Aortic stenosis (AS) and mitral regurgitation (MR) are the most common valvular heart diseases in the adult population. Additionally, they are the most likely to need surgical intervention. Treatment preceding surgical or percutaneous correction of a valvular defect is targeted primarily on symptoms, since causal pharmacological treatment is lacking. A detailed understanding of the mechanisms behind valvular heart disease is necessary to effectively intervene in the pathogenesis. Calcified aortic valve stenosis, currently considered an active process consisting of a wide range of cells, signaling molecules, and transcription factors, is largely consistent with the pathogenesis of atherosclerosis; however, about 50% of patients with calcified AS do not exhibit significant atherosclerosis. Our work was focused on measuring osteoprotegerin (OPG) levels in the tissue of calcified aortic valves. OPG acts as decoy receptor for RANKL and affects calcification and differentiation of various cells during progression of AS. We found significantly higher tissue levels of OPG in patients with isolated AS compared to patients with concurrent AS and coronary atherosclerosis. These findings support the hypothesis that there is a unique pathogenesis for each clinical manifestation of calcified aortic valve disease. MR is another clinical situation, where knowledge of the pathogenesis is key to the management of the disease. MR is very dependent on the hemodynamics as well as the characteristics of the left ventricle. In our study we focused on patients with, at least, moderately severe MR undergoing aortic valve replacement due to AS. Our objective was to evaluate the post-operative decrease in the degree of MR, its dependence on the etiology of MR and other clinical factors, and to assess the prognostic significance of persistent MR. We found a significant decrease in the severity (on average 0.5/4 degrees) of MR regardless of etiology; however, we failed to identify any pre-operative predictors of MR improvement. From a post-operative perspective, persistent MR was associated with a higher frequency of cardiovascular hospitalizations. Another way to directly intervene in the MR pathogenesis mechanism is coronary revascularization (CABG) in patients with ischemic MR. However, not all patients experience a decrease in the degree of ischemic MR after CABG, therefore it is necessary to pre-operatively define factors that increase the probability of ischemic MR improvement. In our work dealing with changes in moderately severe ischemic MR after isolated CABG, we found that a substantial presence of viable myocardium ($\geq$ 5 segments) and an absence of dyssynchrony between the papillary muscles ($<$ 60 ms) were the two main independent predictors of long-term ischemic MR improvement after isolated CABG.