

to pre-ECV (n=15, 42±11%, p=0.015). Atrial EF (aEF) increased immediately after ECV (n=66, from 18±9% to 22±11%, p=0.01). In patients who remained in SR, aEF further increased after 4-6 weeks (n=51, to 36±14%, p <0.001 vs. post-ECV).

**Conclusions:** Immediate improvement in EF after ECV explains about 50% of total EF improvement over time. However, both left ventricular function as well as atrial function continue to increase over 4-6 weeks after ECV.

**P1104 | BEDSIDE**

**Left ventricular vortex following atrial contraction and its interaction with early systolic ejection**

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**Introduction:** Flow inside cardiac chambers organises in vortices, rotational flow structures that could play a role in energy preservation, optimizing cardiac performance. No comparison has been established between the two vortices that appear in the left ventricle (LV) in a normal cardiac cycle, following early ventricular filling (V1) and atrial contraction (V2) and relating their potential differences to their role in cardiac performance.

**Objective and methods:** To describe the potential differences between V1 and V2 and their significance in cardiac function, 50 patients (50% male, aged 53.4±29, LVEF 69.3%±14.8) in sinus rhythm and without valve disease underwent transthoracic echocardiography. LV 3-chamber views were acquired and analyzed using Vector Flow Mapping (VFM), a novel technique allowing intracardiac flow visualization. We measured parameters describing vortex flow intensity, size, location and duration.

**Results:** No significant differences were observed between V1 and V2 in size or flow intensity. However, location and duration differed significantly between V1 and V2. V1 appeared close to the anterior wall, in the mid-basal region of the LV remaining there in 70% of patients. V2 initially appeared in the same region but evolved towards the submitral area in 84% of patients (Figure 1). V1 duration was significantly longer and with significantly higher variation than V2 (18% ± 13% vs. 15% ± 9% of the cardiac cycle, p = 0.017).

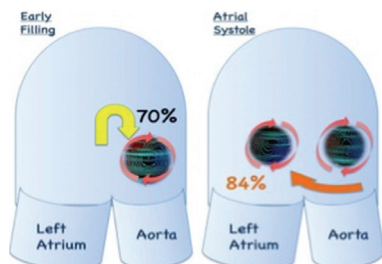


Figure 1. Vortices' location

**Conclusions:** Vortices generated in a normal cardiac cycle are similar in size and intensity, but their duration and position vary significantly. V2's shorter duration, change in position inside the LV and lower variability in its behaviour may be related to the opening of the aortic valve and closure of the anterior leaflet of the mitral valve.

**P1105 | BEDSIDE**

**Inefficient left ventricular inflows in patients with right ventricular apical pacing**

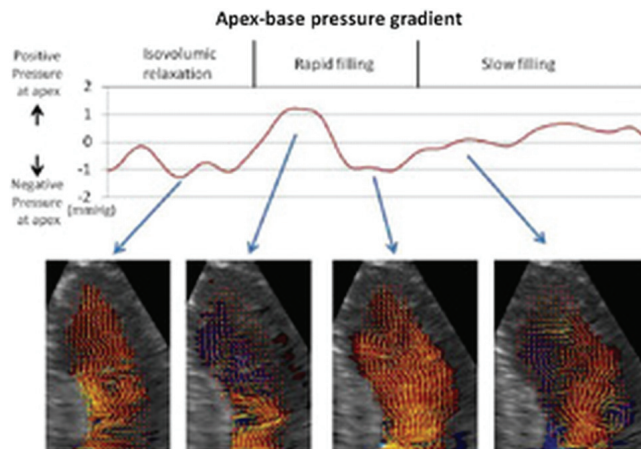
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**Background:** It remains to be clarified whether right ventricular apical pacing (RVAP) affects LV diastolic function.

**Methods:** This study included 20 patients with a RV pacemaker implanted (age 58±13 y/o) and 20 age-matched healthy controls. Inflow pattern and pressure distribution within the LV were evaluated, using VFM. Apex to base pressure gradient within the left ventricle was estimated from the color M mode data by solving the Euler equation, as previously established.

**Results:** In 16 patients (80%) with RVAP, flow was directed toward the apex during the isovolumic relaxation period, but then it reversed toward the base and collided with inflow through the mitral valve during early diastole. These flow patterns reflected insufficient LV suction, with lower apex to base pressure gradient during early diastole, compared with normal controls (1.0±0.5 vs 2.5±1.0 mmHg, p<0.01).

**Conclusions:** LV with RVAP exhibits inefficient LV inflows and suction.



Abstract P1105 – Figure 1

**P1106 | BENCH**

**Left ventricular wave propagation velocity during late diastole: relationship with tissue distensibility, loading and material properties**

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Ventricular inflow generates waves propagating within the chamber and along the myocardial walls, governed by intraventricular pressure gradients and vortex formation/propagation. The faster (pressure) waves may have a mechanism similar to that described in elastic tubes and arteries, in which wave speed is inversely related to distensibility (Db). Here, we measured wave propagation velocity along the myocardial wall in late diastole (VpA) and analyzed its relationship with tissue Db and stiffness.

**Methods:** VpA was measured from 4-5 piezocrystals implanted in line (base to apex) along anterior LV wall in 15 pigs. Additional crystals tracked changes in short-axis at 2-3 levels. Acute myocardial infarction (MI) was induced by coronary artery occlusion followed by reperfusion. Preload and afterload were altered by caval (IVC) and aortic (AoC) constriction and fluids (Vol) at baseline and reperfusion (Figure). Data were acquired at 500-1,000Hz. Db during late diastole and myocardial stiffness (β-coefficient) were calculated. In animals and 3 human volunteers, LV VpA was measured by tissue Doppler (strain rates).

**Results:** VpA ranged from 0.5-8m/s and followed changes in Db with acute interventions (Figure). VpA increased with preload, but more so with alterations in myocardial stiffness post-MI (confirmed by β). VpA was 2.1±1.1m/s vs. 7.2±5.1m/s in subendocardial vs. transmural MI (p<0.05) despite similar end-diastolic LV pressures. The correlation between VpA and 1/Db (measure of stiffness) was modest but significant (r=0.58, p=0.03 at reperfusion). Tissue Doppler data showed similar results.

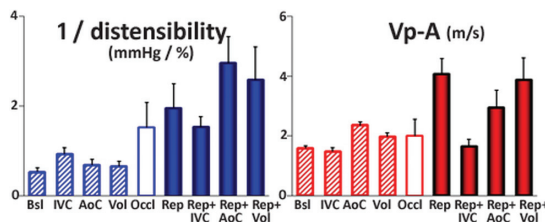


Figure 1

**Conclusions:** This study demonstrates that LV VpA is related to mechanical properties of the myocardium and loading. The results are relevant for the assessment of myocardial elasticity by echocardiography.

**P1107 | SPOTLIGHT 2013**

**Positive end expiratory pressure-PEEP can affects the 4-chamber longitudinal strain analysis**

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**Purpose:** The use of echocardiography in critically ill patients admitted to mixed intensive care unit (ICU) is increasing. Speckle-tracking echocardiography (STE) has now emerged as an ultrasound technique for evaluating cardiac function with longitudinal strain (LS) analysis.

In mechanically ventilated (MV) patients no studies have been performed to evaluate the effects of positive pressure ventilation on the strain of the heart chambers. The aim of the study was to evaluate the effects of positive end-expiratory pressure (PEEP) on 4-chamber LS analysis in MV patients.

**Methods:** We enrolled 25 consecutive MV patients (mean age  $65\pm 17$ ) admitted to a mixed ICU. Inclusion criteria were: hypoxia requiring PEEP titration, invasive arterial pressure monitoring, age  $> 18$ . Exclusion criteria were: cardiac arrhythmias and valvular pathologies. Standard echocardiography (MyLab 70 Xvision, Esaote) was performed by the same operator at three times: (T1) PEEP of 5 cmH<sub>2</sub>O; (T2) PEEP of 10 cmH<sub>2</sub>O; (T3) PEEP of 15 cmH<sub>2</sub>O. Cardiac output (CO) was evaluated using the pulse contour method MostCare (Vygon, Padua, Italy). STE analysis was performed off-line with a dedicated software (XStrainTMMMyLab 70 Xvision, Esaote).

**Results:** Left peak atrial LS (LA-PALS) was significantly reduced from T1 to T2, and from T2 to T3 ( $41.2\pm 11\%$ ,  $40\pm 9$ , and  $27.5\pm 8\%$ ; T1, T2, T3, respectively;  $p < 0.05$ ). Right peak atrial LS (RA-PALS) and right ventricular (RV)-LS showed a significant reduction only at T3 (RA-PALS:  $45\pm 48.2\%$  at T1,  $36\pm 10.6\%$  at T3; RV-LS:  $-20.5\pm 2\%$  at T1,  $-15.2\pm 1.6\%$  at T3;  $p < 0.05$ ). Left ventricular (LV)-LS did not change significantly during PEEP titration. Cardiac chambers' volumes and cardiac output (CO) showed a significant reduction at higher levels of PEEP.

**Conclusions:** The increase of PEEP induces a reduction of LA-, RA- and RV-LS values, without affecting LV-LS values. The fall in CO when using values of PEEP  $> 10$  cmH<sub>2</sub>O seems to be related to preload reduction and not to myocardial contractility changes.

Whenever interpreting data based on longitudinal strain analysis, clinicians should draw attention to different levels of PEEP in MV patients. The higher the PEEP, the more the probability that speckle tracking echocardiography measurements can be affected.

### P1108 | BENCH

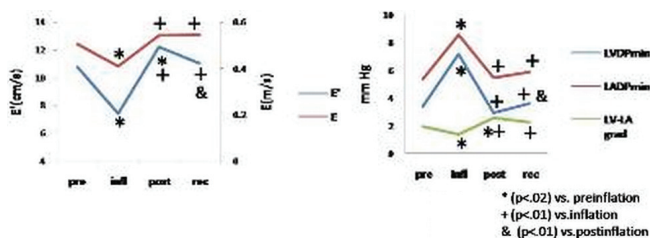
#### How rapidly can diastolic function alter with acute afterloading - insights from an experimental closed chest/closed pericardium acute afterload porcine model

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**Intro:** The speed and timing of changes in diastolic function parameters during acute LV loading/unloading is unknown.

**Methods:** In a closed chest/closed pericardium porcine model, LV systolic pressure was increased by 30% using a transient descending aortic partial balloon inflation. Morphology and mechanical changes were monitored in real time by echocardiography as were changes in left/right heart diastolic pressures by 3 Millar catheters. Simultaneous blood pool and tissue Doppler data (radial+long axis) were acquired during afterload changes.

**Results:** In 7 animals, acute loading consistently induced LV dilatation and rightwards ventricular septal shift due to lateral LV pericardial restraint. This reduced RV volume. Afterload increase caused an immediate decrease in early diastolic filling wave (E) and lateral wall velocity (E'). This was mirrored by an immediate increase in both early diastolic LV (LVDPmin) and LA (LADPmin) pressure (Fig1). Pulmonary vein flow gradually reduced with decreased late flow. The rightwards septal shift induced a small but significant increase in RVDpmin and an increase in mean RA pressure, but failed to increase pulmonary artery pressure. Acute afterload release immediately returned E values to baseline but E' showed a transient further 3 to 5 beat increase before it normalised.



**Conclusion:** Acute afterloading variably elevates all cardiac diastolic pressures and reduces LV relaxation. These early changes in myocardial mechanics occur simultaneous with changes in LVDPmin. Right heart diastolic pressures elevates by septal shift which both reduced RV volume and decreased LV compliance but did not elevate pulmonary pressure. E/E' recovery differed which could be attributed to transient preload changes.

### P1109 | BENCH

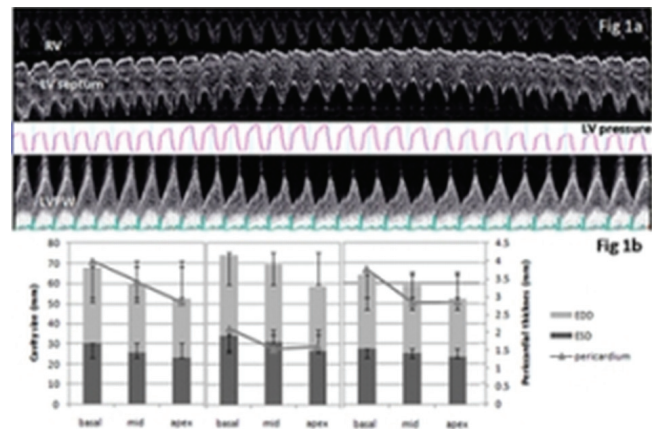
#### Acute cardiac remodeling in short term acute afterload increase - the effect of pericardial constraint

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**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:** Seven pigs were studied. All had an acute 5 beat 30% SBPI induced by a non-occlusive mid-desc aortic balloon inflation and release. Each challenge was continuously monitored for changes in cardiac morphology and function by cardiac ultrasound (2D, Doppler and Strain) and changes 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) (early diastolic preceding late changes) a corresponding systolic increase in LV, LA and RA pressures. Acute LV dilatation resulted in pericardial flattening (= pericardial constraint), a right septal shift and a decrease in RV size with a 30% reduction in LVEF % (Fig1a). During inflation, pericardial excursion flattened  $> 40\%$  in basal, mid and apical LV segments, mostly mid wall (from  $3.4\pm 1.4$  mm to  $1.6\pm 0.7$  mm) (Fig 1b). With balloon deflation, all parameters rapidly returned to baseline.



**Conclusion:** Acute afterload increase induced immediate, profound and consistent changes in LV dimensions and function. Pericardial constraint caused 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 left heart. It remains to be evaluated how these changes affect mechano-electric coupling and as such could play a role in fatal arrhythmias.

### P1110 | BEDSIDE

#### Value of apical circumferential strain at the early post-myocardial infarction period for prediction of left ventricular remodeling

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**Background:** Post myocardial Left Ventricular (LV) remodeling is related to increased morbidity and mortality. The aim of the present study was to examine whether LV deformational and torsional parameters can predict LV remodeling in patients with Acute Myocardial Infarction (AMI).

**Methods:** Forty two patients (age  $57\pm 14$  yrs) presented with an anterior ST elevation AMI treated with primary Percutaneous Transluminal Coronary Angioplasty (PTCA) were included. Four days post MI, LV Ejection Fraction (EF), LV torsion, longitudinal (4-, 3- & 2-chamber) and circumferential strain (CS) of the LV apex were evaluated by conventional and speckle tracking echocardiography. Echocardiographic study was repeated 3 months post-AMI and patients with LV remodeling [increase in LV End-Systolic Volume (LVESV)  $> 15\%$ ] were identified.

**Results:** The 13 patients identified with LV remodeling had significantly more impaired apical CS ( $-7.3\pm 2.2\%$  vs.  $-18.9\pm 5.2\%$ ,  $p=0.001$ ), EF ( $42\pm 7\%$  vs.  $48.9\pm 6\%$ ,  $p=0.005$ ), LV apical rotation ( $6.8\pm 4.8^\circ$  vs.  $11.1\pm 4.0^\circ$ ,  $p=0.027$ ) and LV global longitudinal strain ( $-9.7\pm 1.9\%$  vs.  $-12.9\pm 2.9\%$ ,  $p=0.03$ ) in comparison to those without LV remodeling, at 4th day post-AMI. Apical CS at 4th day post-AMI showed the strongest correlation with the LVESV 3 months post-AMI ( $r=0.76$ ,  $p=0.001$ ) (figure) in relation to EF ( $r= -0.60$ ,  $p=0.001$ ), global longitudinal strain ( $r=0.56$ ,  $p=0.001$ ) and LV apical rotation ( $r= -0.53$ ,  $p=0.001$ ). Furthermore, CS strain demonstrated the highest diagnostic accuracy [area under the receiver op-

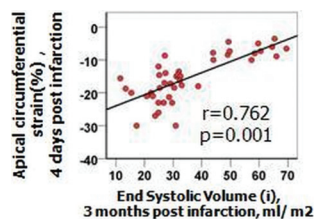


Figure 1