

# A Contrarian View of Ischemic Mitral Regurgitation

Vincent A. Gaudiani, MD

## Introduction

This condition vexes us because even when we perform a reasonable ring annuloplasty and a competent coronary bypass operation, too often, either the MR recurs or the patient does poorly, or both. Naturally this stimulates investigation into fancier annuloplasty rings and more study of valve leaflets and chordae. These areas do deserve attention, but I respectfully propose two areas that have received much less attention and might help us to perform better in the operating room. I pose them as questions to be addressed in every operative candidate with LV dysfunction, central MR, and coronary disease.

1. What is the mechanism for electrical activation of the posterior left ventricular wall?
2. What are the precise anatomic details of the damaged left ventricle?

Most of the papers we rely on completely ignore the electrocardiogram and its profound effect on mitral valve function, particularly in damaged hearts. Furthermore we tend to “lump” impaired left ventricles that demonstrate central MR defining their abnormality in terms of the “restricted leaflet motion” that results, but what about the ventricle itself? Below I offer for your consideration a somewhat different view of this difficult problem.

## Starling’s Law

As we all know Starling’s Law states that increased LV filling causes increased LV contractility, but we must also remember that the Law works in reverse. Decreased filling reduces contractility. Once the ventricle is filled, at end diastole, a failure of mitral valve closure early in systole allows blood to “drool” back into the left atrium thereby reducing LV filling, interfering with isovolemic systole, and reducing contractility. If the anterior leaflet is anatomically normal, failure of the mitral valve to close early in systole must be largely the result of a defect somewhere in what we might call the “posterior apparatus”, that is, the entire mechanism that delivers the posterior leaflet to the anterior leaflet early enough to achieve isovolemic systole before any blood can leak out of the LV. This consists of the posterior leaflet and annulus, the LV summit it sits on, the chordae, the papillary muscles, the LV wall from which they emanate, the overall dimensions of the left ventricle, and the electrical mechanism that activates the posterior apparatus. A failure in any part of this complex in a patient with coronary artery disease and some central MR is likely to be labeled “ischemic mitral regurgitation”, but the treatments may differ vastly with the details of why the posterior apparatus failed to deliver the posterior leaflet to the anterior early in systole.

## Electrical Activation

When and how is this “posterior apparatus” activated? Let’s start here because this area has changed cardiology dramatically, but barely dented cardiac surgery. You may recall from medical school that the Bundle of His quickly divides into a small right bundle and a much larger left bundle as it passes through the central fibrous body. The left bundle then quickly divides again into a small anterior fascicle and a much larger posterior

fascicle. In fact the sum of both the right bundle and the left anterior fascicle is less conduction tissue than the posterior fascicle alone. Why was the posterior fascicle made so large? The posterior fascicle winds around the summit or base of the left ventricle and activates it early in systole. By activating the summit of the free wall early, it starts the posterior leaflet of the mitral valve moving towards the anterior leaflet of the mitral valve early in systole. Early closure of the mitral valve is arguably the most important accomplishment of the entire conduction system because it facilitates Starling's Law, that is, it permits isovolemic systole to occur when the ventricle is as full as it can be given the filling conditions. What electrical conditions can delay early mitral valve closure?

The common culprits are

- Left bundle branch block (LBBB)
- RV apex pacing
- Many QRS durations longer than about 150 ms. This is generally called an intraventricular conduction delay.
- Just to make sure you understand, right bundle branch block is **not** a culprit. Do you see why?

Happily, the system is robust, and if the ventricle is normal in size and contractility and has a nice mitral apparatus, early mitral valve closure may occur even when activation of the posterior wall is delayed. This is why many patients with typical pacing systems or LBBB have little if any MR. At the opposite extreme some combinations of LV size and LV dysfunction cause central MR even when QRS duration is normal. In the middle, some combinations that permit early mitral valve closure when QRS duration is normal will yield MR and heart failure if the QRS duration becomes prolonged.

Delayed left sided activation is the cause of "pacemaker syndrome" and explains why some marginal patients decompensate when paced by a standard RV endocardial lead. It also explains a vexing result in the cardiology literature. Numerous randomized controlled trials comparing VVI and DDD pacing failed to show any benefit of the presumably more "physiologic" DDD devices, a major embarrassment for both practitioners and pacing companies. As it turns out, this happened because the detrimental effects of standard ventricular pacing overwhelm any benefit of AV synchrony. The moral of the story is simple: if the mitral valve is to close early in systole, the posterior summit must be electrically activated early in systole. And of course, the entire complex contractile mechanism of the posterior apparatus must be alive and able to respond to electrical stimulation. What happens if one or more of the contractile elements of the posterior apparatus are infarcted, ischemic and unable to respond?

### **The Anatomy of the Posterior Apparatus**

Leaving electrical activation aside for a moment, the posterior apparatus may impair early mitral valve closure because its blood supply and/or its function is deranged.

Suppose, for instance,

- It is entirely alive, but has a severely compromised blood flow and fails to contract under some systolic loading conditions. The posterior apparatus fails to move forward early enough in systole to prevent central MR.

- It is entirely alive with or without associated arterial compromise, but the patient has sustained a large anterior MI in the past and now has a dilated LV, and therefore a dilated mitral annulus and central MR.
- It is entirely dead from a previous infarction that caused thinning from apex to base, reduced EF, LV enlargement, dilated mitral annulus, dead papillary muscles and posterior wall, and central MR.
- Its base is damaged and bulges somewhat on LV angiogram, the annulus is dilated as a result, the papillary muscles are unaffected, and there is central MR.
- Its base contracts on LV angiogram, but there is severe infero-apical hypokinesia and central MR. The papillary muscles are little white scars when inspected through the left atrium.

Let's assume that there are one or more operable coronary lesions in each of these examples. In which of these situations would you diagnose ischemic MR and which would you treat with the ring of your choice and coronary bypass? Is there a place for MV replacement in any of these cases or is that always an inferior option to ring annuloplasty? Do you want to do any other procedures? And to come back to our first question, what would you do if any of these patients had QRS duration of 200 ms?

### **The Challenge**

I propose that we take the following steps:

- Rethink the “ischemic MR” literature accounting for delayed left sided activation.
- Rethink the “ischemic MR” literature accounting for the precise abnormality of posterior apparatus blood supply and contractile function.
- Learn how to perform permanent and temporary biventricular pacing during cardiac operations.
- Reduce the use of the term “restricted leaflet motion” that merely summarizes the end result of a variety of posterior apparatus malfunctions that may require different surgical solutions.

We may still fail when we treat patients with ischemic mitral regurgitation because this condition signals a potentially profound disruption at the epicenter of left ventricular design, but this approach might identify subgroups that have a better prognosis.