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REVIEW ARTICLE

Pulmonary artery catheterization in anaesthesia and intensive care

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Since its introduction nearly 30 yr ago, the pulmonary artery catheter (PAC) has become part of everyday management in cardiology, anaesthesia and intensive care but remains controversial.[12 37 78 136 164] The apparent failure to demonstrate improvements in patient outcome[28 66 68 192] and the risks associated with its use[15 153] have long been criticized.[139] Failure to explore its full use and redundancy of the catheter after initial placement are additional concerns. More worrying is the incorrect interpretation of data[64 82 97] and consequent misdirection of therapy. Others are disturbed by a reliance on measurements rather than on clinical signs.[140] However, many argue that the PAC enhances bedside understanding of cardiorespiratory interactions, complements the interpretation of clinical signs and benefits management of haemodynamic disorders.[47 53 62 65 77 136 157 170]

The first part of this article reviews the history, technical aspects and practical uses of pulmonary artery catheterization. The second part evaluates the clinical impact of the PAC on anaesthesia and intensive care medicine.

Historical developments

A century and a half ago Bernard, and later Chauveau and Marey, detailed left and right heart catheterization in live animals while studying pulmonary "combustion" (heat production)[6] and the timing of the apical beat.[23]

In 1929, Forssmann catheterized his own right atrium.[55] The following year Jiménez Díaz and Sánchez Cuenca reported the first series of patients to undergo right atrial catheterization.[88] Cardiac output by the Fick method was validated over the following decade, initially sampling from the right atrium but quickly progressing to the pulmonary artery.[34 94] Forssmann, Cournand and Richards shared the 1956 Nobel prize in medicine and physiology.

Pulmonary artery occlusion (without a balloon) was first reported in 1947 for oxygen measurement[42] and in 1948 and 1949 for pressure measurement.[75 99] In 1953, pulmonary artery occlusion with a balloon-tipped, flow-directed catheter was pioneered in dogs.[101]

In 1964, Bradley described the use of he PAC at the bedside[17] and 4 yr later Branthwaite validated thermodilution cardiac output measurement in humans.[18 50] Seventeen years after its first description in dogs, Swan and colleagues described pulmonary artery occlusion with a balloon-tipped, flow-directed catheter in humans.[169]

Practical considerations

INSERTION

Usually the PAC is inserted into a central vein through a large (7.5–9 F) percutaneous sheath. The internal jugular vein is the preferred site of cannulation, with a low risk of pneumothorax. The incidence of carotid artery puncture is 1.5–2% and its inadvertent cannulation is less than 1 in 1000. The subclavian route has a higher risk of pneumothorax and arterial puncture whereas the femoral vein may be a difficult site from which to float the catheter.[90 118 153]

If difficulty in distinguishing venous from arterial puncture is anticipated, the vessel can be entered with a small cannula before percutaneous dilatation; a typical venous waveform after connection to a pressure transducer confirms venous cannulation.

The PAC may be associated with serious complications.[15] Arrhythmias, although frequent, usually resolve spontaneously.[83] However, between 1 and 3% require treatment.[15 153] Thrombosis and haemorrhagic lesions along the path of the PAC have been found in as many as 53% and 78% of patients, respectively, dying with a PAC *in situ*.[27]

Infections related to pulmonary artery catheterization are probably underestimated and have been thoroughly reviewed elsewhere.[112] Infections are more common with internal jugular placement, if catheters remain in place for longer than 4 days and if they are re-inserted at an old site.[25] Colonization of the catheter, usually by organisms from the skin of the insertion site, has been reported in approximately 22% of catheters on removal, the commonest organisms being *Staphylococcus epidermidis* and gram-negative bacilli. In approximately 1% of catheterizations, catheter colonizations may lead to bloodstream infection, commonly with *Staphylococcus epidermidis*, *Staphylococcus aureus* and yeasts. Septic thrombosis (infection of a thrombus which develops near the catheter) can cause a serious generalized infection despite the absence of local inflammatory signs.[91 112] Infective endocarditis exclusively caused by PAC occurs infrequently, although autopsy studies report an incidence of 2–7%.[112 142]

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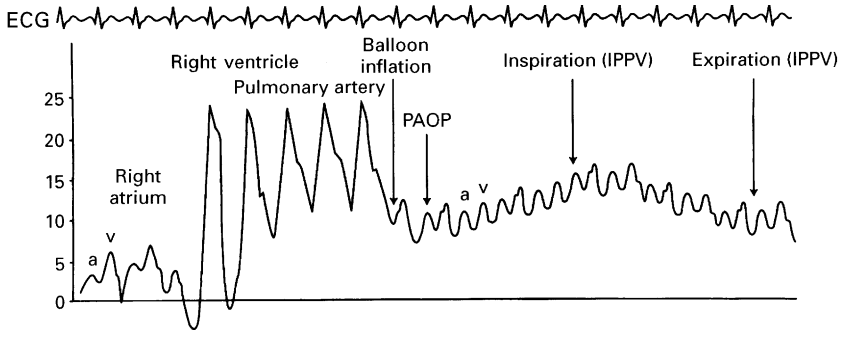

Figure 1 Representative traces during pulmonary artery catheterization and simultaneous real-time ECG recording. Note the occlusion "a" wave, the true PAOP, is in synchrony with the ECG "p" wave. Note also the pressure oscillations caused by a positive pressure respiratory cycle. IPPV = Intermittent positive pressure ventilation. Modified with permission from Wiedemann, Matthay and Matthay.[188]

Distal catheter migration occurs in up to 1% of insertions and may cause pulmonary infarction,[15] often presenting clinically as pulmonary embolism. Distal migration should be suspected if there is a continuous wedge trace with the balloon deflated or if minimal balloon inflation produces a wedge trace. A sudden increase in transduced pressure on balloon inflation suggests catheter abuttal against the pulmonary artery wall.

Pulmonary artery rupture is more common in patients more than 60 yr of age, those receiving anticoagulant therapy or those with pulmonary hypertension.[71] It is caused mostly by balloon overinflation.[71] Less frequently it can be caused by erosion of the balloon tip into the arterial wall.

Massive haemorrhage from pulmonary artery rupture has a reported incidence of 0.031–0.25% with a mortality of 25–83%.[15 93 153] Management by reversal of anticoagulation and high levels of PEEP has been successful occasionally,[151] but other therapies have often been used. These include selective endobronchial intubation with differential ventilation and transcatheter embolization.[51 146] Two reports suggest an apparent benefit from early surgical repair, which may require formal pulmonary resection.[93 177]

CONFIRMATION OF PLACEMENT

Correct placement of a PAC is accompanied by characteristic pressure wave changes leading to the atrial type waveform of pulmonary artery occlusion[188] (fig. 1). When properly wedged, the different pressures follow the relationship:

$$PADP > PAOP$$

where PADP and PAOP = pulmonary artery diastolic and occlusion pressure, respectively.

An important exception is severe mitral valve disease in which PAOP can be greater than PADP but remains less than MPAP.

Correct placement may also be confirmed by aspiration of "arterialized" blood from the catheter in the wedged position, as its oxygen tension Pw_{O_2} should be similar to Pa_{O_2}. However, if the catheter has floated into an area of low ventilation–perfusion match, this may not occur.[20]

Pressures in the pulmonary circulation

The physical characteristics required of systems designed to measure biological pressures have been reviewed extensively by Gardner and Hollingsworth.[59]

Inflation of the balloon isolates a pulmonary vascular segment, and flow in that segment ceases (fig. 2). As there are few precapillary anastomoses[38 75] the occluded segment is also isolated from flow in nearby non-occluded units. Therefore, downstream pressure in this occluded segment, pulmonary artery occlusion pressure (PAOP), equilibrates with pulmonary venous pressure (PVP). The balloon inflation technique does not measure the true pulmonary capillary pressure. In fact "pulmonary capillary wedge pressure" is a misleading hybrid term with no physiological correlate.[182 187] The pulmonary capillaries are not wedged and the pressure recorded when the pulmonary artery is occluded is not that of the capillaries.[184]

INTERPRETATION OF PAOP

At end-diastole, flow between the pulmonary valve and the mitral valve (which is still open) is minimal. End-diastole can be identified because the "a" wave on the wedge trace coincides with the "p" on the ECG (see fig. 1)[117]; sometimes one or both may be difficult to detect or non-existent, as in atrial fibrillation. Effectively there is a continuous column of

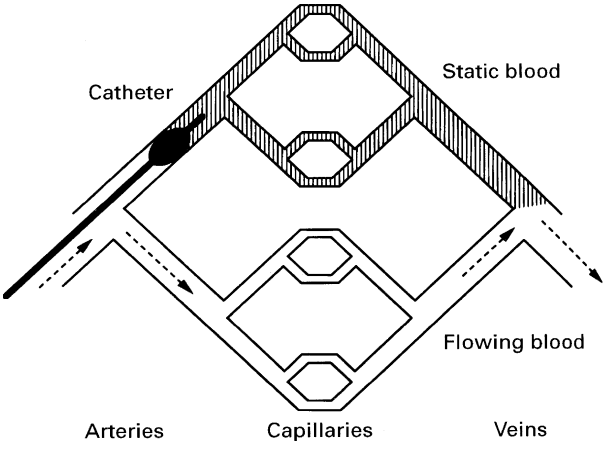

Figure 2 Diagrammatic representation of occlusion of a pulmonary arteriole by a balloon-tipped PAC. Note the absence of precapillary anastomoses, which explains why the pressure of the column of static blood (shaded area) equilibrates with downstream venous pressure. Modified from Weed[182] with permission.

Table 1 Discrepancies between PAOP and true preload (see text for detailed explanation)

Measurement discrepancy (boldface)	Reason	Comment
PAEDP > PAOP > PVP > LAP ≈ LVEDP α LVEDV	Tachycardia/increased PVR	End-diastolic pressure gradient PA → LA
PAEDP < "PAOP" > PVP ≈ LAP ≈ LVEDP α LVEDV	West zones 1: $P_A > P_a > P_V$	catheter senses alveolar pressure, so true PAOP not measured
	West zones 2: $P_a > P_A > P_V$	
PAEDP ≈ PAOP ≈ PVP ≈ **LAP ≠ LVEDP** α LVEDV	Mitral stenosis,	**LAP > LVEDP; large 'a' wave**
	mitral regurgitation	**LAP > LVEDP; large 'v' wave**
PAEDP ≈ PAOP ≈ PVP ≈ **LAP ≠ LVEDP** α LVEDV	Aortic regurgitation	**LAP < LVEDV**
PAEDP ≈ PAOP ≈ PVP ≈ **LAP ≠ LVEDP** α LVEDV	LV dysfunction	**LAP < LVEDP**
PAEDP ≈ PAOP ≈ PVP ≈ LAP ≈ **LVEDP ≠ LVEDV**	Low LV compliance	**LVEDP indicates a greater LVEDP**
	High LV compliance	**LVEDP indicates a smaller LVEDV**

blood from the pulmonary artery to the left ventricle and all the pressures between these points (PAEDP, PVP, LAP and cavitary LVEDP) tend to equilibrate.[117 122] Consequently, end-diastolic PAOP approximates LVEDP. In addition, it has been suggested that PAOP should be measured at end-expiration[122] when the effect of intrathoracic pressure is minimized. Discontinuation of IPPV to measure PAOP can introduce error caused by the variable relationship between PAOP and ventilation.[39]

The force of ventricular contraction is related to fibre length at end-diastole, which is determined by left ventricular end-diastolic volume (LVEDV).[124 167] For a given ventricular compliance, LVEDP is directly proportional to LVEDV[188] so LVEDP and PAOP are indicative of left ventricular preload. It is crucial to understand that the PAC as an index of left ventricular preload depends on this LVEDP-LVEDV relationship remaining intact.

The assumption that PA occlusion always generates an uninterrupted column of blood is not always true and may lead to measurement errors. Table 1 summarizes the clinical circumstances, described below, in which PAEDP, PAOP and LVEDP cannot be assumed to be equal.

PAEDP different from LVEDP

Occasionally when it is difficult to obtain PA occlusion pressure, pulmonary artery diastolic pressure is used instead. When diastole is short (heart rate >115 beat min^{-1}) or there is impairment to flow, such as in pulmonary hypertension, flow may not cease and pressures between the pulmonary artery and left atrium do not equilibrate. Consequently, PAEDP remains greater than LAP[14 87] with PAOP somewhere between the two.[100]

PAOP different from PVP

When the catheter tip is in a West zone 1 or 2,[186] high alveolar pressure may interrupt the channel of blood between the pulmonary artery and left atrium.[188] In these situations the catheter sensor reflects alveolar and not vascular pressure. The erroneous "PAOP" is higher than PADP or may have a marked inspiratory swing.[122] In patients with high airway pressures or hypovolaemia, or both, West zones 1 and 2 occupy a proportionately greater volume of lung.[154] In these situations the PAC is more likely, relative to normal conditions, to float into zones 1 and 2 or to become lodged in areas which convert to zones 1 and 2, for example after patient positioning.

The effect of PEEP on PAOP depends on compliance of the lung.[22] Compliant lungs transmit a greater proportion of the applied pressure to the microvasculare than non-compliant lungs. As an approximation, PAOP should increase by no more than half of the PEEP increment.[117 122] Greater increases suggest catheter tip placement in West zone 1 or 2.

LAP different from LVEDP

In mitral valve disease the increase in left atrial pressure (LAP), reflected in PAOP, overestimates LVEDP. In mitral stenosis the PAOP trace has a characteristic large "a" wave. In mitral regurgitation the "v" wave is large. Other causes of large v wave include any mechanism that increases pulmonary venous flow such as left to right shunt.[117 122]

Conversely, in left ventricular dysfunction, LAP underestimates LVEDP because left atrial ejection into a stiff ventricle causes a greater increase in the latter's pressure.[14 49 33] In aortic regurgitation LAP is also less than LVEDP.[76]

Altered LVEDP/LVEDV relationship (left ventricular compliance)

Myocardial ischaemia, pericardial disease, aortic stenosis, shock and other causes of increased left ventricular stiffness lead to a greater LVEDP for a given ventricular filling volume.[54 58] The reduction in compliance is reflected in an increased PAOP which overestimates true preload.

Conversely, dilated cardiomyopathies are associated with increased ventricular compliance and PAOP may underestimate ventricular filling.[117] Ventricular compliance also varies in response to afterload changes and inotropes.[1]

PULMONARY CAPILLARY PRESSURE

Pulmonary capillary pressure (P_c) is an important determinant of transvascular filtration.[38 167] Although P_c can be measured by direct micropuncture[8] or by the isogravimetric technique in isolated perfused lungs[57] there is no practical method *in vivo*.

In health, P_c is usually 5–10 mm Hg and can be estimated from PAOP using the following relationship:

$$P_c = \text{PAOP} + 0.4\ (\text{MPAP} - \text{PAOP}).^{57\,79}$$

The value 0.4 was obtained from dog experiments in which the resistance from pulmonary capillary to vein accounted for 44% of the total pulmonary vascular resistance whereas the resistance between

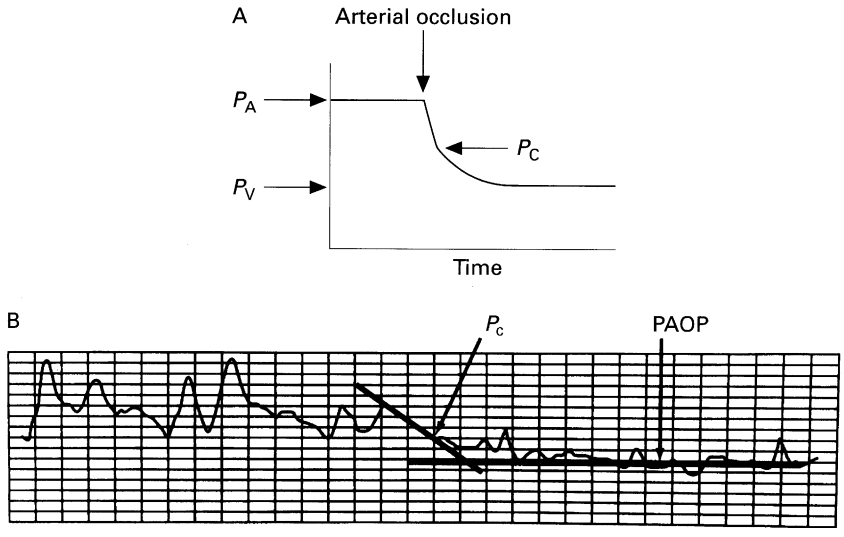

Figure 3 Estimation of pulmonary capillary pressure (P_c) from inspection of the pulmonary artery pressure (PAP) trace immediately after balloon occlusion. A: Theoretical pressure decay curve, with P_c marking the inflection point between the two limbs of the curve; reproduced with permission from Holloway and colleagues.[79] B:Actual bi-exponential pressure drop. Note P_c is significantly greater than PAOP but not "halfway" between mean PAP and PAOP, illustrating the uneven distribution of pulmonary vascular resistance. Reproduced from Levy[102] with permission.

pulmonary artery and capillary accounted for 56% of the total PVR. This is why P_c is normally closer to PAOP than to MPAP.[32]

However, patients with sepsis or acute lung injury may have a normal PAOP but high P_c with the linear relationship between the two being lost.[31 32] In these conditions, pulmonary oedema, which is traditionally attributed to abnormal capillary permeability, may have an important hydrostatic component, shown by an increased P_c. This implies that therapy monitored by PAOP may not always be successful.

In these circumstances it might be useful to estimate P_c by analysis of the curve resulting from pulmonary artery balloon occlusion (fig. 3) which shows biexponential decay. The initial rapid pressure decrease towards P_c is caused by the sudden absence of upstream pressure. The slower pressure decrease from P_c towards PVP is the result of the discharge of blood from the capillary capacitance vessels into the pulmonary venules. The inflection point is taken to be P_c.[79]

This estimated P_c has been validated against a wide range of predicted PCP values based on the isogravimetric technique.[57] The latter has itself been validated against direct occlusion measurements in isolated lungs[173] and is known to correlate well with direct micropuncture measurements.[8 32]

Further, for a given patient, the position of this inflection point (P_c) on the decay curve illustrates the relationship between pulmonary arterial and venous resistances and helps determine the location of the increased PVR. The higher the P_c on the curve, the less arterial resistance contributes to PVR. Recent studies suggest that alveolar hypoxia tends to increase pulmonary vascular resistance predominantly upstream of the capillaries (Ra), whereas sepsis, pulmonary hypertension[143] and acute lung injury usually cause an increase in pulmonary resistance downstream of the capillaries (Rv).[30 160 187]

However, there is concern that these small nuances can be overwhelmed by the magnitude of errors in measuring PAOP.[63] These and other issues in pulmonary capillary pressure are reviewed elsewhere.[30 102]

Thermodilution cardiac output

Measurement of cardiac output using the PAC is based on injection of a tracer into the right atrium and analysis of the change of its concentration over time in the pulmonary artery.[70 168] It is assumed that the mass (M) of the substance injected remains constant and therefore equals the product of the mean tracer concentration (measured in the PA) and its volume of distribution. It can be shown that M is equal to the product of flow and the integral of concentration over time,[18 50 183] expressed by the equation:

$$M=\dot{Q}\times\int C(t)dt$$

where $\dot{Q}$ = flow and represents right ventricular cardiac output.

Currently the preferred method is to inject a cold solution and then monitor pulmonary artery blood temperature. The equation above can be expressed with reference to temperature as:

$$V_i(T_b-T_i)K_1K_2=\dot{Q}\times\int \text{Temp}\ dt$$

and

$$\dot{Q}=V_i(T_b-T_i)K_1K_2/\int \text{Temp}\ dt$$

where V_i and T_i = volume and temperature of injectate, respectively, T_b = blood temperature, Temp = difference between the initial temperature and the new temperature in the pulmonary artery; K_1 unifies units and relates the specific heat and gravity of injectate to blood and K_2 relates the injectate and dead-space volumes.

Despite empirical correction factors and rapid injection of tracer fluid, heat transfer to right atrial blood, catheter wall and surrounding tissues may lead to overestimation of cardiac output.[18 86 185] Baseline fluctuations in pulmonary artery tempera-

ture,[125 183] abnormal haematocrit values and intracardiac shunts are other potential sources of error.[121]

For convenience there has been a trend to use room temperature injectate in everyday practice. The signal-to-noise ratio obtained with 10 ml of injectate at room temperature is adequate for most circumstances with cardiac indices of 2.6–4.2 litre min^{-1} m^{-2}.[98 137 179]

The effect of IPPV on cardiac output measurement throughout the respiratory cycle is complex. Predictably, during IPPV cardiac output measurements are highest at end-expiration. Positive end expiratory pressure (PEEP) decreases preload and mean cardiac output, particularly in hypovolaemic patients[86 109 180] although some have found that the response to PEEP, particularly in ischaemic heart disease, is more heterogenous.[148]

For these reasons variations of up to 25% can occur between single measurements and it is customary to average three measurements taken randomly throughout the respiratory cycle[2] and to be guided by trends rather than absolute values.

Continuous cardiac output and mixed venous oxygen saturation

More recent PAC developments have enabled near continuous thermodilution calculation of cardiac output (CCO).[189] A heating filament is attached to a pulmonary artery catheter such that it comes to lie in the right ventricle. Pulses of heat produced by the filament are detected by the thermistor sited in the distal tip of the PAC. Changes in distal pulmonary artery temperature produce a thermodilution-like washout. Measurements are updated every 30 s and averaged over several minutes.

CCO appears as accurate and reproducible as intermittent thermodilution calculations over a large number of diverse patients.[21 113] Although it is clearly not truly continuous CCO is potentially better than intermittent measurements, typically several hours apart.[119]

CCO has the advantage of eliminating the potential errors associated with variations in volume and speed of tracer injection. Nevertheless, rapid fluid infusions may change PA blood temperature and affect its accuracy.[45 119]

Pulmonary artery catheters capable of measuring continuous $S\bar{v}_{O_2}$ by fibreoptic oximetry have been available since the early 1970s.[110 119] Based on the Fick principle it follows that cardiac output, oxygen consumption and arteriovenous oxygen difference are related such that when oxygen consumption is constant, changes in mixed venous oxygen content (and $S\bar{v}_{O_2}$) are directly proportional to cardiac output.[89 181]

Normally there is good correlation between cardiac output and $S\bar{v}_{O_2}$, although if oxygen consumption fluctuates a decrease in $S\bar{v}_{O_2}$ may be caused by a decrease in cardiac output or by an increase in oxygen extraction.[120] Nevertheless, $S\bar{v}_{O_2}$ reflects the overall balance between oxygen supply and consumption.[119] Other potential problems include measurement errors caused by methaemoglobinaemia, carboxyhaemoglobinaemia and non-linearity of the haemoglobin oxygen dissociation curve.[4 110]

Some of the interpretation difficulties outlined above may be overcome when simultaneous $S\bar{v}_{O_2}$, arterial saturation and cardiac output readings are combined. If cardiac output is unchanged, arterial desaturation with a widening gap between Sa_{O_2} and $S\bar{v}_{O_2}$ suggests an increased extraction ratio (fig. 4A). If Sa_{O_2} remains relatively unchanged, a decrease in $S\bar{v}_{O_2}$ normally represents an increase in oxygen extraction to compensate for the reduced oxygen delivery associated with a low cardiac output (fig. 4B).

Right ventricular ejection fraction (RVEF)

RVEF can be determined from the thermodilution curve by analysis of successive diastolic plateaus using a PAC equipped with a rapid response thermistor.[92] More recently, techniques based on computer algorithms[43] and systolic time intervals[67] have been introduced.

In order to avoid inaccuracies in estimating RVEF it is important to position the PAC carefully. Ideally, the injection port should be in the right atrium as near as possible to the tricuspid valve; the thermistor should be in the pulmonary artery just distal to the pulmonary valve.[165] When heart rate is erratic or very fast, RVEF is technically difficult to obtain. Also, RVEF is too dependent on right ventricular afterload to be a good indicator of right ventricular contractility.[120] However, it can be used to calculate right ventricular end-diastolic volume (RVEDV) and other right ventricular derived variables from the formula: RVEDV = SV/RVEF.

It has been suggested that RVEDV is a more accurate indicator of preload and correlates better with cardiac output and right and left ventricular work indices than RAP or PAOP.[44 138 148] This is understandable when one considers the complex interactions between positive pressure ventilation, PEEP, vasoactive agents, ventricular compliance and venous tone,

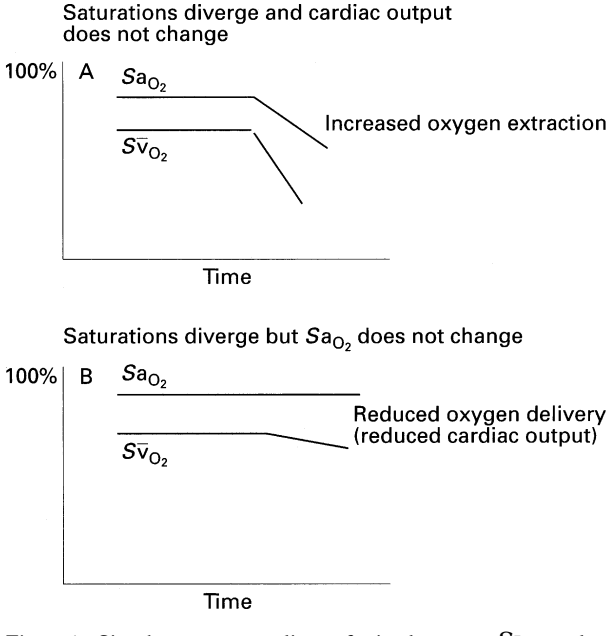

Figure 4 Simultaneous recordings of mixed venous $S\bar{v}_{O_2}$ and arterial (Sa_{O_2}) saturations in two common situations of changing tissue oxigenation. A: Cardiac output is unchanged and tissue oxygenation worsens because of increased oxygen extraction. B: Arterial oxygenation remains constant, but a decrease in cardiac output causes a decrease in oxygen delivery.

all of which can increase PAOP while decreasing actual ventricular volume. However, as the net effect of such factors on overall cardiac output can be monitored directly, it can be argued that calculation of RVEDV, despite being more accurate, contributes little additional information. Further, the mathematical relationship between RVEDV and SV increases the possibility of artefactual coupling as a possible cause of the correlation between the two.[120]

The case for measurement of RVEDV is stronger in sepsis,[95 135] pulmonary hypertension[144 191] and ARDS[159 163] where right ventricular function is a major determinant of cardiac output.[128] In addition, high PEEP levels are normally necessary in the management of these conditions and this makes preload assessment by pressure (PAOP) measurements less reliable.[120]

Pulmonary artery catheterization in practice

Between 1989 and 1994, a prospective observational study was conducted involving 5735 patients treated in 15 intensive care units in five teaching centres in the US.[28] This work was an extension of previous studies restricted to patients with myocardial infarction[66 192] because it included, in addition, patients with congestive heart failure, chronic obstructive pulmonary disease, acute respiratory failure, sepsis, multi-organ failure and four other disease categories. Treatment selection bias was adjusted by case matching, multivariate analysis and a propensity score, itself a source of controversy.[136 176]

It reported a significantly increased risk of death at 30 days in patients managed with a PAC, as follows: odds ratio (OR) of 1.24 in 1008 pairs of patients case matched to adjust for treatment bias, and OR of 1.21 in the total patient group of 5735 after multivariate analysis to adjust for treatment bias. In addition, the PAC group had longer length of stay in the ICU and increased direct hospital costs.[28]

The result of the study by Connors and colleagues[28] raised concern that pulmonary artery catheterization might be partly responsible for an increased mortality, either directly or by leading to potentially harmful treatment.[37 164] A recent task force analysed the evidence from clinical studies involving PAC-guided management in a variety of clinical groups.[129] In many patient groups the evidence was either weak or inconclusive.

Given such controversy, the impact of the PAC on patient outcome might be judged from clinical studies of PAC derived management changes. However, few such studies, mostly limited to perioperative care, include patients managed without PAC-derived information.[5 68 85 175] This is largely because of the ethics of depriving patients of an instrument previously considered as beneficial. The setting appears to be set for large randomized, controlled studies on the issue of PAC-guided management[29 37 129 136] or, at the very least, a re-evaluation of its use.[164 176]

In critical care and anaesthesia there have been principally three indications for pulmonary artery catheterization: diagnosis, guidance of fluid and vasoactive drug administration, and close observation of cardiopulmonary interactions. In practice this includes four areas which are discussed below.

ASSESSMENT OF INTRAVASCULAR VOLUME, ADEQUACY OF CARDIAC OUTPUT AND OXYGEN TRANSPORT

For many years right heart catheterization with the PAC has been standard in assessing left ventricular preload and therefore intravascular volume status.[53] However, PAOP does not always correlate with left ventricular preload, particularly in non-compliant hearts,[58] left ventricular dysfunction,[14 49] myocardial ischaemia,[54 133] valvular heart disease or patients receiving vasoactive therapy,[1] all traditional indications for insertion of a PAC. Nevertheless, subsequent studies revealed inaccurate "bedside" estimation of haemodynamic variables compared with PAC readings.[26 47] This is perhaps inevitable if the PAC is used as the gold standard; the reverse would likely be true were clinical signs taken as the gold standard. These observed inaccuracies were used to support PAC catheterization. But there seems to be poor understanding and incorrect interpretation of PAC-derived data.[64 82]

But even when the PAC data are interpreted correctly and indicate true preload, we do not know if given values are adequate or what is the individual optimum preload. For example, most clinicians would find that a cardiac index of 3 litre min^{-1} m^{-2} is more than expected, immediately after heart surgery, too little for a 20-yr-old trauma victim and possibly adequate for an 80-yr-old with sepsis. Other technologies[104 105 161] are becoming popular but again do not indicate if the cardiac output achieved is effective.

The PAC is also used to calculate systemic and pulmonary haemodynamic data (table 2) and oxygen transport values (table 3). The physiological response to trauma, anaesthesia and major surgery has long been the subject of interest.[24 33 40 152 155 171] Oxygen transport studies in high-risk surgical patients found higher levels of cardiac index (CI), oxygen delivery index [$\dot{D}_{O_2}$I] and oxygen consumption index ($\dot{V}_{O_2}$I) among survivors than in non-survivors. Low oxygen transport indices were proposed as a cause of tissue hypoxia and death.

Shoemaker and colleagues suggested that the median values among survivors be used as therapeutic goals.[11 158] Numerous studies used the PAC to guide augmentation of oxygen transport, with the aim of treating underlying tissue hypoxia.

In prospective studies to test this hypothesis patients treated to achieve target values in CI, [$\dot{D}_{O_2}$I] and $\dot{V}_{O_2}$ had much higher survival rates.[156 157] This was further supported in patients undergoing peripheral limb surgery[5] and in a prospective randomized study of high-risk surgical patients.[16] In septic shock, similar therapy was also associated with improved outcome compared with historical controls[46] or with subgroups created retrospectively.[174 190]

However, these encouraging results were not replicated when predominantly septic patients were randomized after entry to an ICU. Hayes and colleagues found that patients receiving aggressive management with fluids and, when necessary, inotropes to achieve target haemodynamic goals had higher mortality than controls treated to reach acceptable baseline haemodynamic values.[72] Further, in a much larger study, when supranormal cardiac output was the sole goal, patients did no better than those who remained

Table 2 Measured haemodynamic variables

Variable	Normal range (units)
Central venous pressure (CVP)	0–8 mm Hg
Right ventricular systolic pressure (RVSP)	15–30 mm Hg
Right ventricular end-diastolic pressure (RVEDP)	0–8 mm Hg
Pulmonary artery (systolic) pressure (PAP)	15–30 mm Hg
Pulmonary artery diastolic pressure (PADP)	4–12 mm Hg
Pulmonary artery occlusion pressure (PAOP)	2–12 mm Hg
Cardiac output ($\dot{Q}$)	Varies with size, age and sex
Mixed venous oxygen saturation ($S\bar{v}_{O_2}$)	75%

as controls or who were treated to achieve a "normal" $S\bar{v}_{O_2}$.[60]

Unfortunately, even the most rigorous studies have neither separated supportive from definitive treatment (correct antibiotics, timely surgery, etc.) nor established that the latter was balanced between study groups. Overall, those patients with sufficient cardiorespiratory reserve to increase their oxygen delivery[16 174] and utilization[73 74] appear to have a better chance of survival. Those with less cardiorespiratory reserve treated to achieve supranormal targets have, at best, no better outcome than similar patients managed within their physiological limitations.[77] There is no convincing evidence that attemping to increase cardiac index indiscriminantly across a wide cross section of critically ill patients improves outcome[60 72] and some evidence indicates it may increase mortality.[72]

The emphasis in cardiovascular resuscitation has shifted away from "routinely" attempting to obtain goals for which the PAC is a prerequisite, to management adjusted to individual cardiac performance. This might be acomplished by determining if cardiac output is effective, initially predominantly by clinical assessment, and later complemented by pulmonary artery catheterization. An effective cardiac output (ECO) could be defined as that which achieves normal premorbid arterial pressure with warm peripheries at a heart rate of less than 100 beat min^{-1}, irrespective of supportive therapy. The effectiveness of peripheral perfusion can later be assessed by biochemical analyses such as acid–base balance, gastric intra-mucosal acid estimation and blood lactate concentration. However, it is important to consider that these biochemical markers can be deranged for reasons other than haemodynamics (i.e. hepatic or renal failure, or both).

MYOCARDIAL DYSFUNCTION

Patients with acute MI complicated by congestive cardiac failure or hypotension in whom treatment was guided by a PAC had a higher mortality rate than those managed without it, even after multivariate analysis to adjust for disease severity.[66 192] These studies were retrospective and were unable to exclude that sicker patients received PAC, but the direct mortality associated with PAC was a possibility. As a result, reassessment of the PAC was advocated.[36 139]

When the PAC has been used to guide management in elective coronary artery bypass grafting[175] and abdominal aneurysm surgery[85] there has also been no difference in serious end-organ complications or mortality. Furthermore, placement of a PAC electively or after haemodynamic deterioration did not appear to affect outcome, suggesting that conventional indicators of the need for PAC may be unreliable.[175]

Perioperative myocardial ischaemia is an important cause of postoperative cardiac morbidity and mortality.[107 108] Despite conflicting accounts of the role of the PAC in the detection of myocardial ischaemia[69 85 178] it is useful in detecting haemodynamic events, such as decrease in cardiac output, stroke work or mixed venous oxygenation or an increase in PAOP, which normally are associated with myocardial ischaemia. Earlier work indicated that when used as part of a management strategy designed to avoid adverse haemodynamic fluctuations, the PAC helped to reduce perioperative cardiac events.[134] There is, however, no definitive evidence favouring the use of PAC in patients at risk of perioperative cardiac dysfunction undergoing major surgery.[129]

Table 3 Derived haemodynamic variables. DAP = Diastolic arterial pressure; SAP = systolic arterial pressure; Hb = haemoglobin; ($P\bar{v}_{O_2}$) = mixed venous oxygen tension

Variable	Calculation	Normal range
Body surface area (BSA)	Weight $(kg)^{0.425} \times$ height $(cm)^{0.725} \times 0.007184$	
Mean arterial pressure (MAP)	DAP + 1/3 × (SAP–DAP)	70–105 mm Hg
Cardiac index (CI)	$\dot{Q}$/BSA	2.8–4.2 litre min^{-1} m^{-2}
Stroke volume (SV)	$\dot{Q}$/HR	80 ml
Stroke index (SI)	SV/BSA	30–65 ml m^{-2}
Left ventricular stroke work index (LVSWI)	CI × (MAP–PAOP) × 0.0136	44–64 g m^{-1} m^{-2}
Systemic vascular resistance (SVR)	(MAP–CVP) × 80/$\dot{Q}$	1000–1200 dyn s cm^{-5} m^{-2}
Systemic vascular resistance index (SVRI)	(MAP–CVP) × 80/CI; (nb SVRI = SVR × BSA)	1600–2400 dyn s cm^{-5} m^{-2}
Mean pulmonary artery pressure (MPAP)	MPAP + 1/3 × (PAP–PADP)	9–16 mm Hg
Right ventricular stroke work index (RVSWI)	CI×(MPAP–CVP) × 0.0136	7–12 g m^{-1} m^{-2}
Pulmonary vascular resistance (PVR)	(MPAP–PAOP) × 80/$\dot{Q}$	60–120 dyn s cm^{-5} m^{-2}
Pulmonary vascular resistance index (PVRI)	(MPAP–PAOP) × 80/CI; (nb PVRI = PVR × BSA)	250–340 dyn s cm^{-5} m^{-2}
Arterial oxygen content (Ca_{O_2})	Sa_{O_2} × Hb×1.39 + Pa_{O_2} (mm Hg) × 0.003	180 ml $litre^{-1}$
Mixed venous oxygen content ($C\bar{v}_{O_2}$)	$S\bar{v}_{O_2}$ × Hb×1.39 + Pv_{O_2} (mm Hg) × 0.003	130 ml $litre^{-1}$
Oxygen delivery (Do_2)	$\dot{Q} \times Ca_{O_2}$	850–1050 ml min^{-1}
Oxygen consumption ($\dot{V}o_2$)	$\dot{Q}/(Ca_{O_2}/Cv_{O_2})$	180–300 ml min^{-1}
Oxygen delivery index (Do_2I)	Do_2/BSA	520–650 ml min^{-1} m^{-2}
Oxygen consumption index ($\dot{V}o_2$I)	$\dot{V}o_2$/BSA	110–180 ml min^{-1} m^{-2}

ACUTE LUNG INJURY

Acute lung injury (ALI) and its most severe form ARDS[3] are characterized by refractory hypoxaemia, widespread alveolar infiltrates on chest x-ray and respiratory distress not caused by but which may coexist with increased left atrial pressure.[7] The destruction of the alveolo-capillary membrane, inflammatory alveolar exudate[10] and subsequent fibrotic response[35] lead to alveolar oedema, reduced lung compliance, increased venous admixture and hypoxaemia.[96] Pulmonary vasoconstriction can worsen ventilation–perfusion mismatch and cause pulmonary hypertension with increased right ventricular work.[191]

In ALI high capillary permeability and increased capillary pressures coexist.[30 166] There is currently no treatment that can reverse the former.[149] Therefore, it might be more important to reduce pulmonary capillary hydrostatic pressure, in an attempt to minimize alveolar oedema,[111 149] and to decrease pulmonary vascular resistance in order to reduce pulmonary hypertension.

There is no conclusive evidence that pulmonary artery catheterization affects outcome in ARDS. However, especially when high levels of PEEP are used, it might be justifiable to use PAC in the diagnosis of ARDS and to measure the variables to be manipulated; these include PAOP and the pulmonary vascular and right ventricular haemodynamic indices.

Reduction of alveolar oedema

As the early manifestations of ARDS include alveolar and interstitial fluid accumulation, therapy leading to removal of alveolar fluid may improve gas exchange. Indeed numerous studies suggest that fluid restriction may be beneficial.[150] In retrospective analyses a favourable fluid balance[147] and a reduction in PAOP[80] were associated with less ventilator, ICU and hospital days. When the guide to fluid reduction was extravascular lung water (EVLW) (measured by double indicator dilution[103 162]), there was a reduction in ICU and hospital stay[114] and, after retrospective subdivisions, mortality.[48] However, these studies may be taken as markers of less severe illness, in as much as they reflect ability to withstand fluid elimination without detriment.

Nevertheless, there is widespread agreement that in some patients excessive fluid reduction may worsen cardiovascular performance and compromise organ perfusion.[81 150 172] In practice the ability to remove enough fluid to improve oxygenation depends on whether this can be done without life threatening hypoperfusion, particularly if the underlying sepsis remains uncontrolled. Furthermore, as the relationship between PAOP, P_c, alveolar fluid and oxygenation is complex[30 79 102 160] the impact of lowering PAOP on alveolar fluid is unpredictable. On balance it seems appropriate to reduce PAOP in the early exudative phase of ARDS cautiously to a level which does not compromise cardiovascular function or organ perfusion. The possible benefit of cautious fluid restriction and diuresis for more than 3 or 4 days remains to be determined.[96]

Selective pulmonary vasodilatation

Patients with severe ARDS and acute lung injury may develop pulmonary hypertension (PHT) with significant right ventricular dysfunction[159 191] and loss of hypoxic pulmonary vasoconstriction.[19] Traditional i.v. vasodilators used to control pulmonary hypertension can lead to worsening intrapulmonary shunt, systemic hypotension and impaired oxygenation.

In the past few years inhaled nitric oxide has been used as an alternative pulmonary vasodilator. Nitric oxide is an endogenous gas[84 123] with potent vascular smooth muscle relaxant properties.[115] It preferentially vasodilates ventilated lung segments[56 61 127] and can improve ventilation–perfusion matching and reduce pulmonary arterial pressure with little systemic hypotension.[9 13 126 130 132 141] Nitric oxide also reduces right ventricular afterload[52] and pulmonary hypertension attributable in part to permissive hypercapnia.[131]

The results of the first randomized, controlled phase II trial of placebo *vs* nitric oxide at various doses have become available.[41] Although nitric oxide was well tolerated, it did not improve mortality. However, a relatively large proportion (60%) of patients receiving nitric oxide responded with an increase in oxygenation in the first 4 h, but this was small and not long lasting. There was a decrease in mean PAP which was also small (approximately 2 mm Hg) and short-lived.

PULMONARY HYPERTENSION

PHT caused by chronic obstructive lung disease,[106 145] left heart disease and thromboembolic disease[116] is commonly encountered in the intensive care patient. Many general treatments of these conditions and more specific treatments of PHT (discussed above) affect right and left heart performance. Therefore, the PAC becomes useful to monitor pulmonary artery pressure, and ventricular and systemic haemodynamic responses to therapy.

Conclusion

Pulmonary artery catheterization is over half a century old. It is a valuable educational tool and facilitates quantification of the clinical assessment of cardiorespiratory performance. However, the PAC has also taken the clinican away from patients and encouraged decisions based on numerical targets, thus detracting from the concept of individualized supportive therapy.

The PAC still has a significant role in patient management, primarily as a method for diagnosis and monitoring haemodynamic responses to therapy. In common with every other instrument, it has drawbacks. Its insertion delays immediate therapy, introduces infection and may precipitate iatrogenic events. If it allows us to adjust clinical assessment and supportive therapy it will have achieved its goal. On the other hand, if it promotes hands off haemodynamic management it will be criticized unfairly and may be superseded by other technologies which could go through the same cycle.

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