Introduction:
Atrioventricular disruption (AVD) is a rare and grave complication of cardiac surgery. We present a case of AVD in a patient undergoing aortic and mitral valve replacement (AVR and MVR).

Case Presentation:
An 82 year-old female with severe aortic and moderate mitral stenosis presented for AVR and MVR. Intraoperative TEE indicated preserved biventricular systolic function, a calcified and stenotic AV, and significant MV annular and leaflet calcification. Total aortic cross-clamp and CPB durations were 141 and 182 minutes, respectively. Separation from CPB was achieved with inotrope and pressor support. TEE demonstrated that the mechanical prosthetic valves were well positioned and functioned properly. Prior to chest closure, modest but persistent bleeding from behind the heart was noted. The possibility of AVD was discussed, but it was concluded that the patient would not tolerate return to CPB for re-exploration and possible repair. However, concern for AVD prompted administration of rFVIIa and packing of the posterior atrioventricular groove with Surgicel®; the bleeding abated. Approximately eight hours postoperatively, the patient became acutely unstable with increased mediastinal bleeding requiring significant transfusion and high doses of pressors and inotropes. This bleeding proved refractory and the patient expired shortly afterwards.

Discussion:
First described in 1967 (1), AVD refers to dehiscence of the left atrium from the left ventricle, frequently at the posterior atrioventricular groove. AVD most commonly occurs after MVR, although it has been described in other settings. Historically, the incidence of AVD was as high as 0.5-2% following MVR (2). The incidence of AVD has decreased dramatically due to factors such as less calcific MV disease, reduced frequency of MVR, improved techniques of MV repair, better designed MV prostheses, and increased preservation of the posterior mitral valve apparatus (2,3). Risk factors for AVD include advanced age, female sex, thin body habitus, mitral stenosis, first time MV surgery, as well as aggressive debridement of posterior mitral annular calcification and/or the mitral subvalvular structures (2,4). AVD may present as inability to separate from CPB, paravalvular leak with mitral insufficiency, left ventricle (LV) aneurysm or pseudoaneurysm, new wall motion abnormality, bleeding, and/or increased inotrope/pressor need. Ultimately, diagnosis of AVD requires a high index of suspicion. AVD may be treated via topical application of hemostatic materials (5) or LV patching, often requiring return to CPB and replacement of the MV prosthesis (6). Non-surgical interventions include gradual weaning from CPB, minimizing volume loading to reduce LV dilation, and avoiding excessive increases in LV contractility and afterload (7). Prognosis following AVD is grim, with mortality in excess of 50% (4). Although treatment of AVD is primarily surgical, we highlight AVD as a severe albeit now rare complication of cardiac surgery.

References:
1) J Thorac Cardiovasc Surg 1967;54(3):422