INTRODUCTION

One of the biggest challenges for anaesthetists today is the safe conduct of anaesthesia for patients who have pre-existing cardiac disease. The number of patients with cardiac disease presenting for anaesthesia is increasing. These patients present some of the greatest anaesthetic challenges because their cardiac lesions will still exist after the operation ***(Bill, 2009).***

The anaesthesiologist's responsibilities in the care of cardiac patients undergoing surgery extend well beyond the administration of anaesthesia. These responsibilities include knowledge of the pathophysiology of each lesion, the effects of anaesthetic drugs on the circulation, and resuscitation techniques. The anaesthetic care of cardiac patients undergoing surgery demands much more of the anaesthesiologist than the use of anaesthetic techniques and drugs. The anaesthesiologist's primary function under these circumstances often consists of the continuous application of critical intensive care measures throughout the period of anaesthesia and operation. The support of vital functions always takes priority over the administration of anaesthesia ***(Castillo et al., 2011).***

Perioperative adverse cardiac outcomes ensue as a result of pre-existing cardiac disease which is aggravated by the anaesthetic manipulations and the surgical stress. The goal is to optimize perioperatively this fragile group of patients based on the most recent guide lines for the management of patients with cardiac insufficiency **(*Toufektziana et al, 2007).***

The risk of developing cardiovascular complications during and after surgery depends on the nature of the surgery undertaken, and increases with the age of the patient. As many as 10% of all patients undergoing non-cardiac surgery have, or are at risk of having, cardiovascular disease ***(Arrowsmith & Mackay, 2002)***.

Anaesthesiologists have always dreamed of a "magic bullet", a drug or a technique able to protect the patients' heart in the perioperative period to improve the outcome without important side-effects ***(Landoni et al., 2007).***

There are several accepted anaesthetic techniques for cardiac patients undergoing non-cardiac surgery, but the choice of anaesthetic technique or group of drugs is less important than the manner in which they are applied to different clinical circumstances ***(Gothard , & Keogh , 2009).*** Pharmacological balancing remains the cornerstone for optimal results **(*Toufektziana et al, 2007).***

Diseases of the cardiovascular system are often associated with changes in CO, Valvular heart disease, Stenotic lesions, and tamponade lead to a low, fixed cardiac output, which tolerates poorly any changes in rhythm, tachycardia and decrease in preload and vascular resistance ***(Bill, 2009).***

Aortic valvular stenosis remains the most common debilitating valvular heart lesion ***(Billings et al., 2007),*** while mitral stenosis is one of the most common valvular lesions especially in the under developed countries. Mitral stenosis occurs most commonly as a result of rheumatic heart disease ***(Chohan et al., 2006).***

Cardiac tamponade is a cardiological emergency requiring prompt treatment in order to avoid a fatal outcome.

Anaesthetists have to be aware of its presentation, diagnosis and management ***(Spodick , 2003).***

AIM OF THE WORK

The aim of this work is to review the updates in anaesthetic management for patients with low fixed cardiac output states to provide safe and efficient conduct of anaesthesia without any adverse effect for these compromised groups.

Physiology OF Cardiac Output

Definition

Cardiac output 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. Cardiac output is perhaps the most important factor that we have to consider in relation to the circulation. Cardiac output is calculated via the following formula: *Cardiac Output* = Stroke Volume x Heart Rate ***(Brengelmann, 2003).***

Normal Values for Cardiac Output

Cardiac output varies widely with the level of activity of the body. The following factors, directly affect cardiac output:

1. Basic level of body metabolism.
2. Whether the person is exercising.
3. The person's age.
4. Size of the body.

For *young, healthy men*, resting cardiac output averages about 5.6 L/min. For *women*, this value is about 4.9 L/min. The average cardiac output for the resting adult, in round numbers, is often stated to be almost exactly 5 L/min ***(Gaasch and Zile, 2004).***

Cardiac Index

Cardiac output is frequently stated in terms of the *cardiac index*, 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 ***(Koch et al, 2000).***

Control of Cardiac Output

Many factors contribute to control cardiac output: venous return, peripheral resistance, tissue metabolism, heart rate, and contractility.

A. Venous Return

Various factors of the peripheral circulation affect flow of blood into the heart from the veins, called *venous return*, *Frank-Starling law of the heart* states that when increased quantities of blood flow into the heart the following takes place:

1. The increased blood stretches the walls of the heart chambers. As a result of the stretch, the cardiac muscle contracts with increased force, and this empties the extra blood that has entered from the systemic circulation. Therefore, the blood that flows into the heart is automatically pumped without delay.

2. Stretching the heart causes the heart to pump faster, That is, stretch of the *sinus node* in the wall of the right atrium increase heart rate as much as 10 to 15 per cent.

3. The stretched right atrium initiates ***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. *Under most normal*

unstressful conditions, the cardiac output is controlled almost entirely by peripheral factors that determine venous return.

(Rockman et al, 2000)

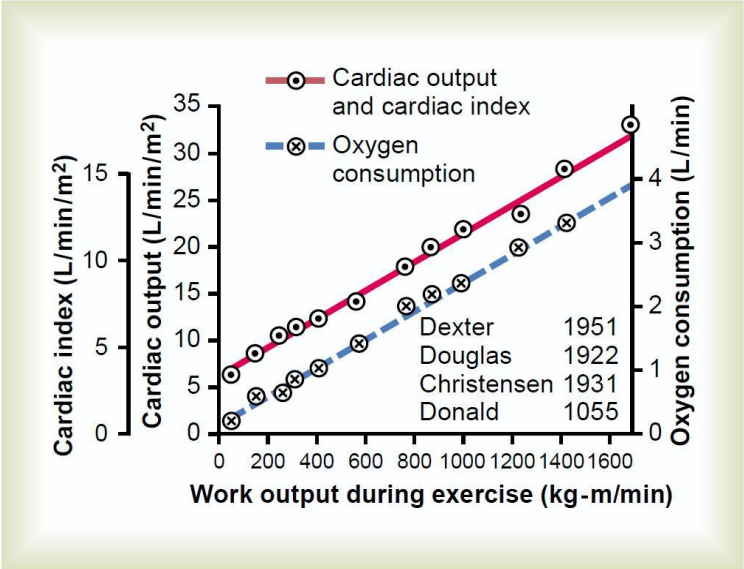

Fig.1: Effect of increasing levels of exercise to increase cardiac output ***(Guyton & Hall, 2006)***.

B. Tissue Metabolism

The venous return to the heart is the sum of all the local blood flows through all the individual tissue segments of the peripheral circulation. Therefore, it follows that cardiac output regulation is the sum of all the local blood flow regulations. In most tissues, blood flow increases mainly in proportion to each tissue's metabolism. For instance, local blood flow almost always increases when tissue oxygen consumption increases; this effect is demonstrated in Figure 1 for different levels of exercise. Note that at each increasing level of work output during exercise, the oxygen consumption and the cardiac output increase in parallel to each other ***(Guyton & Hall, 2006).***

C. Peripheral Resistance

Under most normal conditions, the long-term cardiac output level varies reciprocally with changes in total peripheral resistance:

- ✓ When the total peripheral resistance is exactly normal (at the 100 per cent mark in the figure 2), the cardiac output is also normal.
- ✓ When the total peripheral resistance increases above normal, the cardiac output falls.
- ✓ Conversely, when the total peripheral resistance decreases, the cardiac output increases.

(Uemura et al., 2004)

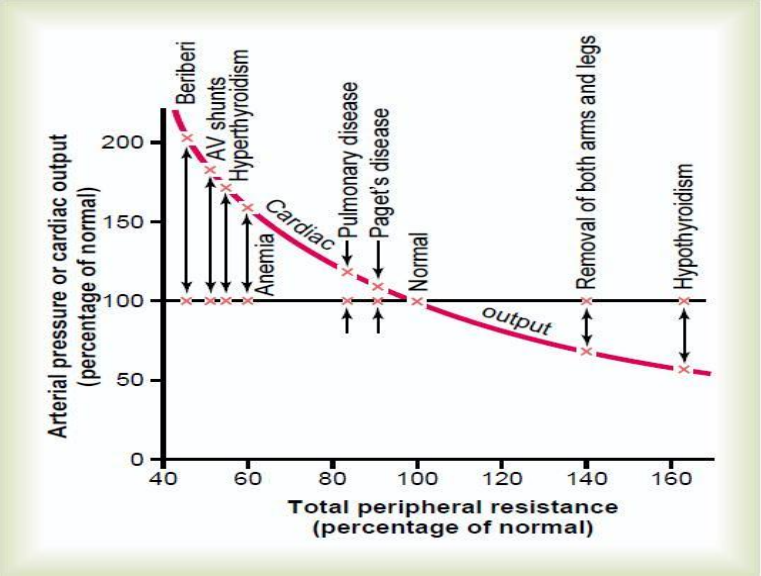

Fig. 2: Chronic effect of different levels of total peripheral resistance on cardiac output, showing a reciprocal relationship between total peripheral resistance and cardiac output ***(Guyton & Hall, 2006)***.

One can easily understand this by reconsidering one of the forms of Ohm's law, Cardiac Output: Arterial Pressure/Total Peripheral Resistance. Any time the long-term level of total peripheral resistance changes (but no other functions of the circulation change), the cardiac output changes quantitatively in exactly the opposite direction ***(Guyton & Hall, 2006).***

D. Heart rate

Defined as the number of beats per minute, and is heavily influenced by autonomic nervous system. Increases in heart rate increase cardiac output up until the point at which rapid heart rate does not permit for adequate ventricular filling during diastole, and cardiac output consequently falls ***(Uemura et al., 2004).***

E. Myocardial contractility

Defined as inherent inotropic ability of the heart, and influenced by the regulation of intracellular calcium concentration and ventricular compliance which determines the ability of ventricle to fill.

Effect of Age on Cardiac Output

Figure 3 shows the cardiac output, expressed as cardiac index, at different ages. Rising rapidly to a level greater than 4 L/min/m2 at age 10 years, the cardiac index declines to about 2.4 L/min/m2 at age 80 years. Cardiac output is regulated throughout life almost directly in proportion to the overall bodily metabolic activity. Therefore, the declining cardiac index is indicative of declining activity with age ***(Guyton & Hall, 2006).***

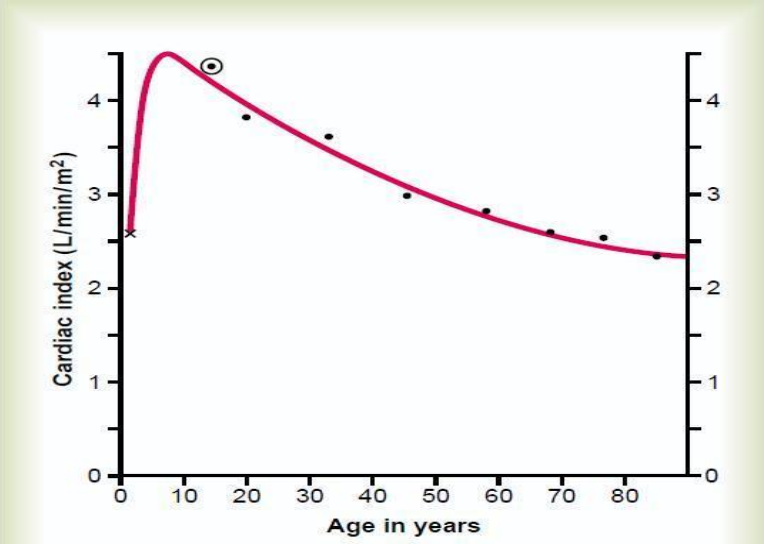

Fig.3: Cardiac index for the human being at different ages ***(Guyton & Hall, 2006)***.

Limits of cardiac output

There are definite limits to the amount of blood that the heart can pump, which can be expressed quantitatively in the form of *cardiac output curves*. Figure 4 demonstrates the *normal cardiac output curve*, showing the cardiac output per minute at each level of right atrial pressure. This is one type of *cardiac function curve*. The plateau level of this normal cardiac output curve is about 13 L/min, 2.5 times the normal cardiac output of about 5 L/min. This means that the normal human heart, functioning without any special stimulation, can pump an amount of venous return up to about 2.5 times the normal venous return before the heart becomes a limiting factor in the control of cardiac output. Shown in Figure 4 are several other cardiac output curves for hearts that are not pumping normally. The uppermost curves are for *hypereffective hearts* that are pumping better than normal. The lowermost curves are for

hypoeffective hearts that are pumping at levels below normal ***(Guyton & Hall, 2006).***

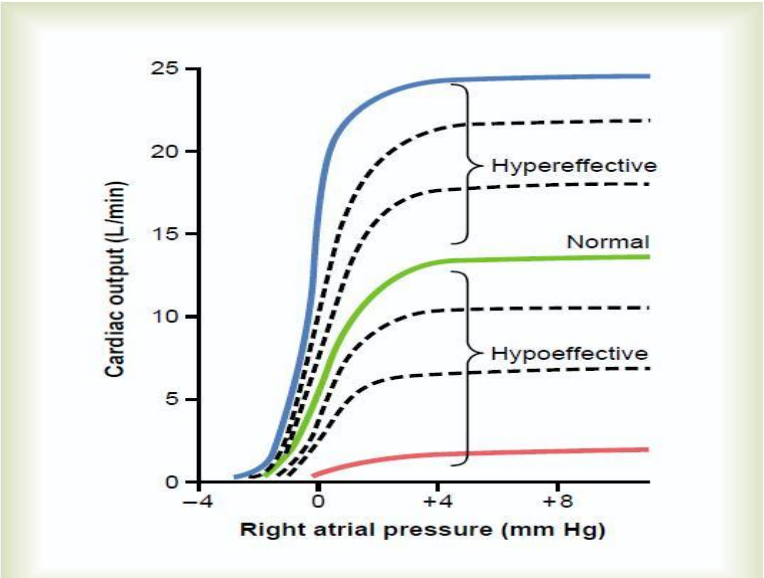

Fig. 4: Cardiac output curves for the normal heart and for hypoeffective and hypereffective hearts ***(Guyton & Hall, 2006).***

Factors increase cardiac output

Only two types of factors usually can make the heart a better pump than normal.

A. *Nervous stimulation.*

B. *Hypertrophy of the heart muscle.*

A. *Effect of Nervous Excitation on cardiac output*

Combination of **sympathetic** ***stimulation*** *and* ***parasympathetic inhibition*** does two things to increase the pumping effectiveness of the heart:

1. It greatly increases the heart rate sometimes, in young people, from the normal level of 72 beats/min up to 180 to 200 beats/min.

2. It increases the strength of heart contraction (which is called increased "contractility") to twice its normal strength. Combining these two effects, maximal nervous excitation of the heart can raise the plateau level of the cardiac output curve to almost twice the plateau of the normal curve, as shown by the 25-liter level of the uppermost curve in figure ***(Gaasch and Zile, 2004).***

B. Increased Pumping Effectiveness Caused by Heart Hypertrophy

A long-term increased workload, but not so much excess load that it damages the heart, causes the heart muscle to increase in mass and contractile strength in the same way that heavy exercise causes skeletal muscles to hypertrophy. For instance, it is common for the hearts of marathon runners to be increased in mass by 50 to 75 per cent. This increases the plateau level of the cardiac output curve, sometimes 60 to 100 per cent, and therefore allows the heart to pump much greater than usual amounts of cardiac output. *When one combines nervous excitation of the heart and hypertrophy, as occurs in marathon runners, the total effect can allow the heart to pump as much 30 to 40 L/min, about 22 times normal*; this increased level of pumping is one of the most important factors indetermining the runner's running time ***(Guyton & Hall, 2006)***.

Factors decrease cardiac output

Any factor that decreases the heart's ability to pump blood causes hypoeffectivity. Some of the factors that can do this are the following:

a. Coronary artery blockage, causing a "heart attack".

b. Inhibition of nervous excitation of the heart.

c. Pathological factors that cause abnormal heart rhythm or rate of heartbeat.

d. Valvular heart disease.

e. Increased arterial pressure against which the heart must pump, such as in hypertension.

f. Congenital heart disease.

g. Myocarditis.

h. Cardiac hypoxia.

(Uemura et al., 2004)

Effect of the Nervous System to Increase the Arterial PressureDuring Exercise.

During exercise, intense increase in metabolism in active skeletal muscles acts directly on the muscle arterioles to relax them and to allow adequate oxygen and other nutrients needed to sustain muscle contraction. This greatly decreases the peripheral resistance, which normally would decrease the arterial pressure also. However, the nervous system immediately compensates. The brain activity sends simultaneous signals into the autonomic nervous centers of the brain to excite circulatory activity, causing :

a. Large vein constriction.

b. Increased heart rate.

c. Increased contractility of the heart.

All these changes increase the arterial pressure above normal, which in turn forces more blood flow through the active muscles. In summary, when local tissue vessels dilate and thereby increase venous return and cardiac output above normal, the nervous system plays an exceedingly important role in preventing the arterial pressure from falling to disastrously low levels. In fact, during exercise, the nervous system goes even further, providing additional signals to raise the arterial pressure even above normal, which serves to increase the cardiac output an extra 30 to 100 % ***(Brengelmann, 2003).***

Methods for Measuring Cardiac Output

Cardiac output can be measured either by direct (invasive) or indirect (non-invasive) methods.

A. Direct (invasive) methods include:

1. Oxygen Fick Principle

The Fick principle involves calculating the oxygen consumed over a given period of time from measurement of the oxygen concentration of the venous blood and the arterial blood. Cardiac output can be calculated from these measurements:

- VO2 consumption per minute using a spirometer (with the subject re-breathing air) and a CO2 absorber
- the oxygen content of blood taken from the pulmonary artery (representing mixed venous blood)

- the oxygen content of blood from a cannula in a peripheral artery (representing arterial blood)

From these values, we know that:

VO2 = (Q×CA) - (Q×CV) where

- CA = Oxygen content of arterial blood
- CV = Oxygen content of venous blood.

This allows us to say

Q = (VO2/(CA – CV)

(Guyton & Hall , 2006)

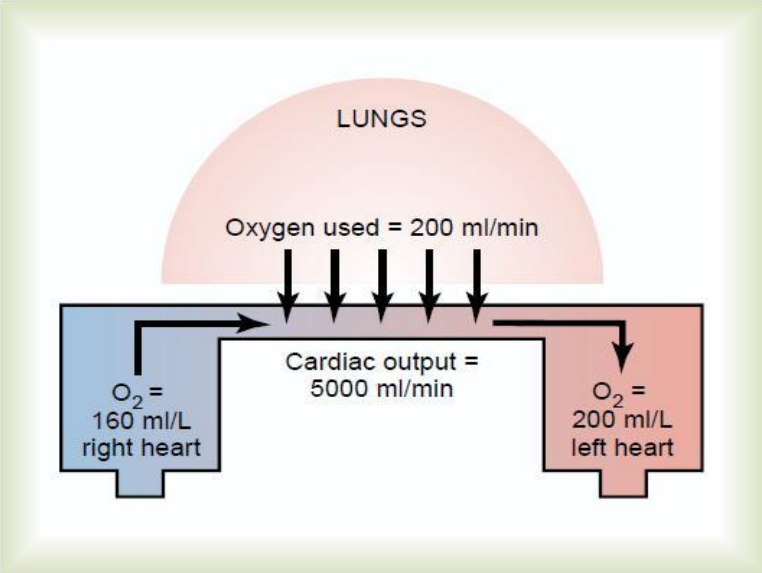

Fig. 5: Fick principle for determining cardiac output ***(Guyton & Hall, 2006).***