

CONGENITAL HEART DISEASE

Haemodynamic calculations in the catheter laboratory

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Many of the calculations used in the evaluation of haemodynamic abnormalities are relatively simple and can be performed rapidly with a hand held calculator or (for the mentally agile) "in the head". Others are more complex and require a more time consuming process of analysis of the recorded data, often performed some time after the actual procedure.

Currently available catheter laboratory equipment for physiological monitoring and analysis will often provide a range of semi automatic calculations which will save time and allow the production of a comprehensive report at the conclusion of the procedure. It is vital, however, that cardiologists continue to have a clear understanding of the basis of such calculations and the limitations/pitfalls intrinsic to them and to some of the data on which they are based. Some of the calculations that can be made are of limited clinical utility while others are potentially misleading unless the data from which they are derived are carefully checked for accuracy and have been obtained using rigorous methodology.

When, as is all too often the case, the data have been acquired largely automatically and have not been carefully scrutinised by someone familiar with the potential errors, the figures for pulmonary and systemic blood flow, shunt flows and resistances may be almost meaningless and can readily lead to inappropriate and potentially dangerous decisions.

In practice most of the important calculations—shunt ratio (Qp:Qs), pulmonary blood flow, and pulmonary vascular resistance—can be estimated, albeit imprecisely, on the basis of straightforward and quick "guesstimates" which provide a rapid and generally useful "cross check" of the figures produced by the computer (or by a more time consuming and comprehensive manual method). While such rapid calculations are not a substitute for a careful and detailed analysis of the data, they are an effective way of understanding how the data relate to the haemodynamic disturbance; they also allow the trainee (or the established cardiologist) to demonstrate his or her mastery of the concepts involved and to avoid being over dependent on the "computer generated" report.

This article will focus on the usefulness of the different calculations in clinical practice and on a number of simple (short cut) methods of performing some of them, in an effort to "cross check" the more complete data obtained by the computer or by more laborious manual methods.

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Shunts

In patients with congenital heart disease in whom there is a communication between the two sides of the heart, or between the aorta and the pulmonary artery, allowing a shunt to exist, a number of calculations may be made. These include:

- (1) left to right shunt;
- (2) right to left shunt;
- (3) effective pulmonary blood flow;
- (4) pulmonary to systemic flow ratio (Qp:Qs).

Of these calculations the only one that is of practical value is probably the pulmonary to systemic flow ratio (Qp:Qs). This provides a simple and reliable estimate of the extent to which pulmonary flow is increased or reduced and provides a useful insight into the severity of the haemodynamic disturbance in most cases. It is also very simple to perform, employing solely the oxygen saturation data from systemic arterial blood, left atrial/pulmonary venous blood, pulmonary artery, and vena caval/right heart samples.

The samples need to be acquired with the patient breathing (or being ventilated with) air or a gas mixture containing no more than a maximum of 30% oxygen. If oxygen enriched gas is being given (> 30% oxygen) then the saturation data may not provide accurate information regarding pulmonary blood flow, as a significant amount of oxygen may be present in dissolved form in the pulmonary venous sample (which will not be factored into the calculation if saturations alone are used). Under such circumstances pulmonary flow will tend to be overestimated and the Qp:Qs ratio will be correspondingly exaggerated.

The calculations to determine left to right shunt, right to left shunt, and effective pulmonary blood flow are all fairly simple. They do not provide particularly useful information, however, and will not be discussed further here.

Pulmonary to systemic flow ratio (Qp:Qs)

The calculation is based on the Fick principle, by which both pulmonary and systemic flow may be estimated. As such factors as oxygen carrying capacity and oxygen consumption are used for each individual calculation (for pulmonary and for systemic flow), they cancel out when only the ratio of the two flows is being estimated. This is very convenient as it removes the more difficult and time consuming parts of the calculation. The resulting equation (after removing the factors which cancel out) is pleasingly simple:

$$Q_p:Q_s = \frac{\text{Sat Ao} - \text{Sat MV}}{\text{Sat PV} - \text{Sat PA}} \quad (1)$$

where: Sat Ao is aortic saturation, Sat MV is mixed venous saturation, Sat PV is pulmonary

vein saturation, and Sat PA is pulmonary artery saturation.

As the arterial saturation (Sat Ao) and the pulmonary artery saturation (Sat PA) are routinely estimated, the only components of this set of data that may present any problem are the pulmonary vein saturation (Sat PV) and the “mixed venous” saturation (Sat MV). If a pulmonary vein has not been entered an assumed value of 98% may be employed for Sat PV. The left atrial saturation can be substituted provided that there is no right to left shunt at atrial level. Similarly left ventricular or arterial saturation may be substituted, provided that there is no right to left shunt.

For “mixed venous” (Sat MV) the tradition is to use the most distal right heart chamber or site where there is no left to right shunt. Thus, right atrium may be used in the absence of an atrial septal defect or right ventricle if there is no shunt at atrial or ventricular level. In practice superior vena cava (SVC) saturation is often used but a value intermediate between SVC and inferior vena cava (IVC) may be preferable as the two may be significantly different. It has been demonstrated that the mixed venous saturation more closely approximates to the SVC than to the IVC. Hence the following formula is often used¹:

$$\text{Sat MV} = \frac{3 \times \text{Sat SVC} + 1 \times \text{Sat IVC}}{4} \quad (2)$$

It is noteworthy that IVC saturation varies depending on where the sample is obtained, and the sampling site should be at the level of the diaphragm to ensure that hepatic venous blood is taken into account.

A very simple way of calculating this (“in the head”) is to use the formula:

$$\text{MV} = \text{Sat SVC} - \frac{\text{Sat SVC} - \text{Sat IVC}}{4} \quad (3)$$

Thus if SVC saturation (Sat SVC) is 78% and IVC saturation (Sat IVC) is 70%, mixed venous (MV) should be 76% ($78 - 70 = 8$; $8/4 = 2$; $78 - 2 = 76$).

As mentioned above, it is important that the samples used for this calculation are acquired with the patient breathing air or an oxygen enriched mixture not exceeding 30%. If higher concentrations of oxygen (50% or greater) are to be used (to test for pulmonary vascular reactivity, for example) then the calculation of pulmonary blood flow (and Qp:Qs ratio) should involve measurement of pO₂ on at least the pulmonary vein sample (preferably also the pulmonary artery sample). This allows inclusion of dissolved oxygen in the calculation (a more complex calculation, which necessitates calculation of the oxygen content of the samples—see below).

Usefulness of shunt ratio in practice

Qp:Qs ratio is very useful in many situations—such as in making decisions about surgery for a child with a ventricular septal defect where, in

Common sources of errors

- Use of inappropriate value/sample for “mixed venous” blood
- Failure to calculate dissolved O₂ when using enriched gas (for example, 100% O₂)

a child beyond infancy, a shunt producing a Qp:Qs $\geq 1.8:1$ is likely to require intervention, while one of $\leq 1.5:1$ may be regarded as insignificant. Qp:Qs is also helpful in assessing the haemodynamics of many more complex defects but it should be recognised that under some circumstances it is of limited practical help. For instance, with an atrial septal defect, if there is evidence of a significant shunt on clinical grounds and non-invasive testing (for example, right ventricular dilatation on echocardiography, with paradoxical movement of the ventricular septum; cardiomegaly on x ray; well developed right ventricular volume load pattern on ECG (incomplete right bundle branch block)), the shunt ratio at catheter should not be used to decide about treatment. This is because of the fact that atrial shunts, which depend on right ventricular filling characteristics, can vary depending on conditions (for example, sympathetic tone, catecholamine concentrations). It is not uncommon for the measured shunt, at the time of catheter, to be small (for example, $< 1.5:1$) despite other evidence of a significant atrial septal defect/shunt.

Cardiac output and pulmonary blood flow

Assessment of cardiac output and of pulmonary blood flow is important in several situations. In the absence of any shunt pulmonary flow and systemic cardiac output are the same and may be measured as part of the investigation of patients with impaired cardiac function for a variety of reasons—notably as part of transplant assessment (for example, in patients with cardiomyopathy). In such patients the simplest methods of measuring cardiac output are by thermodilution or using the Fick method. The latter requires estimation of oxygen consumption, which presents considerable practical difficulties, and assumed values based on age, sex, and heart rate are often substituted (see below).

Thermodilution provides a straightforward and useful alternative,² but will only provide meaningful data when no shunt is present. The principle is similar to that of indicator (dye) dilution methods for measuring cardiac output.

The latter (dye dilution) is now seldom used but involves the injection of a bolus of indicator (dye) into the circulation, which is diluted in the blood stream³. Sampling is done at a site some distance “downstream” and the concentration of indicator is measured continuously, using a cuvette, during its first pass through the circulation, producing a time/concentration

curve. The down slope of the primary curve is projected to the baseline, in order to exclude recirculation of the indicator. The mean concentration of the indicator during this first passage is then used, with the duration (in seconds) of the extrapolated curve (from the time of first detection of indicator) and an estimate of cardiac output can be obtained using the “Stewart-Hamilton” formula:

$$\text{Cardiac output(l/min)} = \frac{I \times 60}{Ct} \quad (4)$$

where I is the quantity of injectate (mg), C is mean concentration (mg/l), and t is time in seconds.

Several dyes have been used, notably Evans blue, Cardiogreen, and methylene blue.

Using thermodilution a catheter with a lumen opening via a side hole in the right atrium and with a thermister at the tip, placed in the pulmonary artery, is employed. A bolus of cooled dextrose solution at either 5°C or at room temperature (22°C) is injected rapidly into the right atrium, and a time/temperature curve is recorded via the thermister in the pulmonary artery. Several determinations are usually made and are averaged.

The technique is now largely automated and a computer does the calculations. The volume and temperature of the injectate are critical and the speed of delivery of the bolus is also important. While the method is generally simple and reliable it is important that operators are familiar with the technique and this necessitates that one or more technologists or cardiologists gain experience with using the method on a regular basis. Results have been shown to correlate closely with both dye dilution and Fick methods, though in low cardiac output states the Fick method is considered to be more reliable.

While methods exist for estimating the size of left to right shunts (for example, Q_p:Q_s) using indicator dilution, the assessment of systemic cardiac output and pulmonary flow is not valid in the presence of shunting.

Common sources of errors

- Slow injection of cooled dextrose
- Operator “selection” of computer results. When the results are “scattered” the operator may elect to reject those that appear to be wide of the anticipated value and to average only those that are closer to that which is expected (it is worthy of note that some degree of “scatter” is frequent with this method)

Calculation of cardiac output and pulmonary blood flow by the Fick method is the routine for use in patients with septal defects and associated shunts. The method depends on the fact that oxygen uptake by the lungs is equal to oxygen consumption in the tissues. Blood flow is calculated by measurement of the oxygen

content of venous blood and of arterial blood (in ml/l) and hence estimating the difference between the two, which represents the tissue oxygen utilisation. In general the difference (pulmonary VA O₂ diff. or systemic AV O₂ diff.) tends to be in the order of 20–50 ml/l, depending on conditions and with considerable variability between individuals. If oxygen consumption (V_{O₂}) is known (in an adult usually around 200–250 ml/min) then blood flow is calculated by the simple equation:

$$Q = \frac{V_{O_2}}{VA \text{ O}_2 \text{ diff.}} \quad (5)$$

where Q = blood flow in l/min.

Thus, in the above example, if the content difference is 50 ml/l and oxygen consumption is 250 ml/min then blood flow is 5 l/min.

The same equation allows calculation of either pulmonary blood flow or systemic cardiac output—by substituting pulmonary VA O₂ diff. or systemic AV O₂ diff.

Thus Q_p (pulmonary flow) is calculated by the equation:

$$Q_p = \frac{V_{O_2}}{\text{Pulmonary VA O}_2 \text{ diff.}} \quad (6)$$

Similarly systemic flow may be estimated employing the difference in oxygen content between the aorta and a “mixed venous” sample (systemic AV O₂ diff.)

$$Q_s = \frac{V_{O_2}}{\text{Systemic AV O}_2 \text{ diff.}} \quad (7)$$

In practice absolute values for pulmonary and systemic flow are less useful than indexed values (corrected for body surface area). Therefore most paediatric cardiologists will take into account surface area; the simplest way of doing this is to employ a figure for oxygen consumption that has been related to body surface area—for example, ml/min/m². Thus for an adult with a body surface area of 2 m² and a V_{O₂} of 240 ml/min the oxygen consumption may be expressed as being 120 ml/min/m². Flow calculations then produce a result in “litres/min/m²”. This correction (for body surface area) is particularly important for estimation of pulmonary and systemic vascular resistance, where the use of indexed flows (pulmonary flow index and systemic cardiac index) produces meaningful resistance calculations without the need for any further “correction”.

The critical parts of these equations are the calculation of the oxygen content of the various samples and estimation of oxygen consumption. Oxygen content is calculated by estimating the oxygen carrying capacity of the patient’s blood, as haemoglobin bound oxygen. This is the volume of oxygen that could be carried on haemoglobin at 100% saturation. This is calculated by: Hb (g/l) × 1.36.

Usually this is in the order of 200 ml/l, though it varies with Hb. The content of each sample is then computed by multiplying by the saturation. Thus if Hb is 140 g/l and saturation in a sample is 70% the oxygen carrying capacity will be $140 \times 1.36 = 190$ ml/l and content will be $190 \times 70\% = 133$ ml/l.

Providing that the patient is breathing air or an oxygen enriched mixture of 30% or less the amount of dissolved oxygen in plasma is sufficiently small as to be unimportant. Each sample needs to have its oxygen content calculated as above. The pulmonary VA oxygen difference and the systemic AV oxygen difference are thus easily estimated. As in the calculation for Qp:Qs ratio the mixed venous saturation is estimated either using SVC alone or by employing a sample from within the right heart (proximal to any left to right shunt), or by a formula using both the SVC and the IVC saturation. The last of these is our preferred method.

The largest source of error is in the assessment of oxygen consumption. Traditionally this has been measured using a hood and gas pump that extracts all exhaled air and passes it through a mixing system before measuring the oxygen content. The difference between inhaled oxygen content and exhaled oxygen content, coupled with the flow maintained by the pump, allows estimation of oxygen consumption.⁴ The method involves several assumptions. Firstly, it assumes that the pump caters for all exhaled air and that none is “lost”. Secondly it assumes effective mixing before the oxygen measurement. Thirdly, it assumes (at least with some equipment) that the volume of exhaled air is the same as that of inhaled air, which is only true if carbon dioxide production is identical with oxygen uptake (in some labs a respiratory quotient—respiratory exchange ratio (RER)—of 0.8 is assumed).⁵ It also requires very accurate measurement of flow through the pump. Additionally it requires very precise measurement of the oxygen level in exhaled air, which has in the past required the use of large and cumbersome equipment (a mass spectrometer). Patients being catheterised under anaesthesia may require a closed circuit method, which is also laborious and time consuming to perform. In either case it is essential that the medical and technical personnel involved be very familiar with the equipment and the methodology, and that they perform such measurements on a regular basis.⁶

Until recently no commercially available system had been produced that allowed simple and reliable measurements to be made routinely by technologists or physicians without substantial and regular experience of the apparatus and its potential problems. For this reason regular measurement of oxygen consumption has been largely restricted to centres in which there are physicians and/or technical personnel with a major interest in oxygen consumption measurements, and usually an ongoing research programme or project that involves them.

There are now several commercially available methods of measuring oxygen consumption, which employ relatively compact and reasonably

simple equipment that eliminates, to some degree, many of the problems detailed above.^{7,8}

In the majority of institutions, even when such equipment is available, oxygen consumption is not measured routinely; when measurements are required it is often difficult or impossible to obtain satisfactory measurements—for example, because those staff who are familiar with the apparatus are unavailable, and the personnel involved with the procedure are unfamiliar with the equipment and lack confidence/competence in obtaining the necessary data.

The availability of nomograms for oxygen consumption obtained from children of varying age and sex and at different heart rates has allowed the use of “assumed oxygen consumption” based on such data.⁹ Several regression equations and tables of “assumed oxygen consumption” are available and produce normal values, ranging from around 180 ml/min/m² in young children (aged 2–3 years) down to around 100 ml/min/m² in adult women.⁴ Males have higher oxygen consumption (by 10–20%) than females and tachycardia above 150 beats/min is associated with a 10% increase compared with heart rates of 120 or lower. Young children (aged 2–5 years) have oxygen consumption values between around 150 and 200 ml/min/m². Older children (for example, adolescents) tend to have values between 120 and 180 ml/min/m². The sex difference is less pronounced in the younger age groups and is largest in adults. Infants younger than 3 months may have somewhat lower oxygen consumption values (130 ml/min/m²) than older infants (170 ml/min/m²), while children of 1–2 years have values close to 200 ml/min/m².

Unfortunately those studies in which direct comparisons have been made between assumed and measured oxygen consumption have shown poor correlation and wide discrepancies in individual cases.⁴

Despite the deficiencies implicit in the use of assumed oxygen consumption this method is employed very widely and is probably adequate for most purposes. A useful practice is to do duplicate calculations—assuming alternative oxygen consumption values—at the upper and lower levels of the likely range for a child of the particular age and sex. Thus, for a 5 year old boy one might use assumed oxygen consumption values of 140 ml/min/m² and 200 ml/min/m². The calculated flow using these two figures should give values at the extremes of the likely range, and the actual figure is most likely somewhere in between.

Common sources of errors

- Assumed O₂ consumption is notoriously unreliable
- Unfamiliarity with O₂ consumption measurement technique—leads to unpredictable/unreliable results
- Failure to calculate dissolved O₂ when using enriched gas (for example, 100% O₂)

Pulmonary resistance

Calculation of pulmonary resistance and assessment of pulmonary vascular reactivity remains a fundamentally important issue in many patients. The calculation becomes extremely simple once the pulmonary blood flow index has been estimated as indicated above. Resistance is the pressure drop across the pulmonary (or systemic) circulation per unit of flow in a specified time period. As flow is usually measured in l/min/m² this is the unit of measurement usually employed. The pressure drop is the difference between mean arterial and mean venous pressure. In the case of pulmonary resistance the equation is therefore:

$$R_p = \frac{P_{Am} - L_{Am}}{Q_p} \quad (8)$$

where R_p is pulmonary resistance, P_{Am} is mean pulmonary artery pressure, L_{Am} is mean left atrium (or pulmonary vein) pressure, and Q_p is the pulmonary blood flow index.

If the left atrium and/or pulmonary veins have not been entered a pulmonary capillary wedge pressure may be used. Alternatively an assumed pressure of around 8 mm may be employed.

The resistance units in this calculation are in “mm Hg/l/min”—referred to usually as Wood units. An alternative is to measure resistance in metric units in “dyne.sec.cm⁻⁵”. The conversion is achieved by multiplying resistance in Wood units by 80 to achieve the metric units in dyne.sec.cm⁻⁵.

It should be appreciated that if the figure for pulmonary blood flow is indexed to body surface area the resistance is also indexed. Values of resistance (in Wood units) are frequently expressed with the simple abbreviation of “u” (units). When indexed to body surface area the appropriate abbreviation is “u.m²”. Unfortunately in much of the published literature this has been misrepresented as “u/m²”, which is misleading as it implies that the calculated resistance in units has been divided by the body surface area to index it. If absolute values for flow (rather than indexed values) are used to calculate resistance it will become clear that smaller patients have much higher levels of resistance (because of the lower flows with smaller surface area). Obviously the use of indexed flows eliminates this disparity. If the value of resistance obtained by using absolute flows is divided by body surface area, however (as the abbreviation “u/m²” would imply) the disparity is exaggerated. For example, a child with a body surface area of 0.5 m² has a pulmonary blood flow (Q_p) of 2 l/min and a pulmonary artery mean pressure of 20 mm Hg with a left atrium mean of 8 mm Hg. His absolute resistance is therefore $(20 - 8)/2$ or 6 u. If this is “corrected” for surface area by dividing by 0.5 the result will be 12 u/m². However, if the flow is corrected for surface area it becomes 4 l/min/m². The calculation will then produce

the correct figure for indexed resistance: $(20 - 8)/4 = 3$ u.m². The same result will be achieved by taking the absolute figure for resistance (6 u) and multiplying (rather than dividing) it by body surface area ($6 \times 0.5 = 3$).

Common sources of errors

- Correction for body surface area by *dividing* by BSA in m²
- Unstable haemodynamics due to hypoventilation or acidosis, leading to pulmonary vasoconstriction and high pulmonary vascular resistance (check pH and pCO₂)

Pulmonary vascular reactivity

The assessment of pulmonary vascular reactivity is sometimes important if the initial value (with the patient breathing air) is greatly elevated, raising concerns about the presence of significant pulmonary vascular disease. The significance of raised levels of pulmonary vascular resistance depends on the patient's age. In the early months of life high resistance is often related to pulmonary vasoconstriction/increased vasomotor tone (with increased medial smooth muscle in the walls of the pulmonary arterioles). It does not necessarily imply significant obliterative pulmonary vascular disease until later in infancy/childhood. Values of pulmonary resistance above 6 u.m² would be a cause for concern on this score in a child above 1 year of age (estimates greater than 10 u.m² would be especially sinister). In interpreting such measurements it should be recognised that hypoventilation or acidosis can produce quite intense pulmonary vasoconstriction and may be associated with artificially (misleadingly) elevated resistance. To exclude this as a potential source of error, blood gas measurements need to be carried out at the time of the pressure and saturation measurements, to ensure that pH and pCO₂ are within the normal range.

In cases in which a high pulmonary vascular resistance is demonstrated, it is customary to allow the patient to breathe an oxygen enriched mixture (80% or 100% oxygen) for 10 minutes and then to repeat the pressure and saturation measurements in order to get a calculation of flow and resistance under these conditions. This is a very important and useful manoeuvre but does introduce a very important potential source of error. With the increased concentration of inspired oxygen the partial pressure of oxygen in pulmonary alveoli and in pulmonary capillary and pulmonary venous blood will rise to supernormal levels. This will result in quite significant amounts of oxygen being transported dissolved in plasma, in addition to that which is bound to haemoglobin. If the calculations do not take this into account the oxygen content difference between pulmonary vein and pulmonary artery blood will be underesti-

mated. The estimated pulmonary blood flow will then be overestimated and pulmonary resistance will appear to be lower than is really the case. To calculate dissolved oxygen is extremely simple. The pO_2 of pulmonary venous blood is measured (in mm Hg) and this value multiplied by 0.03 to provide a volume of dissolved oxygen (in ml/l). Thus if the pulmonary vein pO_2 is 500 mm Hg there will be 15 ml/l of dissolved oxygen ($500 \times 0.03 = 15$). The amount of dissolved oxygen in pulmonary arterial blood should also be estimated by the same method (though in practice it is seldom more than 3 ml/l). Thus there may be as much as 12 ml/l oxygen content difference in the form of dissolved oxygen. In patients with high pulmonary flow this may account for more than 50% of the total oxygen content difference between pulmonary venous and pulmonary arterial blood. Consequently, failure to include dissolved oxygen in the calculations can lead to major errors in the data for pulmonary flow and resistance.

One of the misconceptions concerning the measurements made in 100% oxygen, which is quite widely held, is that patients with significantly labile pulmonary vascular beds (in whom resistance will drop with increased inspired oxygen) will always show a fall in pulmonary artery pressure under these conditions. Thus the assumption may be made that the absence of any fall in pressure demonstrates a lack of lability and implies the presence of advanced pulmonary vascular disease. However, some patients may achieve a substantial increase in pulmonary blood flow, associated with a large drop in resistance, with little change in pulmonary artery pressure. Thus careful assessment of pulmonary blood flow index and resistance (including the calculation of dissolved oxygen) is an essential part of the study in patients being evaluated with 100% oxygen because of pulmonary hypertension.

As an alternative to the use of 100% oxygen (or in addition), other vasodilators may be employed to test vasoreactivity. The most useful of these is probably inhaled nitric oxide—usually given in concentrations between 20 and 80 parts per million (ppm). This is a useful adjunct to (but not a substitute for) use of 100% oxygen. However it should be born in mind that while there is broad agreement about the level of pulmonary vascular resistance which is likely to be “reversible” as demonstrated with the vasodilation and fall in resistance achieved with 100% oxygen (usually a fall to 6 u.m^2 or less), it is not yet clear whether patients who show a similar fall with nitric oxide (but not with 100% oxygen) will prove to have similarly “reversible” pulmonary vascular damage. Thus, in a patient whose resting resistance is calculated at 10 u.m^2 , in whom 100% oxygen produces a fall to around 8 u.m^2 , and nitric oxide produces a further fall to 6 u.m^2 , it is by no means certain that the outcome after surgery would be the same as might be anticipated if 100% oxygen had produced a fall to 6 u.m^2 .

Common sources of errors

- Hypoventilation/acidosis producing pulmonary vasoconstriction
- Failure to calculate dissolved O_2 when using enriched gas (for example, 100% O_2)
- Assumption that no fall in pulmonary artery pressure means no fall in resistance

It may appear from the above brief analysis that the assessment of pulmonary hypertension and pulmonary vascular reactivity is time consuming, complex, and fraught with assumptions that are of doubtful validity. In practice useful assessments can be made rapidly and quite simply. The calculations need to be worked through carefully (usually after the case has been completed), but a rapid estimation can often be made at the time of the procedure, which may produce a useful insight into the severity of the problem.

In a patient in stable haemodynamic state with systemic venous saturations in the normal range (60–75%) it is reasonable to assume that the systemic cardiac index will be in the general range of 3–4 l/min/m². As $Q_p:Q_s$ can be estimated very simply while the patient is under basal conditions (for example, breathing air) a simple “guesstimate” of pulmonary blood flow index is easily made. If $Q_p:Q_s$ is 2.5 then Q_p must be in the general range of 7–10 l/min/m² ($3 \times 2.5 = 7.5$, $4 \times 2.5 = 10$). If the pulmonary artery mean pressure is 35 mm Hg and left atrium is 10 mm Hg then the pressure drop (transpulmonary gradient) is 25 mm Hg. Resistance in this case is likely to fall between 2.5 and 4 u.m^2 . This calculation is simple, quick, and informative—although it does not eliminate the need to do the complete calculations.

A fairly simple “cross check” can be made by doing a quick mental calculation of pulmonary veno-arterial oxygen content difference and using an assumed oxygen consumption of 150–200 ml/min/m².

This depends on having a value for Hb and for the saturation difference between the pulmonary artery and pulmonary vein. Thus if the pulmonary artery saturation is 90% (in the presence of a left to right shunt) and the left atrium is 99%, with a Hb of 120 g/l the following calculation may be made:

$Hb \times 1.36 = 120 \times 1.36 = \text{approximately } 160$;
 $Sat \text{ PV} - Sat \text{ PA} = 99 - 90 = 9$;
 $160 \times 9\% = \text{approximately } 15 \text{ ml/l}$ (oxygen content difference).

Pulmonary blood flow index is then likely to be in the general range of 10–13 l/min/m² ($150/15 = 10$; $200/15 = 13$).

If the transpulmonary gradient is 25 mm, as in the earlier example, then the pulmonary vascular resistance is $2\text{--}2.5 \text{ u.m}^2$ ($25/10 = 2.5$; $25/13 \sim 2$).

A similar piece of mental arithmetic will allow estimation of systemic cardiac index as well as systemic vascular resistance.

Similar calculations may be performed with the patient in 100% oxygen, but here the

dissolved oxygen needs to be taken into account. A fairly simple way to do this is to assume the “worst case scenario”—which would have a difference in dissolved oxygen between pulmonary vein and pulmonary artery of around 12 ml/l (it would very seldom be any greater than this).

Using the same values for saturation and Hb, as well as the same assumed oxygen consumption as in the earlier example, the equation is now:

$Hb \times 1.36 = 120 \times 1.36 =$ approximately 160;
 $Sat\ PV - Sat\ PA = 99 - 90 = 9$;

$160 \times 9\% =$ approximately 15 ml/l; dissolved oxygen difference = 12 ml/l (worst case scenario).

Total oxygen content difference =
 $15 + 12 = 27$ ml/l

This now produces a very different result in the blood flow calculation.

Pulmonary blood flow index is now likely to be in the general range of 5.5–7.5 l/min/m² ($150/27 = 5.5$; $200/27 = 7.4$).

If the transpulmonary gradient is 25 mm then pulmonary vascular resistance is now 3–4.5 u.m² ($25/5.5 = 4.5$; $25/7.5 = 3.3$).

In reality if the dissolved oxygen content difference is lower than the “worst case scenario” the flow will be higher than this (nearer to the value arrived at when the dissolved oxygen is not included in the calculation).

Perhaps surprisingly, considering all the assumptions and approximations contained in these “rough calculations”, the results correlate generally very well with the more laborious calculations performed after the case is complete and with the calculations produced by the computer software which is often employed for automating these estimations. Moreover where major discrepancies arise it is often desirable to go back and carefully check the data and the way in which the calculations have been done. Sometimes the “rough result” is the more correct one and errors have been made in the more detailed calculation.

In any case the ability to perform these quick “mental” calculations in the catheter laboratory is an entertaining exercise and demonstrates an understanding of the data.

Valve (orifice) area

Calculation of valve area is based on the hydraulic formula usually referred to as the “Gorlin formula” and published almost 50 years ago.¹⁰

The calculation depends on obtaining estimates for valve flow in ml/sec during the time that the valve is open.

This is conventionally estimated by measuring the duration (in seconds) of systolic ejection or of diastolic filling, from the pressure wave forms, and multiplying by heart rate—to assess the period of flow through the valve per minute (expressed in secs/min), which is in turn divided into the cardiac output (in ml/min) to obtain flow per second across the

valve for which an area calculation is required (in ml/sec).

The mean ventricular pressure during flow through the valve (systole for arterial valves, diastole for atrioventricular (AV) valves) and the mean pressure proximal or distal to the valve are required, in order to estimate the mean transvalvar gradient. These mean pressure measurements need to relate specifically to the period when the valve is open (during systolic ejection for an arterial valve or during diastolic filling for an AV valve). This conventionally requires planimetry and is potentially time consuming and cumbersome.

The formula includes constants, one of which is an “orifice constant coefficient” (0.8 for mitral valve; 1.0 for aortic, pulmonary, and tricuspid valves).

The final formula is:

$$\text{Valve area (cm}^2\text{)} = \frac{\text{Flow (ml/sec)}}{\text{Oc} \times 44.3 \sqrt{\text{mn Gradient}}} \quad (9)$$

where Oc is orifice constant coefficient (0.8 for mitral valve, 1.0 for other valves); 44.3 is a constant derived from $\sqrt{2g}$ (where g is gravity acceleration = 980 cm/s/s); and mn Gradient is the mean transvalvar gradient (mm Hg), being the difference in mean pressure on each side of the valve during systolic ejection (arterial valve) or diastolic filling (AV valve).

Simplified versions of this formula have been advocated and include the Bache formula for aortic valve area (using peak to peak gradient)¹¹ and the Hakki formula¹²:

$$\text{Valve area} = \frac{\text{CO}}{\sqrt{\text{mn Gradient}}} \quad (10)$$

In practice these formulae all depend on a number of assumptions and approximations. They permit estimations of valve orifice that are, in our opinion, of limited clinical use. We do not rely on such data for clinical decision making, preferring to use other parameters.

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