Functional (also called ischemic) mitral regurgitation (MR) in patients with coronary artery disease (CAD) is a disease of the cardiac muscle instead of a disease of the valve. This MR is produced by left ventricular (LV) global remodeling (dilation and sphericity) or regional remodeling in either case leading to tethering of the valvular apparatus towards a dyssinergic wall. This tethering may lead to restriction of the leaflets and incomplete closure of the valves in systole (Figure 1). Both valve closing force decrease due to LV systolic dysfunction and dyssynchrony may also have a role. This entity has been comprehensively studied in recent years with both resting and exercise echocardiography studies. Exercise echocardiographic studies have consistently shown that the development or worsening of MR with exercise is an important independent predictor of bad outcome in patients with CAD [1-5]. However, in some of these studies, it has been seen that functional (aka “ischemic”) MR may increase significantly during exercise in them, even in absence of ischemia [1-2]. Therefore in our view there is a confusing terminology, as it does not seem appropriate to define as ischemic a condition in which ischemia is not present. Thus, it appears that we are actually talking of two different entities. Entity one would be truly ischemic MR, which would develop during an ischemic burden, for example during an unstable condition at rest or during stress-induced ischemia. We have studied in the past this type of MR in patients with suspected or known CAD that underwent exercise echocardiography [6]. In that study we saw that a new or increased MR during exercise was associated to severe ischemia leading to a decrease in LV ejection fraction, global or regional dysssinergia, increase in LV volumes with exercise, sphericity, and increase in LV filling pressures, as assessed by a change in LV inflow pattern from and altered relaxation to a pseudonormalized/restrictive pattern (Figure 2). As expected, patients with these findings had more commonly multivessel CAD. Entity two should be named necrotic MR, as the condition is due to regional or global wall motion abnormalities that are already present at rest as a result of a previous infarction, even though this kind of MR may also increase or develop with exercise in absence of ischemia due to changes in local remodeling (Figure 3). Besides the more logical terminology, treatment also differs. The treatment of truly ischemic MR would consist of revascularization, whereas mitral valve repair or mitral valve replacement might be considered for necrotic MR. Of course, as always in medicine mixed conditions may exist in some patients and both procedures would need to be used. However in patients with CAD considered for revascularization who also had moderate MR at rest (therefore necrotic MR), the addition of mitral valve repair to the revascularization procedure did not result in any benefit at one year of follow-up in a recent randomized study with 301 patients [7]. In fact although mitral-valve repair was associated with a reduced prevalence of moderate or severe MR after surgery, there were an increased number of untoward events in this arm.
In conclusion, we propose a change in terminology for two conditions that have several differences although both of them may be present in patients with CAD.

**References**


