

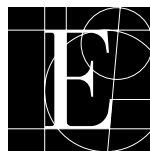
INVASIVE HEMODYNAMIC MONITORING: PHYSIOLOGICAL PRINCIPLES AND CLINICAL APPLICATIONS



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**INVASIVE
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APPLICATIONS**

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INTRODUCTION

During the last 25 years, the art of critical care medicine has greatly changed. This process has been due in part to the formation of specialized units for patient care, advances in technology, and a better understanding of physiology by health care practitioners.

One of the earlier advances in technology that helped to drive this progress was the development in the 1960's of the Edwards Swan-Ganz catheter. In the early 1970's, the addition of a thermistor to the catheter allowed for rapid assessment of cardiac output. At the same time, more sophisticated monitoring systems were also being developed. As a result, more complete hemodynamic assessment could be carried out with relative ease at the patient's bedside.

With advanced technology comes the requirement of advanced clinicians. The critical care practitioner must be better educated in order to practice effectively. The goal of this booklet is to provide the practitioner with an expanded knowledge of basic hemodynamic principles.

This book is divided into sections that discuss the various components of hemodynamic monitoring, including: functional anatomy and applicable cardiac physiology, physiological bases of hemodynamic monitoring, cardiac output determinations, and clinical applications. The book may be read as a whole or each section referred to as a single entity.

FUNCTIONAL ANATOMY

Right vs. Left Heart

In this section, a review of functional cardiac anatomy and applicable physiology is presented. These concepts provide the foundations of hemodynamic monitoring.

When discussing the functional anatomy of the heart in regards to hemodynamic monitoring, the heart is described as two separate pumps. Each side, or pump, has its own function and pressure generation. For this reason, the terms right and left heart are used.

Acting as a single unit, the right heart consists of the right atrium and right ventricle. The right heart's main function is to receive deoxygenated venous blood into the right atrium. From there, the right ventricle needs to generate only a minimal amount of pressure to pump the blood through the pulmonic valve into the pulmonary circulation. It is because of this that the right heart is considered a low pressure system.

The left heart is a similar unit which receives oxygenated blood from the pulmonary system. The left heart is considered a high pressure system since the left ventricle needs to generate a greater amount of pressure to pump blood through the aortic valve, into the aorta, and then through the systemic circulation.

The pulmonary capillary bed lies between the right and left heart. The capillary bed is a very compliant system with a high capacity to hold blood. Changes in this capacity can be detected through pressure changes seen for both sides of the heart.

The circulatory system therefore consists of two circuits in a series: pulmonic circulation, which is a low-pressure system with low resistance to blood flow; and the systemic circulation, which is a high-pressure system with high resistance to blood flow.

Hemodynamic events are classified as either systolic or diastolic. By convention, the terms usually depict ventricular activity. The atria have phases of systole and diastole as do the ventricles. Technically speaking, the left side of the heart is the first to begin and complete the systolic and diastolic phases. For ease of discussion and since the time difference is minimal, we will consider the right and left sides to function at the same time.

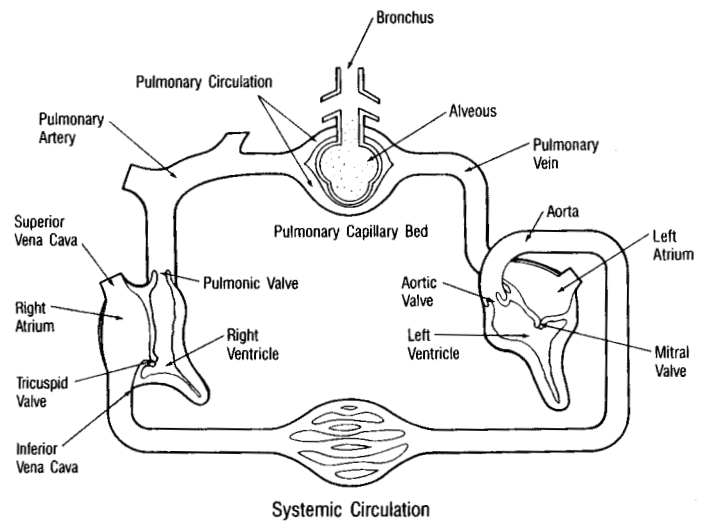


Figure 1
Anatomical Structure Location and Abbreviations

Right Heart

SVC - Superior Vena Cava
IVC - Inferior Vena Cava
RA - Right Atrium
RV - Right Ventricle
TV - Tricuspid Valve
PV - Pulmonic Valve
PA - Pulmonary Artery

Left Heart

LA - Left Atrium
LV - Left Ventricle
Ao - Aorta
MV - Mitral Valve
AoV - Aortic Valve

CARDIAC CYCLE

The cardiac cycle consists of nearly synchronized activity of the atria and ventricles. The sequence is essentially the same for the right and left sides of the heart. For general discussion, systole and diastole are the two basic phases. However, when examining the cycles closer there are many different sub-phases for both. The purpose of this section is to discuss the important phases of the cardiac cycle.

Historically, the ECG has been the basis for noting systole and diastole. For more precise identification of intracardiac waveforms, the delineation of electrical versus mechanical cardiac cycle is addressed here.

The first type of cardiac cycle that must occur is the electrical cardiac cycle. The initial phase is depolarization, which begins from the sinus node and spreads a wave of electrical current throughout the atria. This current is then transmitted throughout the ventricles. Following the wave of depolarization, muscle fibers contract, which produces systole.

The next electrical activity is repolarization which results in the relaxation of the muscle fibers and produces diastole. In the normal heart, initial electrical activity produces the mechanical activity of systole and diastole. There is a time difference between the two called electro-mechanical coupling, or the excitation-contraction phase. When looking at a simultaneous recording of the electrocardiogram and pressure tracing, the ECG will show the appropriate wave before the mechanical tracings will.

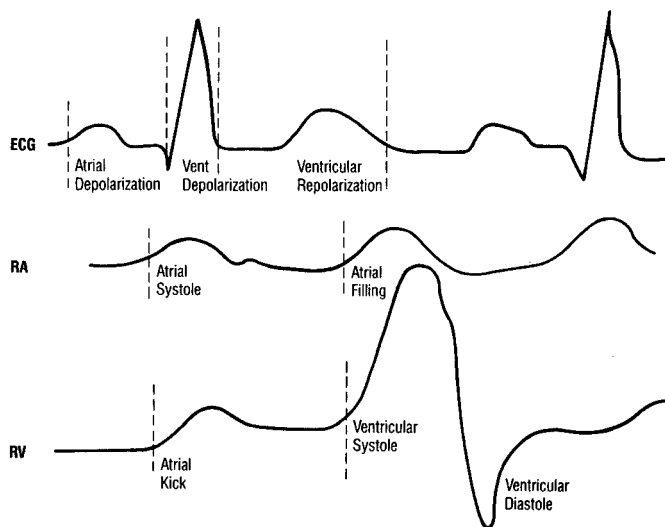


Figure 2
Electrical vs. Mechanical Cycle

As previously mentioned, systole and diastole are generally used in relation to ventricular activity, since it is the ventricles that are responsible for performing the pumping action. It is important to remember that while the ventricles are in systole, the atria are in diastole, and that while the ventricles are in diastole, the atria are in systole. This will become more apparent as we go through the various phases of the cardiac cycle.

The cardiac cycle is a continuous cycle of pressure changes and blood flow. It doesn't matter if the discussion begins with systole or diastole. For our purpose, we will begin with systole.

Systole

The first phase of systole is called the isovolumetric or isometric phase, which is shown on the pressure tracing. This phase occurs after the QRS wave, which is caused by ventricular depolarization of the ECG. All of the valves in the heart are closed at this time. The wave of ventricular depolarization brings about a shortening of the muscle fibers that results in an increase in pressure in the ventricles. Once this pressure exceeds the pressure in the pulmonary artery for the right ventricle and the aorta for the left ventricle, the aortic valve and the pulmonic valve open. It is during the isovolumetric phase that most of the oxygen supplied to the myocardium is consumed.

The second phase of systole is rapid ventricular ejection. Once the pulmonic and aortic valve open, the muscle fibers shorten even more, which may help to propel the blood volume out of the ventricles. It is during this phase that approximately 80-85% of the blood volume is ejected. The ECG correlation is during the ST segment.

As the pressure begins to equalize, the third phase of ventricular systole, or the reduced ventricular ejection phase, begins. This phase is a more gradual ejection with less volume.

During this phase, the atria are in diastole. There is an increase in atrial blood volume from pulmonary and venous inflow. This rise in volume creates a rise in pressure. The resultant rise in pressure is recorded as the "v" wave on the atrial waveform tracing.

At the end of the reduced ventricular ejection phase, most of the volume that will be ejected from the ventricles is now in the pulmonary artery and aorta. During this slower ejection period, aortic pressure and the pulmonary artery pressure are slightly higher than ventricular pressure. Blood begins to flow backwards into the ventricles. At the end of ventricular systole, ventricular pressure drops and semilunar valves close. This produces the S₂ sound that signifies the onset of diastole. The ECG correlation occurs during the T wave.

Diastole

The transition between systole and diastole is a result of the continuum of pressure changes within the heart and great vessels. As with systole, diastole is preceded by an electrical event known as repolarization. Following repolarization, the muscle fibers begin to relax.

The first phase of diastole is similar to the first phase of systole. Instead of isovolumetric contraction, there is isovolumetric relaxation. This phase follows the repolarization phase of the ventricles and is seen on the pressure waveform tracing following the T wave on the ECG.

As tension leaves the myocardium, there is now less pressure in the ventricles than in the atria. With this higher pressure in the atria, the AV valves now open, which leads into the second phase of diastole - rapid ventricular filling. During this phase, approximately two-thirds of the blood volume is passively moved from the atria into the ventricles.

The third phase is also called the slow or passive filling phase. During this phase, more atrial blood volume goes into the ventricle. In sinus or some atrial rhythms, atrial systole, which follows the P wave of atrial depolarization on the ECG, helps to push in the remaining one-third of ventricular volume. This wave is identified as the “a” wave for atrial pressure tracings and is seen only as a result of sinus or some atrial rhythms. At one point there is an equal amount of volume or pressure in the atria and ventricles. This phase is end diastole. Right after end diastole, more blood volume is in the ventricles. Since ventricular pressure exceeds atrial pressure, the AV valves begin to close. Once the AV valves close, the S₁ that is heard begins the next cycle of systole.

Systole

Isovolumetric Phase

- Follows QRS of ECG
- All valves are closed
- Majority of oxygen consumed

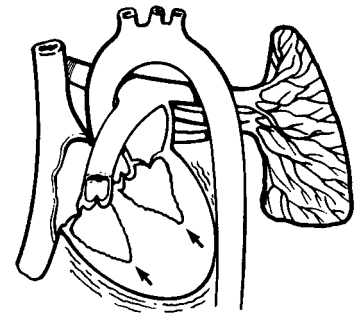


Figure 3a

Rapid Ventricular Ejection

- Occurs during ST segment
- 80% to 85% of blood volume ejected

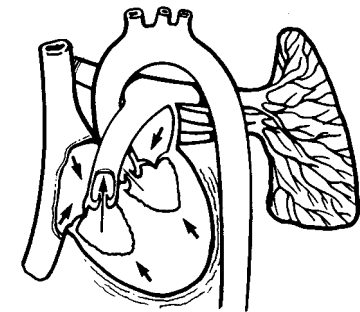
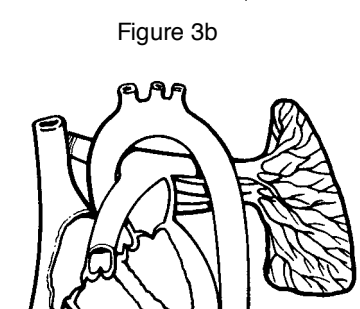


Figure 3b

Reduced Ventricular Ejection

- Occurs during T wave
- Atria are in diastole
- Produces “V” wave in atrial tracing



Diastole

1st Phase

Isovolumetric Relaxation

- Follows T wave
- All valves closed
- Ventricular pressure declines further
- Ends in the ventricular “diastolic dip”

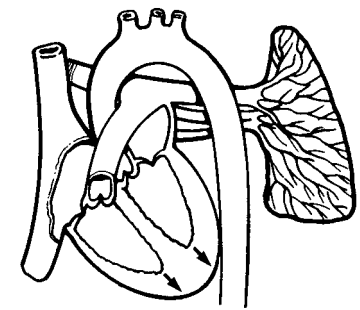


Figure 3c

2nd Phase

Rapid Ventricular Filling

- AV valves open
- Approximately two-thirds goes into ventricle

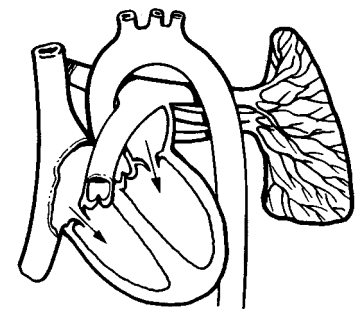


Figure 3d

3rd Phase

End Diastole – “Atrial Kick”

- Follows “P” wave during sinus rhythms
- Atrial systole occurs
- Produces “a” wave on atrial tracings
- Remaining volume goes into ventricle

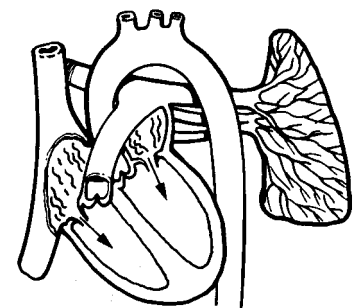


Figure 3e

Figure 3
Mechanical Cardiac Cycle

APPLICABLE CARDIAC PHYSIOLOGY

The heart's ability to act as a pump is closely regulated to ensure adequate supply to meet the metabolic needs of the tissues. Under certain physiological conditions, which can range from illness to exercise, the normal heart should compensate to meet the demands placed upon it.

Cardiac performance has four fundamental components: heart rate, preload, afterload, and contractility. In a diseased heart or altered circulatory system state, one or more of these determinants may be affected or altered in an attempt to maintain adequate cardiac performance.

In this section, each determinant of cardiac function will be discussed as to their regulatory actions and compensatory mechanisms.

Cardiac Output

Cardiac Output (in liters/minute) is defined as the amount of blood ejected from the ventricle (primarily the left ventricle) in a minute. Cardiac output is the term that is used when discussing the pumping effectiveness and ventricular function of the heart – the cardiac performance.

$$\text{Cardiac Output} = \text{Heart Rate} \times \text{Stroke Volume}$$

Where:

$$\text{Heart Rate} = \text{beats/min}$$

$$\text{Stroke Volume} = \text{amount of blood ejected from ventricle in one beat}$$

By altering either heart rate or stroke volume, cardiac output can be manipulated.

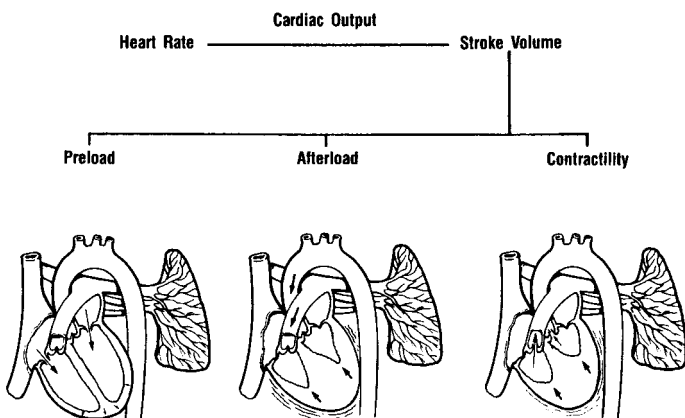


Figure 4

DETERMINANTS OF CARDIAC OUTPUT

Heart Rate

Most non-diseased hearts can tolerate heart rate changes from 40-170 beats per minute. As the cardiac function is more compromised, this range becomes narrower.

Elevated heart rates compromise cardiac output by: an increase in the amount of oxygen consumed by the myocardium, a reduction in diastolic time that can result in less perfusion time for the coronary arteries, and a shortened ventricular filling phase of the cardiac cycle resulting in decreased blood volume to pump on the next contraction.

Slow or decreased heart rate also cause some harmful effects. Initially, there is more filling time that should result in an increased cardiac output. However, even with this increase in volume, the myocardium may be so depressed that the muscle cannot contract strong enough to eject the increased volume. The result is a decreased cardiac output.

Stroke Volume

Stroke volume (SV) is the amount of blood ejected from the left ventricle each time the ventricle contracts. Stroke volume is the difference between end-diastolic volume (EDV), the amount of blood in the left ventricle at the end of diastole, and end-systolic volume (ESV), blood volume in the left ventricle at the end of systole. Normal stroke volume is 60 to 100 ml/beat.

$$SV = EDV - ESV$$

When stroke volume is expressed as a percentage of end diastolic volume, stroke volume is referred to as the ejection fraction (EF). Normal ejection fraction (left ventricle) is 65%.

$$EF = \frac{SV}{EDV} \times 100$$

Stroke volume, as a component of cardiac output, is influenced by the remaining three determinants of cardiac function: preload, afterload, and contractility. All three components are inter-related. In many instances, if one determinant is altered, so will another, and so down the line.

Frank-Starling Law

The relationship between the stroke volume and cardiac performance was described in the late 1890's and early 1900's by Drs. Frank and Starling.

The Frank-Starling law describes the relationship between myocardial muscle length and the force of contraction. Simply stated, the more you stretch the muscle fiber in diastole, or the more volume in the ventricle, the stronger the next contraction will be in systole. This law also states that this phenomenon will occur until a physiological limit has been reached. Once that limit has been reached, the force of contraction will begin to decline, regardless of the increase in amount of fiber stretch.

In the heart, it is this ability to increase the force of contraction that converts an increase in venous return to an increase in stroke volume. Stroke volume must match venous return or the heart will fail.

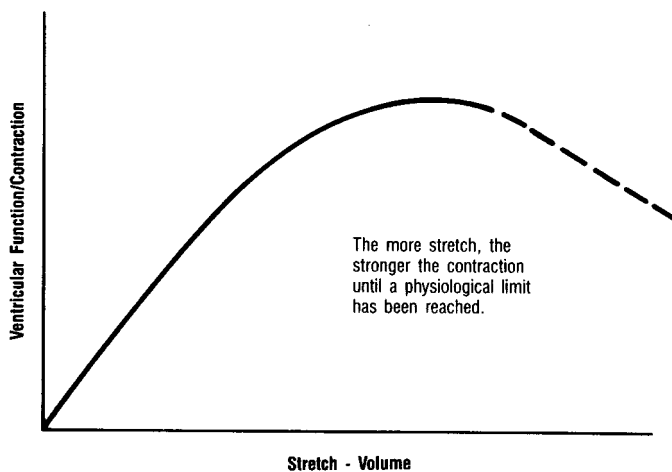


Figure 5
Fran-Starling Curve

Preload

Preload refers to the amount of myocardial fiber stretch at the end of diastole. Preload also refers to the amount of volume in the ventricle at this phase. It is very difficult to actually measure fiber length or volume at the bedside. It has been clinically acceptable to measure the pressure required to fill the ventricles (LVFP) as a measure of left ventricular end diastolic volume (LVEDV) or fiber length.

The actual relationship between end diastolic volume and end diastolic pressure is dependent upon the compliance of the muscle wall. The relationship between the two is curvilinear. With normal compliance, relatively large increases in volume create relatively small increases in pressure. Whereas in a non-compliant ventricle, a greater pressure is generated with very little increase in volume. Increased compliance of the ventricle allows for large changes in volume with little rise in pressure.

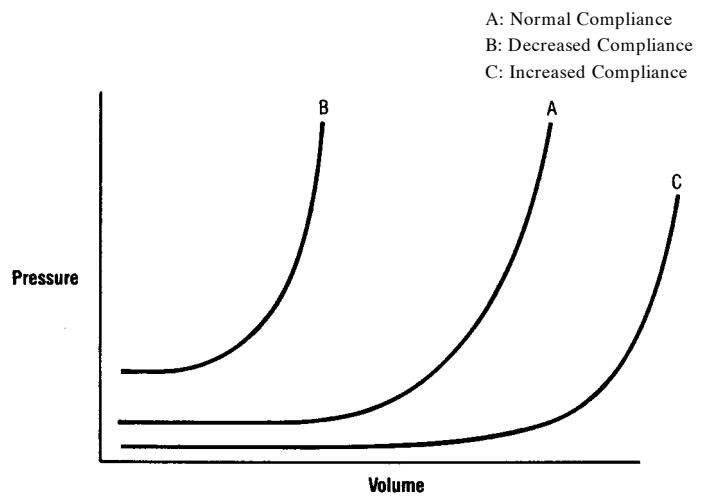


Figure 6
Compliance Curves

Afterload

Afterload refers to the resistance, impedance, or pressure that the ventricle must overcome to eject its blood volume. Afterload is determined by a number of factors, including: volume and mass of blood ejected, the size and wall thickness of the ventricle, and the impedance of the vasculature. In the clinical setting, the most sensitive measure of afterload is systemic vascular resistance (SVR) for the left ventricle and pulmonary vascular resistance (PVR) for the right ventricle. In reality, the resistance of the vascular system is derived from the measurements of cardiac output (CO) and mean arterial pressure (MAP). The formulas for calculating afterload look at the gradient difference between the beginning (inflow) of the circuit and the end (outflow) of the circuit.

$$\text{SVR} = \frac{(\text{MAP} - \text{RAP}) \times 80}{\text{CO}}$$

Normal value:
800 – 1200 dynes/sec/cm⁵

Where:

MAP = mean arterial pressure

RAP = right atrial pressure

$$\text{PVR} = \frac{(\text{MPA} - \text{PAW}) \times 80}{\text{CO}}$$

Normal value:
< 250 dynes/sec/cm⁵

Where:

MPA = mean pulmonary artery pressure

PAW = pulmonary artery wedge pressure

Afterload has an inverse relationship to ventricular function. As resistance to ejection increases, the force of contraction decreases. This results in a decreased stroke volume. The inter-relationship between afterload and stroke volume, as determinants of cardiac performance, are important ones.

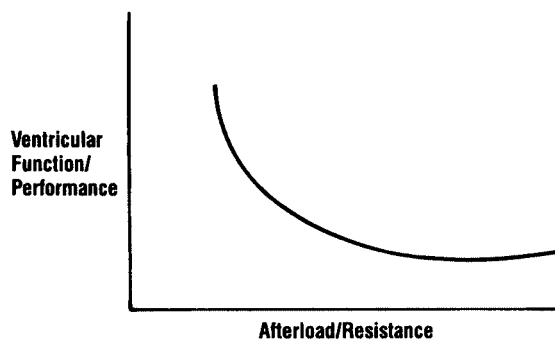


Figure 7
Afterload/Ventricular Function Curve

For the normal heart, if the resistance increases, there is little change in the stroke volume. As myocardial dysfunction increases, the rise in resistance produces a greater decrease in stroke volume. This dysfunction level is frequently a result of decreased contractility of the myocardium itself.

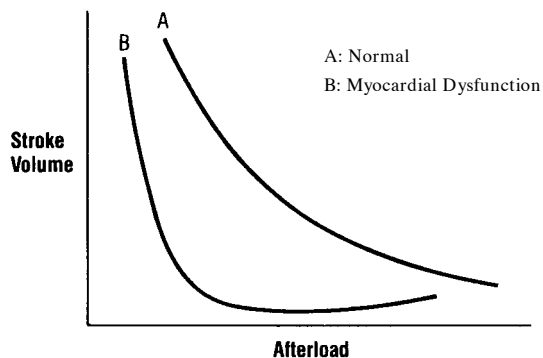


Figure 8
Stroke Volume - Afterload Curves

Contractility

Inotrophism refers to the inherent property of shortening of the myocardial muscle fibers without altering the fiber length or preload. There are multiple factors that influence the contractile state of the myocardium.

The most important of these influences is the effect of the sympathetic nervous system on the heart. There can be an instant increase in contractility, or a slower increase from the release of catecholamines. The increase in heart rate from the sympathetic nervous system may also increase contractility slightly. Other influences including metabolic changes such as acidotic states will decrease contractility. Drug therapy can be provided to elicit either a positive or negative inotropic state, depending on patient conditions or hemodynamic requirements.

As with the effects of changes in afterload on ventricular function, contractility changes can also be plotted on a curve. It is important to note that changes in contractility result in shifts of the curves, but not the underlying or basic shape.

Summary

Since ventricular function can be represented now by a family of curves, the performance characteristics of the heart can move from one curve to another, depending upon the balance of the four primary determinants of cardiac function. When evaluating the hemodynamic status of the patient, the inter-relationship of heart rate, preload, afterload, and the contractile state of the myocardium needs to be considered.

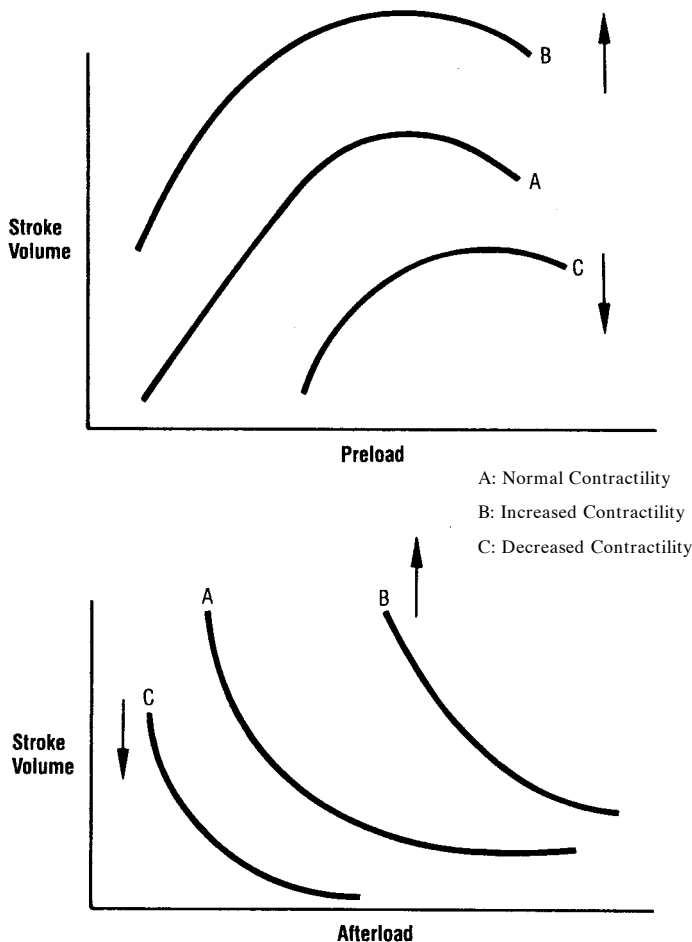


Figure 9

MYOCARDIAL OXYGEN CONSUMPTION

Myocardial oxygen consumption is the amount of oxygen utilized by the heart to function. The workload of the heart is costly, even during periods of rest. Normally, the myocardium consumes approximately 65% -80% of the oxygen it receives. At present, there is not a direct means to measure myocardial oxygen consumption.

Factors that affect myocardial oxygen consumption can be divided into a supply side and demand side. Since oxygen extraction cannot be greatly increased when the demand or workload increases, the only way to compensate is to increase blood flow, or the supply side.

Coronary artery perfusion for the left ventricle occurs primarily during diastole. The increase in ventricular wall stress during systole increases resistance to such an extent that there is very little blood flow into the endocardium. The right ventricle has less muscle mass and therefore less wall stress during systole so that due to less resistance, there is more blood flow through the right coronary artery and into the right ventricle during systole. There must be adequate diastolic pressure in the aortic root for both the coronary arteries to be perfused.

The demand side of myocardial oxygen consumption includes all of the determinants of cardiac performance. Manipulation of cardiac output is not without cost to the heart.

In many disease states, it is difficult to increase supply, whereas the demand factors may be greatly increased. Whenever there is an increase in demand, the risk of increasing myocardial oxygen consumption must be taken into consideration, since the myocardium has relatively no oxygen reserve.

Through hemodynamic monitoring, demand factors such as preload, afterload, contractility, and heart rate can be altered by various therapeutic interventions. These interventions and their effects will be addressed in a later section.

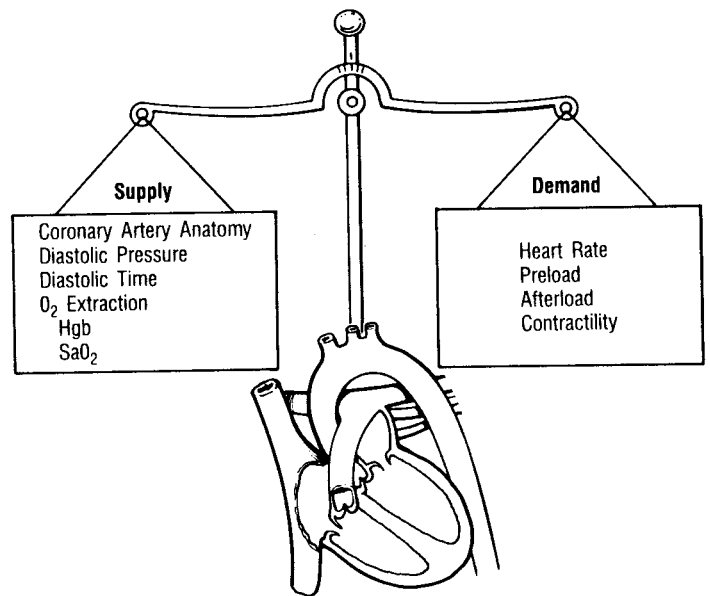


Figure 11

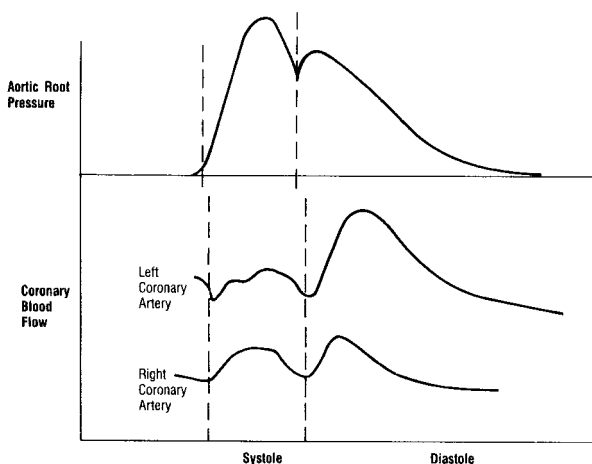


Figure 10
Coronary Artery Perfusion

PHYSIOLOGICAL BASIS OF PULMONARY ARTERY PRESSURE MONITORING

During the late 1960's and early 1970's, Drs. H.J.C. Swan and William Ganz developed a balloon-tipped flotation catheter. The function of the catheter was to be able to continuously measure at the bedside certain intracardiac pressures. Before this, to assess more complete cardiac function, the patient had to be transported to the catheterization laboratory, and only then intermittent values could be obtained.

By floating a flexible catheter into the pulmonary artery and using a balloon to occlude pulmonary artery pressures, left heart pressures could be reflected.

Since the initial catheter design, more advances have been made to allow for additional hemodynamic data such as right atrial pressures to be obtained. A thermistor was later added to allow for cardiac output determinations.

The four figures that follow show the rationale for the Swan-Ganz catheter. The figures represent proper catheter tip placement after insertion and, unless otherwise noted, the pressures recorded from the distal lumen.

Note: For a more detailed description of catheter insertion, please refer to Edwards product capsule, "Procedure for Insertion of Swan-Ganz Catheters."

In Figure 12, the balloon is deflated and the ventricles are in systole. During this phase of the cardiac cycle, the tricuspid and mitral valves are closed, while the pulmonic and aortic valves are open. The higher pressure generated by the right ventricle during contraction is transmitted to the catheter tip located in the pulmonary artery. The catheter records pulmonary artery systolic pressure (PASP), which reflects right ventricular systolic pressure (RVSP), because there is now a common chamber with a common volume and pressure.

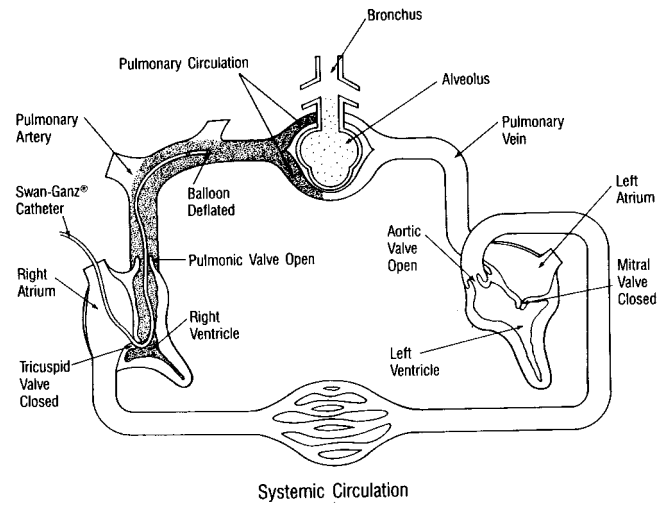


Figure 12
Ventricles in Systole

In Figure 13 during diastole, the tricuspid and mitral valves are open. The ventricles are filling with blood from their respective atria. At this time, the pulmonic valve and aortic valve are closed.

With the balloon still deflated, pulmonary artery diastolic pressure (PADP) is recorded. After the closure of the pulmonic valve, the right ventricle continues to relax. This causes a lower diastolic pressure in the right ventricle than in the pulmonary artery. RVDP is less than PADP.

Since there is normally no obstruction between the pulmonary artery and left atrium, the pressure recorded will be virtually the same as left atrial pressure. Over a pressure range of about 6 mm Hg to 20 mm Hg, this pressure is nearly the same as left ventricular diastolic pressure. $PADP = LAP = LVDP$

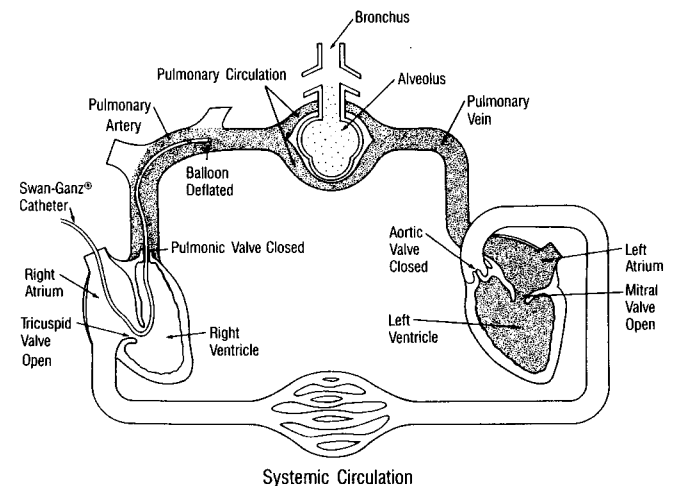


Figure 13
Ventricles in Diastole

As shown in Figure 14, the balloon is now inflated. By inflating the balloon, the catheter floats downstream into a smaller branch of the pulmonary artery. Once the balloon lodges, the catheter is considered “wedged.” It is in this wedge position that right side and PA diastole pressures are effectively occluded.

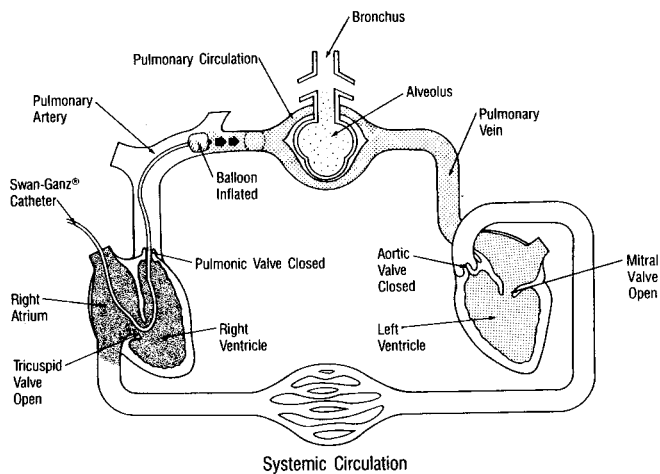


Figure 14
Ventricles in Diastole; Catheter Wedged

Because there are no valves between the pulmonic valve and mitral valve, and the pulmonary capillary bed is a compliant system, there is now an unrestricted vascular channel between the catheter tip in the pulmonary artery through the pulmonary vascular bed, the pulmonary vein, the left atrium, the open mitral valve and into the left ventricle. The distal lumen is now more closely monitoring left ventricular filling pressure or left ventricular end diastolic pressure.

Various terms have been used to describe this pressure. Previously, pulmonary capillary wedge pressure (PCWP) had been most common. In reality, the catheter does not float into a capillary so this has been a misnomer. A more correct term would be pulmonary artery wedge pressure (PAWP) or even pulmonary artery occlusion pressure (PAoP). Many institutions generally term the value, the “wedge.”

The importance of this pressure is that normally it closely approximates the pressure present in the left ventricle during end diastole, and provides a means of measuring ventricular preload.

With the addition of more lumens, such as a right atrial lumen to the catheter, more information regarding cardiac function can be obtained. During this phase of the cardiac cycle, the right atrial reading will reflect right ventricular end diastolic pressure or right ventricular preload due to the open tricuspid valve.

In Figure 15, the balloon is still in the “wedge” position and the ventricles are in systole. Since the balloon is occluding a branch of the pulmonary artery, the right ventricular pressures are effectively blocked. The distal lumen of the catheter now monitors the pressure reflected from filling of the left atrium. Pulmonary artery wedge pressure is also known as left atrial filling pressure (LAFP).

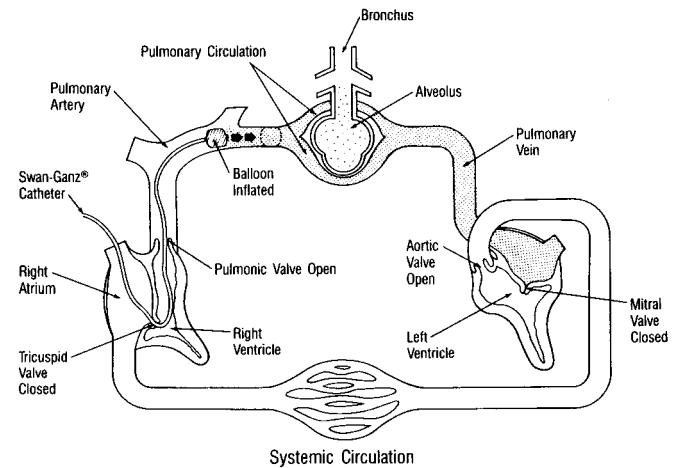


Figure 15
Ventricles in Systole; Catheter Wedged

INSERTION TECHNIQUES FOR THE SWAN-GANZ CATHETER

The previous sections discussed the physiological basis of hemodynamic monitoring. This section will describe insertion techniques, characteristic waveforms seen during insertion, and benefits of continuous pulmonary artery monitoring.

Before insertion of the Swan-Ganz catheter, prepare the pressure monitoring system for use according to the institution's own policies and procedures.

Insert the catheter following recommended guidelines, and advance the catheter towards the thorax.

Common insertion sites for percutaneous approach include the internal jugular vein, subclavian vein, and femoral vein. Either the right or left antecubital fossa may be used for a cut-down approach. The subclavian vein approach may cause kinking of the catheter due to anatomical differences and a bending of the sheath introducer. Kinking of the catheter can cause damped waveforms. Repositioning of the catheter may be necessary.

Catheter Insertion Sites

Location	Distance Until Vena Cava/RA junction (cm)
Internal jugular	15 to 20
Superior Vena Cava	10 to 15
Femoral Vein	30
Right Antecubital Fossa	40
Left Antecubital Fossa	50

Catheter advancement to the pulmonary artery should be rapid, since prolonged manipulation can result in loss of catheter stiffness.

Generally, fluoroscopy is not required for insertion of the Swan-Ganz thermodilution catheter for primarily two reasons. First, the catheter is designed to be flow-directed when the balloon is inflated. During insertion, the inflated balloon allows the catheter to follow the venous blood flow from the right heart into the pulmonary artery.

Second, the chambers on the right side of the heart have characteristic pressures and waveforms. The pulmonary artery also has typical waveforms and pressures. By

visualizing on a monitor the various waveforms and pressures, catheter tip location can be determined.

Next, normal waveform characteristics and pressures noted during insertion of the Swan-Ganz catheter will be described. For clarity, the waveforms are idealized.

Once the catheter tip has reached the junction of the superior or inferior vena cava and right atrium, the balloon is inflated with air or CO₂ to the full volume indicated on the catheter shaft (7 to 7.5F; 1.5 cc). This position can be noted when respiratory oscillations are seen on the monitor screen. The first chamber reached is the right atrium. Pressures are normally low and will produce two small upright waves.

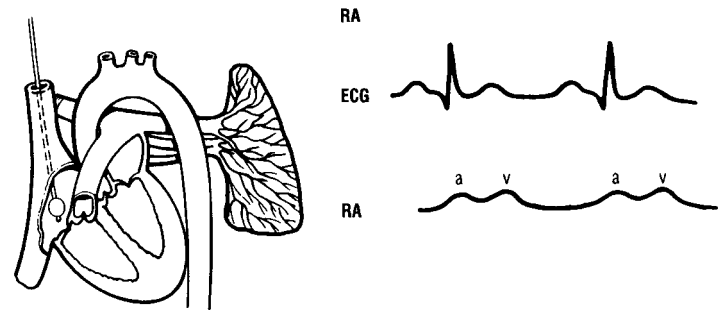


Figure 16
Right Atrial Waveform

The next chamber is the right ventricle. Waveforms show taller, sharp uprisings as a result of ventricular systole and low diastolic dips and values. The systolic pressure is higher in the right ventricle, with the diastolic value being nearly the same as the right atrial pressure value. When the catheter has passed the tricuspid valve, special attention should be paid to the patient's ECG to identify any ventricular ectopy that may occur.

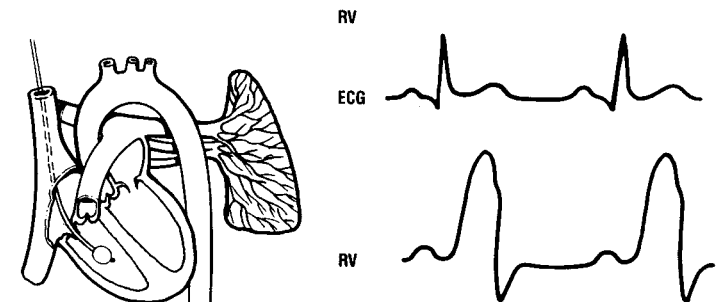


Figure 17
Right Ventricular Waveform

As the catheter floats into the pulmonary artery (**not in a wedge position**), characteristic waveforms can again be noted.

As a result of right ventricular systole, there is a rise in pressure in the pulmonary artery. This pressure is recorded as being almost the same as right ventricular systolic pressure. The waveform produced has a large excursion with the upward slope being more rounded than the right ventricular tracing.

The onset of diastole begins with the closure of the pulmonic valve, which produces a dicrotic notch on the pulmonary artery tracing. Diastole continues in the ventricles. Once the pulmonic valve closes, and since the pulmonary artery does not relax further, the diastolic pressure is higher in the pulmonary artery than in the right ventricle.

Because diastolic pressures will be higher in the pulmonary artery than in the right ventricle, special attention should be paid to observing diastolic pressures during insertion. Right ventricular systolic and pulmonary artery systolic pressures are nearly the same. If monitoring them during insertion, distinguishing catheter tip location between the right ventricle and pulmonary artery may be more difficult. By observing the diastolic pressures, a rise in pressure value will be noted when the pulmonary artery has been reached.

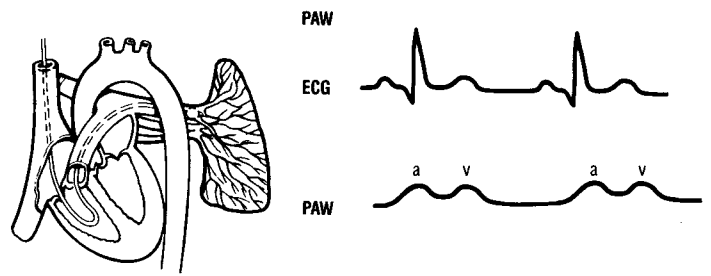


Figure 19
Pulmonary Artery Wedge Waveform



Figure 20
Normal Insertion Tracings

Table 1.

Typical Hemodynamic Pressure Values

Location	Normal Values in mm Hg
Right Atrium	
Right Atrial (RAP)	-1 to +7
Mean (MRAP)	4
Right Ventricle	
Systolic (RVSP)	15 to 25
Diastolic (RVDP)	0 to 8
Pulmonary Artery	
Systolic (PASP)	15 to 25
Diastolic (PADP)	8 to 15
Mean (MPAP)	10 to 20
Wedge (PAWP)	6 to 12
Left Atrial (LAP)	6 to 12

Once the wedge position has been identified, the balloon is deflated by removing the syringe and allowing the back pressure in the pulmonary artery to deflate the balloon. Once the balloon has been deflated, reattach the syringe to the gate valve. To reduce or remove any redundant length or loop in the right atrium or ventricle, slowly pull the catheter

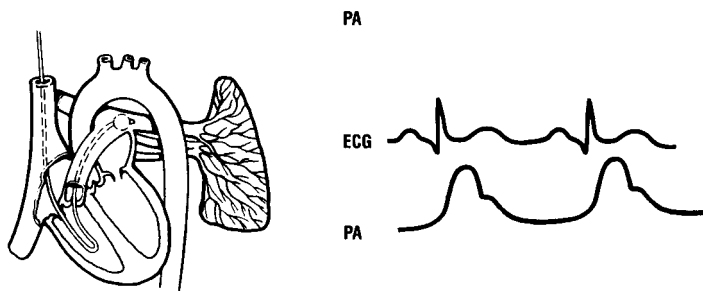


Figure 18
Pulmonary Artery Waveform

The catheter, with the balloon still inflated, is now advanced further until it finally wedges in a central branch of the pulmonary artery. At this point, right heart pressures and pulmonary influences are occluded. The catheter tip is “looking” at left heart pressures. The waveform reflected will be that of the left atrium. The pressures recorded will be slightly higher than the right atrium (6 mm Hg to 12 mm Hg). The waveform will have two small rounded excursions from left atrial systole and diastole.

The value recorded will also be slightly less than the pulmonary artery diastolic pressure. Pulmonary artery diastolic pressure is higher than pulmonary artery wedge pressure by 1 mm Hg to 4 mm Hg, typically.

back 1 cm to 2 cm. Then reinflate the balloon to determine the **minimum** inflation volume necessary to obtain a wedge pressure tracing. The catheter tip should be in a position where the full or near-full inflation volume (1.0 cc to 1.5 cc) produces a wedge pressure tracing.

Continuous pulmonary artery tracings and pressures can now be monitored. Since there is always a potential risk of pulmonary artery damage during wedging, in most situations monitoring of the PAD will reflect PAW values.

Continuous Pressure Monitoring

To obtain reliable values and clearly identifiable waveforms, the pressure monitoring system must be optimal. This includes pressure transducers of high quality, and properly calibrated and zeroed monitors and pressure monitoring systems. For monitor calibration, use procedures recommended by the monitor manufacturer.

To continuously monitor pulmonary artery pressures, a system utilizing heparinized solution is required to maintain catheter lumen patency. Many different types of systems exist that are configured to monitor various combinations of pressure.

Refer to Edwards Transducer Product Information Data Sheet for proper line setup and guidelines.

Continuous monitoring of the pulmonary artery pressure is important for several reasons. First, therapy will depend upon the values obtained from the pulmonary artery and intermittent wedge recordings. Second, catheter tip position changes can present potential risks to the patient.

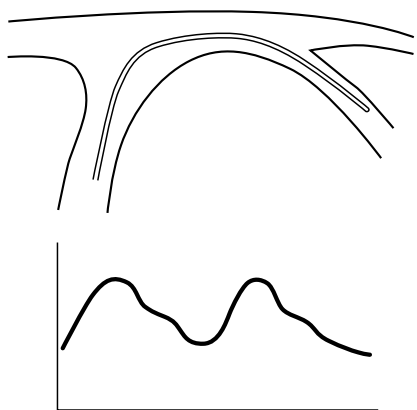


Figure 21
Proper PA Position

The catheter may spontaneously migrate into a more distal pulmonary artery branch when the balloon is deflated. This migration may occur at any time, but more frequently occurs within the first few hours of insertion.

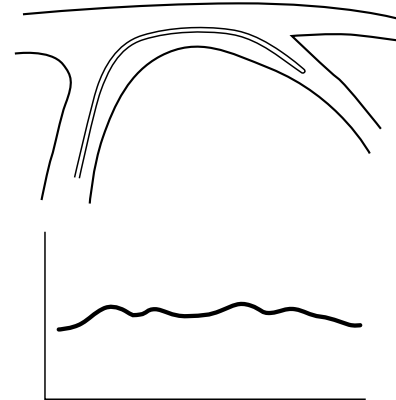


Figure 22
Catheter Migration

This migration is due primarily to the softening of the catheter once it has been warmed to body temperature. During insertion, there may have been an excess of catheter in the right ventricle. This excess, when sufficiently softened, can cause the catheter to float out further.

One technique used to help prevent this cause of migration is to decrease the amount of curl in the right ventricle by pulling the catheter back 1 cm to 2 cm, after obtaining the appropriate wedge. Once this has been done, re wedge to ascertain proper tip position with full or near full inflation volume.

When the catheter is only partially wedged, the pulmonary artery systolic pressure will be lower than previously recorded. The clarity of the tracing may also be lost. If the catheter has become completely wedged, the characteristic wedge tracing will be observed.

If the catheter, with the balloon deflated, is either partially or completely wedged, the catheter should be withdrawn in 1 cm to 2 cm increments until a clear pulmonary artery tracing is recorded. Subsequent wedgings should require near maximal balloon inflation volumes.

Occasionally, the catheter tip may slip back into the right ventricle. This can be recognized by observing characteristic right ventricular tracings on the monitor. The systolic pressures will remain relatively the same, whereas the diastolic pressure will be lower than the previously recorded pulmonary artery diastolic pressure.

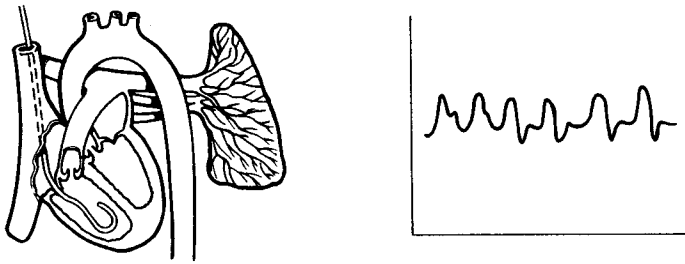


Figure 23
Catheter Tip in Right Ventricle

If this occurs, the balloon should be inflated to protect the right ventricle from irritability. Because this is a flow-directed catheter, merely inflating the balloon may again float the catheter into the pulmonary artery. At times, changing the patient's position may result in obtaining proper catheter tip location.

If the pulmonary artery cannot be reached by these maneuvers, readvancement of the catheter should be attempted only if sterility of the exposed catheter segment has been maintained. If sterility has not been maintained, the catheter should be withdrawn, and if still required, replaced by a second catheter.

Whenever the catheter is withdrawn from the pulmonary artery to the right ventricle, and also from the right ventricle to the right atrium, the balloon should be deflated to minimize valvular trauma.

As with observing proper catheter tip location, proper balloon inflation techniques are also important. Overinflation of the balloon may cause overdilatation of the pulmonary artery, which can cause rupture of the vessel.

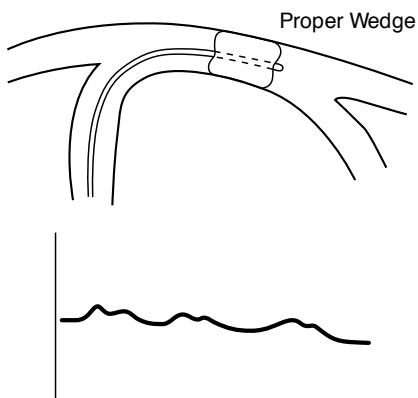


Figure 24
Wedging Techniques - Proper Wedge

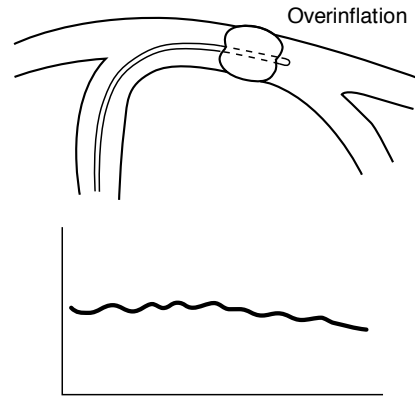


Figure 25
Wedging Techniques - Overinflation

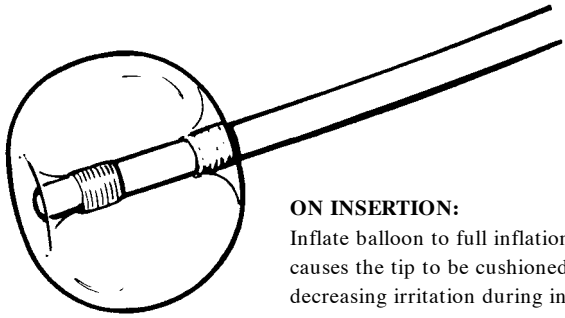
The balloon should always be inflated slowly while the waveform tracing is noted on the monitor. Once the characteristic wedge tracing is observed, inflation of the balloon should cease immediately. **Maximum balloon inflation volume should never be exceeded.**

If the wedge tracing is recorded at a lower balloon volume (typically less than 1.5 cc), the catheter may have migrated into a distal location. Withdrawal of the catheter tip to a central pulmonary artery location is required.

Also, if a pulmonary artery wedge tracing is observed at a low inflation volume, and inflation is continued, the resulting pressure may become progressively higher with a loss of clarity to the waveform. This occurrence is termed "overwedged" and can be related to the pressure from the overinflated balloon being transmitted into the catheter's distal lumen.

A high quality and optimized pressure monitoring system, along with proper catheter tip location in a central branch of the pulmonary artery, provides the means to evaluate pulmonary artery pressures accurately. With these values obtained, appropriate therapeutic interventions can be instituted.

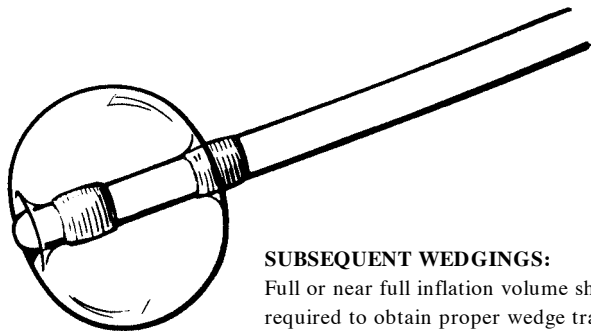
The following summary guidelines are provided to enhance correct and safe use of balloon-tipped pulmonary artery catheters.



ON INSERTION:

Inflate balloon to full inflation volume. This causes the tip to be cushioned to assist in decreasing irritation during insertion. This also helps to position the tip of the catheter in the proper central pulmonary artery location.

Figure 26
Balloon at Full Inflation Volume



SUBSEQUENT WEDGINGS:

Full or near full inflation volume should be required to obtain proper wedge tracings. If less than 1.25 to 1.5 cc of air is used to produce an appropriate wedge tracing, then repositioning of the catheter is required.

Figure 27
Balloon at Less Than Full Inflation Volume

SUMMARY GUIDELINES FOR SAFE USE OF BALLOON-TIPPED PULMONARY ARTERY CATHETERS

1. Keep catheter tip centrally located in a main branch of the pulmonary artery.

- During insertion, inflate the balloon to the full recommended volume (1.5 cc) and advance the catheter to a pulmonary artery wedge position. Deflate the balloon.
- To reduce or remove any redundant length or loop in the right atrium or ventricle, slowly pull the catheter back 1 cm to 2 cm.
- Do not advance the catheter tip too far peripherally. Ideally, the catheter tip should be located near the hilum of the lungs. Remember, the tip migrates towards the periphery of the lungs during balloon inflation. Therefore, a central location before inflation is important.
- Keep the tip at all times in a position where a full or near full (1.0 cc to 1.5 cc) inflation volume is necessary to produce a “wedge” tracing.

2. Anticipate spontaneous catheter tip migration toward the periphery of the pulmonary bed:

- Reduce any redundant length or loop in the right atrium or ventricle at the time of insertion to prevent subsequent peripheral migration (see No. 1).
- Monitor the distal tip pressure continuously to ensure that the catheter is not inadvertently wedged with the balloon deflated (this may induce pulmonary infarction).
- Check catheter position daily by chest X-ray film to detect peripheral placement. If migration has occurred, pull the catheter back to a central pulmonary artery position, carefully avoiding contamination of the insertion site.
- Spontaneous catheter tip migration towards the periphery of the lung occurs during cardiopulmonary bypass (Ref. 18). Partial catheter withdrawal (3 cm to 5 cm) just before bypass should be considered, as

withdrawal may help reduce the amount of distal migration and may prevent permanent catheter wedging in the postbypass period (Ref. 18). After termination of bypass, the catheter may require repositioning. Check the distal pulmonary artery tracing before inflating the balloon.

3. Exercise caution when inflating the balloon:

- If “wedge” is obtained at volumes less than 1.0 cc, pull the catheter back to a position where the full or near-full inflation volume (1.0 cc to 1.5 cc) produces a wedge pressure tracing.
- Check the distal pressure waveform before inflating the balloon. If the waveform appears dampened or distorted, do not inflate the balloon. The catheter may be wedged with the balloon deflated. Check catheter position.
- When the balloon is reinflated to record wedge pressure, add the inflation medium (CO₂ or air) **slowly** under continuous monitoring of the pulmonary artery pressure waveform. Stop inflating **immediately** when the pulmonary artery tracing is seen to change to pulmonary artery wedge pressure. Remove the syringe to allow rapid balloon deflation, then reattach the syringe to the balloon lumen. Air should never be used for balloon inflation in any situation where air may enter the arterial circulation (see **Insertion Procedure**).
- Never over-inflate the balloon beyond the maximum volume printed on the catheter shaft (1.5 cc). Use the volume-limited syringe provided with the catheter.
- Do not use liquids for balloon inflation; they may be irretrievable and may prevent balloon deflation.
- Keep the syringe attached to the balloon lumen of the catheter to prevent accidental injection of liquids into the balloon.

4. Obtain a pulmonary artery occlusion “wedge” pressure only when necessary:

- If the pulmonary artery diastolic (PAD) and the wedge (PAW) pressures are nearly identical, wedging the balloon may not be necessary: measure PAD pressure instead of PAW as long as the patient’s heart rate, blood pressure, cardiac output, and clinical state remain stable. However, in states of changing pulmonary arterial and pulmonary venous tone (i.e., sepsis, acute respiratory failure, shock), the relationship between

PAD and “wedge” may change with the patient’s clinical condition. PAW measurement may be necessary.

- Keep “wedge” time to a minimum (two respiratory cycles or 10 to 15 seconds), especially in patients with pulmonary hypertension.
- Avoid prolonged maneuvers to obtain wedge pressure. If difficulties are encountered, give up the “wedge.”
- Never flush the catheter when the balloon is wedged in the pulmonary artery.

5. Patients at highest risk of pulmonary artery rupture or perforation are elderly patients with pulmonary hypertension. These are usually elderly patients who are undergoing cardiac surgery with anticoagulation and hypothermia. Proximal catheter tip location near the hilum of the lungs may reduce the incidence of pulmonary artery perforation (Ref. 21).

CARDIAC OUTPUT DETERMINATIONS

Capabilities of the Swan-Ganz catheter discussed so far are: pulmonary artery pressure monitoring, ability to monitor right atrial pressures, and obtaining pulmonary artery wedge recordings. Another capability which adds to the hemodynamic picture is obtaining cardiac output determinations by the thermodilution method. By including cardiac output, a more complete assessment of cardiac performance can be obtained.

There are three indirect methods for cardiac output determinations: the Fick method, the dye indicator dilution method, and the thermodilution indicator method. The first two are primarily performed in a controlled catheterization laboratory setting. The last, the thermodilution method, is most readily used at the bedside. This section will discuss the various techniques used for cardiac output determination, with the main focus being on the thermodilution method.

The Fick Method

The “gold standard” for cardiac output determinations is based on the principles developed by Adolph Fick in the 1870’s. Fick’s concept proposes that the uptake or release of a substance by an organ is the product of blood flow through that organ and the difference between the arterial and venous values of the same substance .

The Fick method utilizes oxygen as the substance and the lungs as the organ. Arterial oxygen content and venous oxygen content are measured to obtain the arterial and venous oxygen (A – v O₂) difference. Oxygen consumption can be calculated from the inspired minus expired oxygen content and ventilation rate. The cardiac output can then be determined using this formula:

$$CO = \frac{\text{Oxygen Consumption in ml/min}}{A - v O_2 \text{ difference}}$$

- Normal arterial oxygen content is 20 volume % (volume % = I ml oxygen/100 cc)
- Normal mixed venous oxygen content is 15 volume % (volume % = I ml oxygen/100 cc)
- Normal oxygen consumption is 250 ml/min

Inserting these values into the equation:

$$\begin{aligned} CO &= 250 \text{ ml/min} \times 100/20 - 15 \text{ vol \%} \\ &= 5000 \text{ ml/min or } 5 \text{ l/min} \end{aligned}$$

Even though the Fick method has been described as the “gold standard”, there are many drawbacks to this technique. The patient must be in a steady physiological state at the time of the procedure. Most patients requiring cardiac output determinations are critically ill, which is frequently defined as an “unsteady state.” Other technique related drawbacks are the requirements of simultaneous expired air and blood samples, controlled inspired oxygen content, and arterial blood sampling.

The Fick method is one of the most accurate methods for patients who have especially low cardiac output states. But for technique requirements, it is one of the most clinically impractical.

Dye Indicator Dilution Method

Another indirect method for determining cardiac output is the dye indicator or indicator dilution method. Principles providing the basis for this method were first proposed in the 1890’s by Stewart, and later refined by Hamilton.

The basis of the dye indicator technique is that a known concentration of an indicator is added to a body of fluid, after allowing adequate mixing time, the dilution of that indicator will produce the amount of fluid it was added to. Simply stated, the indicator method can determine cardiac output by means of a downstream device called a densitometer. This device records the dye or indicator concentration in the blood after a known dye sample was injected upstream.

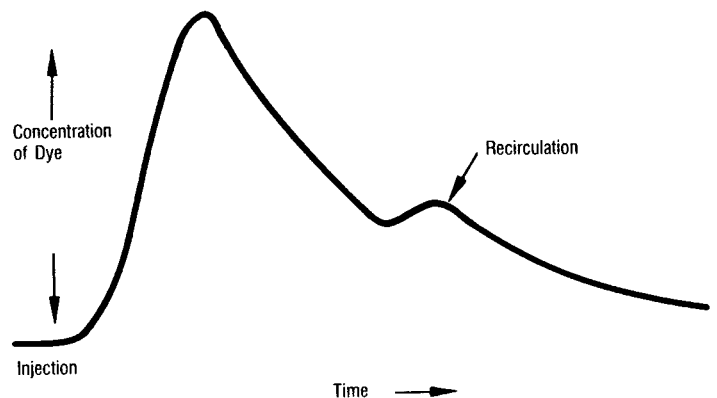


Figure 28
Indicator Dilution Curve

By taking continuous blood samples, a time-concentration plot, called an indicator-dilution curve can be obtained. Once this is plotted, the cardiac output can be calculated using the Stewart-Hamilton Equation:

$$CO = \frac{I \times 60}{C_m \times t} \times \frac{1}{k}$$

Where:

- CO = cardiac output (l/min)
- I = amount of dye injected (mg)
- 60 = 60 sec/min
- C_m = mean indicator concentration (mg/l)
- t = total curve duration (sec)
- K = calibration factor (mg/ml/mm deflection)

This method of cardiac output is more accurate in high cardiac output states. The technique, if it is to be performed accurately, requires complex equipment skills, and, therefore, is also not a clinically practical method.

Thermodilution Method

In the early 1950's, Fegler first described measuring cardiac output by the thermodilution method. It was not until the early 1970's that Drs. Swan and Ganz demonstrated reliability and reproducibility of this method with a special temperature sensing pulmonary artery catheter. Since that time, the thermodilution method of obtaining cardiac output has become a standard for clinical practice.

The thermodilution method applies indicator dilution principles, using temperature change as the indicator. A known amount of solution with a known temperature is injected rapidly into the right atrial lumen of the catheter. This cooler solution mixes with and cools the surrounding blood, and the temperature is measured downstream in the pulmonary artery by a thermistor bead embedded in the catheter. The resultant change in temperature is then plotted on a time-temperature curve. This curve is similar to the one produced by the indicator-dilution method.

A normal curve characteristically shows a sharp upstroke from rapid injection of the injectate. This is followed by a smooth curve and slightly prolonged downslope back to the baseline. Since this curve is representing a change from warmer temperature to cooler and then back to warmer temperature, the actual curve is in a negative direction. For continuity of most graphs, the curve is produced in an upright fashion. The area under the curve is inversely proportional to the cardiac output.

When cardiac output is low, more time is required for the temperature to return to baseline, producing a larger area under the curve. With high cardiac output, the cooler injectate is carried faster through the heart, and the temperature returns to baseline faster. This produces a smaller area under the curve.

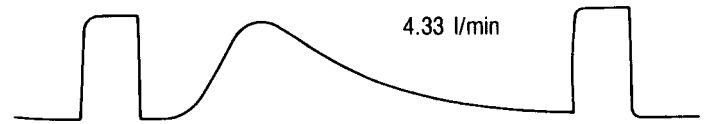


Figure 29
Normal Cardiac Output

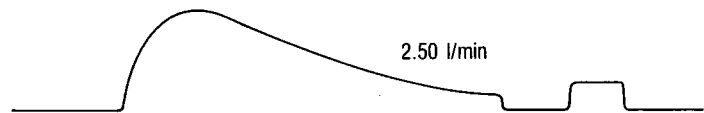


Figure 30
Low Cardiac Output



Figure 31
High Cardiac Output

A modified Stewart-Hamilton equation is used to calculate the cardiac output taking into consideration the change in temperature as the indicator, modifications include the measured temperature of the injectate and the patient's blood temperature, along with the specific gravity of the solution injected.

$$CO = \frac{V \times (T_B - T_I)}{A} \times \frac{(S_I \times C_I)}{(S_B \times C_B)} \times \frac{60 \times C \times K}{1}$$

Where:

- CO = cardiac output
- V = Volume of injectate (ml)
- A = area of thermodilution curve in square mm divided by paper speed (mm/sec)
- K = calibration constant in mm/°C
- T_B, T_I = temperature of blood (B) and injectate (I)
- S_B, S_I = specific gravity of blood and injectate
- C_B, C_I = specific heat of blood and injectate

$$\frac{(S_I \times C_I)}{(S_B \times C_B)} = 1.08 \text{ when 5\% dextrose is used}$$

60 = 60 sec/min

C_T = correction factor for injectate warming

The thermistor port of the catheter is attached to a computer or monitor. Calculations are performed internally with the results displayed on the screen. Some computers and monitors can also display the actual cardiac output time-temperature curve. By observing the actual

thermodilution curve, assessment of injection technique and artifactual influences can be noted.

The temperature of the injectate can be iced or room temperature. Available data suggest that there will be less variability in cardiac output determinations if iced solution is used. The computer is registering a change (signal) in temperature from the patient's baseline (noise). In some conditions, a variation in temperature of 0.05°C may occur with respirations. This decreases the "signal-to-noise" ratio and may produce an abnormally low cardiac output value. Other conditions where an increased signal to-noise ratio may be beneficial is febrile patients, low cardiac output states, and patients with wide respiratory variations.

There are two primary injectate delivery systems. One is an open system that utilizes prefilled syringes, either iced or room temperature. The other is a closed system, either for ice or room temperature, that is maintained in a closed-loop fashion to reduce multiple entry into a sterile system. The user may consult the references on the use of closed versus open injectate delivery systems.

Conditions where the thermodilution method may produce unreliable results are those that have a backward flow of blood on the right side; tricuspid or pulmonic valve regurgitation, and ventricular or atrial septal defects.

The advantages of this technique over the other methods previously mentioned are the reliability and ease of performing at the bedside. Also, serial cardiac outputs are able to be performed rapidly, approximately every minute, without requiring blood sampling.

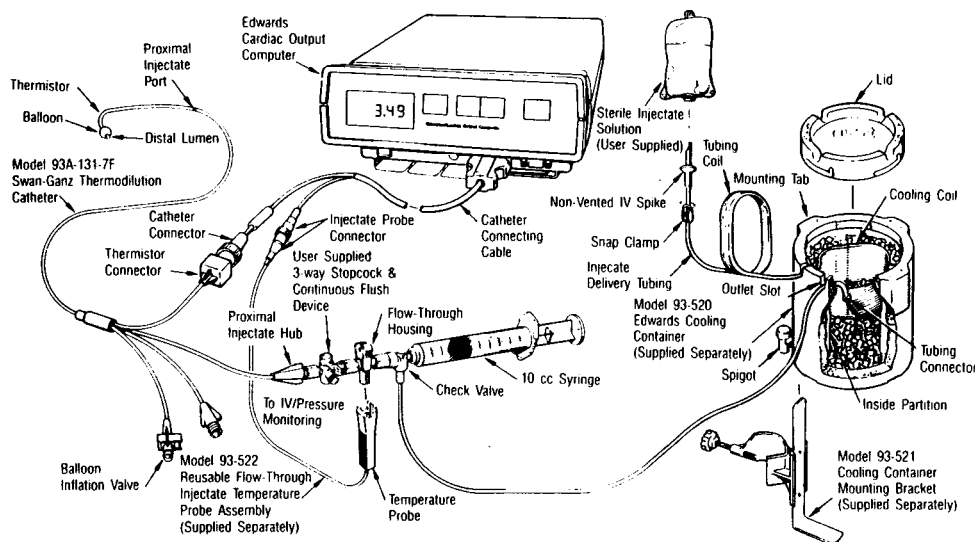


Figure 32
Closed Injectate Delivery System, Cold

CLINICAL APPLICATIONS

The Swan-Ganz thermodilution catheter is a powerful tool for the clinician in assessment and management of the critically ill patient. Use of the catheter by itself is not an intervention. Instead, it is a diagnostic adjunct that if utilized and the data obtained interpreted properly leads to appropriate therapeutic interventions.

Evaluation of Cardiac Performance

The heart's ability to function as a pump can be evaluated through the use of the Swan-Ganz catheter. During end diastole, under most conditions, ventricular preload is indirectly reflected to their respective atria. Left ventricular preload can be evaluated by observing the PADP, or more accurately the PAWP, since while the catheter is wedged, left heart pressures are reflected.

As left ventricular function deteriorates, end diastolic pressure (preload) increases. This increase is reflected back to the atria where for the left heart the PAWP will also be recorded higher. Cardiac output will decline as a result, and clinically the patient will exhibit signs of poor organ perfusion.

It was thought that by obtaining central venous pressures (CVP), the left heart function could be assessed. At that time, the only readily available monitoring means was the CVP catheter. Since the utilization of the pulmonary artery catheter, this concept of CVP for the left heart pressure monitoring has been dispelled.

By using the right atrial (RA) port of a triple-lumen catheter instead of a central venous line, the right ventricular (RV) preload status can be assessed. Increased RV preload, as a result of severe pulmonary disease or RV dysfunction, will be reflected as an elevated RA pressure. There are certain pulmonary conditions and right heart disease states where only right-sided pressures will be abnormal. It is for these conditions that again the CVP catheter is inadequate for assessing left ventricular function.

Both right and left ventricular function can be assessed when using a triple-lumen Swan-Ganz catheter. The information obtained from the PAWP reflects left heart function, while the RA lumen reflects right heart function. Under conditions of severe left heart failure and resultant right heart failure, both values will be elevated.

Significance of Hemodynamic Measurements

The determinants of cardiac performance are heart rate (HR), preload, afterload, contractility. Through hemodynamic monitoring not only can cardiac output be determined, but, by indirect calculations, other performance factors can be assessed.

Direct Measurements

Hemodynamic assessment of the patient can include both invasive and noninvasive parameters. From direct measurements, cardiac performance can be further evaluated by calculating derived parameters.

• Heart Rate

One of the more easily obtained values for assessing hemodynamic status is the heart rate. As a component of cardiac output, the heart rate plays an integral part as to diastolic filling time and therefore end diastolic volume. The heart rate can either be palpated or obtained from the ECG monitor.

• Systolic and Diastolic Blood Pressures

Blood pressure is the measured tension within the blood vessels during ventricular systole and diastole. This measurement can be obtained indirectly with a sphygmomanometer or more accurately with an intra-arterial catheter.

• Pulmonary Artery Pressures

With the use of a Swan-Ganz catheter, pulmonary artery systolic and diastolic pressures can be obtained, as can pulmonary artery wedge pressure values. The PASP reflects the pressure generated by the RV during systole. The PADP reflects the higher diastolic pressure in the pulmonary vasculature. Once the catheter has been wedged, right heart and pulmonary influences are occluded, and left atrial pressures are reflected.

• Right Atrial Pressure

Right ventricular filling pressures, in mm Hg instead of cm H₂O as with a central venous line, can be obtained by using the RA lumen of the Swan-Ganz catheter. This value provides information as to right ventricular function.

• Waveform Analysis

Measurement of the “a” and “v” wave of the RA and PAW tracings can provide valuable information as to filling pressures and disease states.

• Cardiac Output

Through the use of a thermodilution catheter, cardiac output determinations can be made at the bedside with relative ease and accuracy. The amount of blood being pumped by the heart, in l/min, helps provide an overall assessment of cardiac performance.

Derived Parameters

From the direct measurements obtained, derived parameters can be calculated to further assess cardiac performance and normalize values obtained for body size.

• Mean Arterial Pressure

This is the average pressure through the vascular system during systole and diastole. The maintenance of a minimal pressure is necessary for coronary artery and tissue perfusion. This value can be measured electrically or approximately by the following formula:

$$\text{MAP} = \frac{\text{SBP} + (\text{DBP} \times 2)}{3}$$

Normal MAP = 70 to 105 mm Hg

Where:

SBP = Systolic Blood Pressure
DBP = Diastolic Blood Pressure
MAP = Mean Arterial Pressure

• Cardiac Index

The normal range for cardiac output is wide, 4 l/min to 8 l/min. Since the value assesses the function of the ventricle, normalizing the value to the body size can offer more precise information. To index a hemodynamic value, the patient’s body surface area (BSA) is obtained from a nomogram using the patient’s height and weight. Any value that is to be indexed can then be divided by the BSA.

$$\text{CI} = \frac{\text{CO}}{\text{BSA}}$$

Normal CI = 2.5 to 4.0 l/min/m²

Where:

CO = Cardiac Output
BSA = Body Surface Area
CI = Cardiac Index

• Stroke Volume

This is the amount of blood pumped by the ventricle in one contraction. Since stroke volume (SV) is part of the cardiac output equation, the value can be derived at mathematically.

$$\text{SV} = \frac{\text{CO}}{\text{HR}} \times 1000 \text{ ml/L}$$

Normal SV = 60 to 100 ml/beat

Where:

CO = Cardiac Output
HR = Heart Rate
SV = Stroke Volume

• Stroke Volume Index

As with the cardiac output, SV can also be indexed to the patient’s body size. This is also known as Stroke Index (SI). There are two ways to arrive at this value:

$$\text{SVI} = \frac{\text{SV}}{\text{BSA}} \quad \text{or} \quad \frac{\text{CI}}{\text{HR}}$$

Normal SVI = 33 to 47 mm/Beat/m²

By calculating SV or SVI, some indication of the state of contractility can be evaluated.

• Vascular Resistance

Another variable of ventricular function is vascular resistance. Resistance is the relationship of pressure to flow. As blood flows through the vasculature, there is resistance. This value is the clinical representation of afterload, the amount of resistance the ventricle must overcome to eject blood volume. Each ventricle has to overcome the afterload of their respective circuits.

• Systemic Vascular Resistance

Systemic vascular resistance (SVR) measures the afterload or resistance for the left ventricle. Since resistance relates pressure to flow, pressure is arrived at by measuring the gradient between the beginning of the circuit (MAP) and the end (RA). This value is then divided by the flow or cardiac output. A rounded conversion factor of 80 is used to adjust the value into units of force; dyne-sec-cm⁻⁵.

$$SVR = \frac{(MAP - RAP) \times 80}{CO}$$

$$\text{Normal SVR} = 800 \text{ to } 1200 \text{ dynes/sec/cm}^2$$

• Pulmonary Vascular Resistance

The right ventricular afterload is clinically measured by calculating pulmonary vascular resistance (PVR). Again, the gradient of the circuit is measured, MPAP minus the end PAWP, then divided by the flow or cardiac output. The conversion factor of 80 is again multiplied to convert into units of force.

$$PVR = \frac{(MPAP - PAWP) \times 80}{CO}$$

$$\text{Normal SVR} = < 250 \text{ dynes/sec/cm}^5$$

Where:

MPAP = Mean Pulmonary Artery Pressure

PAWP = Pulmonary Artery Wedge Pressure

• Stroke Work

Another way to evaluate ventricular function is by measuring the external work for the ventricle in one contraction. This value can be calculated from obtaining the average pressure generated by a ventricle during one heart beat and multiply that by the amount of blood ejected during one heart beat. Another factor for converting pressure into work, 0.0136 is utilized. Many institutions index this value for BSA and may also evaluate the stroke work index for each ventricle.

$$SW = (MAP - LVEDP) \times SV \times 0.0136$$

Where:

MAP = Mean Arterial Pressure

LVEDP = Left Ventricular End Diastolic Pressure

LVSWI = SVI (MAP - PAWP) x 0.0136

Normal LVSWI = 45 to 75 gm-m/m²/Beat

Where:

SVI = Stroke Volume Index

MAP = Mean Arterial Pressure

PAWP = Pulmonary Artery Wedge Pressure

RVSWI = SVI (MPA - MRA) x 0.0136

Normal RVSWI = 5 to 10 gm-m/m²/Beat

Where:

SVI = Stroke Volume Index

MPA = Mean Pulmonary Artery Pressure

MRA = Mean Right Arterial Pressure

UTILIZING THE SWAN-GANZ CATHETER FOR DIFFERENTIAL DIAGNOSIS

Many newer monitoring systems are now designed to provide the practitioner with the full range of derived parameters. Key direct parameters that provide the basis for most of the derived and calculated ones are cardiac output, mean arterial pressure, and pulmonary artery pressures. Incorporating these values into the data base along with clinical assessment of the patient can assist in fine tuning the physiological response to therapeutic interventions and also assist with differentiation of diagnosis.

Obtaining hemodynamic parameters can assist the clinician with not only assessing the status of ventricular function, but can also provide important information that assists in differentiating disease states where the patient's clinical presentation may be nearly the same. Using the obtained values can also help to further "subset" the patient's condition to guide therapeutic modalities and project patient outcomes.

Low Output States

During low output states, many patients present with a low blood pressure, low cardiac output determinations, and signs of poor tissue perfusion. By use of hemodynamic monitoring, precise identification of the state can be defined. If a patient is hypovolemic, BP and CO will be low. Along with these lower than normal values will be a low PAWP. Another patient who may be in cardiogenic shock will also present with a low BP, low CO, but instead will have a high PAWP. Right ventricular infarction is another condition where a low cardiac output and low BP state exists. Since the dysfunction is on the right side, elevations in RV filling pressures will be recorded as elevated RA pressures. If there is no left ventricular involvement, the PAWP will be normal or even low.

Hemodynamic Subsets of Acute Myocardial Infarction

(Forrester Classification) *

	Indications	PCW	Therapy	Mortality
SUBSET I No Failure	Cardiac Index Greater than 2.2 L/MIN/M ²	Less than 18 mmHg	Sedate	3%
SUBSET II Pulmonary Congestion	Cardiac Index Greater than 2.2 L/MIN/M ²	Greater than 18 mmHg	Normal Blood Pressure: Diuretics Elevated Blood Pressure: Vasodilators	9%
SUBSET III Peripheral Hypoperfusion	Cardiac Index Less than 2.2 L/MIN/M ²	Less than 18 mmHg	Elevated Heart Rate: Add Volume Depressed Heart Rate: Pacing	23%
SUBSET IV Congestion & Hypoperfusion	Cardiac Index Less than 2.2 L/MIN/M ²	Greater than 18 mmHg	Depressed Blood Pressure: Inotropes Normal Blood Pressure: Vasodilators	51%

*With permission of James S. Forrester, M.D., Assistant Director of Cardiology, Cedars-Sinai Medical Center, Los Angeles, California.

Acute myocardial infarction can produce one of four different hemodynamic subsets. James Forrester (Ref. 13) studied the relationship between cardiac index, as the measurement for peripheral hypoperfusion, and pulmonary artery wedge pressure as a means to assess pulmonary congestion. By obtaining these values and placing the patient in proper subset, therapeutic goals could be directed more precisely. The subsets could also be used to make outcome decisions.

Two major complications of myocardial infarction, acute mitral insufficiency and acute ventricular septal defect, present clinically the same with a low cardiac output. These complications can be differentiated using the Swan-Ganz thermodilution catheter. The effect of therapy in these conditions can also be assessed.

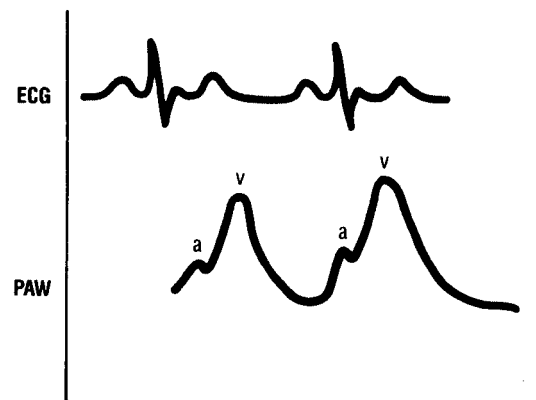


Figure 33
Acute Mitral Insufficiency: Before Nitroprusside

Mitral valve insufficiency can be detected by observing the PAW waveform. As a result of the incompetent valve, a large regurgitant “v” wave will show during the atrial filling phase. Typical drug therapy includes the use of an afterload reducer such as Sodium Nitroprusside. As the afterload is reduced, more forward flow from the ventricle can occur, which in turn causes a decrease in backward flow. This improvement is seen as a reduction in the elevation of the “v” wave.

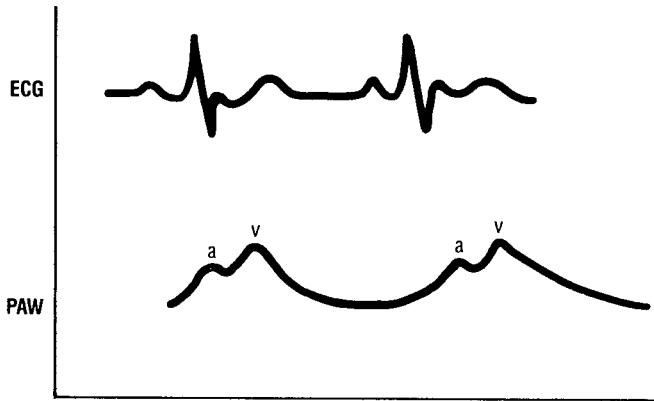


Figure 34
Acute Mitral Insufficiency: After Nitroprusside

Acute ventricular septal defect (VSD) can also produce low cardiac output when the blood volume from the left ventricle shunts over to the right ventricle. This shunting causes a step up of oxygen saturation in the right ventricle and pulmonary artery. By determining saturation values in the PA, a VSD can be detected. In severe cases, a resultant elevation in the “v” wave during a wedge recording may also be seen. This is due to the increase in blood volume from the left ventricle, which during atrial filling, records as an elevation. Timing of the “v” wave is helpful to identify the cause. During mitral valve regurgitation, the “v” appears closer to the “a” wave. With VSD, the “v” wave elevation will be in the normal timing relation to the “a” wave. As with mitral valve insufficiency, afterload reducers may be of benefit to the patients as long as the systemic resistance is above normal.

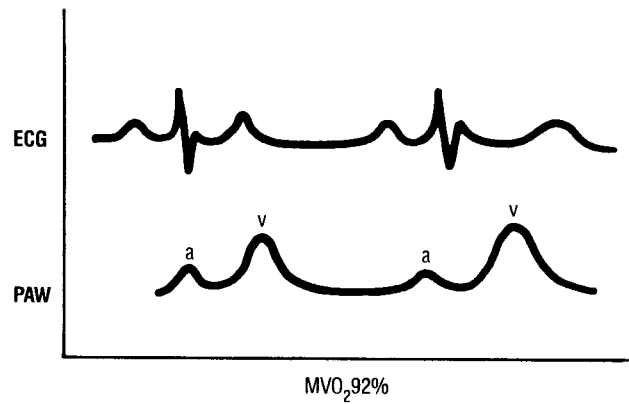


Figure 35
VSD

Hemodynamic parameters can identify cardiac from pulmonary dysfunctions. Conditions such as pulmonary hypertension, regardless of the cause, will show elevations in the PASP and PADP. Since the wedged catheter more accurately assesses left ventricular function, there will be a normal wedge value if there is not concurrent ventricular disease.

Conditions such as cardiac tamponade and constrictive pericarditis may present hemodynamic alterations prior to clinical manifestations. Whereas both conditions may produce equalization of diastolic pressures, waveform identification can assist in differentiating the two. Cardiac tamponade classically shows loss of the “y” descent in a wedge or RA tracing as a result of higher diastolic values. In constrictive pericarditis, there are exaggerated “y” descents from rapid diastolic filling as a result of the rigid pericardium.

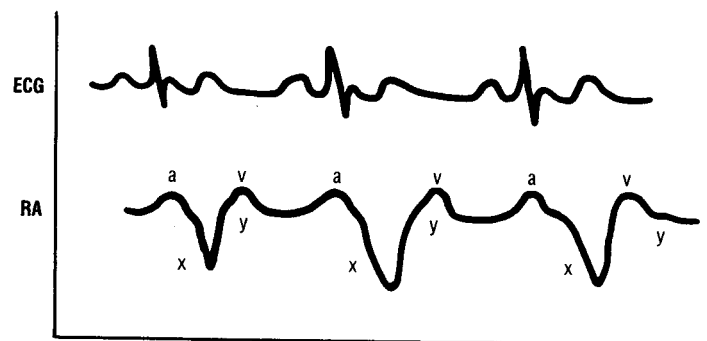


Figure 36
Cardiac Tamponade

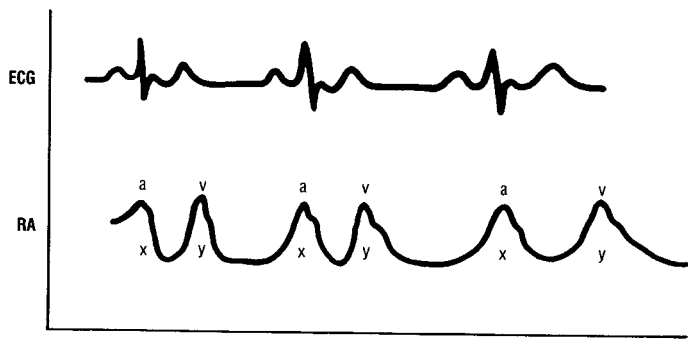


Figure 37
Constrictive Pericarditis

Invasive hemodynamic monitoring has also been used effectively for preoperative assessment of high-risk patients. By obtaining the various parameters, ventricular function can be optimized prior to surgery. Del Guercio (Ref. 11) developed a system of preoperative staging based on invasive hemodynamic monitoring. As a result, patients that fell into a moderate or severe impairment group showed higher survival rates following physiologic fine tuning than patients without moderate or severe impairment.

Patient Outcomes

Assessment	N	%	Treatment	Survival Rate
1. Normal	20	13.5	Surgery as planned	100%
2. Mild impairment	94	63.5	Advanced intraoperative monitoring	91.5%
3. Moderate			Physiological fine tuning and intraoperative monitoring	
4. Severe impairment	34	23	Restricted surgical procedure (n = 7)	100%
			Surgery cancelled (n = 19)	*
			Surgery as planned (n = 8)	0%

*Surgery cancelled; not included in survival rate.

LIMITATIONS OF HEMODYNAMIC MONITORING

In most clinical conditions, the pulmonary artery wedge pressure closely reflects left atrial pressure which in turn closely reflects LVEDP. Clinical assessment of preload, left ventricular end diastolic volume, as measured by the wedge reading, does have some limitations.

It is understood that there are clinical situations where discrepancies between true LVEDV and LVEDP occur. There are also certain conditions where PAW pressures do not accurately reflect LVEDP.

The next section will identify those conditions and situations where using the wedge reading as a true indicator of left ventricular preload may be inaccurate. These conditions of discrepancy are discussed to provide the clinician with a basis to allow the values that they obtain be more realistic and patient specific.

Left Ventricular End Diastolic Volumes vs. End Diastolic Pressure

In the applicable cardiac physiology segment, it was discussed, that due, in part, to ventricular compliance, the relationship between pressure and volume is a curvilinear one. As the ventricle becomes less compliant and, therefore, more stiff, a higher pressure is generated with the same amount of volume. The opposite is true with a more compliant ventricle. As the ventricle becomes less stiff, more volume is able to be held at a lesser pressure. Identifying conditions that alter compliance can place more validity to the values obtained.

Factors Affecting Ventricular Compliance

Decreased Compliance	Increased Compliance
Ischemia	Afterload reducers
Severe LV hypertrophies	Cardiomyopathies
Restrictive cardiomyopathies	Relief of ischemia
Shock states	
Effects of Increased PVR	

Pulmonary Artery Wedge vs. Left Ventricular End Diastolic Pressures

Since the wedge is actually reflecting left atrial filling pressures, there must be no occlusions between the tip of the PA catheter and left ventricle to obtain accurate readings. Conditions such as mitral valve stenosis, left atrial myxoma, and pulmonary diseases produce some form of occlusion between the tip and left ventricle. In these conditions, the wedge will not reflect LVEDP. The wedge value will be recorded abnormally higher than the true LVEDP.

Mitral valve regurgitation is another condition, where due to an elevation in the “v” wave, the monitor will read an abnormally high PAWP. This again will not accurately reflect LVEDP.

Conditions exist where wedge recordings may be lower than actual LVEDP. In conditions where the LVEDP is greatly elevated such as severely decreased compliance with pressures greater than 25 mm Hg, the PAWP is recorded lower. This is due in part to the increased compliance of the pulmonary vascular bed inadequately reflecting the high pressures. Aortic regurgitation can also cause an increase in LVEDP due to backward flow. In the early stages, this backward pressure may not be reflected all the way back to the left atrium and finally the wedge.

Conditions in Which PAW is Greater Than LVEDP

- Mitral stenosis
- Left atrial myxoma
- Pulmonary embolus
- Mitral valve regurgitation

Conditions in Which PAD is Less Than LVEDP

- Decreased left ventricular compliance
- High (> 25 mm Hg) LVEDP
- Aortic valve regurgitation

Conditions in Which PAD Does Not Equal PAW (1 – 4 mm Hg)

- Increased PVR
- Pulmonary hypertension
- Cor pulmonale
- Pulmonary embolus
- Eisenmenger’s syndrome

Respiratory variations, application of PEEP, and lung zone catheter tip location can also be causes of inaccurate values.

Three methods for obtaining pressures with respiratory variations have been proposed. During the normal respiratory cycle, the changes in intrathoracic pressures are transmitted to the Swan-Ganz catheter. As a result, on inspiration the pressures will be recorded lower, while on expiration they will be higher. During end expiration the intrathoracic pressures are nearly equalized. Recordings obtained at this time may be more accurate. Monitors that display a digital reading may be of the type where the value displayed has been averaged over a period of time. It has been recommended that manual recordings be obtained. Another technique described is the averaging of the pressures obtained over a few respiratory cycles. As a general rule, hemodynamic values are assessed as trend values rather than absolute values (Refs. 5 & 35).

Lozman (Ref. 25) has conducted studies to show the correlation of PAWP and LAP under varying centimeters of PEEP. Whereas each patient may respond differently to PEEP, close correlation of PAWP to LAP maintains until approximately 12 cm to 15 cm of PEEP. Over that range, the higher intrathoracic pressure is transmitted to the catheter causing an abnormal elevation in the recorded wedge.

The catheter tip location in lung zones will also play an important role on the validity of the wedge reading, both under normal conditions and under conditions of PEEP. The lung zones are identified by the relationships among the inflow pressure (pulmonary artery pressure), the outflow pressure (pulmonary venous pressure), and the surrounding alveolar pressure.

In Zone 1, the alveolar pressure is greater than both the pulmonary arterial and pulmonary venous pressure. As a result, there is no blood flow from the collapsed pulmonary capillary beds. Since the Swan-Ganz catheter is a flow-directed catheter, more than likely the tip will not be in that location.

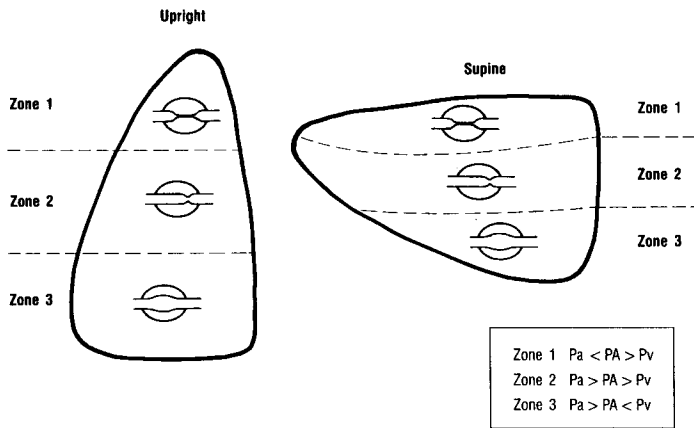


Figure 38

Lung Zone 2 also has a higher alveolar pressure than pulmonary venous pressure, but the arterial pressure is great enough to allow for blood flow. Under most conditions, this location will provide accurate wedge readings. If PEEP is implemented, higher levels of PEEP may increase the alveolar pressure, which will then cause Zone 2 to be more like a Zone 1. The higher surrounding pressure will be transmitted back through to the catheter.

Zone 3 is the best location for recording pulmonary artery wedge readings. In this zone, the pulmonary venous pressure is higher than the sounding alveolar pressure, and all capillaries are open. In this position, the catheter tip is usually below the level of the left atrium and can be verified by a lateral chest x-ray.

Certain mechanical or technical influences may also affect the accuracy of the values obtained. Improper transducer positioning, less than optimal pressure systems along with electrical interferences can alter the recording. As previously mentioned, overwedging of the balloon may also give erroneous values. In many circumstances, optimizing the pressure system and being comfortable with the electronics will eliminate these difficulties.

Although the list of limitations for pulmonary artery monitoring seems great, at present, the use of the Swan-Ganz catheter to accurately assess ventricular function

remains one of the most reliable and easily accessible monitoring tools available. The values obtained, in the knowledgeable clinician's hands, can provide information to better assess the patient's conditions and to guide the therapeutic interventions.

Complications

Even though there are no absolute contraindications for the use of the Swan-Ganz catheter, there are some general risks and complications associated with indwelling catheters. Most of these can either be eliminated or decreased by a thorough familiarization of insertion techniques, catheter maintenance and patient history.

Patients with a history of pulmonary hypertension and advanced age should have extreme care taken during insertion and wedging of the balloon to prevent the possibility of pulmonary artery rupture. It is recommended that inflation time of the balloon should be no more than two respiratory cycles, or 10 to 15 seconds. Monitoring of the PADP instead of wedging is recommended in these conditions.

Particular care to electrocardiographic monitoring during catheter insertion should be maintained for patients with complete left bundle branch block, Wolff-Parkinson-White syndrome, and Ebstein's malformation. Although some rhythm disturbances may occur, the catheter has been designed to be less rigid than standard catheterization catheters and therefore lessens the risks.

INTRA-ARTERIAL MONITORING

Another means for assessing the hemodynamic status of the patient is by direct intra-arterial pressure monitoring. Use of an intra-arterial catheter, pressure monitoring system, and transducer allows a means for continuous observation of the patient's systemic blood pressure. Many monitoring amplifiers have the capability to calculate from the arterial waveform the mean arterial pressure, which is a common value for calculating derived hemodynamic parameters.

Indirect methods of assessing arterial pressures include sphygmomanometer with a cuff and doppler devices. If properly used, these methods accurately reflect the patient's arterial pressure for healthy individuals. However, it is during low cardiac output states that these methods may give erroneous values.

Changes in vascular compliance can alter the transmission of Korotkoff sounds that are typically used to determine blood pressure by auscultation methods. It is thought that the distinctive sounds heard are a result of the vibration of the arterial wall during intermittent flow from the cuff that has compressed the arterial segment. Under optimal conditions, the indirect methods tend to underestimate the systolic pressure and overestimate the diastolic pressure by about 5 mm Hg.

In conditions of high systemic vascular resistance, there is an increased wall tension. This condition may diminish the vibration capability and therefore diminish sound formation. Conditions that produce a low systemic vascular resistance may also diminish vibrations because of the lack of intermittent blood flow through the occluded arterial segment. In both of these situations, an abnormally low cuff pressure may be noted, even though in reality, the arterial pressure may be higher.

The contour of the arterial pulse changes as it travels from the aortic root to the periphery. These changes are due in part to the difference in elastic characteristics of different arterial sites and also the loss of some of the kinetic energy. As the wave becomes more distal, the upstroke becomes sharper with a higher systolic pressure and a lower diastolic pressure. Even with these changes, the mean arterial pressure remains the same.

Components of the Arterial Pulse

As with intracardiac waveforms, arterial waveforms are a result of mechanical function. Arterial waveforms are produced after electrical activation of the heart. When evaluating arterial waveforms at the same time as electrical waves, the electrical activity will be noted first followed by the mechanical activity.

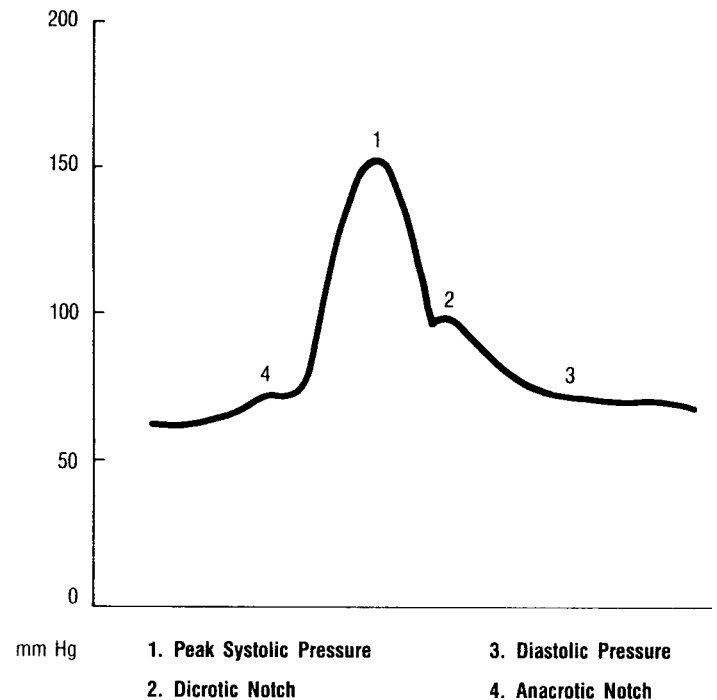


Figure 39
Components of Arterial Pulse

• Peak Systolic Pressure (PSP)

Peak systolic pressure reflects maximum left ventricular systolic pressure. This phase begins with the opening of the aortic valve. A sharp uprise is seen in the tracing that reflects the outflow of blood from the ventricle and into the arterial system. This upward stroke is also referred to as the ascending limb.

• Dicrotic Notch

With a greater pressure in the aorta than in the left ventricle, blood flow attempts to equalize by flowing backwards. This causes the aortic valve to close. On the tracing, aortic valve closure is seen as a dicrotic notch. This event marks the end of systole and the onset of diastole.

• Diastolic Pressure

This value relates to the level of vessel recoil or amount of vasoconstriction in the arterial system. There is also a relation between the diastolic pressure and diastolic time during the cardiac cycle. During diastole, there must be ample time for the blood in the arterial system to drain down into the smaller arteriole branches. If the heart rate is faster and therefore has a shorter diastolic time, there is less time for run off into the more distal branches. The result is a higher diastolic pressure. This decline in pressure during diastole is called the descending limb.

• Anacrotic Notch

During the first phase of ventricular systole (isovolumetric contraction), a presystolic rise may be seen. This rise is called the anacrotic notch, which will occur before the opening of the aortic valve. This wave typically will be seen only in central aortic pressure monitoring, an aortic root tracing, or in some pathological conditions.

• Pulse Pressure

The difference between the systolic and diastolic pressure is called the pulse pressure. Factors that can affect the pulse pressure are changes in stroke volume, as noted in the systolic pressure, and also changes in vascular compliance, as seen in the diastolic pressure.

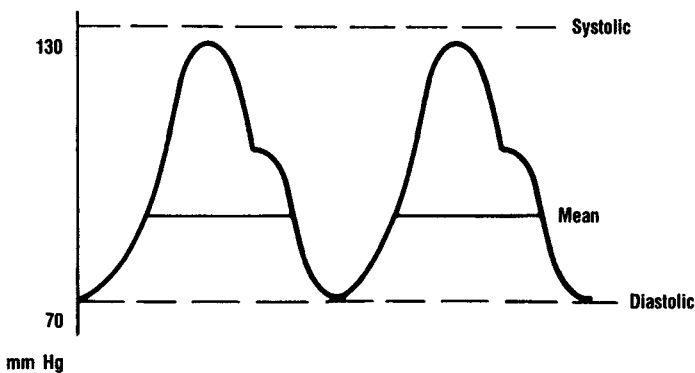


Figure 40
Arterial Waveform

Mean Arterial Pressure

This value reflects the average pressure in the arterial system during a complete cardiac cycle of systole and diastole. Systole requires one-third of the cardiac cycle time, while diastole requires two-thirds. This timing relationship is reflected in the equation for calculating MAP.

$$\text{MAP} = \frac{S + (D \times 2)}{3} \text{ or } D + \text{PP}$$

Where:

MAP	=	mean arterial pressure
S	=	systolic blood pressure
D	=	diastolic blood pressure
PP	=	pulse pressure

ARTERIAL WAVEFORMS FOR DIFFERENTIAL DIAGNOSIS

Continuous arterial pressure monitoring not only provides information in regards to blood pressure, it also provides a means to assess the cardiovascular status by observing waveform characteristics. Certain clinical conditions may not be apparent by observing only the pulmonary artery tracings. Observing the arterial waveform adds another dimension to patient assessment.

Aortic stenosis produces a small pulse wave with a delayed systolic peak. This lower systolic pressure is a result of slowed ventricular ejection through the stenotic aortic valve. The dicrotic notch is often not well defined from abnormal closure of the valve leaflets during the onset of diastole. Since the systolic pressure is lower, these patients have a narrow pulse pressure.

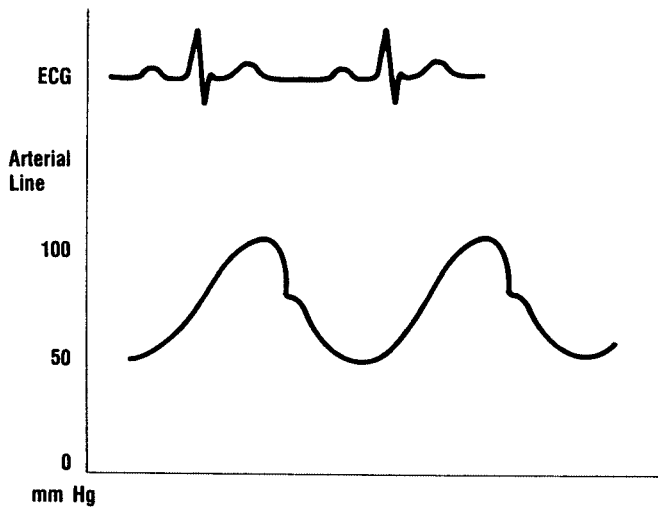


Figure 41
Aortic Stenosis

Aortic insufficiency is also known as aortic regurgitation. This condition is classically identified by a wide pulse pressure. During diastole, the left ventricle receives more backflow blood volume from the incompetent valve. This increase in blood volume is reflected as a higher peak systolic pressure during the next systole.



Figure 42
Aortic Insufficiency

Alterations in heart rhythm also affects the character of the arterial tracings. Atrial fibrillation, with the classic irregularity, produces varying amplitudes in the arterial waveform. During episodes of premature ventricular complexes, the shortening of the diastolic filling time is also noted with a resultant decrease in systolic amplitude.

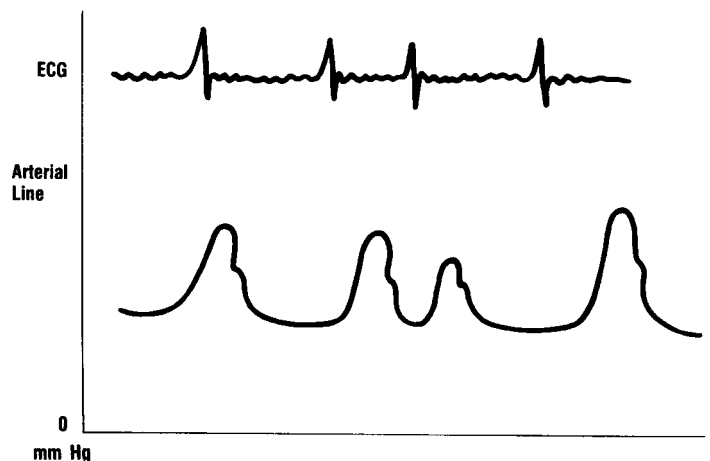


Figure 43
Atrial Fibrillation

Pulses alternans is an abnormality where during a regular sinus rhythm there is regular altering amplitudes of the peak systolic pressures. This condition may be a result of alterations in calcium or myocardial muscle fibers. Pulses alternans is a valuable visual assessment tool for patients with severe left ventricular failure. Clinically, the varying amplitudes may be difficult to palpate at a peripheral artery.

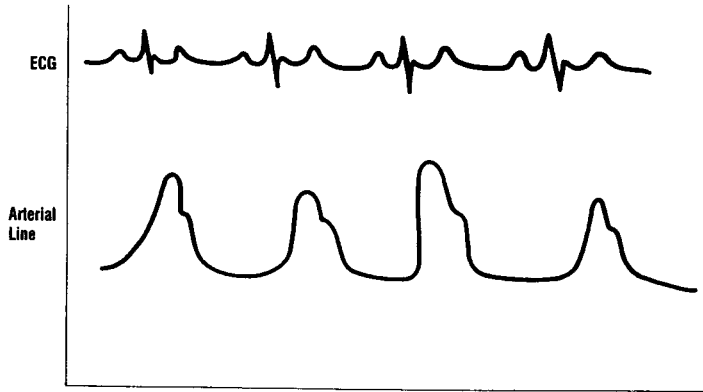


Figure 44
Pulses Alternans

During inspiration, there is a lower intrathoracic pressure. This lower pressure results in an increased pooling of blood in the pulmonary vasculature. There is then less blood volume in the left side of the heart. As a result of this phenomenon, systolic pressures may be 3 to 10 mm Hg lower on inspiration.

On expiration, the blood volume that was pooled in the pulmonary bed during inspiration is now shunted to the left heart. This increase in blood volume is responsible for a higher systolic pressure on expiration.

When there is a greater than 10 mm Hg difference in systolic pressures from inspiration to expiration, the abnormality is referred to as pulses paradoxus. A variety of conditions can cause this phenomenon.

More common conditions include inspiratory exaggerations, either from patient-related causes or pathophysiological-related causes and pericardial diseases. The mechanism for producing pulses paradoxus differs with the underlying etiology. A common mechanism is the alteration in venous return to the right heart and changes in intrathoracic or intrapericardial pressures.

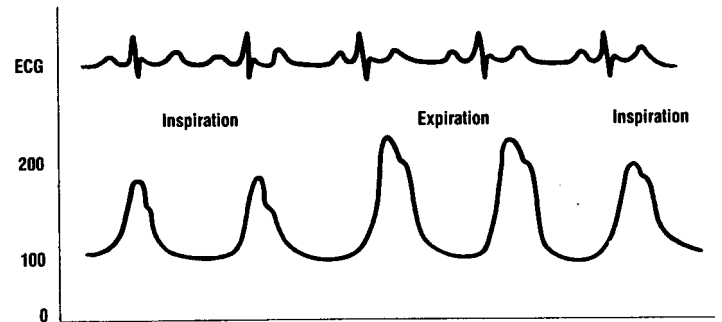


Figure 45
Pulses Paradoxus

For the critically ill patient, not only does intra-arterial pressure monitoring provide easy access for frequent arterial blood sampling, but can also assist with differential diagnosis of certain disease states. This is another tool that the experienced clinician can utilize for optimizing patient management and also for rapid assessment.

SUMMARY

Medicine and technology have advanced greatly over the past few decades. As technology continues to become more sophisticated, so do the requirements for the critical care practitioner.

Developments of the Swan-Ganz catheter from a simple balloon inflation design to the use of fiberoptics for oxygenation assessment, pacing electrodes for heart rate management, and a method for isolating right ventricular performance have helped to drive the need for a better understanding of the cardiopulmonary system. With this better understanding comes an opportunity to evaluate patient conditions and response to therapies more rapidly and with relative ease.

The next few decades will also continue to see rapid advancements in technology. This again will provide a climate for widening the base knowledge of all practitioners caring for the critically ill, and an opportunity to once again review the art and science of hemodynamic monitoring.

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APPENDIX I

Hemodynamic Parameters

<u>Parameter Formula</u>	<u>Normal Value</u>
Cardiac Output (CO)	
$CO = \frac{HR \times SV}{1000}$	4 – 8 l/min
Cardiac Index (C_I)	
$CI = \frac{CO}{BSA}$	2.4 – 4.0 l/min/m ²
Mean Arterial Pressure (MAP)	
$MAP = \frac{SBP + (DBP \times 2)}{3}$	70 – 105 mm Hg
Pulmonary Vascular Resistance (PVR)	
$PVR = \frac{(MPAP - PAWP) \times 80}{CO}$	<250 dynes/sec/cm ⁵
Systemic Vascular Resistance (SVR)	
$SVR = \frac{(MPA - RAP) \times 80}{CO}$	800 – 1200 dynes/sec/cm ⁵
Stroke Volume	
$SV = \frac{CO \times 1000 \text{ ml/l}}{HR}$	60 – 100 ml/Beat
Stroke Volume Index	
$SVI = \frac{SV}{BSA}$	33 – 47 ml/Beat/m ²
Left Ventricular Stroke Work Index (LVSWI)	
$LVSWI = SVI(MAP - PAWP) \times 0.0136$	45 – 75 mg-m/m ² /Beat
Coronary Perfusion Pressure (CPP)	
$CPP = \text{Diastolic BP} - PAWP$	60 – 70 mm Hg
Rate Pressure Product (RPP)	
$RPP = HR \times \text{Systolic BP}$	<12,000
Right Ventricular Stroke Work Index (RVSWI)	
$RVSWI = SVI(MAP - RAP) \times 0.0136$	5 – 10 gm-m/m ² /Beat
Ejection Fraction	
$EF = \frac{SV}{EDV}$	0.67



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