Aim: As acute short-lived increase in afterload may be a contributory mechanism to sudden death, we studied an acute 30% systolic pressure increase (SBPI) in a closed chest, closed pericardium porcine model, closed pericardium being essential to the model clinical applicability.

Methods: 7 pigs were studied. All had an acute 5 beat 30% SBPI induced by a non-occlusive mid-descending aortic balloon inflation and release. Each challenge was continuously monitored for changes in cardiac morphology and function by cardiac ultrasound (2D, Doppler and Doppler Myocardial Imaging) and changes were correlated with pressure data from 3 Millar catheters (LV/Ao; LA; RV/RA). Continuous 12 lead ECG and intracardiac electrograms were also recorded.

Results: Balloon inflation caused an acute diastolic pressure increase in all cavities (except pulmonary artery) with early diastolic preceding late changes and a corresponding systolic increase in LV, LA and RA pressures. Acute LV dilatation resulted in pericardial flattening (which is a visualization of the effects of pericardial constraint), a septal shift towards the RV and a decrease in RV size with a 30% reduction in LVEF % (Figure 1a). During inflation, pericardial excursion flattened >40% in basal, mid and apical LV segments, mostly mid wall (from 3.4 ±1.4 mm to 1.6 ±0.7 mm).

Figure 1a. Parasternal long-axis M-mode showing changes induced by acute left ventricle afterload. A time-aligned left ventricle pressure trace is embedded in the picture.

Figure 1b. Left ventricle cavity size and pericardial excursion changes (at the basal, mid and apical ventricle level) before, during and after acute afterload increase.
p<0.05) (Figure 1b). With balloon deflation, the above rapidly returned to baseline.

**Conclusion:** Acute afterload increase induced immediate, profound and consistent changes in LV dimensions and function. Pericardial constraint caused a marked rightward septal shift which altered right heart diastolic function and reduced filling. Acute loading markedly altered both systolic and diastolic function for both the right and the left heart. It remains to be evaluated how these changes affect the mechano-electric coupling and as such could play a role in fatal arrhythmias.

**KEYWORDS:** acute ventricular afterload, acute ventricular dilatation, experimental cardiovascular models, pericardial constraint, intracardiac pressures.

**Literature**