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بالرسالة صفحات

لم ترد بالأصل

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HAEMODYNAMIC EFFECTS OF DOPEXAMINE HYDROCHLORIDE IN COMPARISON WITH DOPAMINE IN LOW CARDIAC OUTPUT SYNDROME DURING OPEN HEART SURGERY

Thesis submitted for partial fulfilment of M.D Degree in Anaesthesiology

By

Essam Adli Nashed

M.B. B.Ch, M.Sc.

Supervised By

Prof. Dr. Fathy Said Nasr

Professor and Head of Anaesthesiology Department
Faculty of Medicine - El-Azhar University

Prof. Dr. Omar Abd Al-Aleim

Professor and Head of Anaesthesiology Department
Faculty of Medicine - El-Menofia University

Prof. Dr. Sherif Abd El-Hady

Professor of Cardio-Thoracic Surgery
National Heart Institute

Prof. Dr. MamdouH Lotfy

Assistant Professor of Anaesthesiology
Faculty of Medicine - Menofia University

1999

DEDICATIONS

TO

My Parents,

My Wife

and to my lovely daughters

Mary

Klara

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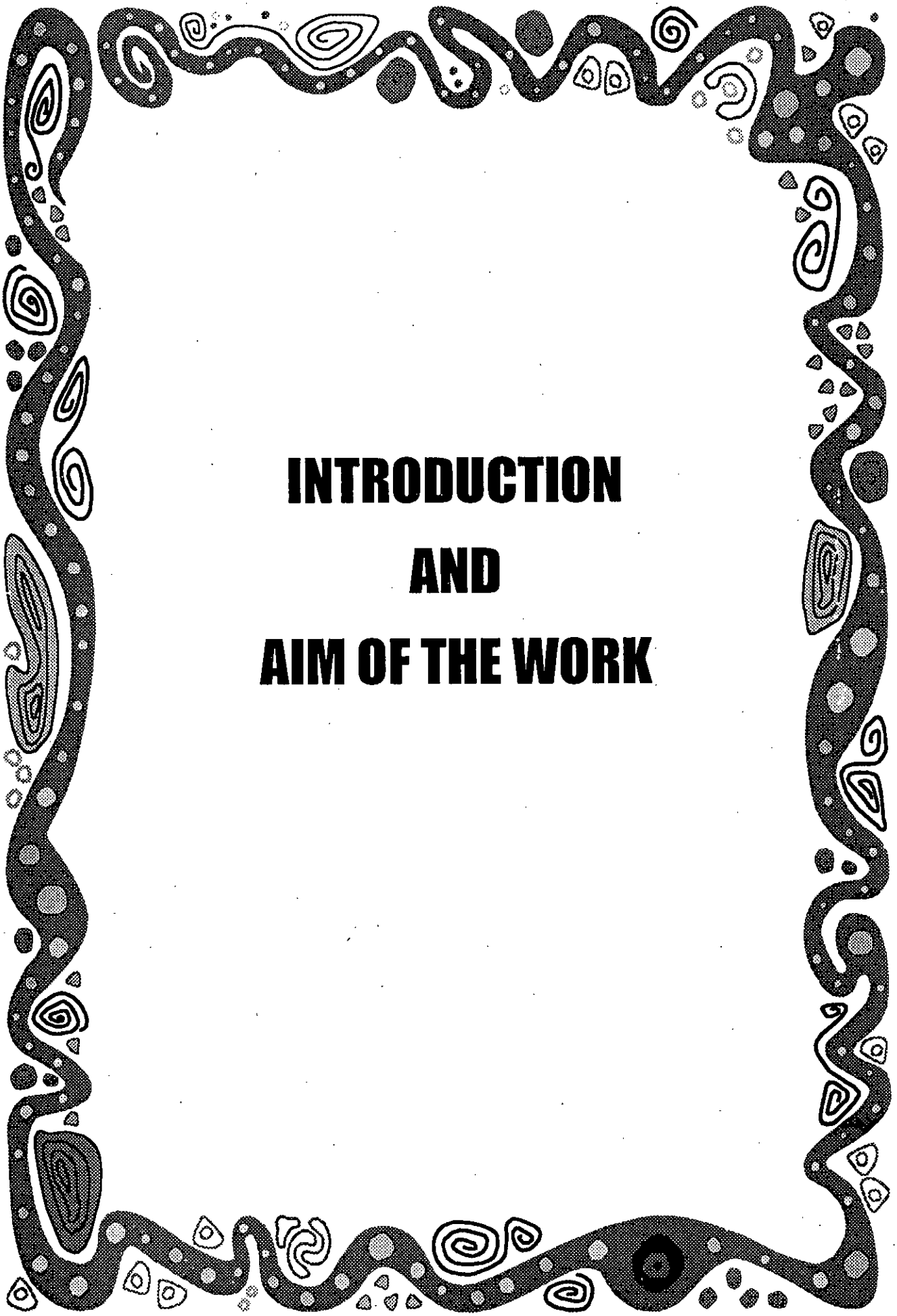
CONTENTS



INTRODUCTION
AND
AIM OF THE WORK

INTRODUCTION

Despite Advances in Cardiac surgery and perioperative care, the prevention and treatment of post operative complications continue to be an integral part of the care of the cardiac surgery patient. As older and sicker adult patients undergo surgery with greater frequency the incidence of complications has increased *(Naunheim, 1988).*

During recent years there has been a distinct decrease in the perioperative mortality associated with open heart surgery. This may be caused by a more effective perioperative drug treatment and better anaesthetic managment. Improved surgical techniques and early preventive medical care may also have contributed to the reduction in mortality *(Gold man, 1995).*

Low cardiac output is defined as that pathophysiological state in which the cardiac output is not sufficient to maintain blood flow to meet the metabolic needs of the body *(Kaplan, 1993).*

Low cardiac output occurs during the early postoperative period in approximatetly 20% of patients who undergo cardiac surgery. The incidence of this complication is dependent on the type and severity of the cardiac lesion undergoing repair, preoperative ventricular function, adequacy of myocardial preservation during the procedure and the adequacy of the surgical repair *(Gus. J. Vlahakes et al., 1994).*

Although intraoperative technical mishaps or inadequate myocardial protection during surgery can cause damage to a previously normal venetricle. The most important factor determining

the incidence of postoperative low output is preoperative ventricular function. Thus preoperative evaluation of ventricular function is important. Carful review of the patient's cardiac catheterization study both the hemodynamic measurment (Cardiac output, left ventricular end diastolic pressure and pulmonary artery pressure) and the left ventriculogram provides a good indication of the presence or absence of preoperative ventricular dysfunction. Other studies such as radionucleide ventriculography multigated angiocardiography or MUGA and echocardiography also provide valuable information about systolic ventricular function. Normal systolic function is associated with an ejection fraction of 60 - 70%. Moderate left ventricular dysfunction is present when the ejection fraction is 35-50% and severe impairment of contractility is associated with an ejection fraction belwo 35%. It must be remembered that the ejection fraction is dependant on pre-load and after load. Therefore it's meaning must be assessed in the cortext of the patient's history and other data. The ejection fraction furthermore reflects only the systolic (ejection) function of the heart. Diastolic dysfunction, reflected as abnormalities in ventricular relaxation and filling, also can result in heart failure and even pulmonary oedema despite the presence of a normal ejection fraction. Patients with ventricular hypertrophy are particulary susecptible to diastolic dysfunction *(Apsteim et al., 1988).*

When low cardiac output is present early after cardiac surgery, managment should follow a logical order of analysis and treatment. First consideration should be given to the possibility of reversible mechanical factors causing the poor cardiac performance. This is especially true for the patient who had good preoperative ventricular

function and who was weaned from cardiopulmonary bypass without difficulty. Left and right heart filling pressure must be determined to rule out hypovolaemia which is the most common cause of low cardiac output. Filling pressure should be compared with those found optimal in the operating room and before surgery. Cardiac tamponade must be rulled out. Considerations should be given to the possibility of technical problems related to the operation *(Doty 1990).*

The treatment of low cardiac output involves consideration of the heart rhythm, rate, status of the ventricular filling pressure (Pre-load), myocardial contractility and peripheral and pulmonary vascular resistance (after - load). Early consideration shoud be given to the patient's cardiac rhythm and rate. Cardiac output decreases with arrhythmias such as heart block, junctional rhyhm, atrial fibrillation and sinus bradycardia *(Hartzler 1977).*

Ventricular function is impaired by hypothermia. Although rewarming of the patient to 36°C or greater is completed befor weaning from cardiopulmonary bypass, this rewarming may be incomplete and further cooling may occur during closure of the incision. Thus the patient frequently becomes cold early and this can contribute significantly to ventricular dysfuntion and haemodynamic deteroriation *(Boldt 1990).*

Optimal cardiac output requires adequate return of blood to the heart (Pre-load) for left ventricular filling. Left ventricular Pre-load may be monitored by measurment of the pulmonary artery diastolic pressure, pulmonary capillary wedge pressure and if a catheter was palced intra operatively, left atrial pressure. Although the normal

heart functions well with a pulmonary wedge pressure of 6-12 mm Hg, failing heart require higher filling pressure because of poor complicane of the left ventricle.

Drug therapy for low cardiac output requires assesment of the hemodynamuc situation based on physical examination and invasive measurments.

* *Inotropic drug treament of low cardiac output.*

Various drugs optimize cardiac output through their effects on myocardial contractility (inotropic effect) or effects on the peripheral resistance (After load effect) or both. Positive inotropic agents act to increase the force and speed of myocardial fiber contraction, and in general they also increase myocardial oxygen consumption. Considerable controversy exists regarding which drug or combination of drugs should be used to treat low cardiac output. Although much experimental and clinical data are avaible, no one drug is best for all pateints with low cardiac output. Rational selection of drugs to treat low cardiac output requires a basic understanding of the agents available, carful analysis of the hemodynamic abnormalities present, tailoring of the therapy to fit the individual hemodynamic setting and realization that the patient's state is daynamic and thus alteration in the type of drug support adminstered may be required with the passage of time *(Disesa 1987).*

The sympathetic amines (dopamine, dobutamine, epinephrine, nor-epinephrime and isoproternol) improve cardiac output by a direct beta-adrenergic inotropic effect and have selectively different effects on the pripheral vasculature. Their effects are mediated

through the adrenergic receptors. Beta-1 reccptors exist predominantly in the myocardium. Their activation results in increased speed and force of myocardial contraction, increased rate of automaticity of the sino atrial node, and faster atrioventricular conduction. The beta-2 receptors are present predominantly in the smooth muscles of blood vessels and Bronchi, their activation results in vasodilation and bronchiodilation. Alpha-adrenegic recpetors exist primarily in the peripheral and pulmonary vasculature. Alpha-receptor stimulation causes vasoconstriction *(Gus J. Vlahakes et al. 1994).*

Dopexamine hydrochloride is a newly developed synthetic catecholamine structurally related to dopamine. It has affinity for both dopaminergic (DA-1) and Beta adrenergic (principally B2) receptors. Reports indicates that dopexamine had a 9.8 fold greater affinity for B2 adrenoreceptors than for B1 adrenoreceptors *(Robertal Hines 1996).*

Dopexamine like dopamine can only be given intravenously. It has been shown to activate post. Junctional dopamine receptors equipotently with dopamine and possesses substantial B2 activity, has no B1 activity and unlike dopamine has no activity at α-recptors. However at higher doses it has been reported to inhibit the neuronal uptake of noradrenaline and therefore mimics the action of dopamine at these receptors *(AG Ramage et al., 1996).*

The actions of dopexamine have been studied in patients with congestive cardiac failure in whom it induces beneficial effects on stroke volume, cardiac output and systemic vascular resistance. It also produces renal vasodilation. Dopexamine appears to be