INVASIVE IMAGING: CARDIAC CATHETERIZATION AND ANGIOGRAPHY

Right heart catheterisation: indications and interpretation

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INTRODUCTION

Significant improvements in the diagnostic power and availability of non-invasive cardiac imaging techniques, in addition to evidence of potential harm associated with pulmonary artery (PA) catheterisation in patients in critical care, have led to a decline in right heart catheterisation (RHC) over recent years.[1] RHC, however, remains an important tool in a cardiologist's diagnostic armoury, providing direct haemodynamic data that can be used to determine cardiac output (CO), evaluate intracardiac shunts and valve dysfunction. It is the gold standard method for diagnosing pulmonary hypertension (PH) and an essential component in the evaluation of patients prior to heart and/or lung transplantation.[2 3] RHC can be also used to assess the haemodynamic effects of treatments directly and provides an entry route for intracardiac biopsy. The European Society of Cardiology (ESC) core curriculum 2013 states that trainees should possess the skills to 'carry out right heart catheterization in the catheterization laboratory and at the bedside, and measure cardiac output, intravascular pressure, and oxygen saturation'.[4]

This article covers the history of RHC, how to perform a complete right heart study and a review of its current place as a diagnostic tool in a range of cardiovascular disorders.

BACKGROUND

The first reported RHC was performed on a horse by the physiologist Claude Bernard in 1844.[5] Glass tubes were inserted via the jugular vein and carotid artery in order to measure the temperature in both ventricles. Bernard *et al* subsequently used the technique to measure intracardiac pressures. The first RHC in a human was performed by Werner Forssmann in 1929 in Eberswalde, Germany. Using an approach that is typically frowned on by research ethics committees, he performed self-catheterisation using a urethral catheter through his left antecubital vein into his right ventricle (RV), and confirmed placement with X-ray.[6] The technique was subsequently refined by Andre Cournand and Dickinson Richards, who were able to demonstrate its safety, even when the catheter was left in situ for over 24 h.[7] They developed the direct Fick method for measuring CO and gained a greater understanding of right heart haemodynamics in both healthy subjects and those with pulmonary disease. Drs Forssman, Cournand and Dickinson received the Nobel Prize in medicine in recognition of their contributions to medical science. Hellems *et al* subsequently developed a way of measuring

Learning objectives

▶ To gain an overview of the history and development of right heart catheterisation.
▶ To learn how to perform a right heart study tailored to answer a specific clinical question.
▶ To gain a better understanding of the role of right heart catheterisation as a diagnostic tool in specific conditions, including pulmonary hypertension, valvular heart disease and differentiating between constrictive pericarditis and restrictive cardiomyopathy.

pulmonary capillary wedge pressure (PCWP) under fluoroscopic guidance, which added to the value of the test.[8]

Use of a self-guiding catheter was first described in 1953 by Lategola and Rahn.[9] Further modifications ultimately led to the development of a balloon flotation catheter developed by Jeremy Swan and William Ganz.[9] The 'Swan–Ganz' catheter had an average time to catheterisation of 35 s, and could be left in situ for prolonged periods, which led to a surge in its use in critical care. At the peak of their popularity in the mid-1980s, Swan–Ganz catheters were used in 45% of American patients admitted to the critical care unit.[10] Although balloon flotation catheters are more reliable than end-hole catheters in making it possible to measure a wedge pressure, the balloon poses some risk of damage to the pulmonary vasculature, particularly if it is left inflated inadvertently. Concerns over the safety of seemingly indiscriminate use of invasive monitoring led to a number of large studies that showed PA catheterisation-guided management was associated with increased mortality and length of intensive care unit stay and has appropriately led to a substantial decline in the use of PA catheters.[1]

TECHNIQUE
Patient preparation

Patients must be fully informed of the indication for, and risks of, the procedure. A large review of complications during RHC, incorporating both retrospective and prospective data, found a serious adverse event rate of 1.1%, and mortality rate of 0.05%.[11] The most common complications are access site haematoma, vagal reaction, pneumothorax and arrhythmias. Where available, individual or departmental complication rates should be quoted.

Catheterisation is frequently performed without interruption of anticoagulation. This is safe in patients with an international normalised ratio of <3.5 undergoing RHC via either the internal jugular vein (IJV) or antecubital veins.[12]

Fasting prior to a procedure will depend on local policy, although it should be borne in mind that overzealous fasting protocols may lead to volume depletion, thus making venous access more challenging.

Venous access

The route of access depends on a number of factors, including operator experience, the presence of cardiac devices and in-dwelling catheters, and prior history of venous cannulation and associated complications. Femoral vein (FV) access is commonly used if left heart catheterisation is performed concurrently, although a number of small studies have demonstrated the feasibility and safety of performing RHC and left heart catheterisation via an antecubital fossa vein and radial artery, respectively.[13–16]

A meta-analysis of ultrasound-guided versus landmark-based venous access demonstrates a clear benefit of ultrasound for IJV cannulation, with a higher success rate, fewer complications and faster access.[17] There are very limited data on the use of ultrasound for FV and subclavian vein (SCV) cannulation.

Strict aseptic measures should be applied. The procedure is performed using local anaesthetic; sedation is rarely required. A Seldinger technique should be used to gain venous access.

Balloon flotation catheters, such as the Swan–Ganz catheter, have a balloon at the distal end to facilitate passage through the right heart. They are designed to be placed without the need for fluoroscopy, although screening is often helpful if the patient has marked right heart dilation or severe tricuspid regurgitation. The catheter is inserted into the right atrium (RA) (15–20 cm via IJV, 10–15 cm via SCV, 25–30 cm via FV) and the balloon is inflated. The catheter then follows the direction of blood flow towards the PAs. Advancing further should allow the performer to obtain the PCWP. It is important to avoid leaving the balloon inflated in the wedge position for longer than necessary, as there is a risk of pulmonary infarction or rupture.

Catheterisation from the FV is commonly performed using a multipurpose end-hole catheter under direct fluoroscopy. It requires greater manipulation than the balloon flotation catheters to navigate through the right heart, and a guidewire may be required to improve steerability. Multipurpose catheters can be used to cross directly into the left atrium (LA) in patients with a patent foramen ovale for direct pressure recordings.

Pressure tracings

It is vital to make sure that pressure lines and transducers are correctly set up, as any inaccuracies in measurement will be magnified in subsequent calculations. Transducers are positioned at the level of the patient's mid-chest. Lines and manifolds should be flushed to remove bubbles that would otherwise result in pressure damping. Each transducer should be zeroed during set up, with zeroing repeated prior to recording pressures. The patient should have ECG monitoring throughout the procedure, ensuring the pressure and ECG traces are timed correctly. The scale and sweep speed should be adjusted for each waveform measurement to facilitate subsequent analysis. Recordings are best taken at end-expiration for consistent and stable traces. Box 1 gives a structured protocol for obtaining RHC measurements that is used at our institution. Clearly, the choice of measurements taken will depend on the clinical question that one wishes to answer, but a plan should be made in advance in order to reduce the risk of a missed trace or saturation measurement.

Oxygen saturation measurements

Direct sampling of blood from intracardiac chambers and great vessels enables the detection and quantification of shunts between the systemic and pulmonary circulations. In RHC via the superior vena cava (SVC), samples are taken from the SVC and main PA (MPA). A difference of >8% may indicate the presence of a left to right shunt and should prompt a full saturation run for more precise localisation. Samples are then taken from high and low inferior vena cava (IVC), high and low SVC, high, mid-RA and low RA, mid-RV, RV outflow, MPA, left and right PAs, left ventricle (LV) and distal aorta. Additional samples are taken from the pulmonary vein and LA when the interatrial septum is crossed. A disadvantage of this method is that it lacks sensitivity to detect small shunts, but the majority of haemodynamically significant shunts will be identified.[18] It also assumes steady state blood flow throughout the entire sampling run, which may not always be the case, particularly if arrhythmias occur. Table 1 provides calculations for the determination of shunt size and figure 1 provides an example saturation run from a patient with a large secundum atrial septal defect.

CO measurement

The two most commonly used techniques for determining CO are the thermodilution and Fick methods. Thermodilution measurements involve injecting a fluid bolus at a known temperature into the proximal port of a PA catheter, and recording the change in temperature at the distal end of the catheter with a thermistor. CO is calculated based on the temperature and specific gravity of blood, and the temperature, specific gravity and volume of injected fluid.

The Fick method for determining CO requires the following measurements:

▶ Oxygen consumption, or VO_2
▶ The oxygen content of arterial blood
▶ The oxygen content of mixed venous blood

The Fick method requires measurement of oxygen consumption during cardiac catheterisation using a spirometer and rebreathing bag, which is

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Box 1 Right and left heart catheterisation pressure recording protocol

- ▶ PA pressure
- ▶ PCWP pressure
- ▶ Simultaneous PCWP and LV pressures
- ▶ Withdrawal from PCWP to PA
- ▶ Simultaneous PA and LV pressures
- ▶ Withdrawal from PA to RV
- ▶ Simultaneous RV and LV pressures
- ▶ Withdrawal from RV to RA
- ▶ Simultaneous RA and LV pressures

LV, left ventricle; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle.

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time-consuming and often impractical. Oxygen consumption is thus often assumed based on age, sex and body surface area. Mixed venous saturation is measured from the MPA, where there is complete mixing of blood returning from the SVC, IVC and coronary sinus. Arterial oxygen saturation can be taken during simultaneous left heart catheterisation, or via pulse oximetry, or peripheral arterial sampling. A recent haemoglobin level is also required for determination of blood oxygen content. The formula for calculating CO using the Fick method is shown in table 1.

Studies have demonstrated a reasonable correlation between thermodilution and Fick methods for the estimation of CO, although there may be significant variation in individual patients.[19] Thermodilution tends to overestimate in low CO states, and it is also inaccurate in patients with significant tricuspid regurgitation.

CO monitoring using Swan–Ganz catheters was once commonplace in critical care patients, but as described earlier, randomised controlled trials subsequently demonstrated that this was associated with harm. This appears to be due to a combination of inaccuracies in measurement, incorrect interpretation of the data and responses to measurements that may have been harmful. PA catheterisation remains useful in specific situations such as acute RV infarction and in patients with PH undergoing surgical intervention.[20]

Pulmonary angiography

Pulmonary angiography can be used to diagnose pulmonary embolism (PE), PA stenosis and arteriovenous malformations. An angled pigtail catheter with side holes is commonly used to deliver contrast, in order to reduce the risk of damage to the pulmonary vasculature. The tip of the catheter is positioned in the proximal left and right PAs, and images should be acquired during a breath-hold at end inspiration. Contrast is injected at a rate of 15–20 mL/s for 2 s. Images should be obtained in at least two projections, typically anteroposterior, and posterior-oblique views (figure 2).

Diagnosis of PE requires the demonstration of thrombus in two radiographic projections.[21] CT pulmonary angiography is now the preferred imaging modality for diagnosing PE, and may be better at identifying sub-segmental emboli.[22] Invasive pulmonary angiography enables the simultaneous collection of haemodynamic data which can help in determining the severity of PE. RHC can also be used for the treatment of life-threatening emboli, particularly when there is an absolute contraindication to thrombolysis.[23] A number of techniques can be used, including mechanical clot disruption and aspiration, which frequently result in acute haemodynamic improvement and early recovery of RV function.[24 25]

Pulmonary angiography is considered the gold standard for diagnosing chronic thromboembolic pulmonary hypertension (CTEPH), and assessing the feasibility of pulmonary endarterectomy.[26] This should be carried out in specialist centres by operators experienced in the management of CTEPH.

Delayed image acquisition once contrast has passed into the LA (termed the levo-phase) can be used to assess pulmonary venous drainage and may be helpful in detecting LA thrombus and myxoma.[27 28]

PULMONARY ARTERIAL HYPERTENSION

RHC is the gold standard for measuring PA pressure. The 2009 ESC guidelines on diagnosis and treatment of PH state that 'RHC is required for the diagnosis of pulmonary arterial hypertension (PAH), to assess the severity of haemodynamic impairment, and to test the vasoreactivity of the pulmonary circulation'.[2] PAH is defined as a mean PA pressure of ≥25 mm Hg at rest or ≥30 mm Hg during exercise. Measurement of PCWP allows differentiation between PH due to left-sided heart disease and pre-capillary PAH—conditions that

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Table 1 Commonly used haemodynamic calculations during right heart catheterisation

Cardiac output (Fick) (L/min)	$\dfrac{O_2 \text{ consumption (mL/min)}}{(SaO_2 - MVO_2\text{ saturation}) \times 1.36 \times Hb \times 10}$
Cardiac index (L/min/m^2)	$\dfrac{\text{Cardiac Output}}{\text{BSA}}$
Stroke volume (mL/beat)	$\dfrac{\text{Cardiac Output}}{\text{HR}} \times 1000$
Stroke volume index (mL/beat/m^2)	$\dfrac{\text{Cardiac Index}}{\text{HR}} \times 1000$
Pulmonary vascular resistance (Wood Units)	$\dfrac{\text{Mean PA pressure} - \text{Mean PCWP}}{\text{Cardiac output}}$
Systemic vascular resistance (Wood Units)	$\dfrac{\text{Mean arterial pressure} - \text{Mean RA pressure}}{\text{Cardiac output}}$
(To convert from Wood Units to dynes/s/cm multiply by 80)	
RV stroke work index (g/m^2/beat)	$SVI \times (\text{Mean PA pressure} - \text{Mean RA pressure}) \times 0.0136$
Intracardiac shunt calculations	
Pulmonary blood flow	$\dfrac{O_2 \text{ consumption (mL/min)}}{PVO_2 - PAO_2}$
Systemic blood flow	$\dfrac{O_2 \text{ consumption (mL/min)}}{SaO_2 - MVO_2}$
Qp/Qs	$\dfrac{\text{Pulmonary blood flow}}{\text{Systemic blood flow}}$
In the presence of a left to right shunt, MVO_2 should be calculated as	$\dfrac{3 \times SVC\,O_2 + IVC\,O_2}{4}$

BSA, body surface area; HR, heart rate; IVC, inferior vena cava; MVO$_2$, mixed venous oxygen saturation; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; SaO$_2$, systemic arterial oxygen saturation; SVC, superior vena cava; SVI, stroke volume index.

Location	Oxygen saturations (%)
Superior Vena Cava	71.1
High right atrium	88.2
Mid right atrium	92.3
Low right atrium	82.7
Inferior Vena Cava	82.2
Right Ventricle	94
Pulmonary Artery	94
Pulmonary vein	99.1
Left Atrium	98.4
Left Ventricle	98
Aorta	96.4
Qp:Qs ratio 4.1:1	

Figure 1 Oxygen saturation recordings from a patient with a large secundum atrial septal defect. This demonstrates a significant 'step up' in saturations at the mid-atrial level, indicating the presence of a left to right shunt.

require markedly different therapeutic approaches (figure 3). A PCWP of >15 mm Hg excludes the diagnosis of pre-capillary PAH.

The transpulmonary pressure gradient (TPG) is defined as the difference between the mean PA pressure and PCWP. In a patient with PH and PCWP >15 mm Hg, a TPG >12 mm Hg suggests 'out of proportion' PH; in other words, the raised pulmonary arterial pressure cannot be fully explained by an increase in LA pressure and is in part due to adaptive changes within the pulmonary vasculature.[29] The diastolic pulmonary gradient (ie, the gradient between pulmonary arterial diastolic pressure and mean PCWP, DPG) is less dependent on changes in CO and PA vessel distensibility than TPG, and might thus be a better measure in diagnosing 'out of proportion' PH. Both a TPG >12 mm Hg and DPG >7 mm Hg are associated with lower median survival in patients with PH due to left-sided heart disease.[30]

Wedge pressure is dependent on volume loading; hypovolaemia may cause an inappropriately low PCWP, leading to a false diagnosis of PAH.
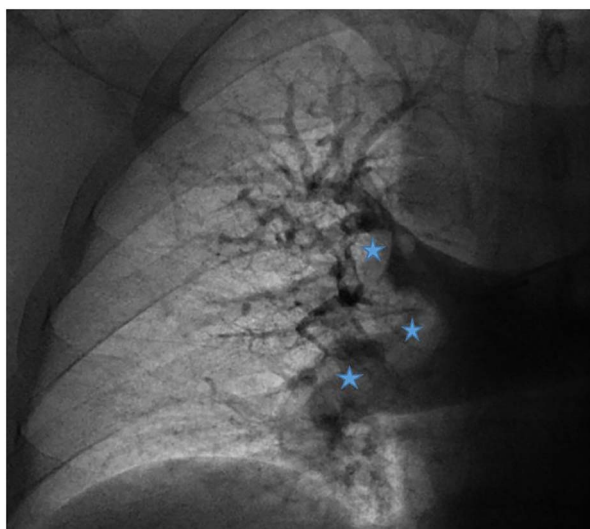

Figure 2 Pulmonary angiography demonstrating multiple filling defects within the right pulmonary artery and lobar arteries (starred), due to acute pulmonary embolism.

A 400 mL fluid challenge will help discriminate between the two conditions.[31]

Acute vasodilator testing should be performed to identify a subset of patients with type 1 PAH that are likely to respond to treatment with long-term calcium channel blockers.[32] Short-acting therapies such as inhaled nitric oxide and intravenous adenosine should be used for testing. A positive response is defined as reduction in mean PA pressure of >10 mm Hg to reach a mean of <40 mm Hg with an increased or unchanged CO. This is seen in approximately 10% of patients with PAH, who have a more favourable prognosis. Vasodilator testing should only be performed in centres experienced in the investigation and management of PH.

The haemodynamic data also provide important prognostic information. Data from international registries consistently show that higher mean RA pressure, low cardiac index and higher mean PA pressure are independently associated with worse outcomes.[33–35] The REVEAL registry of 2716 patients from the USA found that a pulmonary vascular resistance (PVR) >32 Wood units and mean RA pressure >20 mm Hg were associated with reduced survival.[36] These are incorporated into a prospectively validated risk scoring system for newly diagnosed PH.[37] Such elevated PVR measurements were seen in a small number of patients, predominantly with PH secondary to congenital heart disease (R Benza. personal communication).

VALVULAR HEART DISEASE
Advances in echocardiography have resulted in a marked reduction in the use of RHC for the assessment of valvular heart disease. RHC was previously a ubiquitous part of the investigative work-up prior to surgery, but current ESC guidelines recommend that RHC should be reserved for cases in which 'non-invasive evaluation is inconclusive, or discordant with clinical findings'.[38]

Mitral valve disease
The severity of mitral stenosis can be assessed by RHC through measurement of the diastolic pressure gradient across the mitral valve or calculation of valve area. Measurement of mitral valve area by planimetry on echocardiogram is often limited by image quality, and valve area estimation by pressure half time may not be valid in calcific mitral stenosis or in the presence of abnormal LV relaxation.[39] RHC and left heart catheterisations allow simultaneous recording of PCWP (an indirect measure of LA pressure) and LV pressure. The Gorlin formula can then be used to calculate valve area, as shown below.

$$\text{Mitral valve area } (\text{cm}^2) = \frac{\text{Cardiac output (L/min)} \times 1000}{44.3 \times \text{heart rate} \times \text{diastolic filling period} \times \sqrt{\text{mean gradient}}}.$$

The diastolic filling period is measured from the onset of diastole, at the point when the PCWP

Figure 3 Pressure traces taken from a patient with pulmonary arterial hypertension. Trace A shows very high pulmonary artery pressure (mean 75 mm Hg). Trace B shows the normal pulmonary capillary wedge pressure, confirming that the elevated pulmonary pressures are due to precapillary pulmonary vascular disease. Note that the wedge pressure is under-damped with marked artefact.
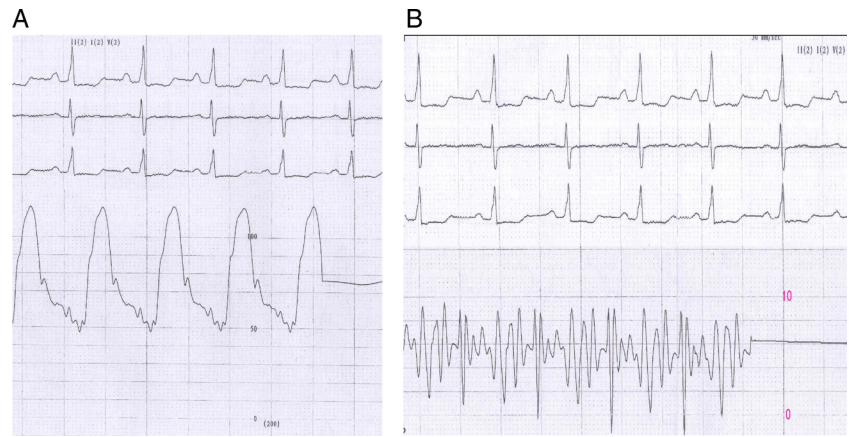
exceeds LV pressure, to the end of diastole, when LV pressure exceeds PCWP, measured in seconds/beat.

The formula incorporates a constant to account for the loss of velocity due to friction, which was calculated based on the difference between the estimated and observed valve areas in the original study by Gorlin and Gorlin.[40] Catheter-based measurements can lead to an overestimation of valve area by up to 50%, due to a delay in the transmission of LA pressure through the pulmonary vasculature.[41] Furthermore, accurate wedge tracings may be difficult to obtain in patients with significant PH or right heart dilation. Direct measurement of LA pressure via trans-septal puncture obviates these problems, but greatly adds to the procedural risk.

PH significantly increases perioperative morbidity and mortality in patients undergoing mitral valve surgery.[42] Right heart catheter measurements may therefore enable a more individualised assessment of operative risk, thus helping informed discussion between patient and clinician.

Mitral regurgitation (MR) can be assessed during a left ventriculogram, with semiquantitative scoring tools used to determine severity. Care should be taken to avoid getting the catheter caught in the mitral valve apparatus, which may accentuate the degree of MR. Large v-waves on the PCWP trace are most commonly due to MR (figure 4), although the differential diagnoses includes atrial septal defects and LV volume overload. A v-wave greater than twice the mean PCWP suggests severe MR, although this is neither a particularly sensitive or specific finding.[43]

Pulmonary and tricuspid stenosis

Valve area calculation using the Gorlin formula has not been validated for the assessment of tricuspid or pulmonary stenosis. Tricuspid stenosis can be assessed using simultaneous RA and RV pressure recordings (figure 5). Pulmonary stenosis is typically measured by catheter pull-back from the MPA into the RV, although simultaneous recordings can

Figure 4 Simultaneous left ventricular (LV) and pulmonary capillary wedge (PCW) traces in a patient with atrial fibrillation and significant mitral regurgitation are shown. Large v-waves are clearly demonstrated on the wedge pressure trace (v), with high LV end-diastolic pressure.
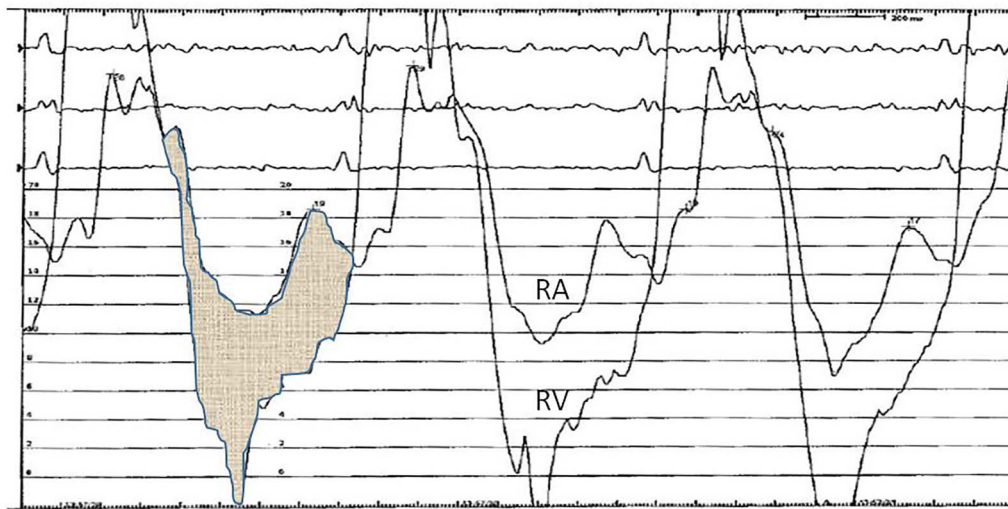
Figure 5 Simultaneous right ventricular (RV) and right atrial (RA) pressure traces from a patient with tricuspid stenosis due to carcinoid syndrome are shown. The shaded area is the pressure gradient between the RA and RV in diastole. The mean gradient is approximately 8 mm Hg, indicating severe tricuspid stenosis.

be obtained through the placement of catheters in both the PA and RV.

CONSTRICTIVE PERICARDITIS

The hallmark feature of constrictive pericarditis is diastolic pressure equalisation in all chambers of the heart. This is due to the global inhibition of diastolic filling from a fibrous, non-compliant pericardial sac.

A classic finding in constriction is a dip-and-plateau pattern in the RV pressure waveform, also known as the square root sign. The dip reflects the unimpaired early diastolic filling of the ventricles, coupled with high LA and RA pressures at the moment the mitral and tricuspid valves open. However, the ventricles then fill rapidly and suddenly meet the constraints of a rigid pericardium—the pressure in the ventricles thus reaches a plateau (figure 6).

The traditional haemodynamic criteria used in diagnosing constrictive pericarditis are shown in box 2. A retrospective review of published data found that the predictive accuracy of each individual measure ranged from 70% to 85%, whereas the positive predictive value if all three criteria are fulfilled is >90%.[44]

Treatment with high-dose diuretics prior to catheterisation may result in low filling pressures and therefore lead to the incorrect exclusion of a diagnosis of constriction. Conversely, low filling pressures due to hypovolaemia, in the absence of constriction, may lead to apparent pressure equalisation and a false-positive result. A fluid challenge can help in improving diagnostic power in both situations.[45]

RESTRICTIVE CARDIOMYOPATHY

The pressure changes in restrictive cardiomyopathy can resemble those of constrictive pericarditis, although the LV diastolic pressure is usually appreciably higher than the right in restrictive cardiomyopathy. Diastolic pressure may, however, be coincidentally nearly identical in both ventricles. The dip-and-plateau pattern is often seen in restriction, but with the diastolic constraint in due to impaired ventricular relaxation rather than pericardial constraint.

RESTRICTIVE CARDIOMYOPATHY VERSUS CONSTRICTIVE PERICARDITIS

Differentiating between a restrictive cardiomyopathy and constrictive pericarditis may pose a diagnostic challenge. It is an important distinction to make as constrictive pericarditis is potentially curable by pericardiectomy. Comparison of the relative merits of the available diagnostic tests is limited by the lack of a gold standard investigation. Studies are typically non-randomised, with retrospective data collection, and therefore prone to bias.[46 47] Simultaneous right and left heart catheterisations are a common step on the diagnostic pathway, the results of which should be interpreted in conjunction with clinical, echocardiographic and CT/MRI findings in order to reach a final diagnosis. Diastolic pressure equalisation can also be seen in restrictive cardiomyopathy, as well as severe dilated cardiomyopathy, cardiac tamponade and advanced pulmonary disease, and should not be used to diagnose constriction in isolation.

A novel diagnostic marker uses respiratory variability in LV and RV pressures to distinguish between restrictive physiology and constrictive physiology.[48] The systolic area index measures the ratio of the area under the RV and LV systolic pressure curves in both inspiration and expiration. During inspiration, systemic venous return increases leading to an increase in RV volume. In constrictive pericarditis, because of ventricular interdependence (the total volume of the heart is constrained by the pericardium), there is a consequent fall in LV volume. Thus, there is an increase in the ratio between RV and LV volume (and pressure-area

Figure 6 Simultaneous left ventricular (LV) and right ventricular (RV) pressures taken from a patient with constrictive pericarditis are shown. Note the classical dip and plateau, or square root sign in diastole (SR), and equalisation of LV and RV end-diastolic pressures to within 5 mm Hg (star).

curve during inspiration compared with expiration). By contrast, in restrictive cardiomyopathy, because there is much less ventricular interdependence, there is no significant change in the LV pressure curve between inspiration and expiration, and thus the systolic area index is normal. A value for systolic area index greater than 1.1 has 97% sensitivity in detecting constriction. The measure has only been evaluated using high fidelity, micromanometer-tipped catheters in a small, selected patient group, and further studies are required to evaluate its usefulness fully.

CARDIAC TRANSPLANTATION

RHC is a key investigation in the work-up of patients being considered for cardiac transplantation. Elevated PVR is associated with an increased risk of post-transplantation RV failure, which leads to significant morbidity and mortality in the early post-transplant period.[49] Any degree of PH is associated with worse outcomes, and the International Society for Heart and Lung Transplantation

considers a PVR >5 Wood units and transpulmonary gradient >15 mm Hg as potential significant contraindications to transplantation. A vasodilator challenge is recommended when PA systolic pressure is >50 mm Hg and PVR is >3 Wood units, with a systolic blood pressure >85 mm Hg. It can be performed using a number of vasoactive drugs, including nitroglycerine, nitroprusside and inhaled nitric oxide although the value of the challenge in selecting patients for transplantation remains unproven.[3] If there is an inadequate response to an acute vasodilator challenge, a further assessment following a minimum of 24 h treatment with inotropes and diuretics is recommended, in an attempt to reduce PCWP, and consequently PVR. Reversible PH is still associated with a higher risk of RV failure in the donor heart following transplantation.[50]

LV assist device (LVAD) implantation in patients with secondary PH frequently leads to significant reductions in TPG and PVR within 1 month of implantation, and is therefore used as a bridge to candidacy for transplantation.[51] Right heart failure is a commonly encountered complication following LVAD implantation. Retrospective studies have found that a lower preoperative RV stroke work index (<600 g/m^2/beat) and mean PA pressure, both indicators of impaired RV contractility, are associated with a higher likelihood of RV failure requiring mechanical support.[52 53]

RHC should be performed prior to transplant listing and annually while the patient is actively

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Box 2   Haemodynamic criteria for diagnosing constrictive pericarditis

▶ Elevated right and left heart end-diastolic pressures equal within 5 mm Hg
▶ Mean right atrial pressure >15 mm Hg
▶ Right ventricular end-diastolic pressure greater than one-third of right ventricular systolic pressure

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listed, or every 3–6 months in those with reversible PH or deteriorating symptoms.[3]

CONGENITAL HEART DISEASE

Widespread availability and use of echocardiography, cardiac CT and MRI means that cardiac catheterisation is rarely required for the diagnosis of congenital cardiac abnormalities. The 2010 ESC guidelines for the management of grown-up congenital heart disease state that continuing indications for cardiac catheterisation include assessment of PVR, LV and RV diastolic function, pressure gradients and shunt quantification when non-invasive evaluation leaves uncertainty.[54] Standard assumptions used in RHC measurements, such as oxygen consumption and pulmonary and systemic blood flow may not be valid in complex cardiac lesions, such as cyanotic heart disease and multi-level shunts and should therefore be undertaken at specialist centres by physicians experienced in their interpretation. A complete review of the role of RHC in congenital heart disease is beyond the scope of this article.

MYOCARDIAL BIOPSY

RHC provides an access route for endomyocardial biopsy, which was first performed using cardiac bioptomes in 1962 by Konno and Sakakbira.[55] It is an important surveillance tool following cardiac transplantation to look for both cell-mediated and antibody-mediated rejection. It remains the gold standard investigation for diagnosing myocarditis and a range of infiltrative/storage disorders, including sarcoidosis and amyloidosis.[56] The procedure itself carries a risk of serious complications and should be performed when the findings are likely to have important implications for prognosis and management.

The 2007 Joint American Heart Association, American College of Cardiology and ESC guidelines on the role of endomyocardial biopsy provide an expert consensus opinion on its usefulness in 14 clinical scenarios (table 2).[56] The strongest recommendations are given for acute heart failure with haemodynamic compromise, and subacute heart failure with conduction system disease and/or ventricular arrhythmias. In both scenarios, it is important to differentiate between lymphocytic myocarditis due to a distinct viral illness, which carries a favourable prognosis, and conditions such as giant cell myocarditis (GCM), which has a mean transplant-free survival of less than 4 months.[57] A confirmed diagnosis will influence decision-making regarding mechanical circulatory support and cardiac transplantation. Patients with GCM and virus-negative myocarditis may respond to immunosuppressant therapy.[58 59]

Major complications of endomyocardial biopsy are predominantly due to cardiac perforation or arrhythmias. The risk of perforation is lower with fluoroscopic versus echocardiographic guidance. Complication rates from single-centre series range from <1% to 4%. A retrospective study of 3048 procedures performed at a high-volume centre (>50 cases/year/operator) found a perforation rate of 0.08% and risk of bradycardia requiring pacing of 0.04%, with no deaths.[60] A further disadvantage of cardiac biopsy is its low yield, particularly in the presence of focal disease processes such as tumours or infiltrative disorders. Echocardiography-guided biopsy may be useful in these situations, and will also enable the operator to guide the bioptome

Table 2	Recommendations for endomyocardial biopsy	
Clinical scenario	**Recommendation**
New-onset heart failure of <2 weeks duration associated with a normal-sized or dilated left ventricle (LV) and haemodynamic compromise	I
New-onset heart failure of 2 weeks to 3 months duration associated with a dilated LV and new ventricular arrhythmias, second-degree or third-degree heart block or failure to respond to usual care within 1–2 weeks	I
Heart failure of >3 months duration associated with a dilated LV and new ventricular arrhythmias, second-degree or third-degree heart block or failure to respond to usual care within 1–2 weeks	II
Heart failure associated with a dilated cardiomyopathy of any duration associated with suspected allergic reaction and/or eosinophilia	II
Heart failure associated with suspected anthracycline cardiomyopathy	II
Heart failure associated with unexplained restrictive cardiomyopathy	II
Suspected cardiac tumours	II
Unexplained cardiomyopathy in children	II
New-onset heart failure of 2 weeks to 3 months duration associated with a dilated LV, without new ventricular arrhythmias or second-degree or third-degree heart block that responds to usual care within 1–2 weeks	IIb
Heart failure of 3 months duration associated with a dilated LV, without new ventricular arrhythmias or second-degree or third-degree heart block that responds to usual care within 1–2 weeks	IIb
Heart failure associated with unexplained hypertrophic cardiomyopathy	IIb
Suspected ARVD/C	IIb
Unexplained ventricular arrhythmias	IIb
Unexplained atrial fibrillation	III

Table 2 is taken from the a scientific statement by the American Heart Association, American College of Cardiology, and European Society of Cardiology on the role of endomyocardial biopsy in cardiovascular disease.[50]
Levels of evidence: I—evidence or general agreement that the procedure is beneficial, useful and effective; IIa—weight of evidence/opinion in favour of its usefulness/efficacy; IIb—weight of evidence/opinion less well established; III—evidence or general agreement that a procedure is not effective or harmful.

Key messages

▸ Meticulous attention to detail is required when preparing equipment and recording pressure traces to ensure that measurements are accurate and reproducible.

▸ Right heart catheterisation (RHC) is the gold standard investigation for diagnosing pulmonary hypertension and provides important prognostic information.

▸ RHC can be very helpful in differentiating between constrictive pericarditis and restrictive cardiomyopathy; the findings should be interpreted in conjunction with clinical and non-invasive imaging data

▸ RHC is an essential investigation when determining eligibility for cardiac transplantation or ventricular assist device therapy. Any degree of pulmonary hypertension is associated with poorer outcomes.

▸ Endomyocardial biopsy should be performed by experienced operators and analysed by appropriately trained cardiac pathologists with access to a full complement of pathological investigations.

toward the septum, to reduce the risk of perforation. Cardiac MRI-guided biopsy may reduce the risk of false-negative results.[61]

A 2011 consensus statement from the Association of European Cardiovascular Pathology and Society for Cardiovascular Pathology recommend that endomyocardial biopsy should be performed with the involvement of referral centres in which the whole armamentarium of pathological investigations are available. Pathologists should be suitably trained in cardiac pathology and pathophysiology, and correlations between the pathological and clinical findings should be made as a multidisciplinary team.[56]

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FUTURE DIRECTIONS

Remote PA pressure measurement is now possible using devices such as the CardioMEMs PA sensor (St Jude Healthcare). The device is implanted into a distal PA via RHC, and pressure measurements are sent wirelessly to a remote monitor. The CHAMPION study was a randomised controlled trial to evaluate the role of PA pressure monitoring to guide management of patients with advanced chronic heart failure. There was a 37% reduction in the primary end point of heart failure hospitalisations at a mean follow-up of 15.6 months. The device was safe with a low rate of complications.[62] It has received Food and Drug Administration approval for use in patients with heart failure with reduced EF, New York Heart Association III symptoms and a recent heart failure hospitalisation.

CONCLUSION

RHC retains an important place in the diagnosis and management of a broad range of cardiovascular disorders. In practice, the diminishing use of RHC makes it difficult for all trainees to become fully proficient in their conduct and interpretation. It is vital, however, that those with particular subspeciality interests, including advanced heart failure, PH and congenital heart disease, receive appropriate training from experienced operators, in order to preserve the art of conducting a thorough, accurate haemodynamic assessment.

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Right heart catheterisation: indications and interpretation

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