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Management of Low Cardiac Output Post Cardiac Surgery

Essay

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in General Intensive Care

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بسم الله الرحمن الرحيم

وقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ عَمَلَكُمْ
ورَسُولُهُ والْمُؤْمِنُونَ

صدق الله العظيم

سورة التوبة آية (105)

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Ahmed Mahmoud Salah

List of Contents

List of Abbreviations

$^{\cdot}O_2^-$	Superoxide anion
$^{\cdot}OH$	Hydroxyl radical
AHF	Acute heart failure
ARDS	Adult respiratory distress syndrome
ATP	Adenosine Tri Phosphate
A-V O_2	Arterial-venous oxygen gradient
CABG	Coronary artery bypass graft
$CaCO_2$	Arterial CO_2 concentration
CHF	Congestive heart failure
CO	Cardiac output
CO_2	Carbon dioxide
CPB	Cardiopulmonary bypass
$CvCO_2$	Venous CO_2 concentration
CVP	Central venous pressure
H_2O_2	Hydrogen peroxide
IABP	Intraaortic Balloon Pump
IL	Interleukins
LBBB	Left bundle-branch block

LCOS	Low cardiac output syndrome
LiCl	Lithium chloride
NHE	Sodium ion-hydrogen ion exchange
NO	Nitric oxide
$ONOO^-$	Peroxynitrite
PA	Pulmonary artery
PCWP	Pulmonary capillary wedge pressure
PDEI	Phosphodiesterase inhibitor
PEEP	Positive end-expiratory pressure
PPV	Pulse pressure variation
RBBB	Right bundle-branch block
ROS	Reactive oxygen species
SIRS	Systemic inflammatory response syndrome
SR	Sarcoplasmic reticulum
SvO_2	Mixed venous oxygen saturation
TNF	Tissue necrosis factor
VAD	Ventricular Assist Devices
VCO_2	CO_2 consumption
XO	Xanthine oxidase

List of Figures

List of Tables

INTRODUCTION

Low cardiac output syndrome (LCOS) can be considered a form of acute heart failure (AHF). In the same way that AHF produces high mortality in non-surgical patients, Low cardiac output syndrome is a major cause of postoperative death in patients undergoing cardiac surgery. Low cardiac output syndrome is, however, a peculiar form of AHF as it differs from the latter in aetiology, prognosis, and treatment, all of which are influenced by the combination of surgery and anaesthesia ***(Álvarez, 2008)***.

Post cardio pulmonary bypass (CPB) low cardiac output syndrome defined as a cardiac index of <2.2 L/min/m2 plus a pulmonary capillary wedge pressure of >15 mmHg during the first 6 hours after aortic declamping, despite adequate control of heart rhythm and in the absence of myocardial ischaemia, valve dysfunction or cardiac tamponade ***(Malliotakis et al., 2007).***

Causes of Low cardiac output syndrome after cardiac surgery are multifactorial, including myocardial ischemia during aortic cross clamping, the effects of cardioplegia, activation of the inflammatory and complement cascades, and alterations in systemic and pulmonary vascular activity. Residual cardiac lesions, even when minor, may also adversely impact the postoperative course ***(Hoffman et al., 2003).***

Patients who have undergone cardiac surgery involving extracorporeal circulation with global myocardial ischemia induced by aortic clamping show different degrees of transitory ventricular dysfunction without myocardial infarction in the immediate postoperative period. This dysfunction can cause postoperative low cardiac output syndrome with a prevalence of about 10%. The mortality rate among those who develop this complication is 17%. Treatment includes the administration of positive inotropic drugs and vasodilators, balloon counter-pulsation, and the use of mechanical devices that assist circulation ***(Malliotakis et al., 2007).***

AIM OF THE WORK

To discuss the aetiology and management of low cardiac output post cardiac surgery.

Chapter (1):

PHYSIOLOGY OF CARDIAC OUTPUT

Cardiac output is the volume of blood pumped by the heart per minute (mL blood/min). Cardiac output is a function of heart rate and stroke volume. The **heart rate** is simply the number of heart beats per minute. The **stroke volume** is the volume of blood, in milliliters (mL), pumped out of the heart with each beat. Increasing either heart rate or stroke volume increases cardiac output**.**

Cardiac Output in mL/min = heart rate (beats/min) X stroke volume (mL/beat). An average person has a resting heart rate of 70 beats/minute and a resting stroke volume of 70 mL/beat, the cardiac output for this person at rest Cardiac Output = 70 (beats/min) X 70 (mL/beat) = 4900 mL/minute, the total volume of blood in the circulatory system of an average person is about 5 liters (5000 mL). According to our calculations, the entire volume of blood within the circulatory sytem is pumped by the heart each minute (at rest). During vigorous exercise, the cardiac output can increase up to 7 fold (35 liters/minute)**.**

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 ***(Richard, 2011).***

Control of cardiac output

Four factors control cardiac output: heart rate, myocardial contractility, preload, and afterload. Heart rate and myocardial contractility are strictly cardiac factors, although they are controlled by various neural and humoral mechanisms. Preload and afterload are factors that are mutually dependent on function of the heart and the vasculature and are important determinants of cardiac output. Preload and afterload are themselves determined by cardiac output and by certain vascular characteristics. Preload and afterload will be called coupling factors because they constitute a functional coupling between the heart and blood vessels.

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 ***(Berne and levy, 2008)***.

Preload

Preload is the degree of myocardial distension prior to shortening. As initially demonstrated by Otto Frank and Ernest Starling, an intrinsic property of myocardial cells is that the force of their contraction depends on the length to which they are stretched: the greater the stretch (within certain limits), the greater the force of contraction. An increase in the distension of the ventricle will therefore result in an increase in the force of contraction, which will increase cardiac output. When heart muscle is stretched, just like skeletal muscle, the increase in length can lead to an increase in tension up to a limit, after which the tension falls. In an individual at rest, heart muscle cells are usually shorter than their optimal length so if they are stretched this can increase the force they can generate. **Starling's law** of the heart states that the energy released during contraction depends on the initial fibre length. The amount of stretch of the heart muscle depends on the **EDV**. This can be affected by two factors:

- The inherent compliance of the muscle. As we age, the heart muscle becomes stiffer and less compliant and therefore it becomes more difficult for the heart to stretch.

- The **end-diastolic pressure** (**EDP**) or **preload**. This is the filling pressure of the heart and is determined by the pressure in the great veins, the **central venous pressure** (**CVP**).

One of the major consequences of Starling's Law is that the output of the left ventricle matches the output of the right ventricle. For example, if there is an increase in the stroke volume of the left side of the heart, this increases the filling pressure of the right side, which increases the right EDV and hence increases the right stroke volume ***(Richard, 2011).***

Afterload

Afterload is the force against which the ventricles must act in order to eject blood, and is largely dependent on the arterial blood pressure and vascular tone. If the pressure in the aorta is increased, the flow of blood out of the heart is reduced. the long-term cardiac output level varies reciprocally with changes in total peripheral resistance. that when the total peripheral resistance is exactly normal (at the 100 per cent mark in the figure 1) the cardiac output is also normal. Then, when the total peripheral resistance increases above normal, the cardiac output falls; conversely, when the total peripheral resistance decreases, the cardiac output increases ***(Guyton & Hall, 2011)***.