Physiological Relevance of Right Heart Catheterization

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Hemodynamics is a branch of cardiology concerned with how blood flows through the heart and vessels. The determinants of such information are oximetry readings and intravascular pressure data; yet proper interpretation relies on understanding the patient's cardiopulmonary physiology, including the pathophysiological properties associated with the right ventricle (RV).[1] Blood tests, exercise testing, and right heart catheterizations (RHCs) are tools cardiologists use to assess the overall health of the cardiovascular system. Fluoroscopy-guided RHC procedures in the cardiac catheterization laboratory are used for evaluating RV function as well as dyspnea (shortness of breath). Because oxygen is necessary to produce adenosine triphosphate, the molecule that stores and releases chemical energy to fuel cellular processes, dyspnea is associated with setbacks in arterial oxygen delivery.[2,3]

Understanding the role and practice of RHCs in the evaluation of dyspnea, including its differential diagnoses and markers of cardiopulmonary functioning, is critical. Cardiac interventional technologists who have knowledge of the processes surrounding right heart evaluations can better communicate with patients before and after an RHC procedure. A proper interpretation of clinical findings demands a simple understanding of physiology and the associated mathematics, including oxygen delivery, cardiac output, and stroke volume.

Parameters Defining Oxygen Delivery

Four parameters define the delivery of oxygen through the arterial system, as managed via heart and lung functions[2-4]:

- cardiac output value
- concentration of serum hemoglobin and the oxygen-binding capacity of hemoglobin
- level of dissolved oxygen gas in arterial blood plasma
- oxygen saturation throughout the arterial system

Cardiac output is expressed in units of liters per minute (L/min) and is obtained by exercise ergometry or by RHC procedures performed independently or in combination with left heart catheterizations (LHCs). Typical resting values are 4.0 to 8.0 L/min, with exercise levels for sedentary individuals increasing 4-fold to approximately 20.0 to 25.0 L/min.[3,5] For comparison, typical exercising cardiac output levels for individuals who are endurance athletes (eg, runners) might rise as high as 32.0 to 40.0 L/min.[6-8] With physical activity, oxygen extraction improves, and the heart's stroke volume is greater than 65 mL.[6] Stroke volume is defined by subtracting ventricular end-systolic volumes from end-diastolic valuations. Oxygen extraction improves per the oxygen-hemoglobin dissociation curve relative to increased body temperature and decreased blood pH (or increased blood acidity values). Also, oxygen consumption increases from an average of 250.0 to 5000.0 mL/min.[9-11]

Obtained via blood tests, hemoglobin levels are recorded in units of grams of hemoglobin per deciliter (g Hb/dL). In general, levels should be greater than 12 g Hb/dL. Typical values are 12.1 to 15.1 g Hb/dL for women and 13.8 to 17.2 g Hb/dL for men.[12] Concerning the oxygen-binding capacity of hemoglobin, this number is a constant equal to 1.39 because each unit of hemoglobin carries 1.39 milliliters of oxygen (O_2 mL/g Hb).[2-4]

The fractional amount for oxygen dissolved in arterial blood plasma in gas form is 0.003 or 0.3%. This parameter marks oxygen delivery as a fractional component of the realized partial pressure of oxygen (PaO_2) in the arterial system. Whereas PaO_2 is determined through oximetry, the fractional value concerning the plasma component of arterial blood is expressed $0.003 \times PaO_2$.[2,13] Therefore, with Q signifying cardiac output, the formula for arterial oxygen delivery is[2]:

$$(Hb \times 1.39 \times Q \times SaO_2) + (0.003 \times PaO_2)$$

This formula accounts for the oxygen carried by hemoglobin in red blood cells and the amount of oxygen dissolved in plasma as a gas.[2]

The oxygen saturation fraction for arterial blood (SaO_2) is found using hemoximetry or pulse oximetry and entered as a decimal number. The test should indicate an SaO_2 level of 0.95 or greater, which means oxygen saturation is 95% or greater.[13,14] Moreover, oxygen content remains the same throughout the arterial system, which implies blood samples for SaO_2 values can be obtained at any arterial site during left heart evaluations, such as from the radial- or femoral-arterial access sites or from the tip of the left heart catheter at the aortic bulb. Pulse oximetry–derived SaO_2 values might be used, however, when conducting RHCs without a subsequent left heart procedure.

Finding Cardiac Output

Clinicians need a distinct method that yields the cardiac output value. This involves dividing oxygen consumption by oxygen content[4]:

$$Q = \frac{VO_2}{Ca - Cv}$$

In the formula, VO_2 represents oxygen consumption in milliliters per minute (or O_2 mL/min); Ca is the oxygen content found in the arterial system; and Cv represents the oxygen content found in the venous system, which is the critical element for accuracy and clinical significance.[4] Created by German physiologist Adolf Fick in 1870, this calculation is called the *Fick principle*.[15]

The units for oxygen content are milliliters of oxygen per liter of blood (O_2 mL/L). Blood samples required for marking venous oxygen saturation should be drawn from the mixed venous return (MVO_2), which is best observed in the pulmonary artery (PA).[4] For example, with a resting MVO_2 of 75% in the PA and a hemoglobin value of 16 g Hb/dL, venous oxygen content is derived:

$$Cv = 0.75 \times 16 \frac{g\,Hb}{dL} \times 13.9 \frac{O_2\,mL}{g\,Hb} \left(\frac{dL}{L}\right) = 166.8 \frac{O_2\,mL}{L}$$

Solving this formula involved the common practice of simplifying it by multiplying the binding capacity of hemoglobin, 1.39 O_2 mL/g Hb, with a 10 dL/L conversion factor to render 13.9 O_2 mL/g Hb (dL/L).

The process to find arterial oxygen content is identical. With an SaO_2 of 99% at rest and a hemoglobin value of 16 g Hb/dL, the resulting saturated oxygen amount is:

$$Ca = 0.99 \times 16 \frac{g\,Hb}{dL} \times 13.9 \frac{O_2\,mL}{g\,Hb} \left(\frac{dL}{L}\right) = 220.2 \frac{O_2\,mL}{L}$$

Therefore, oxygen content (ie, Ca – Cv) is 53.4 O_2 mL/L.

Comparing both equations, the differences are use of mixed venous saturation in the first formula and arterial saturation in the other. Thus, the equations can be combined:

$$(SaO_2 - MVO_2) \times Hb \frac{g\,Hb}{dL} \times 13.9 \frac{O_2\,mL}{g\,Hb} \left(\frac{dL}{L}\right)$$

The name for the term $SaO_2 - MVO_2$ is arteriovenous oxygen difference (A-VO_2). If resting SaO_2 remains at 99% and resting MVO_2 remains at 75%, the arteriovenous oxygen difference is 24%. Naturally, this expression follows:

$$(0.24) \times 16 \frac{g\,Hb}{dL} \times 13.9 \frac{O_2\,mL}{g\,Hb} \left(\frac{dL}{L}\right)$$

Once again, oxygen content (ie, Ca – Cv) is 53.4 O_2 mL/L.

Using the Fick principle for cardiac output (and echoing the value for Ca – Cv), if rough estimates for oxygen consumption are 234.0 O_2 mL/min relative to the heart's rate and stroke volume, then cardiac output equals 4.4 L/min:

$$Q = \frac{234.0 \frac{O_2\,mL}{min}}{53.4 \frac{O_2\,mL}{L}} = 234.0 \frac{\cancel{O_2\,mL}}{min} \times 53.4 \frac{L}{\cancel{O_2\,mL}} = 4.4 \frac{L}{min}$$

Thus, the left and right sides of the heart are balancing the patient's resting oxygen demands by supplying the systemic and pulmonary circulation each with 4.4 liters of blood every minute.

Calculating Oxygen Consumption

In the Fick formula, oxygen consumption was 234.0 O_2 mL/min. In cardiac catheterization laboratories, oxygen consumption is dependent on 2 factors. The key factor is choosing the appropriate indexed rate[4,16,17]:

- 110.0 O_2 mL/min/m^2 – older or obese patients
- 121.0 O_2 mL/min/m^2 – adult sedated patient (with an average valuation or standard reference of 126 O_2 mL/min/m^2)
- 132.0 O_2 mL/min/m^2 – adult nonsedated patient (with an average valuation or standard reference of 126 O_2 mL/min/m^2)
- weight-based approach – takes 125.0 O_2 mL/min/m^2 × 3 and divides the outcome by patient weight in kilograms, rendering units of O_2 mL/kg/min

According to Chase, not only was a standard reference of 126.0 O_2 mL/min/m^2 first reported by Dehmer, Firth, and Hillis in 1982, but, importantly, the practice of rounding down this referenced rate to 125.0 O_2 mL/min/m^2 for an assumed valuation during RHCs stems directly from their results.[17] Moreover, when such data points are entered to produce Fick outcomes in the absence of spirometry, the associated result is called *assumed Fick*.[4] True Fick results occur only when spirometry is performed.

The second factor acknowledges body surface area (BSA) in units of meters squared (m^2). To find BSA, clinicians take the square root of weight times height (in kilograms [kg] and centimeters [cm], respectively) divided by 3600:

$$BSA = \sqrt{\frac{(kg \times cm)}{3600}}$$

For example, if patient weight is 74.1 kg and patient height is 170.2 cm, then BSA is 1.87 m^2.

With both factors appreciated, oxygen consumption (VO_2) rendered in milliliters per minute (O_2 mL/min) is found by multiplying the indexed value by the patient's BSA:

$$VO_2 = standard\ reference \times BSA$$

$$= \frac{125.0 \frac{O_2\,mL}{min}}{m^2} \times 1.87 m^2$$

$$= 234.0 \frac{O_2\,mL}{min}$$

Finally, dividing the outcome by the blood oxygen content worked out earlier restates cardiac output as 4.4 L/min.

Right Heart Catheterization

After ruling out drug interactions as possible causes of dyspnea, physicians look for the most straightforward reasons for depressed oxygen delivery, including low hemoglobin values, hemoglobinopathies, toxicities affecting hemoglobin, and factors affecting cardiac output such as arrhythmias, myocardial infarction, and congestive heart failure. For example, if low hemoglobin values are revealed by laboratory tests, the physician can order a transfusion of packed red blood cells. However, if the blood tests are within optimal range, then cardiopulmonary exercise testing is recommended.[18]

When exercise tests are negative for cardiopulmonary etiologies, dyspnea's root cause is likely deconditioning problems.[2,18,19] For dyspnea that persists after a beginner-to-intermediate conditioning program, however, a differential diagnosis might consider these disease states[2,4,18,19]:

- interstitial lung disease
- myocardial dysfunction
- obesity
- obstructive pulmonary disease
- other cardiopulmonary considerations (eg, heart valve disease vs restrictive or constrictive heart disease, pulmonary arterial hypertension [PAH])

RHCs and the associated hemodynamic data help exclude many of these diseases and give interventional

cardiologists, as well as referring physicians, information to guide further assessment and treatment planning such as:

- heart failure care and remote monitoring
- left-right and right-left shunt detection
- pulmonary disease diagnoses
- valvular disease and restrictive vs constrictive heart disease

If an RHC is carried out in combination with an LHC procedure that includes ventriculography, data obtained from the ventriculogram distinguish angiographic cardiac output and stroke volume compared to RHC-based forward flow calculations. The added information lets the interventionalist or referring physician better target guidelines for an individualized exercise prescription, including physical capacity recommendations. In general, these findings supplement pharmacological or nonpharmacological stress testing results and allow predictions of cardiac and pulmonary rehabilitation patient participation and outcomes.[20]

Heart Failure Care and Remote Monitoring

The CardioMEMS HF System (Abbott), a recent addition to the cardiac catheterization laboratory, allows remote monitoring of heart failure severity after implantation in the PA (preferably the left PA) during an RHC procedure. The U.S. Food and Drug Administration's premarket approval letter asserts, "This device is indicated for wirelessly measuring and monitoring PA pressure and heart rate in New York Heart Association (NYHA) Class III heart failure patients who have been hospitalized for heart failure in the previous year."[21] The acquired hemodynamic data is used clinically to help physicians manage heart failure, with the goal of reducing repeat heart failure–related hospitalizations.

Cardiac Output Parameters, Heart Failure, and Exercise

Demands on heart rate rise during exercise, as do demands levied on stroke volume relative to enhanced ventricular filling and increased contractility from intensifying preload and myocardial wall stretch via the Frank-Starling principle. Adhering to a training effect, the heart first performs less work during resting states and eventually when exercising at submaximal levels.[6] For instance, in a conditioned heart the rate will take longer to rise during exercise and will return to baseline sooner.

Conditioning programs must be well planned for patients with heart failure, and exercise intensity must be kept at the minimum level (eg, $\leq$ 60%-70% of maximum heart rate). The reasons center on the pathophysiological presentation of heart failure; these hearts gradually contain fewer healthy, active myocardiocytes.[13] The risk is that the remaining healthy myocardiocytes might become fibrotic faster through a persisting feedback loop in terms of diminished myocardial efficiency. The loop persists because higher cellular workloads are placed on the remaining heart muscle cells (ie, those currently vulnerable to damage from fibrotic scarring) in challenging states for receiving adequate blood flow and oxygen during exercise relative to previously damaged myocardium (ie, cells that are scarred and contribute to a growing collection of fibrotic heart tissue).[13] Therefore, the functional gains stemming from the training effect in heart failure primarily arise from improving efficiencies in the mechanisms responsible for oxygen exchange at the cellular level in the tissues, which lowers systemic afterload.[22]

Although peak oxygen consumption (rendered now in units of milliliters per kilogram per minute [O_2 mL/kg/min]) poorly correlates with hemodynamic parameters at rest, it correlates well with cardiac output during exercise.[23] Moreover, inadequate cardiac output is historically a primary determinant of physical capacity in asymptomatic or mildly symptomatic heart failure. Conversely, maximal pulmonary wedge pressure does not correlate with peak oxygen consumption.[23] For untrained patients with moderate-to-severe heart failure, skeletal muscle vasodilation (a prime factor affecting afterload) is limited to 50% to 60% compared with 90% in healthy individuals.[23] Such data underscore the complex mechanisms between pulmonary circulation and physical capacity.

Heart failure might be classified into 5 ascending categories of severity based on descending peak oxygen consumption valuations[24]:

- > 20.0 O_2 mL/kg/min – none to mild
- 16.0 to 20.0 O_2 mL/kg/min – mild to moderate
- 10.0 to 16.0 O_2 mL/kg/min – moderate to severe

- 6.0 to 10.0 O_2 mL/kg/min – severe
- < 6.0 O_2 mL/kg/min – very severe

Morimoto et al concluded that 5-year mortality rates were lowest for heart failure patients maintaining peak oxygen consumption at 18.0 O_2 mL/kg/min or greater.[25] Nadruz et al concluded that heart failure–related hospitalizations rose with peak oxygen consumption values of 14 mL/kg/min or less.[26] Swank et al showed that, at 3 months, patients with heart failure who received standard care along with a disease-specific exercise rehabilitation program benefitted from a modest increase in peak oxygen consumption compared with patients receiving standard care only (0.6 O_2 mL/kg/min vs 0.2 O_2 mL/kg/min, respectively).[27] No studies, however, compare the effects of standard care or exercise with the recent addition of remote monitoring of PA pressure changes.

Left-Right and Right-Left Shunt Detection

The circulatory system is a closed system. A resting cardiac output of 4.4 L/min means that 4.4 liters of blood get pumped through the systemic circulation per minute, as well as simultaneously through the pulmonary circulation.[4] Structural heart abnormalities can disrupt blood flow through the heart, leading to imbalances in cardiac output between the systemic and pulmonary circuits. For example, a patent foramen ovale that persists into childhood or adulthood is called an atrial septal defect (see **Figure 1**). Although in infants and children these openings favor right-to-left flow because of typically higher pressures in the pulmonary system, such shunting favors left-to-right flow in adults because left heart and systemic pressures are higher.[4] Similarly, in adults, ventricular septal defects tend to favor left-to-right shunt flow.

With respect to acquiring blood oxygen saturation samples from the superior vena cava (SVC) and the PA, the RHC procedure screens for left-right and right-left shunt defects. Samples having differences of 7% are typical, whereas samples with differences greater than 7% (known as a *7% step up*) suggest the presence of a left-to-right shunt. In such cases, a shunt run is performed to determine significance. A step up greater than 9% is suggestive of a large left-to-right shunt.[28,29] A right-to-left shunt is suspected when desaturated arterial blood does not correct with 100% supplemental oxygen.[28]

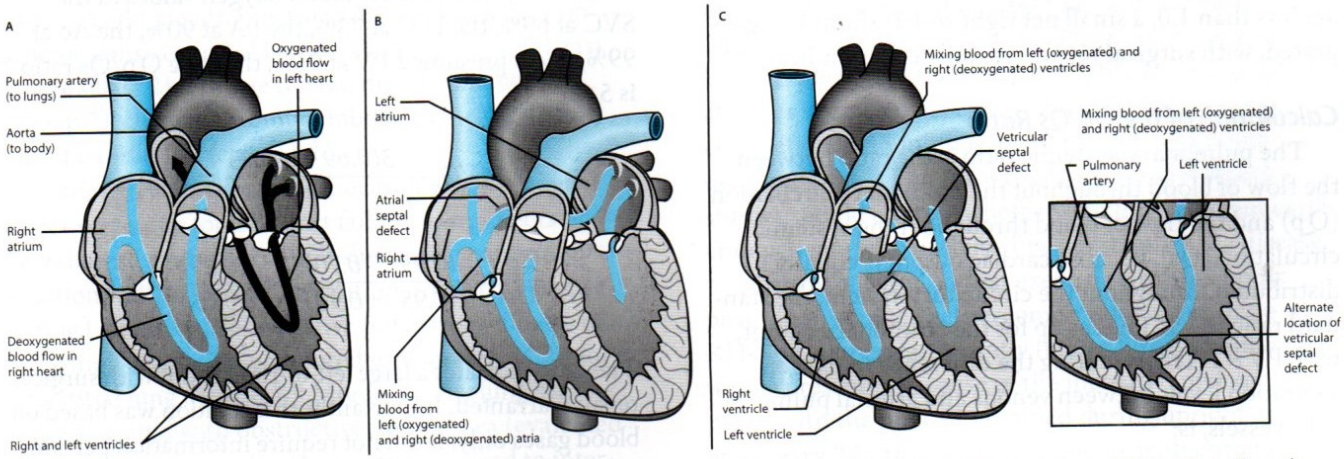

Figure 1. *A. A healthy adult heart with proper blood flow. B. Heart with atrial septal defect (ASD). An ASD is a structural defect that allows fractions of the flow of blood to be shunted from the left atrium (LA) to the right atrium (RA). As a result of this abnormal circulation (ie, a patent atrial left-to-right shunt), some oxygenated blood from the LA mixes directly with the deoxygenated blood in the RA, causing a persistent re-entry loop back to the pulmonary circulation. Unless the ASD is treated, cardiac output remains elevated in the pulmonary circuit compared with the systemic circuit. C. Hearts with ventricular septal defects (VSD). A VSD allows fractions of the flow of blood to be shunted from the left ventricle (LV) to the right ventricle (RV). As a result of this abnormal circulation (ie, a patent ventricular left-to-right shunt), some oxygenated blood from the LV mixes directly with the deoxygenated blood in the RV, causing a persistent re-entry loop back to the pulmonary circulation. Unless the VSD is treated, cardiac output remains elevated in the pulmonary circuit compared with the systemic circuit.*

Shunt Run

During a shunt run, additional blood samples are taken to assess oxygen saturation levels in the inferior vena cava (IVC), the upper right atrium (RA), the middle RA, the lower RA, the RV, the remaining PA, and the pulmonary wedge as a surrogate for the left atrium. Pulmonary vein (PV) saturation is presumed to be 95% when not sampled.[29] Also, for the run, the PA value no longer represents mixed venous oxygen content (or saturation, MVO_2) for the systemic circulation.[29] Instead, saturation values associated with the SVC and the IVC are plugged into the Flamm equation, as follows[29,30]:

$$MVO_2 = \frac{3(SVC) + IVC}{4}$$

The data from a shunt run provide interventionalists with a clearer picture of the clinical situation by permitting calculation of the pulmonary-systemic ratio (ie, the Qp/Qs ratio). Results indicate a small left-to-right shunt if the ratio is less than 1.5, an intermediate left-to-right shunt when ratios fall between 1.5 and 2.0 (with surgical correction justified), and a large left-to-right shunt when the ratio is greater than 2.0 (with surgical correction warranted).[4] When ratios are less than 1.0, a small net right-to-left shunt is suggested, with surgical repair typically contraindicated.[4]

Calculation of the Qp/Qs Ratio

The pulmonary-systemic ratio is the ratio between the flow of blood throughout the pulmonary circulation (Qp) and the flow of blood throughout the systemic circulation (Qs). Because cardiac output is equally distributed throughout the circulatory system, the standard ratio is 1:1. The set-up for the pulmonary circuit, with PV – PA representing the difference in oxygen saturation levels between venous and arterial pulmonary vessels, is:

$$Qp = \frac{O_2\ consumption}{(PV - PA)}$$

Similarly, with Ao – MVO_2 representing the difference in oxygen saturation from the aorta (Ao) to the mixed venous supply, the set-up for the systemic circuit is:

$$Qs = \frac{O_2\ consumption}{(Ao - MVO_2)}$$

A ratio can be produced using the left side of the above 2 equations, creating:

$$\frac{Qp}{Qs} = 1$$

The ratio for the right side of the same 2 equations is:

$$\frac{\left(\frac{O_2\ consumption}{(PV - PA)}\right)}{\left(\frac{O_2\ consumption}{(Ao - MvO_2)}\right)}$$

Like variables can be eliminated, forming:

$$\frac{\cancel{O_2\ consumption}}{(PV - PA)} \times \frac{(Ao - MVO_2)}{\cancel{O_2\ consumption}}$$

Thus, the pulmonary-systemic ratio is:

$$\frac{Qp}{Qs} = \frac{(Ao - MVO_2)}{(PV - PA)} = 1$$

If a shunt run collects blood oxygen values in the SVC at 69%, the IVC at 73%, the PA at 90%, the Ao at 99%, and a presumed PV at 95%, then the Qp/Qs ratio is 5.8:

$$MVO_2 = \frac{3(0.69) + 0.73}{4} = 0.70$$

$$\frac{Qp}{Qs} = \frac{(0.99 - 0.70)}{(0.95 - 0.90)} = \frac{(0.29)}{(0.05)} = 5.8$$

The ratio indicates a large left-to-right shunt with surgical repair warranted. Importantly, this solution was based on blood gases only; it did not require information pertaining to hemoglobin values or cardiac output valuations.

Cardiac Output Parameters and Left-to-Right Intracardiac Shunting

With a left-to-right shunt, the shunt flow is the cardiac output of the pulmonary circuit minus the cardiac output of the systemic circuit (Qp – Qs). For example,

inserting the oxygen consumption level of 234.0 O_2 mL/min computed earlier, the appropriate A-VO_2 difference, and a hemoglobin level of 16 g Hb/dL with the oxygen-binding 1.39 constant multiplied by the 10-point conversion factor, the cardiac output for the pulmonary circuit (Qp), calculated in final units to save space, is:

$$\frac{234.0}{(0.95 - 0.90) \times 16 \times 13.9} = 21.5 \frac{L}{min}$$

Using the remaining A-VO_2 difference, the cardiac output for the systemic circuit (Qs) is:

$$\frac{234.0}{(0.99 - 0.70) \times 16 \times 13.9} = 3.63 \frac{L}{min}$$

Therefore, computing Qp – Qs, the flow of blood through the shunt is 17.9 L/min.

Pulmonary Disease Diagnoses

Five classifications of disorders with various pathophysiological processes that share a common denominator make up the different families of pulmonary hypertension (PH). The common denominator is a mean PA pressure greater than 25 mm Hg.[31] Devised by the World Health Organization, the classification scheme consists of PAH (class 1), PH due to left heart failure (class 2), PH due to lung disease and hypoxia (group 3), chronic thromboembolic PH (class 4), and multifactorial PH (class 5).[31-33]

Although transthoracic echocardiography is helpful for screening for left heart failure, assessments of lung disease are best accomplished using pulmonary function testing (including cardiopulmonary exercise testing) for chronic obstructive pulmonary disease or using high-resolution computed tomography (CT) for interstitial lung disease.[34,35] Moreover, any lung scarring associated with obstructive sleep apnea (evaluated using sleep apnea studies) can eventually lead to interstitial lung disease.[36]

Nuclear medicine lung ventilation perfusion scans, known as *VQ scans*, employ inert gases or radiolabeled aerosols to test ventilation (V) and injectable radionuclides to detect and stage the chronicity of thromboembolism through perfusion (Q).[37,38] The inert gases of choice are krypton Kr 81m and xenon Xe 133; the radiolabeled aerosols use technetium Tc 99.[37] The best choices for injectable radionuclides take advantage of the imaging-friendly properties of technetium Tc 99m.[37,38] As opposed to high-resolution CT, chronic thromboembolism can be detected readily using CT with a pulmonary embolism protocol.[39]

Benefits of Exercise RHC protocols

Given that the resting RHC is the gold standard test required to confirm PAH, this disease is defined by pulmonary arterial pressure greater than 25.0 mm Hg at rest or greater than 30.0 mm Hg with physical activity.[32,40] Confirmation is aided by ensuring that pulmonary wedge pressures are less than 15 mm Hg.[32] To determine exercise-related PA pressures, an exercising RHC protocol might be performed in the cardiac catheterization lab.[4] Keusch et al found that patients who consented to resting and exercising RHC protocols and experienced resting PA pressures ranging from 20.0 to 24.9 mm Hg had PA values greater than 30.0 mm Hg when exercising (ie, exercise PH), and that exercise PH might be a precursor for resting PH in older patients.[33] In this study, exercise during RHC was carried out in a stepwise, incremental fashion (starting at 10 watts or 60 revolutions per minute [60 rpm] and increasing by 3 watts each 3 minutes) using supine cycle ergometry. For this portion of the study, the RHC was terminated at either symptom onset (eg, exertional dyspnea) or completion of 15 minutes of exercising, whichever occurred first.[33] The authors concluded that future studies should address whether starting treatment earlier in patients with exercise PH can stabilize the disease.

Mizumi et al found that exercising-related body position in supine cycle ergometry during exercise RHC promotes significantly higher PA and pulmonary wedge pressures because of the increased preload compared with the preload obtained during upright cycle ergometry.[41] In this study, 17 patients with chronic thromboembolic PH who were treated by percutaneous transluminal pulmonary angioplasty 6 months earlier with near-normal peak PA pressures (< 30 mm Hg at rest) at the time of the RHC experienced mean peak PA pressures at 45 ± 7 vs 40 ± 11 mm Hg during supine vs upright testing. Also, mean peak pulmonary

wedge pressures at 17 ± 4 vs 11 ± 7 mm Hg during supine vs upright testing were recorded for these same patients. Moreover, body position had no influence on pulmonary circulation or peak oxygen consumption.[41] The protocol began with the ergometers at 10 watts (60 rpm) and employed increases of 1 watt every 6 seconds (10 W/min).

In addition to enlisting the cycle ergometer (a form of dynamic exercise), Hanna and Glancy described the benefits of supine RHC-based isometric exercise testing via sustained handgrip tests to reveal left heart failure (ie, left ventricle [LV] dysfunction).[4] Their suggested protocol entails use of a handgrip ergometer to maintain a handgrip at 50% of voluntary maximal capacity sustained for more than 3 minutes or maintaining a handgrip of 75% maximal capacity sustained for more than 1 minute.[4] Patients should not perform the Valsalva maneuver or, because of reduced preload, hold their breath. The authors also corroborated fluid overloading via leg raising, as well as fast atrial pacing for patients with permanent pacemakers, to unmask increases in LV end-diastolic pressure and pulmonary wedge pressure (the effects for patients with pacemakers were observed postpacing).[4]

Valvular Disease and Restrictive vs Constrictive Heart Disease

The Gorlin formula for assessing pulmonic stenosis or tricuspid stenosis must be applied cautiously because the formula has not been validated for these valves.[42] The proper constants have not been identified. Therefore, to evaluate pulmonic or tricuspid stenosis or laxity, special attention is placed on interpretation of pressure tracings. For similar reasons, scrutiny of pressure tracings is used to evaluate restrictive cardiomyopathy vs constrictive pericarditis.

Pressure Waveforms

Waveforms associated with the RA and pulmonary wedge are double humped and squiggly, with mean pressures of 5 mm Hg and 10 mm Hg, respectively (see **Figure 2**). In addition, the pulmonary wedge pressure serves as a surrogate to record the mean pressure for the left atrium. With pressures measuring 25/0 mm Hg, the waveforms of the RV are tall with peaks that correspond to the height of systole then bottom out. In the PA, triangular-shaped waveforms are present with a dicrotic notch representing pulmonic semilunar valve closure, with pressures of 25/10 mm Hg. Mean pulmonary wedge pressure approximates LV end-diastolic pressure, which is an indicator of how well the LV is filling. Likewise, RV end-diastolic pressure indicates how well the RV is filling.

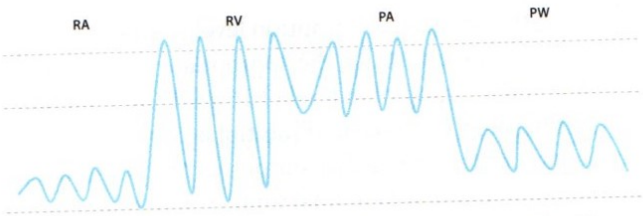

Figure 2. *Generalized pattern for pressure waveforms in a healthy heart. Abbreviations: PA, pulmonary artery; PW, pulmonary wedge. Figure courtesy of the author.*

Valvular Disease

Assessments of pulmonic semilunar and tricuspid valve integrity are routine aspects of RHC procedures. For pulmonic valve stenosis, pullbacks of the right heart catheter from the PA to the RV suffice.[42] A peak-to-peak gradient greater than 30 mm Hg justifies surgical repair in symptomatic patients; surgical consultation is indicated for asymptomatic patients with a peak-to-peak gradient greater than 40 mm Hg.[4] Although asymptomatic, isolated stenosis of this valve occurs in only 8 to 10 out of 100 patients with congenital heart disease, symptomatic stenosis includes exertional dyspnea.[43,44] Angina or cardiac arrest is rare.

Catheter pullbacks from the RV to the RA sufficiently screen for tricuspid stenosis.[42] However, a suitable assessment of tricuspid stenosis requires pressure tracings from the RV and a simultaneous pressure reading from the RA via a separately introduced catheter. Severe tricuspid stenosis is rare but is characterized by a mean diastolic gradient greater than 5 mm Hg.[4] Exertional dyspnea is a common complaint.[45]

Pulmonic insufficiency (or regurgitation) also is rare but is observed most often with endocarditis of this valve or after surgical repair of tetralogy of Fallot. It also might occur secondary to PH.[46] The associated

waveform mimics the RV waveform (ie, the dicrotic notch disappears because of ventricularization of the PA tracing).[4] Although symptomatic pulmonary regurgitation initially presents with exertional dyspnea associated with reduced cardiac output from volume overload to the right heart, worsening cases (untreated, refractory, or nonidentified cases) can lead to right heart failure.[46]

Tricuspid regurgitation can occur as functionally secondary to RV pressure or volume overload, such as that ensuing from a left-to-right shunt.[4] Ventricularization of the RA waveform frequently occurs, yet the regurgitant volume increases with inspiration, which introduces tracings characterized by pronounced V waves and Y descents.[4] Symptoms are consistent with those of right-sided heart failure, such as internal jugular distention (along with the Kussmaul sign) and body edema.[47]

Restrictive vs Constrictive Heart Disease

In the healthy heart, pressure tracings in the RV with a second catheter placed simultaneously in the LV should demonstrate concordant behavior. In other words, tracings of RV and LV systolic waveforms, as well as those of the Ao and PA, will all drop minimally and proportionately on respiratory inspiration because of negative intrathoracic pressure influencing all cardiac and vascular recordings.[4] Moreover, end-diastolic pressures are healthy in either ventricular chamber. Such phenomena are absent in cases of restrictive cardiomyopathy or constrictive pericarditis. RHC procedures performed with LHCs help distinguish between these 2 diseases.

Although restrictive cardiomyopathy is a diastolic-based disease (with a diastolic filling defect) and constrictive pericarditis is a systolic-based disease state, both conditions possess a telltale diastolic filling sign observed during hemodynamic measurements. This diastolic defect is symbolically called the *dip-and-plateau sign* because ventricular filling observed throughout isovolumetric relaxation for these conditions first occurs with rapid onset with the associated hemodynamic pressure data dipping quickly followed by a sharp rise from phasic resistance of continued filling associated with a lack of myocardial pliability. An eventual plateau on the hemodynamic tracing then occurs; again, due to a lack of myocardial pliability.[4] This phasic progression, or stepwise sequence, collectively takes on the shape of a square root sign, which is an alternative name for the dip-and-plateau description.[48]

To distinguish between restrictive cardiomyopathy and constrictive pericarditis, the former's pressure waveforms depicting the RV and the LV demonstrate concordance with inspiration, whereas the latter's associated waveforms display discordant behavior. Discordance is represented during inspiration by disproportionate increases in the RV's systolic tracings together with simultaneous decreases of the LV's tracings at peak systole (known as *ventricular interdependence*).[4,48,49] This associated filling discordance marking constrictive pericarditis not only overwhelms the direct negative inspiratory pressure effect during presentation but engulfs pressure recordings in cases of chronic obstructive pulmonary disease and right heart failure. The hallmark of constrictive pericarditis, however, is a uniform increase in all chambers of the heart by 5 mm Hg.[48]

When left untreated, constrictive pericarditis and restrictive cardiomyopathy commonly lead to right heart failure. Exertional dyspnea can be an initial symptom because of volume overloads associated with weight gain or relative to decreased cardiac output. Furthermore, jugular distention, the Kussmaul sign, and diastolic heart sounds are common symptoms in both conditions. However, jugular pressure (and its distention) does not drop (nor does its associated diminishing of the Kussmaul sign during inspiration) under the constrictive physiology of constrictive pericarditis.[49,50]

Importance of Determining Cardiac Index

In addition to evaluating cardiac output, many clinicians prefer valuations of cardiac index, which normalize cardiac function to the patient's body habitus.[51] With a typical range of 2.5 to 4.0 L/min/m^2, the index is found by dividing cardiac output by the patient's BSA, such that cardiac index equals Q divided by BSA.[5,51] Employing the earlier examples in which a cardiac output value of 4.4 L/min was derived and a BSA of 1.87 m^2 was calculated, cardiac index is:

$$\frac{4.4\ L/min}{1.87\ m^2} = 2.35\ L/min/m^2$$

Carlsson et al found that cardiac index decreases with age by 8 mL/min/m^2 per year, but that no differences existed between healthy individuals and athletes.[52] Furthermore, cardiac index valuations by themselves were not found to be useful indicators of heart failure because values for patients with symptomatic heart failure are within typical limits nearly 50% of the time.[52]

Conversely, when cardiac index is coupled with RA pressures derived by RHC procedures, complementary indices exist with which to best determine heart failure status. Whereas cardiac index is a constraint reflecting right and left heart functions, as well as a parameter that depends on systolic and diastolic ventricular functions, RA pressure indexes compensate for dysfunction related to right and left heart anatomy and physiology.[53] Stroke volume corrected by BSA can be higher in men compared with women.[53,54]

Conclusion

Oximetry readings and intravascular pressure data are instrumental in the study of hemodynamics, the branch of cardiology that examines how efficiently blood flows through the cardiovascular system, including the cardiopulmonary circulation. Cardiac output might be computed using the Fick formula. Cardiac index is cardiac output correlated to BSA, which normalizes cardiac function to body habitus. As complementary indices, cardiac output, cardiac index, and RA pressure can help interventionalists differentiate dyspnea, heart failure, shunt formations, PH, and valvular disease vs constrictive pericarditis.

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