Cath Lab Spotlight

Sanford Health Cardiac Cath Lab

Jana Hart, RN, BSN, Manager Cardiac Cath Lab, Fargo, North Dakota

Tell us about your cath lab.

We have 5 cath lab rooms in total with 3 designated for cardiac cath, vascular, and structural heart procedures, and 2 designated for electrophysiology. We have approximately 40 staff members in our lab. Our team consists of radiologic technologists, registered cardiac electrophysiology specialists (RCES), registered cardiovascular invasive specialists (RCIS), and registered nurses (RNs). Members on our team providing care to patients in the cath lab have from 1 year up to 35 years of experience.

What procedures are performed in your cath lab?

We perform many procedures, including:
- Left and right heart catheterizations along with percutaneous intervention as necessary;
- Intracoronary ultrasound;
- Rotational atherectomy;
- Balloon pump insertion;
- Impella (Abiomed) insertion;
- Peripheral, cerebral, and renal angiograms along with intervention as necessary;
- Valvuloplasty

CATH LAB EDUCATION

Test Your Knowledge: An Overview of Pulmonary Hypertension and an Opportunity to Assess Your Hemodynamic Skills

Richard J. Merschen EdS, RT(R)(CV), RCIS, Mfonobong Ikpim, BS, RT(R), Fahad Alghamdi, BS, Xinmin Liu, BS, RT(R)

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CASE REPORT

Transradial Structural Intervention: Ventricular Septal Defect Closure

Nha Huynh, DO1, Ulrich Luft, MD2, Kintur Sanghvi, MD2
1Deborah Heart & Lung Center, Browns Mills, New Jersey;
2Kaiser Permanente, Portland, Oregon

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Cath Lab Digest talks with Gregory Piazza, MD, MS, Brigham and Women’s Hospital, Assistant Professor of Medicine, Harvard Medical School, Boston, Massachusetts.

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Test Your Knowledge: An Overview of Pulmonary Hypertension and an Opportunity to Assess Your Hemodynamic Skills

Richard J. Merschen EdS, RT(R)(CV), RCIS\(^1\), Mfonobong Ikpen, BS, RT(R)\(^2\), Fahad Alghamdi, BS\(^3\), Xinmin Liu, BS, RT(R)\(^3\)

Pulmonary hypertension (PHTN) is an abnormal elevation in pulmonary artery pressure and may be an idiopathic presentation, the result of left heart failure, pulmonary vascular disease, thromboembolic events, interstitial lung diseases, or a combination of these factors. PHTN is a hemodynamic and pathophysiological condition defined as an increase in mean pulmonary artery pressure greater than 25 mmHg at rest, as assessed by right heart catheterization.\(^1\) It is most commonly caused by left heart disease, particularly diastolic heart failure, and left-sided valvular disease. PHTN classified into one of 5 major groups and is generally a feature of advanced disease, regardless of the underlying causes, because it is often asymptomatic as it progresses. Because there are many potential causes of pulmonary hypertension, it is essential that the etiology underlying the pulmonary hypertension be clearly determined before embarking on treatment. In this regard, right heart catheterization plays a critical role.

According to the World Health Organization, the National Institutes of Health, and other organizations, pulmonary hypertension can be classified into 5 major groupings. They are:

**Group 1:** Primary pulmonary hypertension, which includes idiopathic, inherited pulmonary hypertension, acquired from something such as scleroderma, and disease of the small pulmonary artery and vein branches. It is known as pulmonary artery hypertension.

**Group 2:** Pulmonary hypertension related to left heart disease, such as left heart failure and valvular heart disease. It is the most common cause of PHTN, accounting for over 50% of cases.

**Group 3:** Pulmonary hypertension related to COPD or interstitial lung disease.

**Group 4:** Pulmonary hypertension related to pulmonary emboli\(^\) thromboembolic disease.

**Group 5:** Pulmonary hypertension caused by various other diseases or conditions. Examples include blood disorders such as polycythemia and essential thrombocytopenia, and systemic disorders, such as sarcoidosis.\(^2,\(^3\)

Each of these groups has multiple subcategories, and successful treatment of pulmonary hypertension is centered upon early diagnosis and treatment. Because most types of PHTN have other disease associated with them, treatment of the underlying causes of PHTN is essential in treatment strategies.

Pulmonary hypertension is often a silent disease, like systemic hypertension and chronic kidney disease. Therefore, when a patient has underlying disease that may predispose them to pulmonary hypertension, or a family history of the disease, it is important for them to be treated early and to have their care well managed by medical professionals.

The most important goal in treating pulmonary hypertension is to treat underlying causes, improve the quality of life for patients, and most importantly, prevent right heart failure. Pulmonary hypertension is the main cause of cor pulmonale (right heart failure). Treatment may involve managing acute episodes of PHTN, limiting right ventricle damage and remodeling, and improving exercise tolerance and symptoms of PHTN. If PHTN progresses to the point of causing cor pulmonale, the prognosis is poor. Although treatment for pulmonary hypertension has improved significantly, the long-term prognosis is often poor, because PHTN is often diagnosed after significant damage to the right ventricle has been caused.\(^1\)

The role of the cath lab

There are numerous diagnostic tools to diagnose PHTN and its effect on the pulmonary vasculature and the right ventricle. These include chest radiographs, echocardiography, computed tomography, and nuclear perfusion scans. In diagnosing and assessing pulmonary hypertension, the right heart catheterization is considered the gold standard diagnostic tool. The right heart cath offers the opportunity to assess pressures in all of the right-sided structures and the left atrium. Right heart catheterization is an indispensable tool in the diagnosis and management of patients with pulmonary arterial hypertension (PAH) and those with PH associated with decompensated heart failure (DHF). It allows for differentiation between these conditions, which can appear markedly similar from initial echocardiographic assessments, provides critical input in deriving therapeutic decisions, contributes prognostic information, and figures prominently in patient follow-up. Provocative maneuvers, including vasodilator challenge, volume loading, and exercise, can help uncover the pathophysiology contributing to patients’ symptoms.\(^4\)

The right heart catheterization allows the assessment of left heart disease through wedge pressure measurements. This helps determine whether pulmonary hypertension is the result of left heart disease (primary pulmonary hypertension), or is a right-sided heart disease (pulmonary arterial hypertension).

Right heart catheterization also evaluates gradients between the right and left side of the heart, known as transpulmonary gradients. The transpulmonary gradient is the difference between mean pulmonary artery pressure and mean wedge pressure. A value greater than 12 mmHg is considered significant and diagnosis is “out of proportion right-sided heart failure”, without a left-sided component.\(^5\)

Another gradient, the transpulmonary diastolic measurement, is considered to be superior, according to multiple sources.\(^7,\(^8\)

It evaluates the difference between the diastolic pulmonary pressure and the mean wedge pressure. In determining the severity of a transpulmonary diastolic gradient, a value of greater than 7 mmHg is considered significant. An elevated diastolic pulmonary vascular pressure gradient (DPG) was associated with more advanced pulmonary vascular remodeling and poorer patient outcomes.\(^7,\(^8\)

These gradients require accurate capture of hemodynamic waveforms, which can be seen below, on the post-test review. This includes factoring in respiratory variabilities and using the correct scales to capture the entire waveform. As accurate as computers may be, it is important to verify the results with clinical skills that factor in variabilities and potential operator errors in recording hemodynamic pressures.

The right heart catheterization determines real-time pulmonary vascular resistance by subtracting the mean pulmonary pressure from the mean wedge and then dividing by the cardiac output. A value above 3 Wood units is considered significant. The right heart catheterization can also be used to perform vasoreactivity studies on patients who have idiopathic pulmonary hypertension. A vasoreactivity study is performed on patients using a short-term vasodilator and looking for a 30% drop in pulmonary vascular resistance (PVR). If the test is positive, a patient may benefit from calcium channel blocker therapy. Although the number of patients who respond to vasoreactivity testing is small, the patients who do respond have significant clinical benefits from their treatment.

Calculation of PVR is essential in the management of patients with suspected PH. The formula for measuring pulmonary vascular resistance is mean pulmonary artery pressure – mean wedge pressure/cardiac output. The quality of the right heart study greatly influences the diagnosis of pulmonary hypertension and treatment strategies. When performing a high-quality hemodynamic study, the following elements are necessary to perform an optimal study. High-quality hemodynamics to diagnose PHTN should have the following elements:

1. True zero of the transducer, including proper phlebostatic alignment of the transducer;
2. A high-quality electrocardiogram (ECG) is important for performing calculations and calculate cardiac output;
3. Identification of A and V waves in the atria;
4. Identification of mean atrial pressures;
5. Identification of systolic and diastolic pressures in arteries and ventricles;
6. Proper measurement of mean arterial pressures;
7. Calculation of cardiac output, vascular resistance, and mean pressures;
8. Recording on the correct scales. Keep the measurements in the field so the systolic, diastolic and mean pressures can be accurately determined;
9. If there are respiratory variabilities, measurement should occur on end expiration;
10. Verification of wedge pressures with blood samples, if necessary, to confirm true wedge position of the catheter.

Since patients with elevated pulmonary pressures may also have significant tricuspid regurgitation (TR), the use of thermodilution (TD) cardiac outputs may not be feasible. It is important to evaluate the patient for TR, and then determine whether the Fick method or Swan-Ganz TD catheter should be used for a diagnostic right heart catheterization.

The right heart catheterization confirms pulmonary hypertension, and can distinguish between pulmonary venous pressure elevation caused by left heart disease and a right heart pathology that has a different pathology and treatment regimen. A transpulmonary or diastolic pressure gradient (TPG or DPG) identifies patients with “out-of-proportion” PHTN who have significant pulmonary vascular disease and increased mortality. In patients with postcapillary PHTN and a TPG >12 mmHg, a worse median

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A Note on Tools and Their Use

In the modern cardiac cath lab, computers are able to perform calculations for vascular resistance, cardiac output and index, cardiac shunts, gradients, and perform numerous other tasks. However, the best computer is only accurate and effective when the operator has the necessary skill sets to interpret the data and verify its accuracy. The computer is a tool that should be used like the fluoroscopic unit or intravascular ultrasound machine, and is only effective when used in the hands of highly trained team members. Performing proper hemodynamic assessments requires the technical skills of properly zeroing transducers, correctly aligning the height of the transducer, inspecting the transducer lines for kinks, leaks, and air, and inspecting the wedge catheter before conducting hemodynamic studies.

Skilled staff also factor in respiratory variances, arrhythmias, cardiac output states, valvular disease, and other variables that may skew the results of a study. There is no substitute for a highly trained staff member who can calculate, interpret, and identify proper waveforms in the diagnosis and treatment of disease. It is why hand calculations of core hemodynamics are being reinforced in this article.

The purpose of this article is to strengthen fundamental skills for interpreting hemodynamics. This includes calculating vascular resistance, understanding the key elements of atrial, arterial and ventricular waveforms, calculating mean pressures, and assessing pulmonary hypertension. These are foundational tools for a registered cardiovascular invasive specialist (RCIS), and skills that are rigorously assessed when taking the RCIS exam and its equivalent, offered by the American Registry of Radiologic Technologists. As cath labs perform more complex interventions such as transcatheter aortic valve replacement (TAVR), mitral valvuloplasty, and structural defect repairs, cath staff should apply themselves to become more effective in interpreting hemodynamic data and using it in practice.

4 criterion for good wedge:
1. Balloon stops bobbing on fluoro
2. Distinct A & V waves are seen on the pressure trace
3. The mean pressure is lower than PA
4. O2 sat exceeds 95%

Then wedge = LA = LVEDP = LV filling pressure & preload

Pulmonary Hypertension

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catheterization. It is well tolerated with very few systemic effects. It is expensive and may require a member of a respiratory team to administer. For centers that do not have access to nitric oxide, intravenous prostacyclin and adenosine have also been utilized. These agents can produce systemic side effects such as hypotension and arrhythmia, as well as nausea and vomiting.9 When using adenosine, it is important to watch the ECG as well as the right heart hemodynamics, and to have medications such as aminophylline available for patients who may have adverse reactions to an adenosine challenge. Adverse reactions may include bronchospasm and arrhythmia such as AV block. This study can help determine treatment strategies for PHTN patients, particularly the effectiveness of calcium channel blocker therapy. Around 8% of patients with PHTN are candidates for vasoreactivity studies, and around half of them will get a hemodynamic response, which can be calculated as a 30% drop in the vascular resistance. A positive response to vasoreactivity testing is defined as a reduction of mPAP 210 mmHg to reach an absolute value of mPAP ≤40 mmHg with an increased or unchanged cardiac output (CO).7,10 The goal is to see a significant normalization of pulmonary artery pressures with a vasoreactivity study. In order to measure the response to vasoreactivity studies, the pulmonary vascular resistance is calculated at regular intervals during the study and looks for a drop in pulmonary vascular resistance. The study is performed by recording pulmonary artery pressures and pulmonary wedge pressures, calculating cardiac outputs, and determining pulmonary vascular resistance.

Nitric oxide should be given only to patients with pulmonary hypertension and a normal pulmonary artery wedge pressure, because the nitric oxide may dilate the postcapillary bed and result in a further elevation of left-sided filling pressures in patients with baseline left atrial pressure elevation; a similar deleterious response may be possible with the administration of adenosine. Pulmonary artery capacitance has been shown to have additional prognostic value and should be measured at the time of cardiac catheterization.6

In patients with risk factors for left ventricular (LV) diastolic dysfunction, acute vasoreactivity testing can lead to a significant increase in both LV end-diastolic pressure and pulmonary artery occlusion pressure (PAOP), which unmasks the presence of impaired relaxation of the LV, resulting in acute pulmonary edema. A dramatic V wave pressure increase of the PAOP during vasoreactivity testing will alert the team to the occurrence of this situation.15 Typically, patients require high doses of calcium channel blockers and those who respond favorably usually have dramatic reductions in pulmonary artery pressure and pulmonary vascular resistance. This is associated with improved symptoms, regression of right ventricular (RV) hypertrophy, and improved survival, now documented to exceed 20 years. However, less than 20% of patients respond to calcium channel blockers in the long term. These drugs should not be given to patients who are unresponsive, as they can result in hypotension, hypoxemia, tachycardia, and worsening right heart failure.11 Because calcium channel blocker therapy can have either great benefits or cause worsening of disease, accurate hemodynamics and diagnosis are critical before beginning aggressive calcium channel blocker therapy.

Conclusion
Right heart catheterization is an important tool in the diagnosis and treatment of pulmonary hypertension. It allows for real-time measurement of right heart hemodynamics and also evaluates pulmonary capillary wedge pressure. This helps determine if pulmonary hypertension is exclusively a right heart pathology, or if left heart disease is causing the pulmonary hypertension. Important measurements obtained during right heart catheterization include measurements of transpulmonary gradient, transpulmonary diastolic gradients and vascular resistance. These measurements allow the physician to determine the cause and severity of disease. If the diagnosis is idiopathic (Group 1 pulmonary hypertension), vasoreactivity studies can be performed to assess the effectiveness of short-acting vasodilators such as adenosine and nitric oxide. In a small subset of patients, a reduction in pulmonary vascular resistance during a vasoreactivity study may be an indication for the use of calcium channel blockers. In this subset, mortality and morbidity may be significantly reduced. Therefore, the cath lab plays an important role in diagnosing and treating pulmonary hypertension, and it is important to have excellent knowledge of cardiac hemodynamics when performing these studies. ■

Acknowledgements.
We would like to thank Wes Todd for supplying us with important images to add substance and context to this article. He has been a great resource and is always willing to share materials, knowledge, and time to advance cardiovascular medicine. His lifelong commitment to the field of cardiovascular medicine is greatly appreciated. We consider him to be a friend and an inspiration for the articles that we publish.

Pulmonary Hypertension: Basic Cardiac Hemodynamics Test

The following test offers an opportunity to assess your knowledge of basic cardiac hemodynamics. These questions are designed to identify and calculate the hemodynamics that are necessary to complete a high-quality right heart catheterization, and to make the diagnosis of pulmonary hypertension. At the end of the test, an answer key will be provided to check your answers and to provide brief explanations of each answer.

The following data has been provided to perform the mathematical calculations:

Patient data:
Weight 185 lbs
Height 5’4”
Body surface area (BSA) 1.9cm2

![Figure 1A. Pulmonary capillary wedge pressure (PCWP).](Image 36x73 to 378x288)

1. What is the primary cause of pulmonary hypertension?
   a. Coronary artery disease
   b. Left heart failure/diseases progressing into right heart failure
   c. Tricuspid valve disease
   d. Chronic kidney disease

2. What is the primary cause of pulmonary hypertension?
   a. Coronary artery disease
   b. Left heart failure/diseases progressing into right heart failure
   c. Tricuspid valve disease
   d. Chronic kidney disease

Pulmonary arterial (PA) pressure (mean pressure): see Figure 1A.*
Pulmonary capillary wedge pressure (PCWP) pressure (mean pressure): see Figure 2A.
Right ventricular (RV) pressure: see Figure 3A.
Pulmonary vascular resistance (PVR): see Figure 4A.
Right atrial (RA) pressure (mean pressure): see Figure 5A.
Pulmonary arterial blood oxygen saturation (Pa sat) 53%
Aortic blood oxygen saturation (Ao sat) 94%* Figures are set in the classic wedge to RA pullback sequence.

![Figure 2A. Pulmonary artery pressure.](Image 396x76 to 738x288)

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Pulmonary Hypertension: Test

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2. What is the formula for calculating pulmonary hypertension?
   a. (Mean pulmonary artery pressure – mean pulmonary wedge) / cardiac output
   b. Mean systemic pressure – right atrial mean pressure / cardiac output
   c. Fick cardiac output / body surface area
   d. Ao sat – Pa sat = pulmonary vein sat

3. What is a normal pulmonary artery pressure?
   a. Systolic 25 / end diastolic 5
   b. Systolic 6 / V wave 10 / A wave 6
   c. Systolic 25 / diastolic 10 / mean 15
   d. Systolic 110 / end diastolic 12-16

4. What is a normal pulmonary wedge pressure?
   a. A wave 6 / V wave 4 / mean 2
   b. A wave 12 / V wave 35 / C wave 17
   c. Systolic 35 / mean 17
   d. A wave 12 / V wave 10 / mean 10

5. What is the normal Wood unit range for pulmonary vascular resistance?
   a. 0.3-2.0
   b. 10-20
   c. 24-160
   d. 25/10/15

6. What is the constant to convert Wood units to absolute or metric units (dynes/sec/cm⁻²)?
   a. Divide by 80
   b. Add by 80
   c. Subtract by 80
   d. Multiply by 80

7. What is this patient’s pulmonary wedge pressure?
   a. 32/28/26
   b. 40/20/49
   c. 16/12/10
   d. 6/4/2

8. What is this patient’s pulmonary artery pressure?
   a. 50/20/10
   b. 85/40/55
   c. 120/80/90
   d. 100/6/16

9. What is a normal right ventricular pressure?
   a. 39/23/67
   b. 25/5
   c. 2/2/3
   d. 120/16

10. What is this patient’s right ventricular pressure?
    a. 140/10
    b. 50/23/40
    c. 95/18
    d. 25/5

11. What is a normal mean right atrial pressure?
    a. 2-6 mmHg
    b. 25/10/15
    c. 25/5
    d. 12/16/12

12. What is this patient’s right atrial pressure?
    a. 15/10/7
    b. 120/6
    c. 20/18/14
    d. 2-6

13. Which drugs can be administered to conduct a vasoreactivity study?
    a. Adenosine and nitric oxide
    b. Beta blockers and angiotensin-converting-enzyme (ACE) inhibitors
    c. Heparin and coumadin
    d. Aspirin and clopidogrel

14. What is this patient’s Fick cardiac output?
    a. 45 liters/min
    b. 10 liters/min
    c. 3.5 liters/min
    d. 450 mLs/min

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15. What is this patient’s cardiac index?
   a. 1.75
   b. 4.5
   c. 1.0
   d. 44.3

16. Based off the cardiac output and hemodynamic data, what is this patient’s pulmonary vascular resistance in Wood units?
   a. 63
   b. 6.3
   c. 630
   d. 2.4

17. What is this patient’s pulmonary vascular resistance in metric/absolute units?
   a. 4800
   b. 662
   c. 43
   d. 2.4

18. If pulmonary hypertension is not caused by left heart disease, what is the classic hemodynamic pattern?
   a. All pressures are normal.
   b. Wedge, PA, RV, and RA are all elevated, left-sided pressures may be normal or slightly elevated.
   c. Wedge is relatively normal, while PA, RV, and RA are elevated.
   d. All other hemodynamics are normal, except the RA, which shows severe TR.

19. When advancing a swan catheter, the balloon should be:
   a. Inflated
   b. Deflated
   c. Partially deflated
   d. It doesn’t matter, as long as it is wedged.

20. How many major groupings of pulmonary hypertension are there?
   a. 5
   b. 15
   c. 3
   d. 1

Test Answer Key

1. What is the primary cause of pulmonary hypertension?
   a. Coronary artery disease
   b. Left heart failure / diseases progressing into right heart failure
   c. Tricuspid valve disease
   d. Chronic kidney disease

   Over 50% of pulmonary hypertension cases are related to left heart failure and valvular heart disease. Right heart catheterization plays an important role in identifying PHTN related to left heart disease.

2. What is the formula for calculating pulmonary vascular resistance?
   a. \( \text{Mean pulmonary artery pressure} - \text{mean pulmonary wedge} / \text{cardiac output} \)
   b. Mean systemic pressure – right atrial mean pressure / cardiac output
   c. Fick cardiac output / body surface area
   d. Ao sat – Pa sat = pulmonary vein sat

3. What is a normal pulmonary artery pressure?
   a. Systolic 25 / end diastolic 5
   b. Systolic 6 / V wave 10 / A wave 6
   c. Systolic 25 / diastolic 10 / mean 15
   d. Systolic 110 / end diastolic 12-16

   See Figure 2B and Table 1 to review pulmonary artery pressure waveform interpretation.

4. What is a normal pulmonary wedge pressure?
   a. A wave 6 / V wave 4 / mean 2
   b. A wave 12 / V wave 35 / C wave 17
   c. Systolic 35 / mean 17
   d. A wave 12 / V wave 10 / mean 10

   See Figure 1B and Table 1 to review pulmonary capillary wedge pressure waveform interpretation.

5. What is the normal Wood unit range for pulmonary vascular resistance?
   a. 0.3-2.0
   b. 10-20
   c. 24-160
   d. 25/10/15

   continued on page 20
6. What is the constant to convert Wood units to absolute or metric units (dynes/sec/cm²)?
   a. Divide by 80
   b. Add by 80
   c. Subtract by 80
   d. Multiply by 80
   See Table 2.

7. What is this patient’s pulmonary wedge pressure?
   a. 32/28/26
   b. 40/20/40
   c. 16/12/10
   d. 6/4/2
   See Figure 1B and Table 1 to review pulmonary capillary wedge pressure waveform interpretation.
   Remember to measure during end expiration. The mean wedge pressure is critical in determining the severity of PHTN, so inaccurate values can greatly impact the diagnosis of PHTN and left heart failure.

8. What is this patient’s pulmonary artery pressure?
   a. 50/20/10
   b. 85/40/55
   c. 120/80/90
   d. 100/6/16
   See Figure 2B and Table 1 to review pulmonary artery pressure waveform interpretation.

9. What is a normal right ventricular pressure?
   a. 39/23/67
   b. 25/5
   c. 2/2/3
   d. 120/16
   See Figures 3B-4B and Table 1 to review right ventricle pressure waveform interpretation.

10. What is this patient’s right ventricular pressure?
    a. 140/10
    b. 50/23/40
    c. 95/18
    d. 25/5
    See Figures 3B-4B and Table 1 to review right ventricle pressure waveform interpretation.

11. What is a normal mean right atrial pressure?
    a. 2-6 mmHg
    b. 25/10/15
    c. 25/5
    d. 12/16/12
    See Figure 5B and Table 1 to review right atrial pressure waveform interpretation.

12. What is this patient’s right atrial pressure?
    a. 15/10/7
    b. 120/6
    c. 20/18/14
    d. 2-6
    See Figure 5B and Table 1 to review right atrial pressure waveform interpretation.

13. Which drugs can be administered to conduct a vasoreactivity study?
   a. Adenosine and nitric oxide
   b. Beta blockers and angiotensin-converting-enzyme (ACE) inhibitors
   c. Heparin and coumadin
   d. Aspirin and clopidogrel
   There is a limited application for vasoreactivity studies, and controversy over the effectiveness of adenosine for vasoreactivity studies. However, there is a small subset of patients with idiopathic PHTN that respond well to calcium channel blocker therapy, which improves their symptoms and outcomes.

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**Table 2. Normal vascular resistance.**

<table>
<thead>
<tr>
<th>Vascular Resistance</th>
<th>Wood units</th>
<th>Wood formula</th>
<th>dynes/cm²</th>
<th>Metric formula</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulmonary</strong></td>
<td>0.25-2</td>
<td>PA mean – PCWP mean / cardiac output</td>
<td>20-160</td>
<td>PA mean – PCWP mean / cardiac output x 80</td>
</tr>
<tr>
<td><strong>Systemic</strong></td>
<td>8-20</td>
<td>AO mean – RA mean / cardiac output</td>
<td>600-1400</td>
<td>AO mean – RA mean / cardiac output x 80</td>
</tr>
</tbody>
</table>
Pulmonary Hypertension: Test Answer Key
Continued from page 20

14. What is this patient’s Fick cardiac output?
   a. 45 liters/min
   b. 10 liter/min
   c. 3.5 liters/min
   d. 480 ml/min
   Cardiac output was calculated by using:
   185 lbs = 84 kg x 3 = 252 / (0.95-0.53) x 1.36 x 13 hgb x 10 = 3.5 liters/min

15. What is this patient’s cardiac index?
   a. 1.75
   b. 4.5
   c. 1.0
   d. 44.3
   Cardiac index is calculated by dividing the cardiac output by the body surface area m².
   Normal cardiac index ranges from 2-3.5 and the index is normal for this patient.
   Cardiac index reflects effective cardiac output by factoring in the body surface area
   and output. Obese patients, such as this one, will have lower indexes, even with
   a normal cardiac output. Cardiac index = 3.5 / 2.0 = 1.75.

16. Based off the cardiac output and hemodynamic data, what is this
   patient’s pulmonary vascular resistance (PVR) in Wood units?
   a. 63
   b. 8.3
   c. 630
   d. 2.4
   (Mean PA pressure – mean wedge pressure) / cardiac output = 55-26 / 3.5 = 8.3
   Wood units. This patient’s PVR is extremely elevated, as normal ranges vary between
   0.6 and 2.0 (see Table 2 for normal values).

17. What is this patient’s pulmonary vascular resistance in metric/absolute
   units?
   a. 4800
   b. 662
   c. 43
   d. 2.4
   Absolute units are calculated by multiplying the Wood units by 8.3 x 80 = 662
   dynes/sec/cm².
   See Table 2.

18. If pulmonary hypertension is not caused by left heart failure, what is
   the classic hemodynamic pattern?
   a. All pressures are normal.
   b. Wedge, PA, RV and RA are all elevated, left-sided pressures may be
      normal or slightly elevated.
   c. Wedge is relatively normal, while PA, RV and RA are elevated.
   d. All other hemodynamics are normal, except the RA, which shows severe tricuspid regurgitation.

19. When advancing a Swan-Ganz catheter, the balloon should be:
   a. Inflated.
   b. Deflated.
   c. Partially deflated.
   d. It doesn’t matter, as long as it is wedged.
   The balloon should be inflated. This makes it easier to advance through the
   chambers of the heart and prevents pulmonary rupture, which can occur if the bal-
   loon is inflated in a pulmonary artery branch.

20. How many major groupings of pulmonary hypertension are there?
   a. 5
   b. 15
   c. 3
   d. 1
   In the article, the 5 major groupings of pulmonary hypertension are listed. A
   nd the identification of the correct grouping is essential in treating PHTN, as most cases are
   associated with other disease and medical conditions. In the article, the importance of
determining essential PHTN versus induced left diastolic heart failure is emphasized and
is a major component of right heart catheterization.

Answer Key (repeated):
1b, 2a, 3c, 4d, 5a, 6d, 7a, 8b, 9b, 10c, 11a, 12c, 13a, 14c, 15a, 16b, 17b, 18b, 19a, 20a

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Right heart catheterization is an important tool in the diagnosis and treatment of
   pulmonary hypertension. It allows for real-time measurement of right heart hemodynamics
   and also evaluates pulmonary capillary wedge pressure. This helps determine if pulmonary
   hypertension is exclusively a right heart pathology, or if left heart disease is causing
   the pulmonary hypertension.