What Is That Blip? Using Pulmonary Artery Catheter and Central Venous Pressure Waveforms in the Operating Room

Carl J. Daltner, M.D. Pittsburgh, Pennsylvania

OBJECTIVES

1) To identify normal and abnormal hemodynamic waveforms on central venous and pulmonary artery pressure tracings.
2) To correctly identify to pulmonary artery occlusion pressure and relate it to left ventricular filling and the risk of pulmonary edema.
3) To correctly identify pitfalls in using pulmonary artery occlusion pressure to estimate left ventricular filling.
4) To be able to use waveform morphology to identify specific disease states.

STEM CASE - KEY QUESTIONS

A 72 year old male with a history of atherosclerotic heart disease, syncopy, paroxysmal atrial fibrillation and pacemaker placement presents for coronary artery bypass grafting and aortic valve replacement. Associated diseases include a history of rheumatic fever, benign prostatic hypertrophy and a remote history of smoking. His daily medications include oral metoprolol 25 mg, diltiazem 120 mg and aspirin 81 mg. Prior to admission, the patient had been doing well until awaking acutely short of breath. He presented to the emergency department dyspnic, diaphoretic and complaining of a substernal pressure. After stabilization, laboratory investigation revealed hemoglobin 12 g/dl, hematocrit 36%, platelets 233, BUN 42 mg/dl, Cr 1.8 mg/dl, EKG NSR and CXR with bi-basilar pulmonary effusions. Cardiac catheterization revealed hypokinesis of the antero-lateral wall, overall preserved LV function with an ejection fraction of 50%, aortic valve area 0.9 cm^2, and atherosclerotic disease consisting of 90% lesions of the left anterior descending artery and right coronary artery with a right dominant circulation.

The patient is placed on the operating room table in supine position and standard ASA monitors applied. An indwelling radial artery catheter is inserted. A pulmonary artery catheter (PAC) is placed under aseptic technique.

Key Questions

What are normal tracings seen in the central venous, right ventricle, pulmonary outflow tract and wedge positions? The basic physiologic and anatomic principles that generate hemodynamic waveforms will be reviewed. These will be correlated with transesophageal echocardiographic images. What clues are seen on the tracings that indicate good positioning of the catheter?

During the insertion process, the patient complains of substernal chest discomfort and acute shortness of breath. The EKG has become irregular with a difficult to read baseline. The arterial pressure falls to 85/45 mmHg.

Key Questions

How can the central venous pressure waveform be used to access the EKG irregularities? Waveforms consistent with canon a-waves and pacemaker dysfunction will be reviewed. What therapeutic maneuvers would you contemplate? Therapy including cardioversion, pacemaker adjustment and drug administration will be discussed.

The patient returns to sinus mechanism and after successful placement of the PAC, the patient tolerates induction with minimal vaso-constrictive support. A transesophageal echocardiography probe is placed. A colleague with transesophageal echocardiography (TEE) experience reads the exam as aortic stenosis with mildly depressed LV function. Findings consistent with trace mitral regurgitation are seen. The surgeon performs a sternotomy and initiates dissection of the left internal mammary artery. During this time it is noted that the patient has limited urinary output. The mean pulmonary artery wedge pressure (PAWP) is 19 mmHg.

Key Questions
How and where is PAWP measured? What is the difference between mean and phasic PAWP? What is the filling pressure and does it accurately reflect left ventricular end-diastolic volume in this patient? What PAC waveforms are consistent with aortic stenosis? Why does a patient with aortic stenosis not have heart failure until late in the disease process? Why is the required filling pressure in aortic stenosis elevated? What therapies are appropriate in this scenario? Furosemide, nitroglycerine or volume?

As the internal mammary artery is dissected, the pulmonary artery pressure increases to 57/29 mmHg. Mitral regurgitation is seen on the TEE.

Key Questions
The PAWP tracing shows a large V wave and a high mean value. Why does this situation produce pulmonary edema while waveforms associated with aortic stenosis do not? The differential of elevated mean PAWP compared to PAWP for left ventricular filling will be discussed. How can a V wave be present without structural mitral valve abnormalities? Ischemia induced MR in a patient with aortic stenosis and ASHD will be reviewed. Does the mitral valve need to be replaced? What treatment options exist? If the aortic valve area were 1.1 cm^2, would you recommend replacement of the aortic valve?

The patient is urgently placed on cardiopulmonary bypass. The LAD and RCA are bypassed and the aortic valve is replaced with a bovine bioprosthetic valve. Immediately post-bypass the aortic valve replacement appears to function normally, good LV function is present and no mitral regurgitation is observed. The patient is transferred to the intensive care. Approximately five hours later, you receive a page to the intensive care unit. The surgeon would like to have a TEE performed. He thinks that the LV is failing. No TEE is available at this time. On examination of the patient, you note that the patient is oliguric, tachycardic and hypotensive. The mean PAWP is 24 mmHg.

Key Questions
What is the patient’s volume status? Is the LV failing? What waveform clues can give you the appropriate diagnosis? Waveforms differentiating pericardial constriction, tamponade and right ventricular infarct will be reviewed. These will be correlated with transesophageal echocardiographic images. Waveforms illustrating dip-and-plateau, square root signs, large X and Y descents will be shown. What treatment options exist? Does the patient need to return to the operating room imediately? Should the patient’s stenotomy be opening in the intensive care unit setting?

PROBLEM BASED LEARNING DISCUSSION

Rational for Determination of Filling Pressures

Frank and Starling's original work involved in vitro stretching of canine cardiac muscle and measuring the tension developed at different lengths. The stretch and tension relationship was termed preload. Tension generated by a muscle increases as stretch increases, peaking once the myocytes become maximally distended. During the cardiac cycle, in vivo preload is considered to be the end-diastolic volume of the ventricle. In 1947, Dexter related pulmonary artery wedge pressure to the filling pressure of the left ventricle. During ventricular end-diastole and before mitral valve closure, a static column of blood exists between the catheter tip and left ventricle. At this moment the PAWP estimates left ventricular end-diastolic pressure (LVEDP). Under most conditions, LVEDP is related to left ventricular end-diastolic volume (LVEDV) and subsequently pre-load. During this period, pulmonary artery catheterization provided information for diagnosis of congenital and acquired valvular diseases. Measurement of the pulmonary artery pressure (PAP) determined the need for surgery. The catheters used were semi-rigid and required fluoroscopy for placement. Swan and Ganz developed a flow-guided balloon catheter of flexible construction that would measure PAP. This technology took many diagnostic maneuvers from the cathlab to the bedside.

Normal Wave Forms: The Right Vs Left Heart

During the normal cardiac cycle electrical stimulation produces a contraction of cardiac muscle. Cardiac contraction produces increases in intra-cavitary pressure. The CVP tracing is the pressure representation of the cardiac cycle in the right atrium. To facilitate waveform interpretation, the waveform is compared to a clear reference, such as the EKG. The A-wave is generated by atrial contraction and follows the P wave of the EKG. The C wave is generated by the closure of the tricuspid valve and adjacent carotid artery pulsation. It represents the end of isovolemic contraction. During systole the cardiac skeleton is pulled to the apex increasing atrial size causing the pressure within the chamber to fall. This is represented by the X descent. It is divided into two components. X occurs prior to the C wave and X' follows. The V-wave reflects the increasing pressure in the atrium as it fills against a closed tricuspid valve. The Y descent signals the opening of the tricuspid valve and atrial to ventricular blood flow. In the right heart, wave contractions follow the EKG by 60-80 milliseconds. Due to pulmonary vasculature transmission time, left side waves are delayed. Left sided waves as measured in the pulmonary artery follow EKG events by 160-180 milliseconds. The PAWP is a dampened and delayed reflection of the LAP.

Estimating Left Ventricular Filling Pressure: Preload

With the PAC balloon inflated and the catheter in correct position a continuous column of blood extends from the catheter tip to the left ventricle. Blood drawn from the catheter tip is oxygenated capillary blood. If this does not occur, a continuous column of blood from the catheter tip to the end of the pulmonary veins is not present and PAWP will not reflect true filling pressures. In correct position with the mitral valve open, the PAC can estimate preload. End-diastolic pressure after atrial contraction estimates preload to the ventricle. This is referred to as the Z point. On a PAWP tracing, it is located on the X descent prior to the C wave. In

patients with LV Dysfunction, the peak A wave pressure reflects the end-diastolic kick and is the most accurate measure of LVEDP.

Limitations

Clinical situations arise where PAWP does not accurately reflect ventricular filling pressures. Development of pressure gradients and variations in cardiac compliance alter the PAWP-LVEDP relationship. The substitution of the diastolic Pulmonary Artery Pressure (PAP_{dias}) or Pulmonary Capillary Pressure (PCP) for PAWP illustrates this point.

The Pulmonary Capillary Pressure (PCP) is the pressure within the small vessels of the lung. PCP determines the hydraulic forces acting on fluid within the capillary system. Increased hydrostatic pressure causes fluid to leak into the interstitial space. This causes pulmonary edema. PCP also provides the driving force for forward flow into the left atrium. PAWP and PCP often are not equal. PAWP is a static measurement while PCP is a dynamic assessment. Dynamic measurements include gradients that develop when blood flow occurs. If resistance to flow is present in the pulmonary blood vessels, a gradient will develop across that pathway when flow occurs. The gradient increases PCP. PAWP, a no-flow situation, has no additional gradient and is lower than PCP. Mitral valve replacement, pulmonary veno-occlusive disease, CNS Injury, acute lung injury, septic or hypo-volemic shock may cause this situation to occur. PCP is the determinant of pulmonary edema and PAWP is a measure of LV pre-load. PCP can be estimated in two ways. First, using the pulmonary artery pressure decay technique. The PAC balloon is inflated and the PCP is estimated by the observing the inflection point as the pressure tracing transforms from pulmonary artery pressure to pulmonary artery wedge pressure. PCP also may be estimated by simply obtaining the mean PAWP. A low PAWP may accurately reflect LAP but may underestimate the pulmonary capillary pressure. In this case, the patient is “dry,” that is the LV is under-filled, but the patient has pulmonary edema. Acute mitral regurgitation is a classic example.

Normally, PAWP is similar to PAP_{dias}. Under many conditions, this relationship fails and PAP_{dias} does not represent the PAWP. Things as simple as tachycardia, blood transfusion or positive pressure ventilation cause the PAWP-PAP_{dias} gradient to approach 6 mmHg or greater. In this case, gradients alter the ability of one measurement to estimate LV pre-load.

Additionally, cardiopulmonary compliance alters the PAWP-LVEDV relation. Compliance and elastance define the relation of volume and pressure. While the PAC measures pressures, pre-load is related to chamber volume and stretch of the myocardium. The relation of volume to pressure is termed compliance. Compliance is the change in volume divided by the change in pressure. Elastance is the reciprocal of compliance. Pressure measurements will estimate different volumes as compliance changes.

Compliance=$\Delta V/\Delta P$ Elastance=$\Delta P/\Delta V$

The PAC has other limitations. Hemodynamic waveforms are summation waves. They are sine waves composed of ante grade and reflected pressure fronts. These waves are modified by compliance throughout the vascular tree. Loading conditions, vascular tone, site of measurement, respiratory variation and heart rate modify the wave contour. For these reasons, the peak and

diastolic pressures should be measured from the graph scale directly. The monitor, especially when using the computer-generated mean, may misread phasic measurements. A phasic pressure is a measurement at a specific point in time. Mean pressure is a calculated time weighted average. Changing waveform contour will change the mean pressure but not the systolic or diastolic numbers. PAWP is an example of a phasic pressure. To be accurate PAWP should be measured at the Z-point, not as a mean pressure.

PAWP and LVEDP

Using mean PAWP or PAP_{dias} to estimate filling pressures will lead to errors. In situations of decreased ventricular compliance, mean PAWP may not accurately approximate LVEDP. Atrial contraction will contribute up to 40-50% of LVEDV as ventricular compliance decreases. The A wave will be large due to the compliance change, but the mean PAWP will be low, underestimating LVEDP. To adequately gauge pre-load, PAWP must be measured at the Z-Point. Examples include aortic stenosis, left ventricular hypertrophy and hypertrophic cardiomyopathy. Aortic insufficiency also results in underestimation of the LVEDP. The ventricle fills from two directions. The rapid increase in ventricular pressure causes premature closure of the mitral valve. Filling continues after mitral valve closure but is not transmitted to the catheter tip and underestimation of LVEDP occurs. Pulmonary Regurgitation allows retrograde flow into the right ventricle. Pulmonary artery diastolic pressure will reflect the lower RV pressure and PAP_{dias} will underestimate PAWP, LAP and LVEDP. Right bundle branch block and pulmonary vascular bed reductions also produce underestimation of LVEDP by PAP_{dias}.

Overestimation of LVEDP occurs with positive pressure ventilation, pulmonary hypertension, veno-occlusive disease, mitral stenosis, mitral regurgitation, tachycardia and ventricle septal defects. With positive pressure ventilation, alveolar pressure may rise above pulmonary arterial and venous pressures. Zone one instead of zone three conditions exist and mean PAP reflects alveolar pressure overestimating mean LAP. Pulmonary hypertension causes increased pulmonary vascular resistance as diastolic flow continues. This causes a gradient between PADP and the PAWP. PAWP but not PADP will estimate LVEDP. Mitral stenosis produces a gradient between the catheter tip and LV resulting in PADP and PAWP overestimating LVEDP. Mitral regurgitation elevates mean PAWP and LAP that overestimate LVEDP. Accurate measurement of PAWP at the Z-Point will result in a correct assessment of preload. During tachycardia diastole shortens and pulmonary blood flow has less time to enter the LA. A gradient forms causing overestimation of the LVEDP by the PADP or PAWP. A ventricular septal defect causes left to right shunting. Elevated pulmonary flow causes rapid filling of the LA and a large V wave. The mean PAWP rises and over-estimates LVEDP. Use phasic PAWP to measure LVEDP in patients with a VSD.

PAWP LVEDP Relationships	
Mean PAWP underestimates LVEDP	**Mean PAWP overestimates LVEDP**
Compliance changes (AS,LVH,HCM)	PPV/Non Zone 3
Aortic Insufficiency	Mitral Stenosis/Regurge, Myxoma
Pulmonary Valve Insufficiency	Tachycardia
Right Bundle Branch Block	VSD
	Increased PVR/Pulmonary Embolus

Waveform Analysis

The PAC is used to measure pressures for diagnostic purposes. Analysis of the waveform shape provides additional information. Abnormal CVP and PAWP values and shapes have a differential diagnosis as any lab test. Waveform patterns include large A or V waves, absent A waves, and large or small Y descents.

Absolute Values		
Increased RAP	**Increased Mean PAWP**	**Low PAWP**
Hypervolemia	Hypervolemia	Hypovolemia
RV/LV Failure	LV Failure	
Tricuspid Disease	Mitral Disease	
Constrictive Pericarditis	Constrictive Pericarditis	
Tamponade	Tamponade	
	PEEP/PPV	

Patterns				
Large A Wave	**Large V Wave**	**No A Waves**	**Short Y Descent**	**Large Y Descent**
Tricuspid/Mitral Stenosis	Tricuspid/Mitral Regurge	Afib	Tamponade	Constrictive Pericarditis
Decreased LV Compliance	Supply Ischemia, CHF	AFlutter		Restrictive Diseases
AV Dissociation	VSD	Junctional Rhythm		RV Infarction
Canon A Wave	Increased Afterload			Cardiomyopathy
	Decreased Atrial Compliance			

Cannon A waves
During junctional heart rhythms, the atrial kick occurs against a closed tricuspid valve. This produces a large cannon A wave.

Tricuspid and Mitral Regurgitation

The A wave and initial X decent appear normal. As systole begins, the incompetent atrioventricular valve allows reverse atrial filling. The atrial pressure increases dramatically during the early systolic cycle and a large “C-V” wave is formed.

Tricuspid and Mitral Stenosis

Atrioventricular valve stenosis causes resistance to right atrial outflow. Atrial contraction against an immobile valve generates a high intra-atrial pressure. This high pressure is represented by a large A wave. The immobile valve opens poorly. This is characterized by a severely blunted Y descent. The large A wave occurs due to similar pressure considerations as the cannon A wave in a junctional rhythm.

Ischemia

Ischemia occurs during coronary blood flow supply and demand imbalance. Generally, increases in demand produce diastolic dysfunction and impaired relaxation. Atrial contraction against a poorly compliant ventricle generates a large A wave. Supply ischemia often results in systolic dysfunction. Changes in the valve annulus shape and papillary muscle dysfunction cause mitral valve regurgitation. This produces a large V wave. The sensitivity and specificity of the pulmonary artery catheter for ischemia detection have been questioned. A lack of consensus for absolute changes in A and V wave parameters that indicate ischemia also decrease specificity.

Tamponade and Pericardial Constriction

Anytime right atrial pressure approaches peripheral venous pressure, blood return to the heart decreases. Tamponade and pericardial constriction are two examples. Pericardial constriction produces a rigid box around the heart. Venous return is normal in the early phases of diastole. As the heart fills, it comes in contact with the rigid pericardial shell and late diastolic filling is impaired. Systolic atrial filling is normal. Atrial filling occurs during systole and early diastole so it is termed bi-phasic. The waveform components are exaggerated, producing sharp X and Y descents. This pattern results in a “W” or “M” configuration depending on heart rate. A fast rate forms an “M” pattern and a slow rate produces a “W”. These characteristic patterns are also known as dip and plateau or square root signs. Differential diagnosis includes restrictive cardiomyopathies and RV infarction. Cardiac tamponade produces a characteristic CVP trace. Pericardial fluid exerts a constant pressure on the heart chambers throughout the cardiac cycle. Both early and late diastolic filling are impaired. Venous return to the right heart only occurs during systole so it is termed mono-phasic. A prominent X descent is produced with attenuation of the Y descent.

The Pulmonary Artery Catheter is an extremely useful tool. Proper use requires integration of signs, symptoms and acquired data. Integration of all information is the key to precise assessment of the critically ill patient. In the near future, the results of randomized clinical trials will help to delineate the situations where the pulmonary artery catheter is useful. Improved education in use of the catheter will contribute to satisfactory patient outcome.

REFERENCES

Atlas of Cardiovascular Monitoring. J Mark, Ed. Churchill Livingston, 1998

Courtois, M, Fattal P. Anatomically and Physiologically Based Reference Level for Measurement of Intracardiac Pressures. Circulation. 92(7):1994;1995

LEARNING SUMMARY

1) To identify normal and abnormal hemodynamic waveforms on pulmonary artery pressure tracings.
2) To identify to pulmonary artery occlusion pressure and relate it to left ventricular filling and the risk of pulmonary edema.
3) To identify pitfalls in using pulmonary artery catheter data.
4) To utilize waveform morphology to identify specific disease states.