ANESTHESIA

AND

VALVULAR HEART DISEASE

IN

NON CARDIAC SURGERY

Essay

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of Master Degree in Anesthesiology

by

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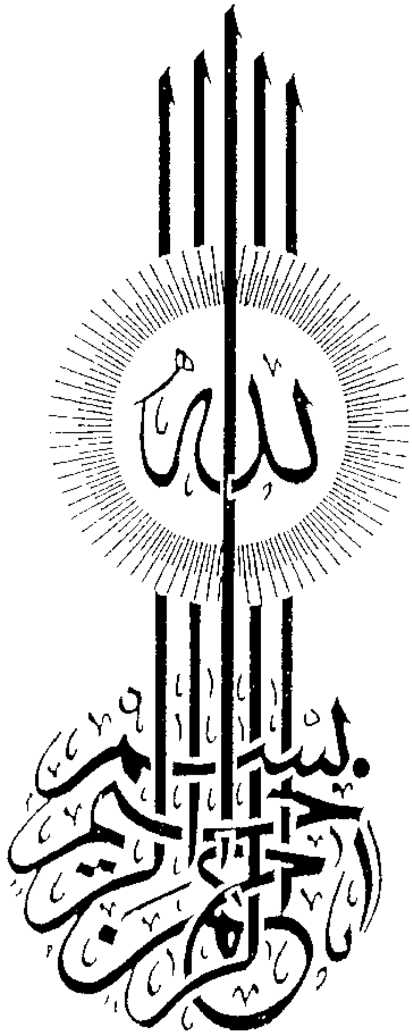
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# Introduction

# INTRODUCTION

Patients with cardiac disease frequently must be anesthetized for non-cardiac surgery . These patients may be just as sick or sometimes sicker than patients undergoing cardiac surgery . Numerous complications involving the cardiovascular system ( C.V.S.) may occur during surgery , anesthesia or postoperative period . The incidence of risks associated with such complications are determined by the type and severity of the cardiovascular disease, the surgical procedure , and the expertise of the operative team ( Kaplan and Dunbar , 1979 ) .

The morbidity and mortality of anesthesia and surgery are substantially increased by cardiovascular diseases . In the elderly , postopertive mortality is high and relates directly to abnormalities of the cardiovascular function that are often underestimated . Relatively silent cardiac or vascular diseases are often first diagnosed during the preoperative visit . Where the diagnosis has already been made it is essential to evaluate its influence on the cardiac reserve and on the risk of cardiac complications of anesthesia and surgery . Decisions may have to be made concerning further medical treatment to improve cardiac function . Optimal anesthetic management and appropriate monitoring rest on the correct diagnosis of the disease and on a detailed knowledge of its pathophysiology . Medical history , clinical examination , interpretation of ECG and chest X-ray form the basis of preoperative evaluation and may need to be complemented by echocardiography , cardiac catheterization and angiography .

( Foëx , 1982 )

# Anatomical Considerations

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# CHAPTER I

## ANATOMICAL CONSIDERATIONS

### Anatomy of the mitral valve :

The mitral valve is funnel - shaped with the apex within the left ventricle . It is composed of two leaflets described well by Roberts and Perloff , ( 1972 ) . The two leaflets are of unequal size , but of identical surface area. The anterior leaflet does not have a true annulus and is continuous with the wall of the ascending aorta . The posterior leaflet is attached ( and is part of ) the mural endocardium of the atrium . The two mitral leaflets are attached by 120 chordae to the two papillary muscles. ( Roberts and Perloff,1972).

### The tricuspid valve :

The anatomic distinction between the mitral valve and the tricuspid valve is largely artificial since both valves consist fundamentally of two large opposing cusps and small intermediary cusps at each end . However , the chordae tendineae of the tricuspid valve usually insert on three fairly distinct groups of papillary muscles , while only two principal papillary muscles serve the mitral valve ( Rushmer , 1976 ) . Another difference is that the tricuspid valve admits the tips of three fingers while the mitral valve admits the tips of only 2 fingers ( Last,1984).

The Semilunar valves :

The aortic and pulmonary valves are similar , each consisting of three symmetrical valve cusps . Two cusps of equal size could close tightly but would not open completely without considerable elastic strech , while three cusps can theoretically open to the full dimension of the valve ring and yet produce a perfect seal when closed.

( Rushmer , 1976 )

The edge length of each of the three leaflets is equal to one diameter of the aorta . In their fully opened position , the total edge length of the three leaflets equals the circumference of the aorta .

( Ewy and Temkin , 1982 )

Behind the aortic valve cusps are three outpuchings , the sinuses of valsalva , which prevent obstruction of the cronary ostia . If a valve leaflet came in contact with the coronary orifice , shutting off the flow of blood from the aorta , coronary pressure would fall repidly as blood left the coronary arterial system , and the valve cusps would sealed against the coronary ostium by a high differential pressure . This unfortunate accident is presumably prevented by the presence of adequate space behind the open valve cusps. Lastly , the heart valves are so simple and effective that the best available man-made substitutes are gross caricatures by comparison .

( Rushmer , 1976 )

# Physiological Considerations

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# CHAPTER II

## PHYSIOLOGICAL CONSIDERATIONS

### Normal Left Ventricular Function

It is divided into 4 phases :

(1) Isovolumic contraction .

(2) Left ventricular ejection .

(3) Isovolumic relaxation .

(4) Diastolic filling .

During isovolumic contraction , ventricular muscle contraction increases wall tension , raising the pressure of blood in the ventricle . When the ventricular blood pressure equals the aortic's , the aortic valve opens and ejection begins . Ejection involves rapidly transferring energy stored in the ventricular wall to the blood . Interia is overcomed and blood is rapidly accelerated into the aortic root to a high peak flow rate . Once the blood is given this " Kick " , it moves to a large extent , of its own accord , reflected by a higher aortic than ventricular pressure in the latter half of systole . The important concept here is of a ventricular " Kicking " ejection as opposed to a slower " squeezing " ejection . Flow into the aorta then slows and pressure falls untill a slight reversal of flow closes the aortic valve .

Normally , about two-thirds of ventricular volume is ejected with each systole .

Like contraction , isovolumic relaxation is an energy dependent process . It is often prolonged with ischemia or ventricular hypertrophy . Incomplete relaxation increases ventricular stiffness , hindering diastolic filling .

( Schneider , 1985 )

Diastolic filling occurs in two phases , passive and active ( during atrial systole ). 80 - 90 % of ventricular filling normally occurs during passive filling . Atrial systole completes ventricular filling and positions the A-V valve for efficient closure during the next ventricular systole .

Ejection requires tension (T) development in the ventricular wall. This is related both to pressure (P) developed during ejection, size ( radius,R ) and shape of the ventricle . For a spherical ventricle, T is proportional to P times R ( $T \propto P \times R$ ) . The whole thickness (h) of the ventricular wall contributes to developing tension ; this is described as stress (S)

$$S \propto \frac{T}{h} \quad , \text{ or } S \propto \frac{PR}{h} \qquad \text{La Place's Law}$$

Thus , changes in either P ( Aortic stenosis , Pulmonary stenosis ) or R ( Aortic incompetence , mitral regurgitation , pulmonary incompetence , tricuspid regurgitation )