Paradigm Shift in Hemodynamic Monitoring

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Citation

Abstract
Since the beginning of modern anesthesia, in 1846, the anesthesiologist has relied on his natural senses like finger on the pulse to monitor the patient, aided recently by sophisticated instruments like stethoscope, sphygmomanometer, ECG etc. The first paradigm shift in hemodynamic monitoring can be traced back to the development of cardiac catheterization by Werner Forssmann in 1929 and subsequent introduction of pulmonary artery catheterization and thermodilution techniques, in 1970, by Swan, Ganz and colleagues. For more than three decades pulmonary artery catheter thermodilution method has generally been accepted and is still the clinical standard to which other methods are compared. The long history of use has led to much experience with its technology, clinical application and inadequacies. Recent advances in technology have led to the development of minimally invasive and non-invasive methods. The development of impedance cardiography and advances in electronics and signal processing has led to the development of completely non-invasive monitors which can provide continuous measurement of hemodynamic parameters and can be considered as the recent paradigm shift in critical care monitoring. The present article deals with the various invasive, minimally invasive and non-invasive techniques currently in use and their physiological basis.

INTRODUCTION

The overthrow of Ptolemaic cosmology by Copernican heliocentrism, and the displacement of Newtonian mechanics by quantum physics and general relativity, is examples of major paradigm shifts. Both movements eventually changed the world view.

Paradigm shift can be defined as movement from one thought system to another. It just does not happen, but rather is driven by agents of change. For example, agriculture changed early primitive human beings from wandering hunters to social beings. Likewise, the printing press, the making of books and the use of vernacular language changed the culture of a people and had a direct effect on scientific revolution. Johann Gutenberg's invention of movable type, in the 1440's, was an agent of change. Books became readily available, smaller and easier to handle and cheap to purchase. Masses of people acquired direct access to the scriptures, that heightened their awareness and attitudes began to change, as people were relieved from clutches of domination. Similarly, agents of change are driving a new paradigm shift today. The signs are all around us. For example, the introduction of the personal computer and the Internet have impacted both personal and business environments, and is shifting us from a mechanistic, manufacturing, industrial society to a service based, information centered society, and increases in technology will continue to impact globally. Change is inevitable. It's the only true constant.

Hemodynamics is defined as the study behind the forces involved with blood circulation. Hemodynamic monitoring started with the estimation of heart rate using the simple skill of ‘finger on the pulse’ and then moved on to more and more sophisticated techniques like stethoscope, sphygmomanometer, ECG etc. The concept of measuring the pressure directly with in the veins, heart and arteries, originated in the minds of a German physician named Werner Forssmann in 1929, and can be considered as the first paradigm shift in hemodynamic monitoring. Working alone in 1929, Dr. Forssmann, threaded a urologic catheter
through a vein in his arm and into his heart. This procedure was done clandestinely since he did not have official approval for such a daring experiment. Dr. Forssmann took a chest X-ray, which demonstrated the catheter's position, and he published the procedure as a brief report. His primary purpose was to develop a technique for direct delivery of drugs to the heart. Cardiac catheterization did not become a clinically useful test, however, until the late 1940's, following the work of Dr. Dickinson W. Richards and Dr. Andre Cournand. With this paradigm shift, the definition of hemodynamic monitoring itself has changed. It is defined as the direct measurement of blood pressure inside the veins, heart and arteries. It consists of observing how the cardiovascular system responds to injury, illness and therapeutic intervention. For cardiac catheterization's revolutionary effect on cardiac diagnosis, all three physicians shared the 1956 Nobel Prize for Medicine.

The hemodynamic status of critically ill patients can be assessed either from non-invasive single parameter indicators or various invasive techniques that provide multi-parameter hemodynamic measurements. As a result, comprehensive data can be provided for the clinician to proactively address hemodynamic crisis and safely manage the patient instead of reacting to late indicators of hemodynamic instability. Because of the risks and costs associated with invasive monitoring techniques, a large fraction of high-risk patients are left underserved.

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Hemodynamic instability is defined as global or regional perfusion that is not adequate to support normal organ function. This definition recognizes the anesthesiologist's or the intensivist's obligation to ensure adequate organ perfusion during the perioperative period. The two variables that most directly reflect organ perfusion are blood pressure and cardiac output. Cardiac output and blood pressure are intimately intertwined. Because of limits on coronary and cerebral auto-regulation, hypotension may compromise adequate perfusion of the brain and heart.

The basic tenet of hemodynamic monitoring is the control of adequate oxygen delivery to tissues. The primary physiologic response to an increased demand in tissue oxygen or to a reduced content in arterial oxygen is to increase cardiac output. However, most cardiovascular disorders limit the heart's ability to respond to those needs. As a result, tissues rely on a second compensatory mechanism by drawing on the venous oxygen reserve. Consequently, when the tissue oxygen supply becomes inadequate, even for brief episodes, lactic acidosis and tissue damage may arise. The primary objective in the management of critically ill patients is to prevent such tissue hypoxia.

While an adequate blood pressure may insure adequate coronary and cerebral perfusion, it does not ensure renal and mesenteric perfusion. While a good cardiac output cannot assure us that the kidneys and lungs are being perfused, it certainly seems evident that a poor cardiac output will put renal and mesenteric perfusion in jeopardy. Inadequate mesenteric perfusion with breakdown of gut endothelium may allow translocation of bacteria, initiating gram negative sepsis. It is prudent to look at multiple indicators of adequate perfusion, such as cardiac output, mixed venous oxygen saturation and lactate concentration. A normal lactate level is a fairly specific indicator of adequate tissue perfusion. An elevated lactic concentration may reflect problems other than those related to perfusion (eg. sepsis, hyper metabolic states, hepatic dysfunction).

Indications of hemodynamic monitoring are decreased cardiac output due to dehydration, hemorrhage, gastrointestinal bleed, burns, all types of shock – hypovolemic, septic, cardiogenic, neurogenic, anaphylactic, any deficit or loss of cardiac function as in acute myocardial infarction, cardiomyopathy, congestive cardiac failure.

Multiple organ dysfunction syndrome accounts for most deaths in the intensive care units. Patients who develop these complications place an enormous burden on all hospital services especially intensive care. Although the exact pathophysiology of multiple organ dysfunction syndrome is not yet definitely known, alterations in systemic hemodynamics, organ perfusion and tissue microcirculation resulting in tissue hypoxia appear to play a key role in the onset and maintenance of this syndrome. Introduction of hemodynamic monitoring had a revolutionary impact on evaluation and management of critically ill. However, optimal monitoring of the critically ill patient remains a challenge. Controversy continues as to whether the patient will benefit from a more aggressive monitoring.

TECHNIQUES OF HEMODYNAMIC MONITORING

HEART RATE MONITORING

The simplest and the least invasive form of cardiac monitoring remains the measurement of heart rate. Electronic monitoring devices are used to provide a continuous display of heart rate.

ARTERIAL BLOOD PRESSURE MONITORING

Of all the hemodynamic variables, monitoring of blood
pressure is the most straight forward. As with heart rate, blood pressure is a fundamental cardiovascular sign which reflects the force that maintains the perfusion of the body. In addition, it is the most important determinant of left ventricular afterload and workload of the heart. Techniques of arterial pressure monitoring can be divided into indirect and direct methods.

The indirect methods are manual intermittent technique using the sphygmomanometer, automated intermittent technique using the automated non invasive (NIBP) device, and automated continuous technique using non-invasive finger blood pressure measurement device.

Direct measurement of arterial blood pressure is by direct arterial cannulation. Indications for and advantages of direct arterial blood pressure are arterial blood sampling, continuous real time monitoring, intentional pharmacological or mechanical cardiovascular manipulation as in cardiac surgery with cardiopulmonary bypass, intra-aortic counter balloon counter pulsation, administration of vasoactive drug infusion, failure of indirect blood pressure measurement as in morbidly obese patients, supplementary diagnostic clues by critical analysis of the arterial pressure wave form 7,8,9.

Complications of direct arterial pressure monitoring include equipment faults on mis-assembly, kinked or disrupted arterial line, vasospasm, arterial line being mistaken for intravenous line, fatal hemorrhage following difficult femoral artery cannulation, upper extremity compartment syndrome following brachial artery cannulation, retrograde arterial embolism when forceful flushing of a peripheral arterial catheter is employed 10,11.

CARDIAC FILLING PRESSURE MONITORING

Cardiac filling pressures are monitored to estimate cardiac filling volumes, which, in turn, determine the stroke outputs of the left and right ventricles. According to Frank-Starling principle, the force of cardiac contraction is directly proportional to end-diastolic muscle fibre length at any given level of intrinsic contractility or inotropy 12,13. This muscle fiber length or preload is proportional to end-diastolic chamber volume.

Cardiac filling pressures are measured directly from a number of sites in the vascular system. Central venous pressure (CVP) monitoring is the least invasive method, followed by pulmonary artery pressure (PAP) monitoring and left atrial pressure (LAP) monitoring.

CENTRAL VENOUS PRESSURE

CVP reflects the balance between systemic venous return and cardiac output. It is best used for patients without pre-existing cardiac disease as an indicator of the adequacy of venous return and cardiac output. Indications for central venous cannulation are CVP monitoring, pulmonary artery catheterization and monitoring, transvenous cardiac pacing, temporary hemodialysis, drug administration, rapid infusion of fluids, aspiration of air emboli and inadequate peripheral intravenous access. Among the numerous sites for central venous cannulation, the most popular are the right internal jugular vein and subclavian veins. The alternate sites are left jugular vein and subclavian veins, femoral veins and axillary veins. Peripherally inserted central venous catheters (PICC) have become a popular alternative to centrally inserted catheters in patients requiring long term intravenous therapy. Venous access for a PICC is obtained through an antecubital vein.

In the normal heart, the right ventricle is more compliant than the left. The use of CVP to assess the left-sided pre-load causes difficulty because CVP primarily reflects changes in pulmonary venous and left sided pressures 14,15. The normal range of CVP is between 4 and 8 mm Hg. Measurements of CVP are affected by ventilation because transthoracic pressure is transmitted through the pericardium and the thin walled vena cavae. During spontaneous ventilation, inspiration lowers CVP while expiration increases it. The situation is reversed in patients being mechanically ventilated, in whom inspiration increases intrathoracic pressure and elevates CVP. The degree of this elevation depends on the compliance of the lungs and intravascular volume and varies between patients. When positive end – expiratory pressure is applied, the positive pressure is transmitted through to the right atrium, causing a decrease in venous return and a rise in CVP. In critical care situations, an esophageal probe can be inserted to estimate the transthoracic pressure. Subtracting the transthoracic pressure from CVP provides transmural pressure, which is a better estimate of right atrial pressure in the presence of elevated transthoracic pressure 16.

Complications of CVP monitoring are inadvertent arterial insertion, pneumothorax, nerve injury, perforation of superior venacava, guide wire induced arrhythmias, air embolism, venous thrombosis, hydrothorax, hydromediastinum, infection 17,18,19.
PULMONARY ARTERY PRESSURE.

Although the concept of using a balloon-assisted catheter had been published in the mid-fifties, a serendipitous observation by a noted cardiologist, H.J.C. Swan in 1970, led to its further development.

On a rare day at the beach in California, H.J.C. Swan noticed a sailboat moving quickly despite the calm weather. This observation led to the initial idea of devising a catheter with a parachute-like or sail-like device attached to its tip that will carry the catheter effortlessly along with the blood flow. It was not that he had never been to the beach before or it was not the first time that he was seeing effortless quick movement of a sailboat. In fact he was a frequent visitor to the Californian beach and had seen effortless cruising of sailboats, in a calm weather, many a times. However, the observation on that particular day was serendipitous that a novel idea burgeoned in his mind, which ultimately gave birth to the present day PA or SG catheter.

Initial testing was conducted with a balloon-tipped catheter because it was easier to fabricate. It proved so successful that the original parachute idea was abandoned. At the same time, the work of William Ganz on the thermodilution method of measuring cardiac output (CO) was incorporated into the catheter’s use. This basic design remains in use today. Interestingly, despite the widespread use of their names for the flow-directed balloon-tipped PA catheter (also known as the Swan-Ganz catheter [SGC]), neither the physicians nor the original manufacturer could obtain a patent.

Swan, Ganz and colleagues introduced pulmonary artery catheterization (PAC) for hemodynamic monitoring into clinical practice in 1970 using the balloon-tipped, flow-directed, pulmonary artery catheter. At present about 2 million catheters are used per year in North America. Despite its widespread use, no formally reviewed recommendations exist for its general use. Many publications present only individual authors’ suggestions for indications and contraindications to the use of the SGC. However, the American College of Cardiology has developed a consensus statement regarding its use. Indications are broadly classified to diagnostic and therapeutic. The diagnostic indications are: diagnosis of shock states, differentiation of high-pressure versus low-pressure pulmonary edema, diagnosis of primary pulmonary hypertension, valvular disease, intracardiac shunts, cardiac tamponade, and pulmonary embolus, monitoring and management of complicated acute myocardial infarction, assessing hemodynamic response to therapies, management of multiorgan failure, severe burns, and hemodynamic instability after cardiac surgery, assessment of response to treatment in patients with primary pulmonary hypotension. The therapeutic indication is aspiration of air emboli. PAC is contraindicated in patients with tricuspid or pulmonary valve mechanical prosthesis, right heart mass (thrombus, tumor) and tricuspid or pulmonary valve endocarditis.

PAC is performed to measure hemodynamic variables like PAP, pulmonary artery wedge pressure (PAWP), mixed venous oxygen saturation and cardiac output in critically patients. These pressure measurements are used to estimate left ventricular filling pressure (LVP) and help guide fluid and vasoactive drug administration when clinical signs, symptoms or other monitored variables are felt to be inadequate or unreliable.

The SGC is inserted percutaneously in to a major vein (internal jugular, subclavian) via an introducer sheath. When the catheter is inserted through either the subclavian or the internal jugular vein, the typical distances required are as follows: right atrium 20 to 25 cm, right ventricle 30 to 35 cm, pulmonary artery 40 to 45 cm and pulmonary capillary wedge 45 to 55 cm. As the balloon floatation catheter is advanced through the heart, characteristic pressure waveforms are obtained that indicate the position of the catheter’s distal port (Fig. 1).

Figure 1

Figure 1: Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances.

Simultaneous ECG monitoring is essential to ensure that
ventricular arrhythmias will be detected as the catheter traverses the right ventricle. When a pulmonary artery catheter floats to the wedge position, the inflated balloon at its tip isolates the distal pressure monitoring from upstream PAP. Blood flow ceases between the catheter tip and a junction point where pulmonary veins draining the occluded pulmonary vascular region join other veins in which blood still flows towards the left atrium. A continuous static column of blood now connects the wedged pulmonary artery catheter tip to this junction point in the pulmonary veins near the left atrium. Thus wedging the pulmonary artery catheter functionally extends the catheter tip to measure the pressure at the point at which blood flow resumes on the venous side of the pulmonary circuit (Fig. 2).

**Figure 2**

Figure 2: With the balloon inflated the PAC floats and wedges into a capillary of the pulmonary artery. When wedged the PAC creates an unrestricted channel from the catheter tip to the left ventricle, thus allowing the distal lumen to indirectly measure left ventricle pressure.

Because resistance to flow in the large pulmonary veins is negative, PAWP provides an accurate, indirect measurement of both pulmonary venous pressure and LAP.

The final position of the catheter tip within the pulmonary artery is critical. This may be described with reference to the physiological model of the pulmonary vasculature which is divided into three zones based on the gravitationally determined relation between PAP, pulmonary venous pressure ($P_v$) and alveolar pressure ($P_a$) (Fig. 3).

**Figure 3**

Figure 3: Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3.

A pulmonary artery catheter positioned in zone 1 and 2 will be influenced by alveolar pressure and bear little relation to the downstream pulmonary venous pressure or left ventricular filling pressure. Under these circumstances, alveolar or airway pressures are being monitored rather than the intended vascular pressure in the left atrium or ventricle. In general, zones 1 and 2 become more extensive when LAP is low, when the pulmonary artery catheter tip is vertically above the left atrium or when the alveolar pressure is high. Only in zone 3 is there an uninterrupted column of blood between the catheter and the left atrium. Decreased airway pressures change the ventilation-perfusion relationship, producing a relative increase in zone 3. Correct placement of pulmonary artery catheter should be ensured by chest X-ray.

Indicators of proper tip placement include a decline in pressure as the catheter moves from the pulmonary artery into the wedged position, ability to aspirate blood from the distal port, and a decline in end-tidal CO$_2$ concentration with inflation of the balloon (produced by a rise in alveolar dead space).

The PAWP estimates left ventricular end diastolic pressure.
and thus serves as an estimate of left ventricular preload. Because the pulmonary vasculature forms a low resistance circuit, the pulmonary end diastolic pressure is only 1 to 3 mmHg higher than the mean PAWP and can be used to estimate left ventricular pressure when the PAWP is not available-in the absence of severe lung disease 27.

Pulmonary capillary filtration pressure ($P_{cap}$) is a measure of the potential difference that drives fluid from the pulmonary vasculature to the perivascular interstitial and alveolar spaces. The equation relating mean PAP, PAWP and $P_{cap}$ is:

$$P_{cap} = PAWP + 0.4 \times (PAP - PAWP)$$

Acute respiratory distress syndrome widens the PAP to PAWP gradient and increases $P_{cap}$, contributing to pulmonary edema 27.

Correlation between CVP and PAWP may be poor in critically ill patients with cardiopulmonary disease because of differences between right and left ventricular function.

PAWP correlates best with LAP when the latter is less than 25 mm Hg. When LAP increases to more than 25 mm Hg which may occur after acute myocardial infarction-PAWP tends to underestimate left ventricular end-diastolic pressure (LVEDP). As left ventricular function deteriorates, the contribution that atrial contraction makes to left ventricular filling is increased, and LVEDP can be significantly higher than PAWP. High positive airway pressures (PEEP more than 15 mmHg) result in pulmonary vascular collapse, causing PAWP to reflect airway pressure instead of LAP 26.

**CARDIAC OUTPUT MONITORING**

Apart from its pressure monitoring capabilities, undoubtedly the most important feature of PAC is its ability to measure cardiac output using the thermodilution method. Cardiac output is the primary compensatory mechanism that responds to an oxygenation challenge. It is thus a clinical parameter ensuring the adequacy of tissue oxygenation. It provides a global assessment of the circulation including the neurohumoral influences on it. Cardiac output is the product of stroke volume and heart rate. Stroke volume is determined by preload that is the left ventricular end-diastolic volume, myocardial contractility and afterload that is the resistance against which the left ventricle ejects. Cardiac output measurements are combined with other hemodynamic measurements to calculate systemic and pulmonary vascular resistance.

**INVASIVE METHODS**

**INDICATOR DILUTION**

In this method a bolus of dye is injected intravenously at the same time that peripheral arterial blood is withdrawn. Arterial sample is continuously passed through a densitometer that calculates dye concentration as function of time based on light absorption. This method requires a catheter, arterial blood withdrawal and a densitometer. The area under the concentration – time curve (Fig. 4) is calculated and Stewart-Hamilton equation is applied.

**Figure 5**

*Figure 4: Variation of dye concentration as a function of time.*

This is approximated to: Cardiac Output = $60 \times$ indicator dose(mg) / (average concn. x time) where $I$ is the amount of indicator and $C_0$ is indicator concentration 34,35. Indicator dilution method is complicated due to the need to simultaneously infuse dye and withdraw blood. It is also not the recommended method for any patient with intracardiac
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shunt since an intact cardiac chamber is necessary for the blood and indicator dye to mix before the downstream sample is obtained.

THERMODILUTION

The thermodilution technique has become the de facto clinical standard for cardiac output determination. This technique relies on principle similar to indicator dilution but uses heat instead of colour as an indicator.

Figure 6

Figure 5: A normal thermodilution curve

A pulmonary artery catheter with multiple ports is advanced into the pulmonary artery. Ice cold saline bolus of known volume (5 – 10 ml) is injected through the proximal (RA) lumen and a thermistor at the end of the SGC measures the change in blood temperature as a function of time. The temperature – time curve (Fig. 5) so obtained allows calculation of blood flow. This method is simpler and its accuracy is reasonable. It requires a Swan-Ganz catheter. The area under the time - temperature curve is inversely proportional to cardiac output. A high cardiac output will have a small area under the curve. This is due to the rapid blood flow through the heart. In low cardiac output the opposite is true. Cardiac output is calculated using the modified Stewart-Hamilton indicator dilution equation:

\[
\text{Cardiac Output} = \frac{(T_B - T_I) \times K}{0^l \int_0^\infty \Delta T_B(t) \, dt}
\]

The constant \( K = \frac{V_I \times S_I \times C_I \times 60}{S_B \times C_B} \)

where \( V_I \) is the volume injectate (ml) and \( T_B, T_I, S_B, S_I, C_B, C_I \) are temperature, specific gravity, specific heat of blood (B) and indicator (I) respectively. Either iced or room temperature thermal boluses can be used although the use of iced fluid improves the signal – to – noise ratio. For best results the difference between the temperature of the blood and that of the injectate should be at least 12°C. Bolus injection speed and warming of the indicator as it passes through the catheter have only minimal effects 36, 37. Common to both indicator and thermo dilution techniques are the requirements for stable blood flow, constant blood volume, absence of recirculation and constant indicator distribution time.

CONTINUOUS CARDIAC OUTPUT MONITORING

The usefulness of cardiac output monitoring has now been further enhanced by the recent development of reliable continuous cardiac output monitoring devices. These methods utilize electrical impulses to generate heat in coils mounted on the right ventricular portion of the pulmonary artery catheter. Multiple small heat signals are generated that result in a corresponding pattern of changes in pulmonary arterial blood temperature, measured by the pulmonary arterial thermistor. Utilizing computer algorithms, the monitoring devices are programmed to analyze these patterns of temperature to calculate a cardiac output value, which reflects the average cardiac output for the previous 3 – 5 minutes. The continuous data may be coupled with arterial and mixed venous oximetry to provide continuous data reflecting oxygen delivery and oxygen consumption.

FICK’S CARDIAC OUTPUT MEASUREMENT

This is a form of indicator dilution method in which exogenous indicators are not required but, instead transported oxygen serves this purpose. The conventional Fick method is based on the principle that \( O_2 \) consumption is proportional to the rate of blood pumped by the heart through the lungs, and monitoring gas exchange and invasively sampling blood gases can measure it. Cardiac
output may be calculated by relating oxygen consumption to arterial and mixed venous oxygen content using the equation

\[ \text{Cardiac Output} = \frac{\text{VO}_2}{(C_{a\text{O}_2} - C_{v\text{O}_2}) \times 10} \]

\( \text{VO}_2 \) is the oxygen consumption, \( C_{a\text{O}_2} \) is oxygen content of arterial blood and \( C_{v\text{O}_2} \) is oxygen content of mixed venous blood. Calculation of cardiac output using Fick's equation is a reference with which all other techniques are compared. The arterio-venous oxygen content difference requires that a pulmonary artery catheter be placed to obtain mixed venous blood. Oxygen consumption is calculated by measuring the oxygen content difference between inspired and exhaled gas. A disadvantage of Fick's method is that the patient must remain in a stable metabolic state, outside the sphere of sudden intervening variables like shivering, pain, extreme emotional stress etc. Such variables can affect oxygen consumption and arterial and mixed venous oxygen content, thereby, interfering with an accurate reflection of cardiac output. Though it is not a practical bedside method, it is the most accurate method available to evaluate patients with low cardiac output.

**MINIMALLY INVASIVE METHODS**

**DOPPLER ULTRASOUND**

The transesophageal Doppler cardiac output monitoring is the most widely applied method of minimally invasive cardiac output monitoring performed currently in critically ill patients and can be considered as a paradigm shift in hemodynamic monitoring. The transesophageal ultrasound monitoring combining echographic (M-mode) and pulsed Doppler measurements provide a minimally invasive approach to real-time continuous monitoring of the cardiac function. This provides an immediate, comprehensive, left ventricular flow based assessment of the net effects of changes in the fundamental hemodynamic component i.e. stroke volume, heart rhythm, preload, contractility, and afterload.

The volumetric flow rate of an established flow as a function of time passing through a blood vessel is expressed as the product of blood vessel cross-sectional area (at time t) and the spatial average velocity of blood over the entire cross-section. Therefore, at each instance, the two simultaneous local measurements required are a geometric parameter determined by anatomy (vessel cross-section) and a physiologic parameter (blood velocity) reflecting the heart's performance as a pulsatile pump as modified by vascular tone.

The Doppler probe is inserted in to the esophagus to a depth of approximately 35 cms from the incisors in an intubated patient. Probe position is adjusted to optimize the audible Doppler flow sound from the descending aorta. The transesophageal probe is equipped with two ultrasonic transducers one of which is an M-mode echograph, which measures the blood vessel cross-sectional area, and the other is a pulsed Doppler transducer, which measures blood velocity. Because the esophagus and the descending aorta lie in close proximity and run essentially parallel to one another, the ultrasound transducer is mounted at a fixed angle, such that the ultrasound beam is oriented as much as possible in a direction that is parallel to the blood flow, that is known by the cardiac output computer and is used to correct the resulting Doppler shift frequency to provide an accurate blood velocity measurement. The current clinical role for esophageal Doppler cardiac output monitoring is not as a replacement for PAC, but as an additional circulatory monitor in patients who do not warrant invasive monitoring.

The other methods for measuring cardiac output using ultrasonic techniques are: suprasternal and transtracheal Doppler where blood velocity in the ascending aorta is measured and Doppler PAC where a PAC is equipped with three ultrasound transducers at the catheter tip. Doppler PAC determines space averaged pulmonary artery blood velocity and vessel cross-section throughout the cardiac cycles. However, this technique has limited clinical acceptance because of its requirement for PAC.

**PARTIAL CO REBREATHING**

The partial CO rebreathing technique uses the differential form of the Fick principle, applied to CO produced by the body and eliminated through gas exchange in the lungs, for non-invasive measurement of cardiac output. With partial rebreathing, a change in VCO and an associated change in end-tidal CO, in response to a change in ventilation, is used in the Fick calculation. The NICO system accomplishes the required change in ventilation by using the rebreathing valve and NICO rebreathing loop. By temporarily adding a rebreathing volume to the breathing circuit, the patient inhales only a portion of the exhaled gases. The resulting changes in VCO and end-tidal CO are used to calculate the
cardiac output. With total rebreathing, the patient inhales his or her own exhaled gas from a bag attached at the mouth. During the total rebreathing maneuver, no CO\(_2\) is eliminated from the lungs and the concentration of exhaled CO\(_2\) approaches the mixed venous concentration, allowing it to be estimated non-invasively from respiratory gas measurements. End-tidal CO\(_2\) is used as a non-invasive estimate of the arterial CO\(_2\) concentration. Even though the total-rebreathing CO\(_2\) technique allows non-invasive cardiac output estimation based on routinely obtained respiratory gas measurements, it is impractical for use in critically ill patients because it requires a skilled operator and patient cooperation for reliable results.

**PULSE DYE DENSITOMETRY**

It is a method for monitoring cardiac output and circulating blood volume. It detects arterial indocyanine green dye concentration following its intravenous injection. The method relies on the measurement of arterial blood need for blood withdrawal. The new pulse dye densitometry method is a less invasive version of the dye dilution method that does not require calibration before measurement. Dye is injected into a vein close to the atrium but no catheter is required. No blood sampling is required; dye concentration is measured continuously and noninvasively by pulse spectrophotometry using a clip-on probe.

**LIDCO INDICATOR DILUTION**

In this technique a bolus of isotonic LiCl solution (150mM) is injected via a central or peripheral venous route and the concentration of LiCl as a function of time is recorded using an ion-selective electrode attached to the arterial line manometer system. The cardiac output is calculated from the Li dose and the area under the concentration – time curve prior to recirculation using the formula

\[
CO = \frac{\{Li \text{ dose (mmol)} \times 60\}}{\text{Area } \times (1-\text{PCV}) \text{ (m mol/sec)}}
\]

where PCV – packed cell volume (Hb (g/dl))/34. The technical innovation of the LiDCO system is the design and application of the ion-selective electrode, which comprises a Li sensitive electrode situated in a flow through cell. Blood flows into the sensor assembly at a rate that is controlled by a battery operated peristaltic pump. Beat by beat cardiac output can be obtained by calibrating an arterial blood pressure trace analysis algorithm. It can also be obtained by coupling LiDCO device to another device (Pulse CO) which calculates continuous cardiac output following LiDCO calibration, by analysis of the arterial waveform.

**PULSE CONTOUR ANALYSIS**

Pulse contour methods use the pressure waveform as an input for a model of the systemic circulation in order to predict instantaneous flow. The pressure waveform is not obtained from the aorta itself but rather from a peripheral artery (radial or femoral), which requires assumptions to be made regarding the changes in pulse shape between these different locations. The models used to represent the systemic circulation may vary according to specific pulse contour device. The values attributed to model parameters (resistance, compliance and characteristic impedance) are initially estimated according to the patient's sex and age, and from the pressure waveform. They are then refined following a calibration of mean cardiac output using an indicator dilution technique. By providing a reference value for peripheral resistance (ratio of mean arterial pressure to mean systemic flow), this calibration allows the system to compute more precisely the other parameters that represent arterial mechanical properties and to obtain a better estimation of cardiac output.

**NON-INVASIVE METHODS**

**ELECTRICAL IMPEDANCE CARDIOGRAPHY**

In the mid 1960s, the National Aeronautical and Space Administration (NASA) researchers and William Kubiceck developed the first practical method of impedance cardiography using the thoracic electrical bio-impedance to study the effects of zero gravity on the cardiac hemodynamics of astronauts. The technique measures electrical resistance changes through the thorax as aortic blood volume increases and decreases during systole and diastole. Continuous measurement of the change in impedance caused by the fluctuation of blood volume through the cardiac cycle make it possible to measure, calculate and continuously monitor stroke volume, cardiac output, myocardial contractility and total thoracic fluid status. The technique is a major paradigm shift in hemodynamic monitoring.

Impedance is the resistance to the flow of electrical current. Blood and other fluids are excellent conductors of electricity and have low impedance, particularly compared to bone, tissue and air. Blood and fluid in the lungs are the most conductive substances in the thorax. Thus larger quantities of thoracic fluid or blood lower impedance and smaller quantities result in increased impedance.
In impedance cardiography, four pairs of electrodes and a set of ECG leads measure hemodynamic parameters. Each pair of electrodes is comprised of a transmitting and sensing electrode. Two pairs are applied to the base of the neck on directly opposite sides and two pairs are placed at the level of the sternal-xiphoid process junction, again directly opposite from each other (Fig. 6).

Figure 9
Figure 6: Application of electrodes in impedance cardiography

The dot electrodes define the upper and lower limits of the thorax and the distance between them is measured to obtain the thoracic length (L).

A high frequency, low amplitude alternating current is introduced through the transmitting thoracic electrodes and the sensing thoracic electrodes measure impedance associated with the pulsatile blood flow in the ascending aorta which occurs during the cardiac cycle. By measuring the impedance change generated by the pulsatile flow and the time intervals between the changes, stroke volume can be calculated. Increased blood volume, flow velocity and alignment of red blood cells during systole reduces impedance. Conversely, decrease in blood volume and flow and more random configuration of red blood cells during diastole causes an increase in impedance.

The change in impedance is measured from the baseline impedance \( Z_0 \) that is the overall thoracic resistance to flow of electrical current. \( Z_0 \) predominantly reflects total thoracic fluid volume. The magnitude and rate of the impedance change is a direct reflection of left ventricular contractility. This change in impedance related to time \((dZ/dt)\) generates a waveform that is similar to the aortic flow curve.

Simultaneous recording of the ECG creates a timing window for the evaluation of each cardiac cycle. During the systole a volume of blood is ejected into the lungs subsequently blood flows away from the lungs to the left atrium. The stroke volume can be determined from the impedance curve by extrapolating to the impedance \((\Delta Z)\) that would result if no blood were to flow out of the thorax to during systole. If no blood were to flow away from the thorax during systole, the impedance would continuously decrease during systole at a rate equal to the maximum rate of decrease of \( Z \). Thus \( \Delta Z \) can be approximated by drawing a tangent to the impedance curve at the point of its maximum rate of change. Then the difference between the impedance values of the tangent line at the beginning and the end of the ejection time is \( \Delta Z \). The value of \( \Delta Z \) is easy to determine with the help of the first derivative curve of the impedance signal. Hence \( \Delta Z \) is the product of the ejection time and absolute of the maximum value of the first derivative of the impedance signal. The stroke volume is calculated using the formula,

\[
\text{Stroke Volume} = \frac{L^2}{\rho} \times \frac{(dZ/dt)_{\text{max}} \times \text{VET}}{Z_0^2}
\]

where \( \rho \) – resistivity of blood, \( L \) – mean distance between the inner electrodes (the thoracic length), VET - ventricular ejection time, \((dZ/dt)_{\text{max}}\) - the absolute of the maximum value of the first derivative during systole and \( Z \) - basal thoracic impedance. VET is obtained from the \( dz/dt - t \) curve (Fig. 7).
Paradigm Shift in Hemodynamic Monitoring

Figure 11
Figure 7: Variation of ventricular, aortic and atrial pressure, aortic flow, thoracic impedance change and first derivative of impedance (dz/dt) as a function of time (t). ECG and phonocardiogram taken simultaneously is also shown. The curve depicts the cardiac events / performance. B = Opening of the Aortic Valve, X = Closure of the Aortic Valve, Y = closure of pulmonary valve, O = mitral valve opening/rapid ventricular filling, B-X = Ventricular Ejection Time (VET), C = Maximal deflection of dz/dt (Peak Flow), B-C slope = Acceleration Contractility Index, A = Atrial Systole, Q = Start of ventricular depolarization

Heart is an electro-mechanical pump and ECG represents the electrical and impedance the mechanical activities. The mechanical events viz. the ventricular, aortic and atrial pressure variations and aortic blood flow during the cardiac cycle are shown along with $\Delta Z$, dz/dt, ECG and phonocardiogram in Fig. 7.

Cardiac output is then calculated from the stroke volume and heart rate. In addition to providing continuous measures of stroke volume and cardiac output, impedance cardiography also measures thoracic fluid content, left ventricular ejection time, systemic vascular resistance and left cardiac work index. Thoracic fluid content (TFC) is a measurement of the net fluid content of thorax, including intravascular, intra-alveolar, and interstitial. The TFC measurements can be used in conjunction with stroke volume and cardiac output to give much of the same information provided by PAWP.

**DERIVED PARAMETERS**

Cardiac output measurements may be combined with systemic arterial, venous, and PAP determinations to calculate a number of variables useful in assessing the overall hemodynamic status of the patient. They are,

Cardiac index = Cardiac output / Body surface area

Systemic vascular resistance = $[(\text{Mean arterial pressure} - \text{CVP}) / \text{Cardiac output}] \times 80$

Pulmonary vascular resistance = $[(\text{PAP} - \text{PAWP}) / \text{Cardiac output}] \times 80$

Stroke volume index = Stroke volume / body surface area

Right ventricular stroke-work index = 0.0136 (PAP – CVP) x Stroke volume index

Left ventricular stroke-work index = 0.0136 x (Mean arterial pressure – PAWP) x Stroke volume index

**MONITORING OF RIGHT VENTRICLE**

The focus of interest in hemodynamic monitoring has been on the dominant left side of the heart. The tendency to overlook the right ventricle as an important part of the circulatory system occurred because it was traditionally regarded as a passive conduit, responsible for accepting venous blood and pumping it through the pulmonary circulation to the left ventricle. Maintenance of normal circulatory homeostasis depends on an adequate function of both ventricles. Changes in dimension and performance of one ventricle influences the geometry of the other. There is growing interest in the importance of the neglected right side of the heart, particularly in patients suffering from sepsis and acute respiratory distress syndrome, and in heart-transplanted patients. CVP, right atrial pressure or right ventricular pressure have been demonstrated to be invalid for judging right ventricular function or right ventricular loading conditions. Hoffmann et al demonstrated no correlation between CVP and right ventricular end-diastolic volume (RVEDV) and they emphasized that the preload factor in the original Frank-Starling hypothesis had nothing to do with pressure but concerned volume. Measurement of right ventricular ejection fraction (RVEF) is based on the thermodilution technique. The ratio between the temperature change of two successive plateaus represents the fraction of blood remaining in the right ventricle (residual
fraction RF). The RVEF is calculated from the equation EF = 1-RF. Further development of computer techniques allow continuous monitoring of right ventricular hemodynamics. However, no large clinical trials are available showing a beneficial impact of RVEF monitoring on patient outcome.

MEASUREMENT OF EXTRAVASCULAR LUNG WATER AND INTRATHORACIC BLOOD VOLUME.

The most common method for evaluating lung water content is based on the double indicator dilution technique using indocyanine green as the non-diffusible indicator prepared in ice-cold dextrose (a diffusible indicator) s2. Intrathoracic blood volume appears to be a more reliable indicator of preload than the cardiac filling pressures.

MONITORING OF ORGAN PERFUSION AND MICROCIRCULATION

Inadequate tissue perfusion and oxygenation are likely to contribute to the development of organ failure and increased mortality in critically ill patients s3. The net balance between cellular oxygen supply and oxygen demand determines tissue oxygenation. Since continuing tissue hypoxia can persist despite the presence of an apparently adequate systemic blood flow, pressure, and arterial oxygen content, monitoring of specific indices of oxygenation at tissue level is essential.

OXYGEN DELIVERY AND OXYGEN CONSUMPTION

The total amount of oxygen delivered to the peripheral tissue per minute(DO2) can be calculated as DO2 = CO x CaO2 where CaO2 = Hb x 1.39 x SaO2.

Uptake of oxygen from the arterial blood (VO2) can be measured directly by the use of metabolic carts and the analysis from expired gas, or calculated CO and arterial and mixed venous blood samples. The ratio VO2/DO2 is the oxygen extraction ratio. This relationship can be used to assess the adequacy of tissue oxygenation s4.

MIXED VENOUS OXYGEN SATURATION (SVO)

Mixed venous oxygen saturation, or the saturation of the blood in the pulmonary artery, can be measured intermittently or continuously using PA catheter. A specially designed PA catheter can provide SvO2 reliably and continuously. Fiberoptic bundles incorporated into the PA catheter determine the oxygen saturation in the pulmonary artery blood based on the principles of reflectance oximetry.

A special computer connected to this PA catheter displays SvO2 continuously and allows standard cardiac output measurements. Blood in the pulmonary artery is normally unoxygenated, having not yet traveled through the lungs, with a saturation of 60 – 80%. SvO2 is clinically important as a function of supply and demand; how much oxygen is being extracted from the blood by the organs, before this blood is returned to the right heart. SvO2, therefore, serves us in evaluating the supply and demand of oxygen to the tissues 55.

It is influenced by oxygen delivery (hemoglobin, SaO2, CO) as well as oxygen consumption (VO2) and is expressed as SvO2 = SaO2 – [VO2 / (1.36 x Hb x CO)].

To the extent that arterial oxygen saturation, oxygen consumption and hemoglobin remain stable, mixed venous oxygen saturation may be used as an indirect indicator of cardiac output. When cardiac output falls, tissue oxygen extraction increases and the mixed venous blood will become more desaturated. As shown in the equation, an increase in VO2 and a decrease in Hb, CO and arterial oxygenation will result in a decrease in SvO2.

BLOOD LACTATE

A normal lactate level is a fairly specific indicator of adequate tissue perfusion. Lactate concentrations above 2mmol/l are generally considered a biochemical indicator of inadequate oxygenation s6. Circulatory failure with impaired tissue perfusion is the most common cause of lactic acidosis in intensive care patients. A number of mechanisms other than impaired tissue oxygenation may cause an increase in blood lactate, including activation in glycolysis, reduced pyruvate dehydrogenase activity, or liver failure. The presence of elevated lactate levels should prompt the intensivist to initiate diagnostic procedures for assessment of the circulatory status.

GASTROINTESTINAL TONOMETRY

The hypovolemic patient is at risk of experiencing splanchnic hypo perfusion with subsequent development of bacterial translocation and systemic inflammatory response syndrome s7. Abnormalities of splanchnic perfusion may coexist with normal systemic hemodynamic and metabolic parameters. Measurement of gastric intramucosal pH has emerged as an attractive option for diagnosis and monitoring of splanchnic hypo perfusion, and it appears to have more relevance for predicting postoperative complications s8.

Gastrointestinal tonometry measures gut mucosal PCO2, a clinically useful variable that is sensitive to alterations in splanchnic perfusion and oxygenation. Tonometer
measurements might provide an insight in a region that is among the first to develop an inadequacy of tissue oxygenation in circulatory shock and is the last to be restored by resuscitation. It has been shown that prolonged acidosis in the gastric mucosa might be a sensitive, but not specific, predictor of outcome in critically ill patients.

NEAR-INFRA RED SPECTROSCOPY

Near-infra red spectroscopy (NIRS) is a continuous non-invasive method applying the principles of light transmission and absorption to determine tissue oxygen saturation. Although NIRS may be applied to almost any organ, it has mainly been used in studies investigating cerebral or muscle oxygenation after different types of hypoxic injuries. The main limitation of NIRS in the clinical setting is the inability to make quantitative measurements because of the contamination of light by scatter and absorption.

TISSUE OXYGEN TENSION

Measuring tissue oxygen tension has become feasible for clinical use by the development of miniaturized implantable Clark electrodes. The polarographic oxygen sensors enable us to measure partial pressure of oxygen in tissues (pO₂), organs and body fluids directly and continuously. The pO₂ values correspond to oxygen availability on a cellular level and provide information about oxygen supply and utilization in specific tissue beds.

CONCLUSION

There are many monitoring instruments available and there have been some outstanding developments in the past. None of them emerges as more accurate than the others, although no formal comparisons have yet been attempted. Most of the techniques are still relatively invasive, requiring either sedation and mechanical ventilation as in esophageal Doppler and Fick / carbon dioxide methods, or arterial and central venous access for pulse contour techniques. Esophageal Doppler is operator dependent, training is required to obtain 'optimal' aortic velocity signals, and probe repositioning is mandatory if reliable results are to be obtained. The pulse contour methods also require frequent calibration, and the need for both arterial and central venous catheters preclude their routine use in the operating room. Unlike Doppler and pulse contour, the Fick / carbon dioxide method does not provide an instantaneous measure of cardiac output, but rather a mean value every 3 min. No visible, real-time signal allows the operator to make a critical judgment based on the cardiac output values obtained. These techniques do not exclude each other because their advantages and limitations are quite different. They also are not intended to replace the pulmonary artery catheter, which remains quite unique in providing pressures (right atrial, pulmonary artery and pulmonary 'wedged' pressures) as well as venous oxygen saturation, in addition to cardiac output. Recent technological advances have allowed the development of completely noninvasive cardiac output monitoring using impedance cardiology. This technique is ideal for continuous online and intermittent monitoring of stroke volume, heart rate and derived parameters such as stroke volume index, cardiac index, systemic vascular resistance, left cardiac work index and ejection fraction. Since impedance cardiology uses baseline impedance, Z, for determining cardiac output, large amount of thoracic fluid may interfere with the impedance signal making the hemodynamic data unreliable. No single method stands out or renders the others obsolete. By making cardiac output easily measurable, however, these techniques should all contribute to improvement in hemodynamic management.

References

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