Central venous blood gas analysis

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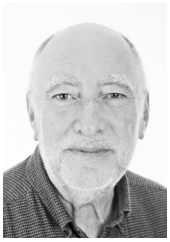

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Blood gas analysis (BGA) is a laboratory and point-of-care test routinely used to assess acid-base status along with adequacy of ventilation and oxygenation among predominantly critically/acutely ill patients.

The "gold standard" sample for BGA is arterial blood collected anaerobically by needle puncture of an artery or via an indwelling arterial catheter. BGA is unique among blood tests in its requirement for arterial blood; all other tests are performed on venous blood, collected usually by needle puncture of a peripheral vein (venepuncture); or less commonly on capillary blood obtained by finger prick.

In intensive care settings most patients who require frequent blood gas monitoring have a central venous catheter inserted that allows easy and safe sampling of venous blood for laboratory testing, obviating the need for repeated venepuncture. It would be logistically convenient for clinical staff, and more comfortable and safer for the patient if this kind of venous blood sample could also be used for BGA.

This article addresses the question: is central venous blood an acceptable alternative to arterial blood for blood gas analysis? The main focus of the article will be results of clinical studies that have compared BGA results derived from arterial blood with BGA results derived from simultaneously sampled central venous blood. Consideration will also be given to mathematical corrections that are intended to allow prediction of arterial blood gas values from measured venous blood gas values.

The article begins with a very brief discussion of relevant physiological differences that distinguish arterial and venous blood.

The arterio-venous (A-V) difference

Blood gas analysis (BGA) involves measurement of three parameters: the amount of free (unbound) oxygen (O_2) and carbon dioxide (CO_2) dissolved in blood, and the pH (acidity/alkalinity) of blood.

The partial pressure (p) exerted by the two gases is what is actually measured so the three measured parameters are: pO_2, pCO_2 and pH. A further parameter, bicarbonate (HCO_3^-) concentration is generated during blood gas analysis but this is calculated from pH and pCO_2, rather than directly measured.

pO_2 is used to assess patient oxygenation status; pCO_2 is used to assess ventilation; and pH, pCO_2 and HCO_3^- results together allow assessment of acid-base status. Another calculated parameter, base excess (BE), is also helpful, although often not necessary in this regard. Clearly, if the pO_2 of arterial blood were the same as the pO_2 of venous blood, then it would be immaterial which sample were used to assess oxygenation.

Likewise, if the pH, pCO_2 and HCO_3^- of arterial blood were the same as the pH, pCO_2 and HCO_3^- of venous blood, then it would be immaterial which sample were used to assess ventilation and acid-base status.

Of course these equalities between arterial and venous blood do not exist because of the physiological exchange of oxygen and carbon dioxide that occurs as blood flows through the capillary bed of all tissues and the capillary bed of the alveoli of the lungs.

It is this two-site gaseous exchange that fulfils a principal function of blood: delivery of inspired oxygen from lungs to all tissue cells and delivery of carbon dioxide (a waste product of cellular metabolism) from all tissue cells to lungs for excretion in expired air.

Veins convey blood from all tissues to the right side of the heart before onward journey via the pulmonary artery from heart to the lungs. This blood (venous blood) is relatively lacking in oxygen and relatively rich in carbon dioxide due to the gaseous exchange that has occurred in the capillary bed of tissue cells.

As this blood flows through the alveoli of the lungs it gains oxygen (becomes oxygenated) and loses carbon dioxide before onward journey via the pulmonary veins to the left side of the heart. Non-pulmonary arteries convey blood from the left side of the heart via the aorta to the capillary bed of all tissues. This blood (arterial blood) is oxygenated but relatively lacking in carbon dioxide due to the gaseous exchange that has occurred in the alveoli of the lungs.

The differences in the oxygen and carbon dioxide tensions of venous and arterial blood are reflected in the reference ranges of parameters generated during blood gas analysis (Table I).

Of particular note for the following discussion it is evident from Table I that normal arterio-venous (A-V) difference is much greater for the measure of oxygenation (pO_2) than for the measurements used to assess ventilation and acid-base status (pH, pCO_2, HCO_3^-).

	Arterial	Venous	Arterio-venous (A-V) difference
pH	7.35-7.45	7.31-7.41	~ 0.04
pCO_2 (kPa)	4.7 - 6.0	5.5 - 6.8	~ 0.6
pCO_2 (mmHg)	35 -45	41 - 51	~ 6
Bicarbonate (mmol/L)	22-28	23 - 29	~ 1
pO_2 (kPa)	10.6 - 13.3	4.0 - 5.3	~ 8.0
pO_2 (mmHg)	80-100	30 - 40	~ 55
sO_2 (%)	> 95	75	> 20

TABLE I: Arterial and venous blood gas reference range

Ever since BGA was first introduced to clinical care in the 1960s, arterial blood has been the standard sample; it reflects alveolar (pulmonary) gas exchange and all parameters generated by BGA are constant throughout the non-pulmonary arterial system.

The great body of research that underlies the clinical application of BGA is based for the most part on studies conducted using arterial blood. Published reference ranges used to interpret patient blood gas values have been extensively validated using arterial blood, and clinicians are familiar with these rather than reference values derived from venous blood which, in any case, are less well validated.

Despite this, over the past decade or two there has been an increasing level of clinical interest in the notion that it is worth investigating if venous blood might be a valid substitute for arterial blood in some circumstances.

The impetus for this clinical interest centres largely on the practical disadvantages associated with sampling arterial rather than venous blood, but validation and development of pulse oximetry as an alternative means of assessing arterial oxygenation has been a significant factor in driving that interest.

Difficulties associated with use of arterial blood for BGA

Collection of arterial blood for BGA is usually by needle puncture of a peripheral artery. The most common puncture site is the radial artery in the wrist; alternative sites include the brachial artery in the forearm and the femoral artery in the groin.

Compared with venepuncture, arterial puncture is technically more demanding and significantly more painful and hazardous for the patient [1-3]. Specialist training in arterial puncture is essential for patient safety and comfort, and in many countries, obtaining arterial blood by arterial puncture remains the almost exclusive preserve of medically qualified staff.

By contrast, venepuncture is a very commonplace procedure that can be easily and safely performed, after minimal training, by ancillary staff with no medical or nursing education.

In an intensive care setting patients often have an indwelling arterial catheter fitted principally to enable continuous blood pressure monitoring. These catheters also allow convenient and painless sampling of arterial blood for BGA.

Although this method of arterial blood sampling obviates the need for repeated needle puncture of patients requiring frequent BGA, fitting of an arterial catheter is itself an invasive and technically difficult procedure [4] that is associated with risk of serious complications including systemic infection, haemorrhage, thrombosis and ischemia [5, 6].

So common and serious are these complications that some have recently questioned whether the benefit of continuous blood pressure monitoring among the critically ill outweighs the considerable risk of arterial catheterization [7].

These concerns suggest that there might be more restricted use of the arterial catheter in the future. If so, then the only means of obtaining arterial blood for BGA, even in an intensive care setting, would be arterial needle puncture.

Patients who require BGA also require regular venous blood sampling for other blood tests. It would clearly be convenient, safer (for patients and staff) and more economic if a single venous sample could be used for all blood tests, including BGA.

Impact of pulse oximetry

The contribution that BGA makes to the assessment of patient oxygenation status is measurement of pO_2. sO_2 determines the % of haemoglobin that is saturated with oxygen (sO_2) and thereby the total amount of oxygen in blood.

The relationship between pO_2 and sO_2, described graphically in the familiar sigmoidally shaped oxyhaemo-

globin dissociation curve, allows calculation of sO_2 from measured pO_2. Arterial blood gas analysis thus allows measurement of arterial pO_2 (pO_2(a)) and calculation of arterial sO_2 (sO_2(a)).

In practice modern blood gas analyzers have an incorporated CO-oximeter that allows direct measurement of sO_2(a).

Pulse oximetry, which has become ubiquitous in all areas of clinical medicine since the mid-1990s, provides an alternative entirely safe, non-invasive means of continuously monitoring arterial oxygen saturation and thereby roughly predicting pO_2(a).

Although there is clinically acceptable agreement between arterial oxygen saturation measured by pulse oximetry (SpO_2) and arterial oxygen saturation measured (or calculated) during BGA (sO_2(a)) for most patient groups [8], this is not necessarily the case [9].

There is for example conflicting evidence that SpO_2 is a less-than-reliable measure of sO_2(a) among critically ill patients with anemia, hypoxemia or acidosis [10]. Still, for many patients in whom the only reason for performing BGA is assessment of oxygenation status, pulse oximetry is a very convenient, reliable and safe alternative.

With pulse oximetry now providing an alternative means of assessing arterial oxygenation, studies aimed at consideration of the reliability of venous blood as a substitute for arterial blood have been able to focus principally on those blood gas parameters (pH, pCO_2 and bicarbonate) that have lowest A-V difference (Table I) and therefore most likely to show agreement when arterial and venous values are compared.

Peripheral venous blood, central venous blood and mixed venous blood

Many (probably most) clinical studies investigating the validity of using venous blood for BGA have been conducted using venous blood obtained by conventional venepuncture of a peripheral vein (i.e. peripheral venous blood) [11-20].

This article is concerned only with studies [21-28] that have utilized central venous blood samples for comparison with arterial blood.

Central venous blood is the blood that is sampled via a central venous catheter (CVC). In addition to facilitating the means for easy sampling of venous blood for diagnostic testing, CVCs allow continuous monitoring of central venous pressure (vital in the haemodynamically unstable patient), and vascular access for administration of drugs, blood transfusion and other fluids.

Most patients (up to ~80 %) in intensive care have an indwelling CVC, but CVC use is not confined to this patient population so these studies [21-28] have relevance outside the intensive care unit, in emergency rooms, recovery rooms and some medical wards.

CVCs are usually inserted cutaneously via the jugular vein in the neck or subclavian vein in the upper chest to the superior vena cava, with the tip sited close to the point where the superior vena cava opens to the right atrium of the heart (Fig. 1), so that the blood sampled is the mixed venous blood from the upper half of the body.

The inferior vena cava conveys mixed venous blood from the lower half of the body to the right atrium. Central venous blood is thus not truly mixed venous blood because it does not include that returning via the inferior vena cava.

Mixing of venous blood from all parts of the body occurs as it flows from the right atrium to the right ventricle before journey from the heart via the pulmonary artery.

Catheterization of the pulmonary artery provides the only means of sampling true mixed venous blood.

Peripheral blood obtained by venepuncture is different from central ("mixed") venous blood and true mixed venous blood with regard to blood gas parameters (pH, pCO_2, pO_2) because as venous blood returns from the periphery back to the heart, it becomes mixed with venous blood from other tissues having differing levels of metabolic activity and therefore potentially differing pH, pO_2, and pCO_2.

Unlike arterial blood, which remains constant with regard to these values until it reaches the capillary bed of tissues, venous blood gas values can potentially differ to some extent with site of sampling.

Studies comparing central venous and arterial blood gas results

All clinical studies [11-28] investigating the validity of using venous blood for BGA share a simple and common design. In essence BGA results derived from arterial blood are compared with BGA results derived from simultaneously collected venous blood among a defined cohort of patients requiring BGA.

It is of course vital for the validity of the comparison that both arterial and venous samples are collected anaerobically and analysed within a common short time frame, using the same analyser.

Of seven studies [21-27] that have examined the validity of using central venous blood for blood gases, all compared central venous and arterial pH; six [21-23, 25-27] compared central venous and arterial pCO_2; four [21, 24, 26, 27] compared central venous and arterial bicarbonate (HCO_3^-); two [23, 24] compared central venous and arterial base excess; and just one [25] compared central venous and arterial pO_2.

Some details of these seven studies along with summary of the results are contained in Tables II-VI. The two most significant columns in these tables are the mean arterio-venous (A-V) difference along with range or SD of that difference; and the 95 % limits of agreement (LOA) on a Bland-Altman plot.

A Bland-Altman plot is the accepted method for assessing the agreement between two tests and represents a clinically relevant measure of comparison. The difference between two paired (arterial and central venous) values are plotted against the mean of those two values.

The derived 95 % LOA allows estimation of the range of difference that can be expected between central venous and arterial values for all patients represented by the study population.

Central venous pH versus arterial pH

In all seven studies mean arterial pH was higher than the mean central venous pH (see Table II). The magnitude of this positive bias (mean A-V difference) ranged from 0.027 [26] to 0.05 pH units [21], but in most studies [23-27] mean bias was close to 0.03 pH units.

Four of the seven studies provided 95 % LOA data. For the study showing best agreement [25] 95 % LOA was 0.008 to 0.063. This indicates that if measured central venous pH is 7.40, then in 95 % of patients arterial pH would lie within the range of 7.408 to 7.463, with most close to 7.43.

For comparison, the study [23] showing the worst level of agreement, with 95 % LOA –0.03 to 0.09 indicates that for a measured central venous pH of 7.40 arterial pH would lie within the range of 7.37 to 7.49 for 95 % of patients, again with most close to 7.43.

Given the narrow 95 % LOA and the consistency of mean A-V difference across nearly all studies, there is general agreement [23-27] that central venous pH is a clinically acceptable substitute for arterial pH after taking account of the systematic positive bias of ~0.03 pH units.

Central venous pCO_2 versus arterial pCO_2

In all six studies mean arterial pCO_2 was found to be less than mean central venous pCO_2 (see Table III). The magnitude of this negative bias (mean A-V difference) ranged from 0.52 [26] to 1.22 kPa [21] (i.e. 3.9 to 9.2 mmHg) with the four most recent studies [23-27] indicating a negative bias in the narrower range of 0.52 to 0.79 kPa (3.9 to 5.9 mmHg).

Three of the six studies provided 95 % LOA data. For the study showing best agreement [25] 95 % LOA was–1.3 to –0.28 kPa. This indicates that if measured central venous pCO_2 is 5.0 kPa (38mmHg), then in 95 % of patients, arterial pCO_2 would lie within the range of 3.70 - 4.72 kPa (28-35 mmHg) with most close to 4.2 kPa (31 mmHg).

Patient number and type	No. of paired samples	Mean Arterial (range or ±2SD)	Mean Venous (range or ±2SD)	Mean A-V diff (range or ±2SD)	Bland-Altman 95 % LOA	Reference
55 "seriously ill" surgical patients	55	7.39 (7.15 to 7.55)	7.34 (7.12 to 7.48)	0.05 (0 to 0.13)	NR	21 (1967)
41 Critically ill adults in ICU	41	7.40 (6.97 to 7.56)	7.36 (6.95 to 7.51)	0.04 (-0.01 to 0.1)	NR	22 (1969)
25 adult trauma patients in ICU	99	7.39 (±0.14)	7.36 (±0.14)	0.032 (±0.052)	-0.03 to 0.09	23 (2005)
110 adult patients in ICU	168	7.37 (7.12 to 7.50)	NR	0.03 (NR)	-0.01 to 0.07	24 (2006)
73 adults from thoracic ICU, general ICU and pulmonary ICU	73	7.39 (7.24 to 7.54)	7.35 (7.21 to 7.45)	0.036 (±0.028)	0.008 to 0.063	25 (2008)
40 adults medical ICU, 72 % with sepsis	190	7.37 (±0.276)	7.34 (±0.268)	0.027 (±0.054)	-0.028 to 0.081	26 (2010)
187 adults medical and surgical ICU and cardiac catheterization lab.	187	7.41 (±0.14)	7.37 (±0.14)	0.035 (±0.04)	only venous adjusted LOA recorded - see text	27 (2010)

TABLE II: Arterial versus central venous pH

NR - not recorded

For comparison, the study showing worst level of agreement [26] with 95 % LOA –1.63 to +0.64 kPa, a measured central venous pCO_2 of 5.0 kPa predicts an arterial pCO_2 in the range of 3.37 to 5.64 kPa (25 to 42 mmHg) for 95 % of patients with most close to 4.5 kPa (34 mmHg).

There is general agreement [22, 25-27] that central venous pCO_2 is a clinically acceptable substitute for arterial pCO_2 in most clinical contexts so long as the systematic negative bias of ~0.6 kPa (5.0 mmHg) is taken into account.

The authors of one study [23] consider the 95 % LOA too wide for general substitution of central venous values but concede that central venous pCO_2 provides clinically valuable information that, for example, can guide weaning of trauma patients (the population they were studying) from mechanical ventilation.

In general one can be 95 % certain that after correction for systematic bias, central venous pCO_2 is within ±0.52 kPa (i.e. ±3.9 mm Hg) of arterial pCO_2 [25].

Central venous bicarbonate versus arterial bicarbonate

Since bicarbonate (HCO_3^-) generated during blood gases is calculated from pH and pCO_2 , it would be expected that if central venous pH and pCO_2 are clinically acceptable substitutes for arterial pH and pCO_2,then central venous HCO_3^-, too, would be an acceptable substitute for arterial HCO_3^- (see Table IV).

This is borne out by the results of the four studies [21, 24, 26, 27] that compared central venous and arterial HCO_3^-. All studies indicate that mean central venous HCO_3^- concentration is slightly higher than mean arterial HCO_3^- concentration.

Patient number and type	No. of paired samples	Mean Arterial (range or ±2SD)	Mean Venous (range or ±2SD)	Mean A-V diff (range or ±2SD)	Bland-Altman 95 % LOA	Reference
55 "seriously ill" surgical patients	55	4.28 (1.99 to 9.31)	5.50 (2.66 to 10.3)	-1.2 (range/ SD -NR)	NR	21 (1967)
41 Critically ill adults in ICU	41	4.52 (2.66 to 8.88)	5.58 (2.93 to 9.71)	-1.06 (-2.39 to +0.27)	NR	22 (1969)
25 adult trauma patients in ICU	99	5.45 (±1.96)	5.98 (±1.83)	-0.58 (±0.89)	-1.44 to -0.29	23 (2005)
73 adults from thoracic ICU. general ICU and pulmonary ICU	73	5.80 (3.98 to 10.81)	6.61 (4.64 to 10.9)	-0.79 (±0.52)	-1.30 to +0.64	25 (2008)
40 adults medical ICU, 72% with sepsis	190	5.10 (±0.276)	5.62 (±0.268)	-0.52 (±0.054)	-1.63 to +0.64	26 (2010)
187 adults medical and surgical ICU and cardiac catheterization lab.	187	5.32 (±0.14)	5.98 (±0.14)	-0.59 (±0.04)	only venous adjusted LOA recorded - see text	27 (2010)

TABLE III: Arterial versus central venous pCO_2 (kPa) ‡

NR - not recorded

‡ to convert kPa to mmHg divide by 0.133

Patient number and type	No. of paired samples	Mean Arterial (range or ±2SD)	Mean Venous (range or ±2SD)	Mean A-V diff (range or ±2SD)	Bland-Altman 95 % LOA	Reference
55 "seriously ill" surgical patients	55	NR	NR	-2,2	NR	21 (1967)
110 adult patients in ICU	168	25 (14.6 to 42.2)	NR	-0.52 (NR)	-2.85 to +1.85	24 (2006)
40 adults medical ICU, 72 % with sepsis	190	22.4 (±15.2)	23.2 (±15.6)	-0.8 (±3.16)	-4.0 to +2.4	26 (2010)
187 adults medical and surgical ICU and cardiac catheterization lab.	187	25.4 (±8.4)	26.6 (±13.2)	-1.13 (±8.6)	only venous adjusted LOA recorded - see text	27 (2010)

TABLE IV: Arterial versus central venous HCO_3^- (mmol/L)

NR - not recorded

The magnitude of this negative bias (A-V difference) ranged from 0.52 mmol/L in one study [24] to 2.2 mmol/L in another [21]. Of the four studies, three returned negative bias of <1.2 mmol/L, which is clinically insignificant. Two studies [24, 26] provided 95 % LOA data.

The study showing best level of agreement with 95 % LOA –2.85 to +1.85 indicate that if measured central venous HCO_3^- is 25 mmol/L, then in 95 % of patients predicted arterial HCO_3^- would be in the range of 22 to 27 mmol/L with most close to 26 mmol/L.

There is general agreement that central venous bicarbonate is a clinically acceptable substitute for arterial bicarbonate, especially if the small systematic positive bias of ~1mmol/L is taken into account.

Central venous base excess versus arterial bases excess

Just three studies [22-24] compared central venous and arterial base excess (BE) (see Table V). Mean A-V difference was small (–0.19 mmol/L and –0.18 mmol/L) and 95 % limit of agreement was sufficiently narrow for one study author to conclude that central venous and arterial values are interchangeable [24].

Central venous pO_2 versus arterial pO_2

Just one study [25] compared central venous O and arterial O (see Table VI). The large mean and range of A-V difference of 8.33kPa ± 7.88 (2SD) (i.e. 63 ± 59 mmHg) confirms that it is not possible to use central venous O as a reliable substitute for arterial O.

There is no correlation between arterial O and venous O (irrespective of the sampling site). The only reliable sample for accurately determining arterial oxygenation is arterial blood. Pulse oximetry provides an alternative means of assessing patients' oxygenation status that requires no blood sampling.

Patient number and type	No. of paired samples	Mean Arterial (range or ±2SD)	Mean Venous (range or ±2SD)	Mean A-V diff (range or ±2SD)	Bland-Altman 95 % LOA	Reference
110 adult patients in ICU	165	-0.1 (-12 to +16)	NR	-0.19 (range/SD -NR)	-2.24 to +1.86	24 (2006)
25 adult trauma patients in ICU with sepsis	99	-0.01 (±7.76)	-0.34 (±7.44)	-0.34 (±2.06)	-2.20 to +1.80	26 (2010)

TABLE V: Arterial versus central venous base excess (mmol/L)

NR - not recorded

Patient number and type	No. of paired samples	Mean Arterial (range or ±2SD)	Mean Venous (range or ±2SD)	Mean A-V diff (range or ±2SD)	Bland-Altman 95 % LOA	Reference
73 adults from thoracic ICU. general ICU and pulmonary ICU	73	11.32 (6.6 to 28.3)	5.41 (3.86 to 7.16)	8.33 (±7.88)	Not calculated	24 (2008)

TABLE VI: Arterial versus central venous pO_2 (kPa) ‡

‡ to convert kPa to mmHg multiply by 0.133

Patients in severe circulatory failure - a special case

The studies discussed thus far [21-27] have confirmed that the normal arterio-(central)venous (A-V) difference for pH and pCO_2 (~0.03 pH units and ~ –0.6 kPa respectively) are maintained within broadly clinically acceptable limits for the generality of patients requiring BGA.

That is not the case for patients with severe circulatory failure (for example those suffering cardiac arrest). Adrouge *et al* [28] found much larger A-V differences in this small subset of very critically ill patients.

His study revealed that mean difference between arterial pH and central venous pH ranged from 0.10 to 0.35 pH units depending on the severity of the circulatory failure, rather than ~0.03 pH units.

Mean difference between arterial pCO_2 and central venous pCO_2 for the same group ranged from –3.2 to –7.4 kPa, rather than –0.6kPa. According to the authors of this report assessment of acid-base status in these patients requires consideration of both arterial and central venous blood gas results.

Two further studies [29, 30] confirm the much larger difference between arterial and central venous pH and pCO_2 for patients in circulatory collapse.

Mathematical corrections

There are three methods for mathematically converting measured central venous blood gas results to give "arterial" blood results. The first and most simple, which has already been hinted at, is to use the systematic differences between arterial and central venous blood that have been derived from the seven studies [23-27] thus:

"arterial" pH = measured central venous pH + 0.03

"arterial" pCO_2(KPa) = measured central venous pCO_2 –0.6

"arterial" HCO_3^- (mmol/L) = measured central venous HCO_3^- + 1.0

The capacity of this simple approach to improve diagnostic accuracy has been demonstrated by Walkey *et al* [27].

A second approach is to use regression equations generated during studies comparing central venous and arterial values. Treger *et al* [26] derived the following regression equations from their data:

"arterial" pH = –0.307+1.05 × measured central venous pH

"arterial" pCO_2 (mmHg) = 0.805 + 0.936 × central venous pCO_2 (mmHg)

"arterial" bicarbonate = 0.513 + 0.945 × central venous bicarbonate

The validity (accuracy) of these two approaches depends on the assumption that the generality of patients are represented by the study population from which the systematic differences and regression equations are derived.

Toftegaard *et al* [31] have recently developed a novel much more sophisticated, patient-specific method of converting venous (either central, peripheral or mixed) to arterial values that depends on measuring arterial oxygenation by pulse oximetry at the time that venous blood is sampled for blood gases.

The principle of the method is "to calculate arterial values by simulating, with the help of mathematical models, the reverse transport of blood from the veins to the arteries until the simulated arterial oxygenation matches that measured by pulse oximetry"– effectively, a mathematical arterialization of venous blood.

The complex mathematical transformation requires input of the following measured venous parameters all available on modern blood gas analyzers: pH, pCO_2, pO_2, sO_2(a), hemoglobin, methemoglobin and carboxyhemoglobin; along with SpO_2 determined by pulse oximetry.

A validation study of this method [31] indicates that calculated arterial values for pH and pCO_2 by this method are essentially the same as measured arterial values.

The transformation also allows for the first time a clinically useful estimation of arterial pO_2 from central venous blood, although this clinical utility only applies to patients with SpO_2 <96 %. For those with SpO_2 >96 %, arterial pO_2 cannot be estimated within an acceptable clinical range by this method.

The imprecision in estimating arterial pO_2 when SpO_2 is >96 % is due to the flat shape of the oxyhemoglobin dissociation curve at high sO_2 values where small changes in sO_2 result in large changes in pO_2.

Although this limits the usefulness of this way of calculating pO_2(a), the authors of this study observe that it is encouraging that the method is able to predict pO_2 (a) within clinically acceptable limits for patients with low SpO_2 because these are the clinically interesting patients.

Previous study [32] has shown this method of estimating pO_2(a) to be highly sensitive to error in SpO_2 measurement. In the validation study [31] comparison of patients' SpO_2 and sO_2(a) revealed a mean SpO_2 bias (SD) of 0.4 %± 1.0 %.

This favorable degree of accuracy/precision in SpO_2 measurement allows calculation of pO_2(a) within ±1.85 kPa (2SD) of measured value, if SpO_2 is <96 %. This is judged clinically acceptable. Error ≥2 % (SD) in SpO_2 measurement results in inaccurate (clinically unacceptable) pO_2(a) estimation, even if SpO_2 is <96 %.

Summary

- Central venous blood is unsuitable for determining patient oxygenation status. For many patients this can be determined sufficiently accurately using non-invasive pulse oximetry. If this is not the case, arterial blood must be sampled for measurement of pO_2(a) and sO_2(a).
- Although central venous pH, pCO_2(a) and bicarbonate are not interchangeable with arterial values, there is excellent correlation between the two for all three parameters.
- With the exception of patients in severe circulatory failure, on average central venous pH is 0.03 pH units lower than arterial pH; central venous pCO_2 is 0.6 kPa (5 mmHg) higher than arterial pCO_2; and central venous and arterial bicarbonate are essentially the same.
- Corrected central venous pH, pCO_2 and bicarbonate provide results that are, in many cases, clinically insignificantly different from those obtained using the"gold standard" arterial blood sample.
- Acidosis and alkalosis can be correctly diagnosed using central venous blood but severity may be under- or overestimated in some patients.
- Central venous blood gases provide clinically useful information about patient acid-base status that can in some cases obviate the necessity for arterial sampling. Certainly the finding of corrected central venous pH, pCO_2 and bicarbonate values within the normal arterial reference range is reliable evidence of normal acid-base status.
- A recently developed, highly sophisticated mathematical conversion allows the most precise calculation of arterial pH, pCO_2 and bicarbonate from measured central venous values. The conversion requires input of oxygen saturation measured by pulse oximetry (SpO_2). The conversion also allows a clinically useful estimation of arterial pO_2 so long as SpO_2 is <96 %.

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