Evaluating hypotension during anesthesia

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Hypotension is a common side effect that often occurs during the course of an anesthetic. Proper utilization of information obtained from a pulmonary artery catheter can aid the anesthetist in recognizing the reasons for hypotension. Individual determinants of cardiac output can be evaluated and a specific treatment can then be chosen. This article provides a basic approach for anesthetists beginning to use pulmonary artery catheters. It is intended as a basic approach to developing the ability to interpret hemodynamic parameters.

Every anesthetist hopes that all his or her patients will undergo a smooth anesthetic course. Clearly, though, this won't be the case 100% of the time. Individual patients have individual cardiovascular systems with individual hemodynamic responses.

The healthy patient who becomes hypotensive after induction or during the procedure usually responds well to a fluid challenge or a lightening of the anesthetic. But is a fluid challenge needed by the patient with a history of congestive heart failure? Is decreasing the anesthetic agent all that is needed for a patient with a history of myocardial infarction? Maybe, but how do we know for sure? There is no room for guessing in these patients.

The Swan-Ganz® catheter

Since the invention of the Swan-Ganz® catheter in the early 1970s, hemodynamic monitoring of the critically ill or injured patient has become much more involved. The catheter supplies valuable information that will aid the clinician in diagnosing, differentiating complications, and guiding and optimizing therapy. Many of these patients are candidates for emergency or elective surgery. The Swan-Ganz® catheter is a natural choice to monitor and guide the anesthetic management of these high risk patients.

The Swan-Ganz® catheter is a flow-directed catheter that is introduced into the pulmonary artery. A balloon at the tip of the catheter occludes the pulmonary artery, blocking off all blood from the right side of the heart into that segment of the pulmonary artery. The distal lumen then measures any pressure distal to the catheter tip. This pressure comes from the left side of the heart, which explains why pulmonary artery wedge (occluded) pressure is an indicator of left ventricle filling pressure.1

What are the indications for a Swan-Ganz® catheter? Dr. Swan summed it up nicely by saying that a pulmonary artery catheter is indicated in "... any situation in which a physician would consider placing a central venous line for the purpose of cardiovascular monitoring."2 Central venous pressure (CVP) is only a measurement of right ventricular filling pressure. It lends very little informa-
tion on left heart function. Utilizing the pulmonary artery catheter’s proximal lumen, right atrial pressure can also be monitored. The insertion technique has become so refined that the Swan-Ganz® catheter does not present much greater risk than a CVP line insertion. When one looks at the information gained by the pulmonary artery catheter as compared to that gained by the right atrial catheter, Dr. Swan’s statement seems reasonable.

**Catheter data and derived parameters**

The Swan-Ganz® catheter is utilized in obtaining pulmonary artery (systolic, diastolic, and mean) pressure, right atrial pressure (RAP) and pulmonary capillary wedge pressure (PCWP).\(^1\) Accurate measurements of cardiac output can be achieved in seconds utilizing a thermodilution technique along with a cardiac output computer.

Taking this data along with heart rate, peripheral blood pressure and body surface area, the CRNA can easily calculate several “derived parameters.” Mathematical equations can be used to determine stroke volume, cardiac and stroke index, coronary perfusion pressure, pulmonary and systemic vascular resistance, and right and left ventricular stroke work index. Parameters can be determined by using a slide rule or by feeding the equations into a programmable calculator.

What do these numbers mean to the anesthetists? How will they affect anesthetic management? First, cardiac output must be considered. This is the amount of blood, in liters per minute, that is ejected from the heart.\(^1\) We can take this one step further and look at cardiac index. Cardiac index is cardiac output divided by body surface area. Cardiac index thus is an individual measurement in each patient. For example, a small elderly lady with a 4 liters/minute cardiac output may be in a hypoperfusion state. But a 6' 4” 280 lb. athlete with a 4 liters/minute cardiac output may be in a hypoperfusion state.

Cardiac output is dependent on four variables: heart rate, pre-load, after-load, and myocardial contractility.\(^8\) Any change in one of these factors will influence myocardial performance. Hypotension occurs when cardiac output decreases.

Utilizing information from the pulmonary artery catheter, we can determine if cardiac output is optimal. If it isn’t, we can tell which variable has changed, determine the cause, and select appropriate and specific therapy. Not only can we determine changes in myocardial performance, we can also differentiate changes in right heart function versus left heart function.

**Heart rate**

Many athletes have normally slow heart rates with an adequate blood pressure while some people require a fast heart rate to maintain a normal cardiac output. Is this really a determinant of cardiac function? If we look at a simple equation for cardiac output, \(CO = SV \times HR\), we can easily see that if stroke volume remains constant, heart rate does directly affect cardiac output.

If bradycardia occurs with hypotension, a small dose of atropine may be all that is needed to return the patient to an optimal state. One may think that tachycardia should then increase cardiac output. This may be true in some cases. An increase in heart rate is one of the body’s first sympathetic responses to hypotension. But remember, as heart rate increases, stroke volume may decrease as ventricular filling time decreases. Slowing the heart rate by deepening the anesthesia or with medication (such as propanolol) may be necessary to increase ventricular filling time and return cardiac output to normal.

**Pre-load**

Starling’s Law is the basis of pre-load. This law may be simply stated as: cardiac output is directly proportional to venous return. Or in other terms, the heart, like any pump, can only pump out what is brought into it. Pre-load is filling pressures of the ventricles.\(^1\)

Pre-load of the right ventricle is measured by right atrial pressure. Certainly if CVP is low, left ventricular filling pressure will probably be low. But with a normal CVP and increased pulmonary vascular resistance, left ventricular filling pressure also could be low.

The left ventricular filling pressure is best determined by pulmonary artery wedge (occluded) pressure. Though not as exact as PCWP, pulmonary artery diastolic pressure (PADP) has also been used to assess left heart pre-load.

Crystalloids and colloids are best used to increase pre-load. Diuretics and IV nitroglycerine have been commonly used to lower pre-load. Position and high intrathoracic pressure (PEEP) may also affect pre-load.

**After-load**

After-load is the resistance the ventricle faces as blood is ejected.\(^4\) This resistance is regulated by the impedance generated by the arterial system.

Right heart after-load is measured by determining pulmonary vascular resistance. This may be increased in congestive heart failure and may be reduced by several anesthetic agents.\(^4\) Morphine
sulfate has been used in treating congested heart failure for its ability to reduce pulmonary vascular resistance.

Left heart after-load is determined by the systemic vascular resistance equation. Systemic vascular resistance has also been called peripheral vascular resistance and total peripheral resistance.

An increased systemic vascular resistance (SVR) and tachycardia are often the body's first physiological responses to hypovolemia. This is one of the reasons that all four of the determinants of cardiac output should be evaluated. Looking at the pre-load alone would probably indicate that fluids would stabilize the patient and allow SVR to return to normal. Without evaluating pre-load, lowering SVR in the hypovolemic patient could be disastrous.

Hypotension, with normal heart rate, pre-load, and myocardial contractility, would indicate a need to lower SVR. A vasodilator such as sodium nitroprusside (Nipride®) will decrease vascular resistance, thus reducing after-load.4

In the case of a low SVR, systemic vasodilation, as seen in anaphylaxis for example, would be the reason for hypotension. If an inhalation anesthetic is being used, obviously lowering the concentration or switching to narcotics might be indicated. IV infusions of epinephrine, norepinephrine (Levophed®), or dopamine could be used to increase SVR.4

Myocardial contractility

The force of the myocardium during systole may be the prime determinant of cardiac output. If the pump itself is damaged or failing, cardiac output cannot be maintained. This is commonly true in cardiogenic shock and/or congestive heart failure.

Even though myocardial contractility may be the prime determinant, it is not easily measured.4 Coronary angiographers can easily measure ejection fractions and myocardial wall tensions in the cath lab. But there is no easy formula to calculate contractility. Measuring the right and left ventricular stroke work index is an indirect way of measuring myocardial contractility and differentiating both ventricles. As these indexes fall, therapeutic intervention using digoxin, calcium, dopamine, or dobutamine should be considered. Plotting a Starling Curve, using right and left ventricular stroke work index with CVP or PCWP, may be more desirable.

Another way of assessing myocardial contractility is by using the process of elimination. Some feel that if cardiac output and index fall when pre-load, heart rate, and after-load remain constant, it is reasonable to assume that myocardial contractility has fallen.4 It is wise to remember that some anesthetic agents, especially nitrous oxide and inhalation agents, may depress myocardial contractility and may be the cause of a low cardiac output.4

Summary

Hypotension during an anesthetic has been a common side effect throughout the evolution of modern anesthesia. In the healthy patient it is sometimes well isolated. It may be relieved by trying to lighten the anesthetic or by administering a bolus of intravenous fluid.

Patients with compromised cardiovascular systems, major surgical candidates, or high risk patients often do not tolerate hypotension. In these patients, there is no margin for error; there is no time to try various therapies. Specific treatment administered as soon as possible is the best management.

The Swan-Ganz® catheter provides information that allows the anesthetist to determine the reason for the hypotension so that an appropriate therapy can be chosen. By evaluating the four determinants of cardiac output independently, changes in myocardial performance are easily distinguished. Evaluation of the right heart function is assessed by measuring CVP, heart rate, right ventricular stroke work index, and pulmonary vascular resistance. Pulmonary artery wedge or diastolic pressure, heart rate, left ventricular stroke work index, and systemic vascular resistance are the four determinants of left heart function.

The ability to evaluate independent variables of both right and left heart function allows the anesthetist to correlate clinical assessment, hemodynamic parameters, and pharmacodynamics. Then specific alternatives are available to return the high risk patient to an optimal hemodynamic state. Dr. Jastremski sums up the manipulation of hemodynamics in high risk patients: "Focus your efforts on 'optimal' rather than 'normal' as the goal of monitoring patients."12

The anesthetist's goal is to keep all patients in optimal condition throughout their intraoperative period. Through the use of hemodynamic parameters, anesthetic management is more specific and there is no "guess work" required in maintaining an optimal condition.

REFERENCES


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