratory rate greater than 35 cycles/min, decrease in left ventricular stroke volume or tissue oxygen delivery by 15%.

Arterial and mixed venous blood samples were analyzed for blood gases and pH within 5 min of sampling. Oxygen saturations, oxygen contents, and Q_{sp}/Q_t were calculated as described by Ruiz [5]. The conventional, oxygen tension based gas exchange indices were calculated using the following formulae:

$$PFI = PaO_2/F_IO_2$$
$$AAI = PaO_2/PAO_2$$
$$AAD = PAO_2 - PaO_2$$

The ventilation-perfusion index was calculated from fractional saturations obtained by oximetry as described earlier.

Statistical analysis

Pearson's correlation coefficients were used to assess the strength of correlations between the measured and calculated variables. Within patient correlations between Q_{sp}/Q_t and its estimates were calculated only for the nine patients in whom CPAP produced a minimum change of 10% in Q_{sp}/Q_t. Inter-subject correlations were calculated using one randomly selected value for each patient in order to avoid the confounding effect of repeated measures. The sample size for these correlations, therefore, was 17.

Table 1. Variables reflecting initial cardiorespiratory function of the 17 patients with respiratory failure

Variable	Mean ± SD	Range
CPAP (cmH_2O)	14 ± 5	5–20
F_IO_2	0.38 ± 0.08	0.30–0.60
PaO_2 (mmHg)	78 ± 18	54–114
$PaCO_2$ (mmHg)	37 ± 6	30–50
Q_{sp}/Q_t (%)	24 ± 10	8–45
pHa	7.42 ± 0.05	7.30–7.52
Cardiac output (l/min)	6.9 ± 2.4	2.7–11.4
$C(a-\bar{v})O_2$ (ml/dl)	4.0 ± 1.3	2.4–6.8
$S\bar{v}O_2$ (%)	69 ± 9	56–83

Results

The study group consisted of 11 males and six females with a mean age of 57 ± 15 years (mean ± SD). Eight patients had acute respiratory failure associated with abdominal sepsis, one had fat embolism syndrome, one had pulmonary contusion, and seven patients had postoperative pulmonary failure without apparent systemic cause. Seven levels of CPAP were studied in 11 patients, 6 levels in 2, 5 levels in 3, and 9 levels in

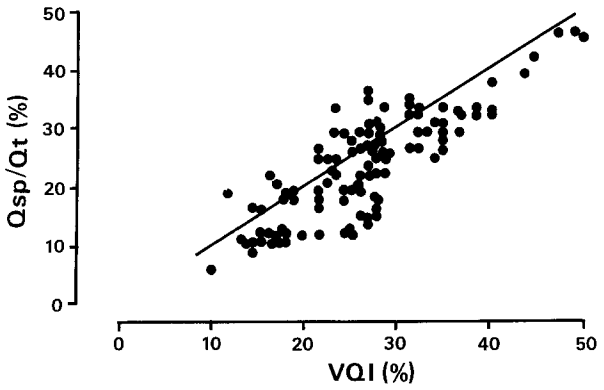

Fig. 1. Relationship betwen Q_{sp}/Q_t and oximetrically derived VQI in 17 patients with respiratory failure

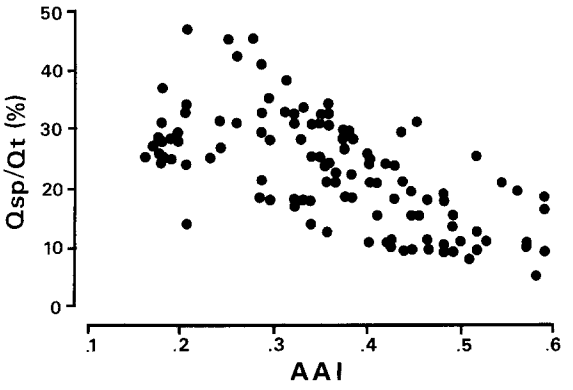

Fig. 3. Relationship between Q_{sp}/Q_t and AAI (PaO_2/PAO_2) in 17 patients with respiratory failure

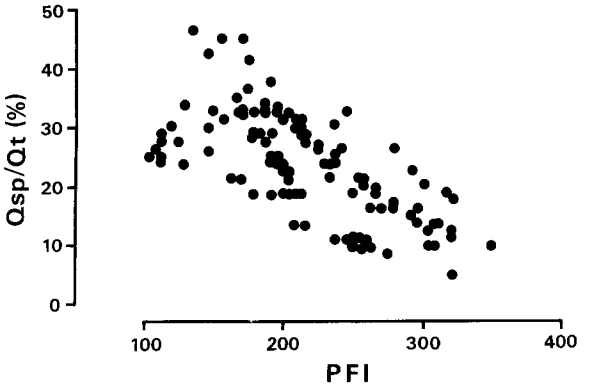

Fig. 2. Relationship between Q_{sp}/Q_t and PFI (PaO_2/F_IO_2) in 17 patients with respiratory failure

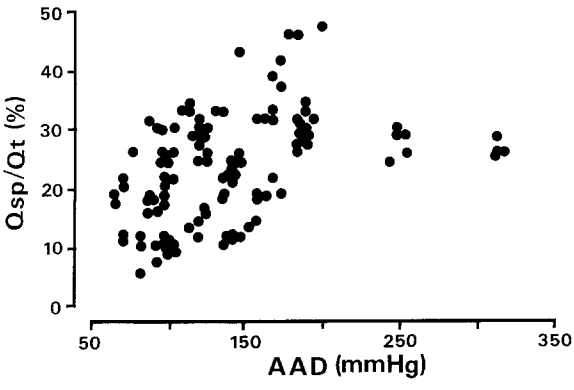

Fig. 4. Relationship between Q_{sp}/Q_t and AAD (PAO_2-PaO_2) in 17 patients with respiratory failure

1 patient, a total of 113 data points. The study was completed within 90 min in all patients. No technical problems with pulse or pulmonary artery oximetry were encountered in any patient.

Inter-subject comparisons of Q_{sp}/Q_t and the conventional oxygen tension derived indices revealed poor correlations (PFI: $r = -0.51$, AAI: $r = -0.47$, AAD: $r = 0.23$). In contrast, VQI correlated well with Q_{sp}/Q_t ($r = 0.78$). The relationship between Q_{sp}/Q_t and VQI appeared linear over the full range of measured values (Fig. 1), while the relationship between Q_{sp}/Q_t and the conventional indices demonstrated increasing scatter as Q_{sp}/Q_t increased (Figs. 2–4). Within subject correlation coefficients for the nine patients who responded to alteration in CPAP with a 10% or greater change in Q_{sp}/Q_t, were similar for all indices (VQI: $r = 0.94 \pm 0.05$, PFI: $r = -0.95 \pm 0.06$, AAI: $r = -0.95 \pm 0.006$, AAD: $r = 0.94 \pm 0.05$). Changes in peripheral circulation were small for individual patients at different levels of CPAP, but there was a large variation between subjects. Within patient change in arteriovenous oxygen content difference averaged 1.1 ml/dl with a range of 0.5–2.5 ml/dl, the total range of arteriovenous oxygen content values was from 2.0 to 7.2 ml/dl. Inspired oxygen concentration was maintained constant for individual patients, and averaged 0.38 ± 0.08 (0.30–0.60) between patients.

Discussion

This investigation was designed to determine the utility of continuous measurement of arterial and mixed venous oxygen saturations to assess pulmonary gas exchange. The results demonstrate that Q_{sp}/Q_t can be monitored accurately on a real-time basis using dual oximetry.

Until now, accurate assessment of gas exchange has depended on complex and time consuming calculation of Q_{sp}/Q_t from arterial and mixed venous

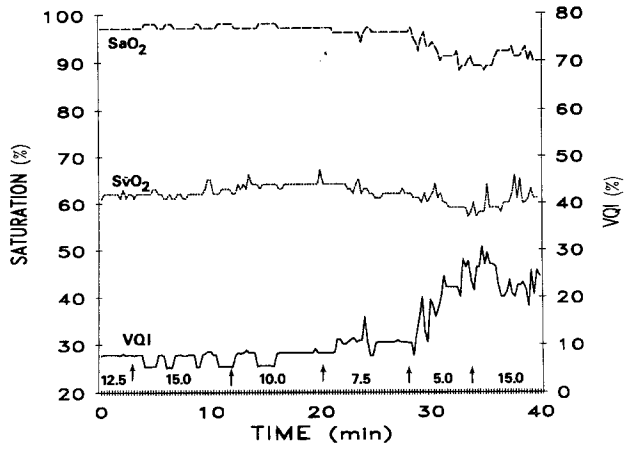

Fig. 5. Continuous recording of SaO_2, $S\bar{v}O_2$, and VQI at various levels of CPAP (arrows) in a patient with respiratory failure. The rapid increase of venous admixture upon reducing CPAP to 5 cmH_2O can be appreciated by examining the VQI trace despite the baseline fluctuation

blood samples. Even conventional bedside estimates of Q_{sp}/Q_t, PFI, AAD, and AAI, involve a delay. Furthermore, the conventional indices have been shown to be accurate only if F_IO_2 is constant, SaO_2 is sufficiently high, and if peripheral oxygen utilization remains unchanged, because they do not account for the nonlinearity of the oxyhemoglobin dissociation curve or for changes in mixed venous oxygen content [1–4, 6]. In this study, change in CPAP produced little effect on peripheral oxygen utilization, and F_IO_2 was not changed during data collection. Therefore, change in Q_{sp}/Q_t in individual patients mainly depended on changes of the oxygenation of arterial blood. As a result, within patient correlations between Q_{sp}/Q_t and the oxygen tension based indices were equally high as those between Q_{sp}/Q_t and VQI. Variations in F_IO_2 and arteriovenous oxygen content difference between patients, however, resulted in unacceptably low overall correlations between Q_{sp}/Q_t and the conventional indices, thus revealing their vulnerability to changes in peripheral circulation and oxygen therapy. Since VQI is calculated from arterial and mixed venous saturations, it responds linearly to changes in Q_{sp}/Q_t if SaO_2 is less than 100%, and it is relatively insensitive to changes of peripheral oxygen utilization and F_IO_2. Thus, good correlation between Q_{sp}/Q_t and VQI was maintained, regardless of whether measurements made in different patients or sequential values from one patient were considered. If arterial blood is 100% saturated with oxygen, VQI will only be effected by changes in dissolved oxygen and $S\bar{v}O_2$, and will no longer reflect changes in Q_{sp}/Q_t. Thus, VQI is less useful in patients with a high PaO_2 and SaO_2. However, in this situation, decrease in F_IO_2 often is appropriate, and with the subsequent decrease in SaO_2, VQI will regain its accuracy.

In the current investigation, appropriate oxygen therapy allowed VQI to remain in its accurate range at all times, thus producing a nearly linear relationship with Q_{sp}/Q_t. Therefore, VQI derived from oximetric saturations provided an accurate, real-time estimate of Q_{sp}/Q_t without the need for blood sampling. During data collection, we were able to study seven levels of CPAP within 90 min by primary commitment of a respiratory therapist and blood gas analyzer to blood sampling and analysis. Time, labor, and cost only rarely allows such assessment in clinical practice. Had we relied upon dual oximetry alone, the optimization of CPAP could have been accomplished in 30 min, the limiting factor then being the length of the equilibration period. Once a fiberoptic pulmonary artery catheter and pulse oximeter probe are in place, the response of gas exchange to changes in respiratory support can be assessed instantly. Rapid titration of therapy may be repeated as necessary without additional expense. Moreover, real-time monitoring of gas exchange possibly will reveal sudden changes in lung function that may not be detected, even by frequent, but of necessity, sporadic blood sampling.

Oximetric data for the present analysis was recorded intermittently, at the time of blood sampling. Therefore, we were unable to determine the variability of VQI resulting from fluctuation of the SaO_2 and $S\bar{v}O_2$ signals in this investigation. The magnitude of such fluctuation, however, is illustrated by a continuous recording of SaO_2, $S\bar{v}O_2$, and VQI (Fig. 5) in one patient, in whom automatic data aquisition was employed. It is currently not known to what extent these rapid alterations represent true changes in oxyhemoglobin saturation as opposed to random 'noise' generated by measurement error. However, clinically significant change in gas exchange is discernible even with present equipment, and accuracy can be expected to improve with further development of oximetric monitoring techniques.

Pulse oximetry is noninvasive and causes no adverse effects to the patient. The complications of inserting a fiberoptic pulmonary artery catheter are similar to those of routine pulmonary artery catheterization. The risk of dual oximetry, then, is no greater than that of routine monitoring with a pulmonary artery catheter. Adding oximetric monitoring capability to a pulmonary artery catheter currently involves an additional expense of $ 100, equivalent to charges for three blood gas analyses at our institution. Other components of the monitoring system are reusable. Therefore, we believe that considering the advantages of continuous monitoring of gas exchange, further de-

velopment of this method likely will result in improved quality and cost effectiveness of patient care.

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Book reviews

K. F. MacDonnell, P. J. Fahey, M. S. Segal. Respiratory Intensive Care. Edinburgh London New York: Churchill Livingstone 1987. 478 pp, hardback. £50.00. ISBN 0-316-54193-1

This book covers very thoroughly the topic of respiratory intensive care. It is written in a cohesive style with a multiauthor text. I have often found with Churchill Livingstone books the quality of the text, diagrams and colour prints to be extemely high and the book is a pleasure to the eye, extremely readable and well referenced. This enables it to be used both as a reference text and for someone coming fresh to an intensive care unit it is an introduction to the subject. Certain transatlantic topics do not travel well. The difference in drug usage is often a problem and this is noted in the chapter on the management of asthma. The chapter on legal implications makes interesting and alarming reading, but at this present moment in time is not directly relevant to the UK practice of medicine. In summary, this book can be recommended at £50 per copy for the book shelf of any hospital library with an intensive care unit, if not in the intensive care unit itself. However, few individuals would wish to spend £50 themselves to have it on their own book shelf.

A. B. Millar (London)

C. Hinds. Intensive Care – A Concise Textbook. Eastbourne: Ballière-Tindall 1987. 99 Diag., 15 Tables. 378 pp., soft cover. ISBN 0-7020-1150-9.

In "Intensive Care – A Concise Textbook" Dr. Charles Hinds aims to "summarise the important theoretical aspects of intensive care practice ... be comprehensive enough to provide sound working knowledge yet concise enough to read in its entirety." At a glance it appears to be exactly what the junior doctor ordered, paperback, fits in a white coat pocket, less than an inch thick and costs less than £ 20. Flicking through, it is more than a list but less than a reference; the chapters have all the right headings and include plenty of photographs, diagrams and tables. On closer examination Dr. Hinds would seem to have achieved his aims. In 17 well-structured chapters all major aspects of intensive care are covered from cardiac and pulmonary physiology to psychiatric disturbances. Accepting that you should not be in a position to need such a book unless you already understand basic physiology we are offered a brief reminder of the theory, then allowed to concentrate on its practical application. Thankfully in only a few places, 'Respiratory Physiology' for example, does derivation get the upper hand and for me only the 'Fluid and Electrolyte Balance' chapter contained far too little theoretical background, even for "a concise textbook". The prose is remarkably crisp and eloquent making it a pleasure to read from cover to cover and the use of a single author avoids repetition or confusion. However, with intensive care being such a controversial speciality it might be kinder to the newcomer to present things in more representative shades of grey than quite so black and white. I suppose the latter is the price one must pay for the former. All chapters are well referenced and good indexing allows rapid access to information if one is looking for moral support. In conclusion, this is an ideal book for doctors or nurses embarking on a career in intensive care, good post-graduate revision material especially for the FFARCS and a valuable addition to the library of anyone involved in the care of severely ill patients irrespective of their environment.

M. Mythen (London)