Invasive and Non-invasive Monitoring Of Cardiac Output

Essay

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INTRODUCTION

Cardiac output (CO) is the quantity of blood pumped into the aorta each minute by the heart. This is also the quantity of blood that flows through the circulation. It is the product of stroke volume and heart rate. CO can thus be manipulated by alteration in heart rate or rhythm, preload, contractility and afterload. Moreover it gives important information about tissue perfusion and oxygen delivery ***(Guyton and Hall, 2010).***

Cardiac output monitoring is an important tool in high risk critically ill surgical patients in whom large fluid shifts are expected along with bleeding and hemodynamic instability. It is an important component of goal directed therapy (GDT), i.e., when a monitor is used in conjunction with administration of fluids and vasopressors to achieve set therapeutic endpoints there by improving patient care and outcome ***(Alhashemi et al., 2011).***

Cardiac output cannot be measured reliably by clinical examination and routine assessment. There are various methods which are broadly classified as follows: (1) Invasive pulmonary artery catheterization (2) Minimally invasive (3) Non-invasive ***(Mehta and Arora, 2014).***

Invasive COP monitoring by the Swan Ganz catheter has become an important tool for calculating cardiac output using Pulmonary artery thermodilution and dye dilution are valid and

reliable intermittent techniques which can be used in awake and anesthetized patients. However, its use has been associated with various complications ***(lee et al., 2011).***

The search for a new, less invasive method of measuring cardiac output has led to the introduction of many new devices into clinical practice. Minimally invasive cardiac output monitors have varying degrees of 'invasiveness' with some being totally non-invasive and others only marginally less invasive than a pulmonary artery catheter (PAC) ***(Drummond and Murphy, 2012).***

Several completely noninvasive technologies are available that allow for continuous cardiac output measurement such as Doppler-based methods and thoracic bioimpedance ***(Saugel et al., 2015).***

Derangements in the circulation are a common feature of sepsis, trauma, major surgery and other critical illnesses. Detailed evaluation of the circulation is therefore an essential aspect of the clinical management of such patients. The use of cardiac output monitoring technology is an increasingly important aspect of evaluating patients in the operating theatre and critical care unit. ***(Jhanji et al., 2008)***

AIM OF THE WORK

The aim of this study is to focus the light on conditions affecting cardiac output, the physiological principles used to measure cardiac output and their application in different techniques of cardiac output monitoring including invasive, minimally invasive and non-invasive methods.

Chapter I

PHYSIOLOGY AND CONDITIONS AFFECTING CARDIAC OUTPUT

Cardiac output (CO) is the quantity of blood pumped into the aorta each minute by the heart. This is also the quantity of blood that flows through the circulation. It is the product of stroke volume and heart rate. CO can thus be manipulated by alteration in heart rate or rhythm, preload, contractility and afterload. Moreover it gives important information about tissue perfusion and oxygen delivery ***(Guyton and Hall, 2010).***

For young, healthy men, resting cardiac output averages about 5.6 L/min. For women, this value is about 4.9 L/min. When one considers the factor of age as well—because with increasing age, body activity diminishes—the average cardiac output for the resting adult, in round numbers, is often stated to be almost exactly 5 L/min ***(Guyton and Hall, 2010).***

Cardiac output is frequently stated in terms of the cardiac index (CI), which is the cardiac output per square meter of body surface area. The normal human being weighing 70 kilograms has a body surface area of about 1.7 square meters, which means that the normal average cardiac index for adults is about 3 L/min/m2 of body surface area ***(Mehta and Arora, 2014).***

CARDIAC OUTPUT DETERMINANTS

PRELOAD

Preload is determined by myocardial fiber length before contraction. According to the Frank-Starling law, there is a direct relationship between the degree of fiber elongation in diastole and posterior shortening of the myocardial fiber in systole. This is because stretching of the fiber involves slight elongation of the sarcomere, thereby increasing the number of sites of interaction between actin and myosin. In the clinical setting we can define preload as the ventricular dimension in telediastole (end-diastole). Left ventricle pressure could be used as a measure of preload, provided the relationship between pressure and ventricular volume is constant ***(Mohrman and Heller, 2013)***

According to the Frank-Starling law, at a constant heart rate, cardiac output is directly proportional to preload, at least up to a certain point, beyond which CO would not increase despite further elevations in ventricle filling pressure, and could in fact decrease under certain conditions ***(Katz 2002).***

Two phases can be defined in the Frank-Starling curve: a first phase in which increasing preload is linearly correlated to increasing systolic volume (the preload-dependent phase or zone), and a second phase in which increasing preload is practically no longer correlated to increased cardiac output (the preload-independent phase or zone). In effect, upon stretching, the myocardial fibers show increased affinity for Ca2+ and

therefore increased contractility, though a point is eventually reached where the heart is unable to distend any further, due to anatomical and structural limitations ***(García et al., 2013)*** **(Figure 1).**

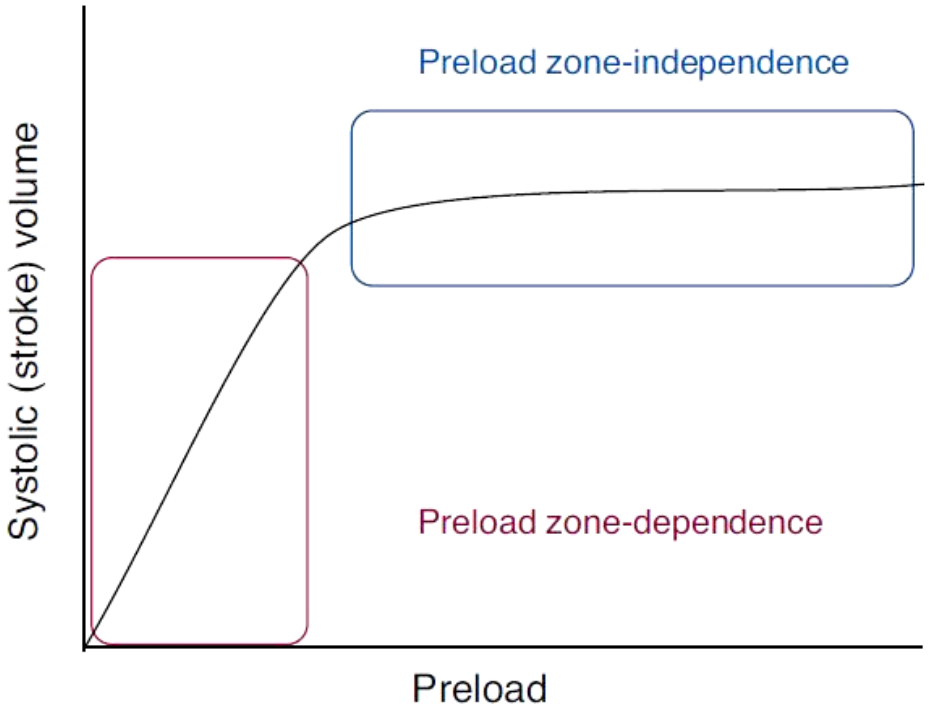

Figure (1): Frank-Starling curve ***(García et al., 2013)***

Myocardial over-distension, as seen for example in dilated cardiomyopathy, gives rise to structural alterations of the myocytes; this in turn results in a loss of contractility, and therefore a drop in cardiac output ***(García et al., 2013).***

Preload is therefore directly related to ventricle filling the main determinant of which is venous return to the heart ***(Mou et al., 2015).***

The relationship between venous return and cardiac function determines the values of venous pressure and cardiac output at each given point in time. An increase in venous return will give rise to an increase in cardiac output in a healthy heart, and the venous pressure values will remain within normal limits. In contrast, under conditions of heart failure, the heart is unable to assimilate all the blood that reaches it; increased right atrial pressure (central venous pressure, CVP) therefore results, with a drop in venous return due to the rise in pressure gradient between the venous system and the right atrium, as well as a reduction in cardiac output ***(Sequeira and van der Velden, 2015).***

Another important factor is that stretching the heart causes the heart to pump faster at an increased heart rate. That is, stretch of the sinus node in the wall of the right atrium has a direct effect on the rhythmicity of the node itself to increase heart rate as much as 10 to 15 per cent. In addition, the stretched right atrium initiates a nervous reflex called the Bainbridge reflex, passing first to the vasomotor center of the brain and then back to the heart by way of the sympathetic nerves and vagi, also to increase the heart rate ***(Crystal and Salem, 2012).***

AFTERLOAD

Afterload is a force that opposes myocardial contraction. It is equal to the tension across the ventricular wall during systole. The Law of Laplace describes the relationship between the determinants of ventricular wall tension, or afterload (Equation 1). In other words, tension (afterload) is directly proportional to ventricular pressure and size (radius) and indirectly related to wall thickness. Ventricular dilation increases afterload, and left ventricular hypertrophy (LVH) is a compensatory mechanism that attempts to decrease afterload ***(Vincent, 2014).***

$$T = (P \text{ r})/ 2h$$

Equation (1): LaPlace law where T is tension, P is chamber pressure, r is the radius of the chamber, and h is the wall thickness ***(Vincent, 2014)***

When the Laplace relationship is applied to the heart, the relevant pressure is the peak transmural pressure across the ventricle during systole, and the relevant radius is the end diastolic radius of the ventricular chamber ***(Marino, 2014).***

Components of Afterload

The forces that contribute to ventricular afterload can be identified by their relationship to the variables in the Laplace equation. This is demonstrated by the flow diagram in Figure 2. The component forces of ventricular afterload include end-

diastolic volume (preload), pleural pressure, vascular impedance, and peripheral vascular resistance ***(Marino, 2014).***

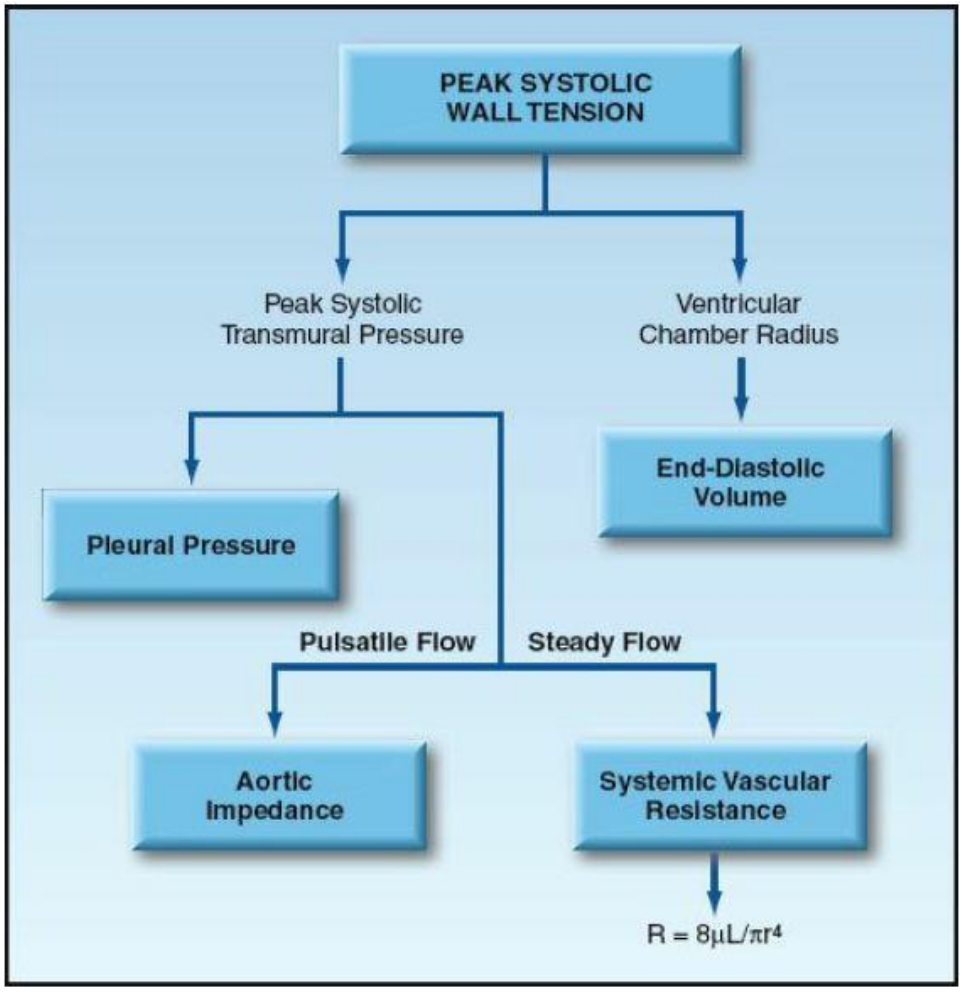

Figure (2): The forces that contribute to ventricular afterload ***(Marino, 2014)***

1. Pleural Pressure

Since afterload is a transmural wall tension, it will be influenced by the pleural pressure surrounding the heart ***(Feihl and Broccard, 2012).***

Negative pleural pressure surrounding the heart will impede ventricular emptying by opposing the inward movement of the ventricular wall during systole. This effect is responsible for the transient decrease in systolic blood pressure that occurs during the inspiratory phase of spontaneous breathing. When the inspiratory drop in systolic pressure is greater than 15 mm Hg, the condition is called "pulsus paradoxus" (which is a misnomer, since the response is not paradoxical, but is an exaggeration of the normal response) ***(Feihl and Broccard, 2012).***

Positive pressures surrounding the heart will promote ventricular emptying by facilitating the inward movement of the ventricular wall during systole ***(Boehmer and Popjes, 2006).***

2. Vascular Components

The stroke output of the left ventricle produces pulsatile pressure and flow patterns in the aorta and major arteries, but the phasic changes in pressure and flow are progressively dampened as the blood moves peripherally, and by the time the blood reaches the small peripheral arterioles, pressure and flow are steady and non-pulsatile. The force that opposes pulsatile

flow is known as impedance, and the force that opposes steady flow is resistance ***(Davidson and Giraud, 2012).***

Vascular impedance is the force that opposes the rate of change in pressure and flow, and it is expressed primarily in the large, proximal arteries, where pulsatile flow is predominant. Impedance in the ascending aorta is considered the principal afterload force for the left ventricle, and impedance in the main pulmonary arteries is considered the principal afterload force for the right ventricle. Vascular impedance is a dynamic force that changes frequently during a single cardiac cycle, and it is not easily measured in the clinical setting ***(Mou et al., 2015).***

Resistance in a vessel is described by the Poiseuille equation:

$$R = 8 \times \mu \times L / p\, r\, 4$$

Equation (2): the Hagen Poiseuille equation where R is resistance, μ represents the viscosity of blood, L is vessel length, and r is vessel radius ***(Vest and Heupler, 2013).***

Thus it can be seen that elevated viscosity, such as in polycythemia, will increase resistance to flow. Importantly, resistance is inversely proportional to the fourth power of the radius, enabling small changes in arteriolar diameter to have profound changes on total peripheral resistance ***(Vest and Heupler, 2013).***

MYOCARDIAL CONTRACTILITY

Myocardial contractility 'often referred to as inotropy" is the inherent capacity of the myocardium to contract independent of preload and afterload. Thus for a given preload and afterload, contractility is a manifestation of all other factors that influence the interactions between contractile proteins ***(Solaro, 2011).***

Accurate clinical measurement of contractility is difficult. The ejection fraction (EF), defined as the ratio of stroke volume to end-diastolic volume, is widely used clinically as a noninvasive index of cardiac contractility. It is measured noninvasively with echocardiography. Ejection fraction normally ranges from 55 to 80% at rest, with an EF of less than 55% suggesting depressed myocardial contractility ***(Dunn and Heupler, 2013).***

HEART RATE

The heart rate is controlled by chronotropic influences on the spontaneous electrical activity of SA nodal cells. Cardiac parasympathetic nerves have a negative chronotropic effect, and sympathetic nerves have a positive chronotropic effect on the SA node ***(Vincent, 2008).***

PATHOLOGICALLY HIGH AND PATHOLOGICALLY LOW CARDIAC OUTPUTS

In healthy human beings, the cardiac outputs are surprisingly constant from one person to another. However, multiple clinical abnormalities (Figure 3) can cause either high or low cardiac outputs ***(Guyton and Hall, 2010).***

- **High Cardiac Output Caused by Reduced Total Peripheral Resistance.**

1. Beriberi. This disease is caused by insufficient quantity of the vitamin thiamine (vitamin B1) in the diet. Lack of this vitamin causes diminished ability of the tissues to use some cellular nutrients, and the local tissue blood flow mechanisms in turn cause marked compensatory peripheral vasodilation ***(Dabar et al., 2015).***

Sometimes the total peripheral resistance decreases to as little as one-half normal. Consequently, the long-term levels of venous return and cardiac output also often increase to twice normal ***(Dabar et al., 2015).***

2. Arteriovenous fistula (shunt) occurs between a major artery and a major vein, tremendous amounts of blood flow directly from the artery into the vein. This, too, greatly decreases the total peripheral resistance and, likewise, increases

the venous return and cardiac output ***(Stern and Klemmer, 2011).***

3. Hyperthyroidism. In hyperthyroidism, the metabolism of most tissues of the body becomes greatly increased. Oxygen usage increases, and vasodilator products are released from the tissues.Therefore, the total peripheral resistance decreases markedly because of the local tissue blood flow control reactions throughout the body; consequently, the venous return and cardiac output often increase to 40 to 80 per cent above normal ***(Biondi, 2015)***.

4. Anemia. In anemia, two peripheral effects greatly decrease the total peripheral resistance. One of these is reduced viscosity of the blood, resulting from the decreased concentration of red blood cells. The other is diminished delivery of oxygen to the tissues, which causes local vasodilation. As a consequence, the cardiac output increases greatly ***(Anand et al., 2004).***

- **Low Cardiac Output**

These conditions fall into two categories:

(1) Those abnormalities that cause the pumping effectiveness of the heart to fall too low and

(2) Those that cause venous return to fall too low ***(Guyton and Hall, 2010).***