

**NON-INVASIVE DETERMINATION OF CARDIAC OUTPUT BY
DOPPLER ECHOCARDIOGRAPHY AND ELECTRICAL
BIOIMPEDANCE IN PATIENTS WITH ACUTE
MYOCARDIAL INFARCTION**

THESIS

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Presented BY

OSAMA IBRAHIM SALAH

M.B.B.Ch.

Supervised By

PROF. DR. RAMEZ RAOUF GUINDY

Professor of Cardiology

Ain Shams University

Dr. MOHAMED TAREK ZAKI

Lecturer of Cardiology

Ain Shams University

Dr. MAIY HAMDY EL-SAYED

Lecturer of Cardiology

Ain Shams University

Dr. ASSEM MOHAMED FATHY

Lecturer of Cardiology

Ain Shams University

1992

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INTRODUCTION
AND
AIM OF THE WORK

INTRODUCTION

Knowledge of the cardiac output is valuable in the management of patients with acute myocardial infarction because it identifies patients at greatly increased risk and also provides a rapid means of monitoring the response to treatment (George and Winter, 1985).

Although the thermodilution method is considered a gold standard for cardiac output measurement, it has many disadvantages. Most importantly, it is highly invasive and carries potential risk. Also, it needs experience, it is expensive, and data acquisition is only intermittent (Bernstein, 1989).

An accurate non-invasive method of measuring cardiac output would therefore have many attractions : two of the most promising techniques are Doppler echocardiography and thoracic electrical bioimpedance (Northridge et al, 1990).

Doppler echocardiography is time consuming, technically demanding, and also data acquisition is intermittent (Mark et al, 1986).

Thoracic electrical bioimpedance is highly reproducible, simple to use, and give instant results. It has the additional advantage of allowing continuous monitoring of the cardiac output which can be helpful in as-

sessing the response to treatment (Northridge et al, 1990).

AIM OF THE WORK

The aim of this study to compare cardiac output determined by Doppler echocardiography with that obtained by thoracic electrical bioimpedance in patients with acute myocardial infarction, and to assess the feasibility and ease of use of both non-invasive techniques inside the coronary care unit.

REVIEW
OF
LITERATURE

CHAPTER I

PHYSIOLOGY OF CARDIAC OUTPUT

Definition

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 and is responsible for transporting substances to and from the tissues (Guyton, 1991).

Normal Values

CO varies widely with the level of activity of the body. Therefore, such factors as the level of body metabolism, whether the person is exercising, age, and size of the body as well as a number of other factors can influence the CO.

For normal young healthy adult men, in whom the greatest number of CO measurements have been made, the resting CO averages about 5.6 liters/min. For women, this value is 10 to 20% less. When one considers the factor of age as well - since with increasing age body activity diminishes - the average CO for the adult, in round numbers, is often stated to be almost exactly 5 liters / min.

Cardiac Index

Because the CO changes markedly with body size, it has been important to find some means by which the cardiac outputs of different sized persons can be compared with each other. Experiments have shown that the cardiac output increases approximately in proportion to the surface area of the body. Therefore, it is frequently stated in terms of the cardiac index, which is the CO per square meter of body surface area. The normal human weighing 70 kilograms has a body surface area of approximately 1.7 square meters, which means that the normal average cardiac index for adults is approximately 3 liters /min per square meter of body surface area.

Effect of Age on CO

Rising rapidly to a level greater than 4 liters per minute per square meter at 10 years of age, the cardiac index declines to about 2.4 liters / min at the age of 80 years.

The CO 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.

Regulation of Cardiac Output

There are two primary factors concerned with CO regulation:

- 1- The pumping ability of the heart.
- 2- The venous return.

There are 3 principal factors that affect venous return to the heart from the systemic circulation, these are:

- (A) The right atrial pressure
- (B) The mean systemic filling pressure.
- (C) The resistance to blood flow between the peripheral vessels and the right atrium .

The Pumping Ability of the Heart

There are a number of factors that lead to hypereffective hearts [i.e. pumping better than normal], and other factors that cause a hypoeffective heart [i.e. pumping at levels below normal].

Factors that cause a hypereffective heart

- 1- Nervous stimulation.

2- Hypertrophy of the heart muscle.

Nervous Stimulation

A combination of sympathetic stimulation and parasympathetic inhibition will do two things to increase the pumping effectiveness of the heart :

- (1) Greatly increase the heart rate - sometimes to as much as 180 to 200 beats per minute .
- (2) Increase the strength of heart contraction, which is called increased contractility, to as much as two times the normal strength. Combining these two effects, maximal nervous excitation of the heart can raise the cardiac output to as much as 25 liters / min.

Hypertrophy of the Heart Muscle

A heart that is subjected to increased workload, but not so much excess load that it damages the heart, will cause 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 as much as 50 to 75 percent. This allows the heart to pump much greater than usual

amounts of cardiac output.

When one combines both nervous excitation of the heart plus hypertrophy, as occurs in marathon runners, the combined effect can allow the heart to pump as much as 30 to 35 liters/min. (Guyton, 1991).

Factors that cause a hypoeffective heart

Any factor that decreases the ability of the heart to pump blood will cause hypoeffectivity. Some of the numerous factors are:

- Inhibition of nervous excitation of the heart.
- Pathological factors that cause abnormal rhythm or rate of heart beat.
- Valvular heart disease.
- Increased arterial pressure against which the heart must pump, such as in hypertension.
- Congenital heart disease.
- Myocarditis.
- Cardiac anoxia.

- Diphtheritic or other types of myocardial damage or toxicity.

The venous return and right atrial pressure

As the right atrial pressure increases, it causes back pressure on the systemic circulation and thereby decreases venous return of blood to the heart.

If all circulatory reflexes are prevented from acting, venous return decreases to zero when the right atrial pressure rises to about 7 mmHg. Such a slight rise in right atrial pressure causes a drastic decrease in venous return because the systemic circulation is a very distensible bag, so that any increase in back pressure causes blood to dam up in this bag instead of returning to the heart. When the right atrial pressure falls below zero - that is, below atmospheric pressure - venous return does not increase significantly. By the time the right atrial pressure has fallen to about - 2 mmHg, the venous return will have reached a plateau, then it remains at this plateau level even though the right atrial pressure falls as low as -20 to -50 mmHg. This plateau is caused by collapse of the veins entering the chest. Low right atrial pressure sucks the walls of the veins together where they enter the chest, which prevents the negative pressure from sucking blood through the veins. Instead, the pressure in the veins immediately outside the chest remains almost exactly equal to

atmospheric pressure (zero pressure) because that is the pressure on the outsides of the very flaccid veins causing them to collapse.

Therefore, the venous pressure where the large veins empty into the chest never falls below a value of 0 mmHg despite the fact that the right atrial pressure may fall to very negative values. Consequently, even very negative pressures in the right atrium cannot increase venous return significantly above that which exists at a normal atrial pressure of 0 mmHg.

The mean circulatory filling pressure, the mean systemic filling pressure, and their effect on venous return

When the heart pumping is stopped by causing ventricular fibrillation or in any other way, the flow of blood everywhere in the circulation ceases a few seconds later. Without blood flow, the pressures everywhere in the circulation become exactly equal after about a minute. This equilibrated pressure level is called the mean circulatory filling pressure.

The mean systemic filling pressure is slightly different from the mean circulatory filling pressure. It is the pressure measured everywhere in the systemic circulation after blood flow has been stopped by clamping the large blood vessels at the heart, so that the pressures in the systemic