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Original Article

Assessment of operability for common arterial trunk without cardiac catheterisation

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Abstract Pulmonary vascular disease is a risk factor for the surgical management of common arterial trunk. Surgical repair, therefore, is usually performed in early infancy, before irreversible changes can occur in the pulmonary vasculature. Because of this, there has been an increasing tendency to dispense with cardiac catheterisation as a means of assessing pulmonary vascular disease. Cardiac catheterisation, nonetheless, is still performed when there is a risk of pulmonary vascular disease, such as in older children. There are no clear guidelines, however, as to who should be catheterised. We have developed a simple screening test to help make this decision.

Keywords: Truncus arteriosus; pulmonary vascular disease; pulmonary vascular resistance

Introduction

Surgical repair of common arterial trunk is associated with significant risk. One risk factor is the presence of pulmonary vascular disease.[1–3] The recent trend, therefore, has been to undertake surgical repair early, before pulmonary vascular disease becomes established.[4–6] Although this has improved surgical outcomes, operative mortality still remains significant. One explanation for this is that pulmonary vascular disease may occur earlier than previously thought.[7]

Pulmonary vascular disease is diagnosed when there is an irreversibly elevated pulmonary vascular resistance. This can be measured during cardiac catheterisation. Catheterisation, however, has increasingly been dispensed with in the management of common arterial trunk, partly because of the policy of early surgical correction when pulmonary vascular resistance is unlikely to be irreversibly elevated. Catheterisation is still undertaken in older patients, or when a high pulmonary vascular resistance is suspected. There are no clear guidelines as to who should or who should not be catheterised. A simple test that would differentiate between these two groups of patients would be of great value. We believe that such a test can be provided in the setting of common arterial trunk, since in most patients, who have unrestricted flow of blood to the lungs, the tensions and saturations of oxygen are similar in the systemic and pulmonary circulations. This allows calculation of pulmonary vascular resistance without catheterisation. Such calculations are made intuitively by many cardiologists, but as far as we are aware, a formal method has not previously been reported. Our aim is to present such a method.

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Methods

Pulmonary vascular resistance is calculated by subtracting the left atrial pressure from the mean pulmonary arterial pressure, and dividing by the flow of blood to the lungs (Table 1 – Equation 1).

In most patients having a common arterial trunk, pulmonary arterial pressure is the same as the mean systemic pressure. Assuming that the mean left atrial pressure would be between 6 and 10 millimetres of mercury, the calculation shown above can be made by taking a left atrial pressure of 6 millimetres of mercury, this being the worst case scenario, with the highest drop in pressure across the lungs, and using the Fick principle to calculate the flow of blood to the lungs. Thus, the flow is calculated by dividing the consumption of oxygen by the difference in content of oxygen between the pulmonary venous and arterial bloods (Table 1 – Equation 2).

The content of oxygen in these blood streams is the sum of that bound to haemoglobin plus the dissolved

Table 1. Equations used in the calculations.

Equation 1
Pulmonary vascular resistance is calculated as:

$$\frac{\text{Mean pulmonary artery pressure} - \text{Left atrial pressure}}{\text{Pulmonary blood flow}}$$

Equation 2

$$\text{Flow} = \frac{\text{Consumption of oxygen}}{\text{Plumonary venous oxygen content} - \text{Pulmonary arterial oxygen content}}$$

Equation 3

$$\text{Oxygen bound to Haemoglobin} = \text{Haemoglobin} \times 10 \times 1.36 \times \text{oxygen saturation}$$

Equation 4
Flow of blood to the lungs is equal to:

$$\frac{\text{Consumption of oxygen}}{\text{Haemoglobin} \times 13.6 \times (\text{Pulmonary venous saturation} - \text{Pulmonary arterial saturation})}$$

oxygen. In room air, the dissolved oxygen is small, and can be discounted. One gram of haemoglobin, when fully saturated, combines with 1.36 millilitres of oxygen at 37 degrees. Haemoglobin is usually measured in grams per decilitre, rather than grams per litre. The amounts of oxygen bound to haemoglobin, therefore, is 10 times the concentration of haemoglobin multiplied by 1.36, and multiplied by the saturation of oxygen (Table 1 – Equation 3). Combining this information with the calculation made using the Fick principle, we can see that the flow of blood to the lungs is equal to the consumption of oxygen divided by the concentration of haemoglobin multiplied by 13.6, and multiplied by the difference in pulmonary venous and arterial saturations of oxygen (Table 1 – Equation 4).

In patients with a common arterial trunk, as already discussed, the pulmonary arterial saturation is similar to the systemic saturation. Pulmonary venous saturation can be assumed to be close to 1, for example 0.98 in room air. Consumption of oxygen is readily obtained from standardised charts.[8] Typical values would be between 180 and 200 millilitres per minute per square metre body surface area in a premature newborn, with values of 150 in a normal newborn, and between 120 and 130 in children. Using these values, it is a simple matter to calculate the flow of blood to the lungs, which then, having taken left atrial pressure as 6 millimetres of mercury, permits calculation of the pulmonary vascular resistance.

Using this method, it is also possible to estimate the reversibility of a high pulmonary vascular resistance without the need for catheterisation. This involves repeating the calculations, but presuming the presence of 100 per cent oxygen. When making these assumptions, however, the amount of dissolved oxygen is more important and cannot be discounted. Dissolved oxygen can be calculated from the partial pressure of oxygen, in that 0.03 millilitres of oxygen are dissolved in each litre of blood for each millimetre of mercury partial pressure of oxygen. We do need to know, however, the partial pressures of oxygen in the pulmonary arterial and venous bloods. Pulmonary arterial partial pressure of oxygen is the same as that in the systemic circulation. Pulmonary venous partial pressure of oxygen will need to be estimated. This will not be higher than 650 millimetres of mercury, a value giving the highest difference in arterio-venous oxygen, and hence the lowest estimated flow of blood to the lungs, and if anything overestimating the pulmonary vascular resistance. It is difficult, nonetheless, to predict left atrial pressure in 100 per cent oxygen, but taking a mean pressure of 8 millimetres of mercury is unlikely to be an overestimate, and will therefore also tend to overestimate the pulmonary vascular resistance.

Example

A 9-month-old infant with a common arterial trunk is known to have concentrations of haemoglobin of 16.5 grams per decilitre. His consumption of oxygen, as taken from standardized charts, is known to be 120 millilitres per minute per square metre body surface area. When he is breathing room air, his systemic saturations of oxygen, equivalent to the pulmonary arterial saturation, is 82 per cent. The mean aortic pressure, also equal to the mean pulmonary arterial pressure, is 45 millimetres of mercury. Pulmonary venous saturation is assumed to be 98 per cent, and mean left atrial pressure to be 6 millimetres of mercury. Using our calculations, we obtain a flow of blood to the lungs of 3.3 litres per minute per square metre body surface area. This equates to a pulmonary vascular resistance of 11.8 units. Administration of 100 per cent oxygen resulted in the arterial saturation of

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oxygen increasing to 98 per cent, with a partial pressure of oxygen of 99 millimetres of mercury. Assuming a partial pressure of oxygen of 650 millimetres of mercury in the pulmonary veins when the saturation of oxygen is 100 per cent, we can calculate the flow of blood to the lungs at 5.7 litres per minute per square metre body surface area. Mean aortic, and mean pulmonary arterial, pressure was 40 millimetres of mercury, and mean left atrial pressure was assumed to be 8 millimetres of mercury, giving a drop in pressure of 32 millimetres of mercury across the lungs. Pulmonary vascular resistance calculates out at 5.6 units.

The degree of reversibility of the pulmonary vascular resistance in this example suggests this patient may be suitable for surgical repair. Had there been no fall in the pulmonary vascular resistance, surgery would have been contraindicated. In borderline cases, it would be wise to undertake cardiac catheterisation so as to measure the reversibility of the pulmonary vascular resistance before making a final decision as to whether or not the patient should undergo surgical repair.

Discussion

We present our method as a simple test for screening. It involves several issues and assumptions which require clarification. The upper limit of the partial pressure of oxygen in pulmonary venous blood is taken as 650 millimetres of mercury when breathing 100 per cent oxygen. This is because we know that atmospheric pressure is 760 millimetres of mercury. Since air contains 21 per cent oxygen, the partial pressure of oxygen in dry air would be $0.21 \times 760 = 160$ millimetres of mercury, while that of 100 per cent oxygen would be 760 millimetres of mercury. Inspired air, nonetheless, is saturated with water vapour by the time it reaches the lungs. The partial pressure of water vapour at normal body temperature is 47 millimetres of mercury. The partial pressure of oxygen in inspired air, therefore, would be $0.21 \times (760 - 47) = 150$ millimetres of mercury. Similarly, the partial pressure of inspired 100 per cent oxygen would be 713 millimetres of mercury. These figures represent the theoretical maximum partial pressures. In practice, they are never reached due to variable degrees of mismatch between ventilation and perfusion. Practical experience shows that, with 100 per cent inspired oxygen, partial pressures of 550 to 600 millimetres of mercury are rarely exceeded. We chose the figure of 650 in order to exaggerate the problem, as this would give us the highest arterio-venous difference in oxygen, and hence the lowest estimated flow of blood to the lungs, thus overestimating pulmonary vascular resistance. It would, therefore, represent the scene for the worst case.

Consumption of oxygen can be measured by various techniques, such as timed collection of expired gas in a Douglas bag, or the open-circuit method where a sensitive oximeter continuously monitors the expired fraction of oxygen.[9] These methods are often technically not possible because of lack of cooperation. For this reason, consumption of oxygen is often calculated on anthropomorphic variables available from standardised charts. These charts provide a range of possible consumptions for each age group, rather than a single figure. We recommend the use of the lowest figure in the range, because this would give us the lowest calculated flow of blood to the lungs, and would therefore overestimate the calculated pulmonary vascular resistance.

The method we have suggested is subject to several systematic errors. This is because assumptions are made regarding left atrial pressures, as well partial pressures of oxygen when breathing 100 per cent oxygen, and the use of anthropomorphic variables to calculate consumption of oxygen. To compensate for these weaknesses, values were chosen for these three variables that would overestimate the pulmonary vascular resistance. Thus, the left atrial pressure is assumed to be 6 millimetres of mercury, since this is the lower end of the range that is usually seen, and would correspond to the highest drop in pressure across the lungs. The partial pressure of oxygen when breathing 100 per cent oxygen is assumed to be higher than is usually seen, at 650 millimetres of mercury, thus exaggerating the arterio-venous difference in oxygen across the lungs, and hence the lowest estimated flow of blood through the lungs. The value chosen for the estimated consumption of oxygen from standardised charts is also such that would correspond to the lowest flow of blood through the lungs, and therefore the highest pulmonary vascular resistance, being taken from the lower end of the range. Taking all these considerations into account, which represent the scene of the worst case, we contend that our method will provide a useful test for screening.

Our present strategy is that if the calculated pulmonary vascular resistance in 100 per cent oxygen falls to 5 Wood units per meter squared or below, the patient can be considered not to have such severe elevation of pulmonary vascular resistance as to preclude surgery. When our test produces estimates of pulmonary vascular resistance above 5 and below 8 units, we proceed to formal cardiac catheterisation, with evaluation of the pulmonary vascular resistance breathing 100 per cent oxygen and/or nitric oxide. We take values above 8 units, despite 100 per cent oxygen, to indicate inoperable pulmonary vascular disease. We consider such values to represent a contraindication for the need for formal haemodynamic assessment.

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