Methods: We enrolled 25 consecutive MV patients (mean age 65±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 cmH2O; (T2) PEEP of 10 cmH2O; (T3) PEEP of 15 cmH2O. Cardiac output (CO) was evaluated using the pulse contour method MostCare (Vygon, Padua, Italy). STE analysis was performed off-line with a dedicated software (XStrainTMMyLab 70 Xvision, Esaote).
Results: Left peak atrial LS (LA-PALS) was significantly reduced from T1 to T2, and from T2 to T3 (41.2±11%, 40±9, and 27.5±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±48.2% at T1, 36±10.6% at T3; RV-LS: -20.5±2% at T1, - 15.2±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 cmH2O 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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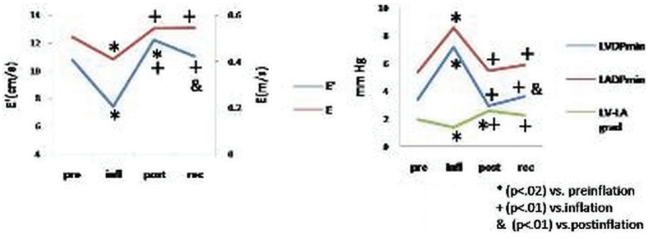
Intro: The speed and timing of changes in diastolic function parameters during acute LV loading/unloading is unknown.
Methods: In a closed chest/closed pericardium porcine model, LV systolic pressure was increased by 30% using a transient descending aortic partial balloon inflation. Morphology and mechanical 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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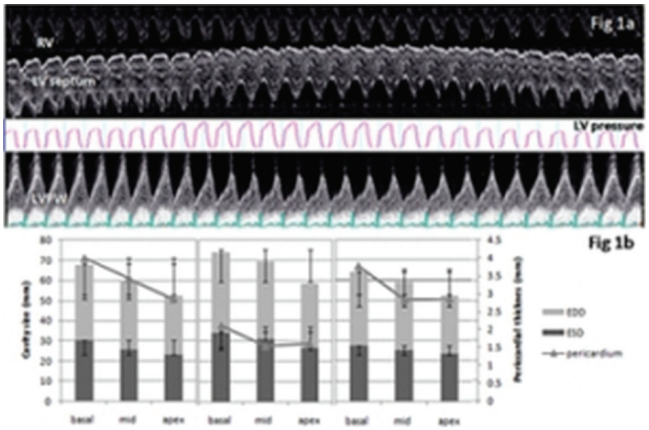
Aim: As acute short-lived increase in afterload may be a contributory mechanism to sudden death we studied an acute 30% systolic pressure increase (SBPI) in a closed chest, closed pericardium porcine model, closed pericardium being essential to the model clinical applicability.
Methods: Seven pigs were 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 preceeding 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±1.4 mm to 1.6±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 nfarction (AMI).
Methods: Forty two patients (age 57±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±2.2% vs. -18.9±5.2%, p=0.001), EF (42±7% vs. 48.9±6%, p=0.005), LV apical rotation (6.8±4.8° vs. 11.1±4.0°, p=0.027) and LV global longitudinal strain (-9.7±1.9% vs. -12.9±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-

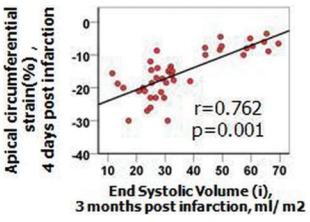

Figure 1