Introduction: Left ventricular outflow tract (LVOT) obstruction is a well-recognized postoperative complication after mitral valve replacement surgery. We present a case of LVOT obstruction following a mitral valve replacement that was not relieved by reseating of the valve.

Case Presentation: A 68 year old female with mitral valve (MV) endocarditis presented for a MV replacement. Preoperative cardiac workup showed an EF of 65%, mild to moderate mitral stenosis, severe mitral regurgitation, and a vegetation seen on the posterior leaflet of the mitral valve. Induction of anesthesia was uneventful and invasive monitoring was established without complications. Intraoperative TEE examination prior to cardiopulmonary bypass (CPB) showed an EF of 55%, severe MR, mild mitral stenosis, and left ventricular hypertrophy. The patient required CPB for 138 minutes during which time a 27mm stented bioprosthetic was placed in the mitral position. Post-CPB TEE examination revealed a strut from the bioprosthetic valve impinging upon the left ventricular outflow tract (LVOT). The peak LVOT gradient was 84mm Hg and the mean LVOT gradient was 34mm Hg. CPB was resumed in order to reposition the bioprosthetic valve. A second CPB run of 126 minutes ensued. Post CPB TEE examination showed the LVOT peak gradient to be 30mm Hg with a mean gradient of 16mm Hg.

The immediate postoperative course was complicated by sepsis with high inotrope and vasopressor requirements. She required mediastinal exploration for hemodynamic instability on POD 3. Intraoperative TEE examination during the exploration showed a hypovolemic, hyperdynamic heart with an EF of >65%. There was evidence of dynamic LVOT obstruction by the MV strut with a peak gradient across the LVOT of 80mmHg and a mean gradient of 30mmHg. Fluids were administered and the obstruction diminished. Repeat TEE performed 2 months postoperatively showed an EF >65%, calcification of the aortic valve, small paravalvular MV leak, and a LVOT peak gradient of 20mmHg.

Discussion: LVOT obstruction after MV replacement is a well-known complication. This obstruction can be fixed or dynamic. Dynamic obstruction can be caused by a narrowed mitral-aortic angle in association with a thickened interventricular septum, a reduction of LV dimensions, a hypercontractile LV, or atrial fibrillation. The risk is increased with the preservation of the native MV apparatus. Due to this patients septic picture with a hyperdynamic LV, in addition to her LVH, she was at high risk for LVOT obstruction. Her LVOT gradient diminished as her sepsis resolved.

References:
1) Tex Heart Inst J 2206; 33: 399-401
3) Am Heart J 1991; 122:483-487