therapy including a vitamin K antagonist added to the classical dual antiplatelet therapy. One patient died from a recurrent subdural haematoma, another one had a peripheric embolism.

Conclusions: In this prospective multicenter study, despite a dual antiplatelet therapy, LV thrombus occurred in 26% of patients after an anterior MI complicated with LV dysfunction. Focused TTE has a high accuracy for their detection. CMR-DE should be performed only in patients in whom the apex is not clearly seen.

Dobutamine-related coronary spasm among patients with false positive dobutamine stress echocardiography: prevalence and predictors

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Background and objective: Dobutamine stress echocardiography (DSE) is being consistently used as an exercise-independent stress modality aimed at the detection of coronary artery disease (CAD) and the evaluation of myocardial ischemia. It may though occasionally induce coronary vasospasm. In this study, we aimed to evaluate the prevalence and predictors of dobutamine-related coronary spasm in patients without known CAD and false positive DSE (positive DSE but no significant coronary lesions on angiogram).

Methods: 3952 patients referred to our echocardiography laboratory for DSE between January 2010 and May 2012 were prospectively investigated. Those with positive DSE underwent coronary angiograms with systematic methylergometrine intracoronary injection in case of absence of significant coronary stenosis or spontaneous occlusive coronary spasm. Patients with spontaneous occlusive coronary spasm or positive methylergometrine test but no significant stenoses were enrolled and compared with those with positive DSE but no coronary lesions nor spontaneous or induced spasm (= true false positive DSE)

Results: 29 patients with DSE-related vasospasm (19.4% of positive DSE without known CAD) were compared with 56 patients with no lesions and no spasm (= true false positive DSE). They were more frequently smokers (72.4% vs 37.5%; P=0.003); they had more frequently dyslipidemia (79.3% vs 43%; P=0.001); they also had a larger ischemic area at peak DSE (3.4 vs 2.7 segments; P=0.05).

On multivariate analysis, dyslipidemia (HR=10.7; 95% Cl= [2.7-42.1]; P=0.001) and active smoking (HR=6.1; 95% Cl=[1.7-21.1]; P=0.004) were found to be independent predictors of spasm-related DSE rather than ≠ true ≠ false positive DSE.

Conclusion: DSE-related coronary spasm is present in a significant proportion of patients with erroneously labelled ‘false’ positive DSE and should systematically be ruled out. Dyslipidemia and active smoking were independent predictors of spasm rather than ≠ true ≠ false positive DSE.

Echocardiographic estimate of pulmonary vascular resistances: a validation in a non-selected population with pulmonary hypertension

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Introduction: Invasive hemodynamic procedures are still necessary to appreciate pulmonary hypertension characteristics. A few studies have addressed the issue of echocardiographic evaluation, but lack an external validation of their results with various settings and patients. The aim of our study was to appreciate the value of 2 published echocardiographic evaluations of pulmonary vascular resistances (PVR) based on tricuspid regurgitation maximal velocity (TRV) and right ventricular outflow tract systolic time velocity integral (TVI).

Methods: All of the patients presenting during 2013 for invasive haemodynamic evaluation with a suspected pulmonary hypertension were prospectively included, with echocardiography being done within 24 hours of catheterization. The TRV/TVI and TRV²/TVI ratio and echographic estimations of PVR were compared with catheter values; predictive values to detect elevated PVR were assessed.

Results: 43 patients were included, 12 with atrial fibrillation, 16 with valvular disease, 14 with hypertrophic cardiomyopathy. Mean pulmonary pressure was 41±11 mmHg, TRV/TVI and TRV²/TVI were correlated with PVR (r=0.71 and r=0.78 respectively, P<0.01). Using TRV²/TVI, a cutpoint value of 0.98 had a specificity of 86% and a sensitivity of 89% to predict PVR over 6 UW. Estimation of PVR with the previously published formula PVR = 5,19 TRV²/TVI – 0.4 was 100% correct to predict an PVR over 6 UW and 93% correct to predict an PVR under 3 UW.

Conclusion: Our results confirm the value of echocardiographic measurement of TRV²/TVI to evaluate PVR in a routine fashion for a non-selected population with high pulmonary pressure (figure next page).