

## Chapter 24

# Surgical Management of Mitral Valve Disease

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### ABSTRACT

*Surgery for the mitral valve has increased over the last decade, with a focus on an increasing number of valve repairs for degenerative mitral valve disease. This chapter discusses the surgical management of mitral valve disease with a focus on the pathology of mitral valve stenosis and regurgitation. With an examination into the pathophysiology of the lesions. Subsequently a discussion regarding the various surgical techniques for mitral valve surgery followed by the major and minor complications of surgery are reviewed to provide the Intensivist with an overview of possible complications. Finally a look at the future direction of the field is briefly examined.*

### INTRODUCTION

With the advent of reliable valve repair, and better bioprosthetic valve replacement technology the incidence of mitral valve surgery has increased over the last decade.

Mitral valve lesions can broadly be divided into two main categories – those being regurgitation and stenosis. Trends in mitral valve surgery have shown an increase in the number of regurgitant lesions being repaired and for those valves requiring replacement a movement to the increasing use of bioprosthetic valves over mechanical valves (Gammie, Sheng et al., 2009). Indeed the management of patients after surgery on the mitral valve can be challenging especially in cases of left ventricular dysfunction and dilatation. In this chapter we will review a basic understanding of mitral valve anatomy and etiologies of mitral valve lesions and their importance in managing the postoperative patient. In addition an understanding of surgical indications and techniques will be discussed including valve replacement, and repair in a variety of conditions and their effect on postoperative care. A major focus will be placed on intraoperative and postoperative considerations and potential complications that the Intensivist must be cognizant of when managing patients after mitral valve surgery. Finally a focus on future research and novel strategies for addressing mitral valve disease will be reviewed.

DOI: 10.4018/978-1-4666-8603-8.ch024

## **EPIDEMIOLOGY**

The incidence of the spectrum of mitral valve disease etiologies has changed over the last two decades with a fall in the number of cases of mitral stenosis secondary to rheumatic disease (Olson, Subramanian et al 1987). The incidence of mitral stenosis in the United States is the least common of all cardiac valve lesions and is estimated to be 0.1% with the frequency being evenly distributed among decades of life after the age of 50 (Nkomo, Gardin et al., 2006). The incidence of mitral regurgitation on the contrary is the most common cardiac valve lesion with an estimated incidence of 1.7% in the United States and having an increasing incidence with age. Surgical treatment for mitral stenosis has predominantly been mechanical valve replacement in the younger to middle-aged population, however this trend has changed towards an increasing use of bioprosthetic valve replacement (Gammie, Sheng et al., 2009). In addition there has been a trend toward increasing mitral valve repair for mitral regurgitation secondary to degenerative (organic) mitral valve disease.

## **VALVE ANATOMY**

The mitral valve is composed of a number of structures that function together allowing the valve to open in diastole and close in systole. There are five main components to allowing the mitral valve to function correctly: The valve leaflets, annulus, chordae tendineae, papillary muscles and the left ventricular wall. As the heart progresses through the cardiac cycle these components move together allowing for a competent valve. Anatomic or physiological changes to one or more of these components may lead to valve dysfunction.

There are two leaflets that comprise the mitral valve - anterior and posterior (Ranganathan, Lam et al., 1970). The region where the leaflets meet in continuity is referred to as the commissures. Although the leaflets are shaped differently they comprise the same surface area. The leaflets coapt along the rough zone which is thicker compared to the thinner atrial zone. Where the leaflets insert into the atrium is known as the annulus of the valve. The anterior aspect of the annulus is supported by the fibrous skeleton of the heart and as one moves towards the posterior annulus in a counterclockwise direction the fibrous support diminishes and eventually is absent leading to a more fragile region along the posterior annulus which is prone to dilatation (Wilcox & Anderson, 2004).

The chordae tendineae of the valve attach the leaflets to the papillary muscles. The chordae are divided into three main types depending on where they attach onto the leaflets (Lam, Ranganathan et al., 1970).

The primary chordae insert very close to the free margin of the leaflet edge. Secondary chordae attach to the leaflets usually in their mid portion in the ventricular side. Finally basal chordae extend from the ventricular wall or papillary muscle and insert at the base of the posterior leaflet.

Two large groups of papillary muscles are usually present – the anterolateral papillary muscle and posteromedial papillary muscle. Both muscle heads insert into the ventricular wall. These papillary muscles provide anchoring of the chordae that are also attached to the valve leaflets. The papillary muscles allow the left ventricle to play an important role in the function of the mitral valve. As the left ventricle contracts the papillary muscles also contract to avoid excess slack in the chordae and hence preventing the leaflets from prolapsing into the atrium resulting in mitral regurgitation.

In adults the mitral valve orifice is 4-6cm<sup>2</sup> when measured in cross-section (Iung, Gohlke-Barwolf et al., 2002).

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