Cardiopulmonary Monitoring: Right Ventricular Function

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The purpose of this presentation is to review some of the important methods of monitoring heart and lung function in patients with cardiac disease who are scheduled for cardiac surgery as well as non-cardiac surgery. A special emphasis will be placed upon the recent resurgence of the recognition of the importance of the right ventricular function. Findings of our current clinical study related to the evaluation of the right ventricular ejection fraction (RVEF) in patients who are scheduled for either coronary artery bypass graft (CABG) surgery or valve replacement surgery during anesthesia and cardiac surgery will also be presented.

There will be some review of the literature and discussion of the indications, risks and benefits of invasive methods of cardiopulmonary monitoring during anesthesia and surgery in cardiac patients.

SECTION 1

BASIC CONSIDERATION

It seems to be appropriate to define the term 'monitoring'. In accordance with Webster's New Collegiate Dictionary, the word monitor means to watch, observe or check especially for a special purpose and/or to keep track of, regulate or control the operation of. Therefore, in medicine, we are to use the word monitor as keeping track of the important vital functions which are relevant to our lives.

We will not discuss here the non-invasive methods of monitoring heart and lung function since there has been recent reviews on this subject.1)

DEFINITIONS

In order to establish the common ground, the definitions of the terms and methods essential in studying the right ventricular function will be described at first:

Preload:

In the isolated muscle preparation, the initial length of the muscle is established by a small

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load, and may be kept constant during contraction. This small load is called 'preload'. In an intact heart, the preload is related to the volume of blood in a ventricle at the end of the diastole. There is a curve linear relationship between the intra-ventricular pressure and intra-ventricular volume during diastole. Measurements of end-diastolic ventricular pressure (EDVP) is frequently used to indicate end-diastolic ventricular volume (EDVV), which is used as an index of 'preload' in the intact heart. However, it is important to note that the volume of the ventricles, rather than the pressure within their cavities, is what determines the amount of energy set free during the contraction. The law of the heart states that the energy of contraction, however measured, is a function of the length of the muscle fiber prior to contraction (preload). An increase in the resting tension (preload), will cause more forceful contraction (length-tensions relationship, heterometric autoregulation, or Starling's effect). The ventricular action is directly related to the volume of blood that expands it in preparation for it's systolic contraction. Normally, a ventricle, it's distension progressively increased by augmented venous return, will increase its stroke volume (SV) and/or stroke work (SW).

**Diastolic Properties:**

Most data indicate that in the resting cardiac muscle, the relationship between strain (or extension) and the change in stress is essentially exponential. However, the resulting linear relation between the stiffness (the ratio of the increment in stress to the corresponding change in length [ΔT/ΔL]) and the stress generally has been utilized to describe the resting properties of the isolated heart muscle:

\[
\Delta T/\Delta L = aT + b
\]

**Afterload:**

In isolated muscle preparation, establishment of preload, added load will be supported by a mechanical stop and encountered only when there is shortening of the muscle. Hence, the additional load that the muscle must move during contraction is called the 'afterload'.

In the intact heart, afterload is determined by intraventricular systolic tension during ventricular ejection. It is important to realize one must consider that the afterload is proportioned to both the transmural presence and the radius of a ventricle, and is in inverse proportional to the thickness of ventricular wall (Laplace's formula).

**Ventricular Function Curve (VFC):**

Some hemodynamic indecies of cardiac performance, usually ventricular SW, SV or cardiac output (CO), is plotted on the vertical axis. The VEDP (preload) is plotted on the horizontal axis. Figure 1 shows the left VFC and the right FVC. The left VFC is obtained by plotting the left VEDP in cm of water against the left VSW in gm-meters. The right VFC is obtained by plotting the right VEDP in cm of water against the right VSW in gm-meters. Thus, the VFC,
as described by Sarnoff and Bergland\(^3\)), indicates the relationship between the preload and the external useful work performed by the ventricle. It is important to emphasize that the VFC shows an initial steep rise. A rise of 1 cm H\(_2\)O in the VEDP in the steep portion of the curve may be related to an increase in the VSW by as much as 300 percent. Note, the left VFC shows a curve linear relationship while the right shows almost a straight line relationship.

**Ventricular End-Diastolic Volume (EDV):**

The amount of blood exists in a ventricle at the end of diastole.

**Ventricular End-Systolic Volume (ESV):**

The amount of blood remains in a ventricle following the completion of ejection.

**Ejection Fraction (EF):**

This is the ratio between the \( SV = (EDV - ESV) \) and end-diastolic volume:

\[
EF = \frac{SV}{EDV} \quad \text{or} \quad EF = 1 - \frac{ESV}{EDV}
\]

**SECTION 2**

**CARDIOPULMONARY MONITORING**

In this section, we will discuss the arterial lines, pulmonary artery lines and right ventricular function.

**Arterial Lines**: 

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*Figure 1*

Families of the left and right ventricular function curves, showing the inotropic effects. Changes in the inotropic state produce different VFCs. Curves-A are obtained during the control state. During a negative inotropic intervention (e.g., anesthetic agents), the same heart exhibits a depressed VFCs (curves-B), resulting in reductions of SW at any given filling pressure for both ventricles. A positive inotropism (administration of norepinephrine, digitalis etc.) is shown by the shifts in VFCs to the left and upward, depicted by curves-c.
Arterial lines are indicated for direct, continuous monitoring of blood pressure and for frequent sampling of arterial blood. Insertion of arterial line is relatively safe. However, one should be aware of possible complications. There are infection and thrombosis. The rate of catheter-associated infection is about 4 percent. The rate of arterial thrombosis is considerably high (about 40 percent in one study). Thromboembolic complications are more likely with an 18 gauge than with a 20 gauge catheter. One must perform Allen's test to ensure the potency of the ulnar circulation in order to minimize the risks related to arterial cannulation. The significance of arterial pulse contours has been described elsewhere. In brief, it is important to see the changes in pulse contours rather than to observe only changes in pressures.

**Pulmonary Artery Lines:**

The balloon-tipped catheter enables one to measure central venous pressure, pulmonary artery pressures, cardiac output and mixed venous blood samples. Thus, the physiologic data to evaluate pulmonary and right and left ventricular function becomes available in clinical patients at bedside and operating room conditions. The major indications for pulmonary artery catheterization are shown in Table 1. It is important to realize that the potential benefits of catheterization must outweigh the risks and complications related to the use of the catheter. This is especially true of critically ill patients with pre-existing cardiovascular or pulmonary disease or increased susceptibility to bleeding and infection.

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**Table 1. Indications for Pulmonary Artery Catheterization**

1. Complicated myocardial infarction or myocardial ischemia
   a. Hypotension
   b. Congestive heart failure
   c. Sinus tachycardia
   d. Hypertension
   e. Acute mitral regurgitation
   f. Ventricular septal defect
   g. Pericardial tamponade
   h. Right ventricular infarction
   i. Evaluation of pharmacologic agents
   j. Assessment of interventions to decrease MI size
2. Shock
3. Pulmonary
   a. Cardiogenic pulmonary edema
   b. Respiratory failure
   c. Respiratory distress of unknown cause
4. Assessment of intravascular volume
5. Vasodilation
6. Surgical
   a. High-risk patient
   b. Proposed surgical procedure; surgery associated with a high mortality anticipates large volume requirements
   c. Postoperative complication

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The major complications of pulmonary artery catheterizations are shown in Table 2. Therefore, one must always remember that complications do occur and that sometime they may be fatal. Complications of pulmonary artery catheterization can be minimized by meticulous attention to detail particularly during its placement. Many serious complications are related to peripheral migration of the catheter tip as a result of cardiac contractions. Balloon inflation should be done gradually and with constant monitoring. The length of time that the catheter is kept in the wedge position should be minimized, especially in patients with pulmonary hypertension in order to avoid rupture of pulmonary artery.

RIGHT VENTRICULAR FUNCTION

The essential function of the right ventricle (RV) has been described in detail. It has been suggested that the RV may not be essential to maintain a normal pulmonary blood flow. Much evidence has been accumulated to demonstrate that the perfusion of both the pulmonary and systemic circulations may be amply provided by the left ventricle.

It has been shown that all the pumping function of the RV can be bypassed with the appropriate surgical manipulation (e.g. right ventricular destruction, right ventricular infarction and partial or complete surgical bypass of the RV). There is a view that the essential function of the right half of the two-pump system is to maintain pressure in the very distensible systemic venous system at a low level. The normal LV has the capability of providing an adequate flow through both the systemic and pulmonary circuits, even if the pumping action of the RV has been eliminated.

It seems very appropriate to state that we are beginning to learn about the essential functions of the RV in health and disease. Particularly information related to influences of anesthetic agents on the RV function is needed.

**Assessment of Ventricular Performance:**

The performance characteristics of the right ventricle (RV) differ in many ways from those

<table>
<thead>
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<th>Table 2. Complications of Pulmonary Artery Catheterization</th>
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<tr>
<td>1. Arrhythmias</td>
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<td>2. Right bundle branch block</td>
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<td>3. Pulmonary infarction</td>
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<td>4. Pulmonary artery rupture</td>
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<td>5. Cardiac complications (Lesions of right atrial endocardium, tricuspid valve, chordae tendinae and pulmonic valve)</td>
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<td>6. Knotting</td>
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<td>7. Infections</td>
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<td>8. Balloon rupture</td>
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<td>9. CVP placement (bleeding, hematoma, pain and swelling, inflammation, infection, thrombophlebitis, arterial puncture, pneumothorax, hemothorax, air embolism, thoracic duct injury and brachial nerve injury).</td>
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<td>10. Thrombosis</td>
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...
of the left ventricle (LV). With the first, one must consider anatomical differences between RV and LV. The LV resembles a prolate ellipsoid while the RV resembles a triangular and crescent-shaped chamber. The RV chamber is a structure bounded by the convex interventricular septum and the concave free wall. Its surface-area to volume ratio is quite large as compared with that of the LV. The manner of contraction of RV chamber, therefore, involves three independent phenomena. At first, the trabeculae and papillary muscles push the tricuspid valve plane down toward the apex of the RV. This motion shortens the longitudinal axis of the RV chamber. This is quite in contrast with the contraction of the LV where the major changes in the circumference occur at the equator along the minor or horizontal axis of the ellipsoidal configuration of the LV. There is little change in the circumference of the ventricular wall along the major or longitudinal axis of the LV. It is interesting to note that the shortening of ventricular wall along the longitudinal axis of the RV has little effect on actual ejection of blood.

Secondly, the free wall of the RV chamber moves toward the convex surface of the septum. This movement is responsible for the major portion of right ventricular contraction.

Thirdly, when the circular fibers of the thicker LV contract, the curvature of the interventricular septum increases. While this contractile movement enhances the bellows action of the free wall, its relative significance in overall RV performance remains unknown.

The next important consideration is related to the functional difference between RV and LV. Due to its configuration, the RV is ideal for ejecting relatively large amounts of blood with minimal myocardial shortening. Thus, the RV may be considered as a ‘volume-pump’. From the standpoint of energetics, the volume-work is more efficient than the pressure-work of the heart. Therefore, the myocardial oxygen consumption (MVO₂) of the RV is relatively low as compared with that of the LV.

In contrast, the RV chamber is not particularly well equipped with generating the high intra-cavity pressures. Due to the geometrical configuration, the tension of the right ventricular myocardium would have to be far greater than that of the LV under similar conditions. Under normal conditions, the pulmonary vasculature provides relatively moderate vascular resistance to blood flow from the RV. However, a sustained increase in pulmonary arterial pressure (e.g. pulmonary arterial hypertension) inevitably may result in cor pulmonale, and right heart failure. In comparison, it should be noted that the surface of the left ventricle is small in relation to its intracavitary volume. Thus, a decrease in the diameter of the main axis, resulting from contraction of the circumferential muscles, is responsible for approximately 85% of the volume changes in LV. Unlike RV, the LV is an excellent ‘pressure-pump’ capable of ejecting against the high-resistance system of the systemic circulation.
RV Function:

As stated previously, the RV is a flow generation pump. The reason for this is that the primary function of the pulmonary circulation is the exchange of oxygen and carbon dioxide between blood and alveolar gas at rates that depend upon the metabolic demands of the systemic tissues. The performance of this task, i.e. a flow generation, unlike that of the LV which is more or less primarily a pressure generation pump. The RV performs this task at minimal cost in energy expenditure and with maximal efficiency. In other words, under normal conditions, the pulmonary circulation functions with the input pressures less than 20% of the systemic circulation. It is of importance to realize that the low pressures render the pulmonary vasculature susceptible to mechanical influences that modify the relation between pressure and blood flow in the lung. These passive effects have practical implications for the assessment of pulmonary vascular status.

In order to maximize gas exchange in the pulmonary circulations, the pulmonary vasculature has the ability to control the distribution of blood flow within the lung by hypoxic pulmonary vasoconstriction to improve the matching of blood flow to ventilation (ventilation-perfusion ratio) in lung units. It is of great importance to note that the active mechanisms that affect the pulmonary vasculature are of our major interest since the potential pharmacological intervention by anesthetic agents may have significant consequence in treating the pulmonary heart disease.

In assessing RV function, it is important to emphasize that Frank-Starling's curve of the RV is different from that of the LV. This is due to at first, the geometry of the RV results in large exchanges in volumes with little muscle fiber shortening and also the relatively thin wall system of the RV is not capable of developing high pressures equivalent to those of the LV.

Increased afterload is probably the earliest abnormality in the development of pulmonary heart disease. Under these conditions, right ventricular hypertrophy (RVH), impaired myocardial contractile state, cardiac dilation, and increased preload may ensue. However, the cardiac output (CO) is often maintained at normal level until severe increases in afterload develops. This is due to the fact that large increases in diastolic volume of the RV result in small changes in end-diastolic pressure and that the RV output increases as end-diastolic volume is increased.

Therefore, the assessment of the RV function must be done in terms of both CO and RV end-diastolic pressure (RVEDP). The use of RV function curve by relating CO with various RVEDP becomes very important.

Right ventricular function appears to be preserved in most patients with COPD. Both CO and RVEDP are normal at rest and during exercise in these patients. The change in CO during exercise is appropriate for the change in oxygen consumption (VO₂). The CO increases
primarily as the result of increasing stroke volume (SV) not by increasing heart rate (HR). Patients with normal CO and RVEDP at rest but elevated RVEDP during exercise are considered to have latent right ventricular failure.

Overt RV failure is characterized at rest by a low CO and elevated RVEDP, and during exercise by a blunted increase in CO and SV, further increases in RVEDP, increased oxygen consumption relative to cardiac output, widening the A-V O₂ difference implying that any increases in CO are inadequate. There are accompanying marked increases in the pulmonary vascular resistance (PVR).

While marked elevation of RVEDP is generally accepted as hemodynamic evidence of right ventricular dysfunction, elevations of RVEDP at rest can be misleading in some patients. For example, fluid retention or increased circulating blood volume can increase RVEDP in the absence of RV failure. On the other hand, in certain clinical situations, RVEDP may be normal in patients with frank RV failure if ventricular compliance is increased due to cardiac dilation.

Increases in RVEDP during exercise are considered to be a more sensitive indication of RV dysfunction, and are more common in patients with electrocardiographic evidence of RVH, even in the absence of other clinical findings. The vectorcardiogram may be even more sensitive of early hemodynamic abnormalities, even in the absence of the RVH.

The SV is usually normal in patients with COPD, and is maintained in the setting of increased afterload during exercise by an increase in end-diastolic volume. The SV is abnormal in patients with severe RV failure, but there is some confusion about SV responses during exercise due to the effects of deconditioning, body position (supine versus upright) and the work rates at which measurements were made.

Oxygen pulse, which is related to SV (VO₂/HR = SV × aVO₂), may provide a noninvasive means to assess this parameter. The O₂ pulse is generally normal in patients with COPD. It is not certain this also holds true in patients with cor pulmonale.

A reduction in O₂ pulse may be useful in separating patients with COPD from those with cor pulmonale. But it probably will not distinguish right from left ventricular dysfunction because a diminished cardiac reserve exists in both conditions.

**Right Ventricular Ejection Fraction :**

A recently developed special purpose pulmonary artery catheter (American Edwards Laboratories; Ejection Fraction/Volumetric catheter) with a rapid response thermister enables us to measure beat-to-beat temperature variations and allows calculation of ventricular volume. With the recent increasing interest in the function of the RV and its relationship to that of the LV, it has become relevant to have access to the method that can reliably quantify the right ventricular function. Thus, we have recently designed a study to determine the “pump-
performance” of the RV in patients who are undergoing coronary artery bypass graft and mitral valve and/or aortic valve replacement surgery. In addition, another study was designed to evaluate critically the thermal technique to calculate ejection fraction as well as cardiac output in order to demonstrate the absolute necessity of actual recording of the thermodilution curve during measurements. We found that the accuracy and reproducibility of both cardiac output (CO) and right ventricular ejection fraction (RVEF) measurements demand recordings of the actual thermodilution curve with normal exponential decay5). We found also the RV-pump performance determined by RVEF in patients who underwent coronary bypass graft surgery and mitral and/or aortic valve replacement surgery is maintained at the normal level. The RVEF decreases only as LV failure develops during anesthesia and cardiac surgery6).

Like elevations in RVEDP, a depressed RVEF also is seen more commonly during stress and/or exercise. Some patients with normal RVEF at rest may decrease during stress and/or exercise.

SUMMARY

The importance of the cardiopulmonary monitoring, particularly the right ventricular function, in patients who are suffering from COPD, pulmonary heart disease, mitral and/or aortic valve disease and coronary artery disease with emphasis on the basic consideration of the right ventricular performance characteristics are discussed. The indications, risks and complications related to the cardiopulmonary monitoring are also presented.

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REFERENCES

3) Sarnoff SJ, Berglund E. Ventricular function: I. Starling’s law of the heart studied by means of simultaneous right and left ventricular function curves in the dog. Circulation 9: 706, 1954