079 "MITRACLIP, A SUMMIT WELL WORTH REACHING FOR". OUTCOME OF TRANSCATHETER MITRAL VALVE CLIP FOR THE MANAGEMENT OF MITRAL REGURGITATION IN HIGH RISK GROUP PATIENTS UNSUITABLE FOR SURGICAL INTERVENTION

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Introduction Mitral regurgitation (MR) can cause or complicate heart failure, affecting patient quality of life and survival. Surgical mitral valve (MV) repair or replacement is effective but associated with substantial morbidity and mortality in high-risk groups. MV clipping is a potential alternative, minimally invasive and lower risk procedure that has been shown to have similar benefits to surgical repair in randomised trials. The aim of this study was to assess the impact of the MitraClip procedure on MR and functional class in clinical practice in patients who were refused MV surgery.
Method Patient demographics including age, gender, cardiac / valve structure and function, aetiology of MR, duration of the procedure, complications, duration of hospital stay and the NYHA class pre and post procedure were recorded. SPSS statistical tool was used to analyse the data.
Results 30 patients unsuitable for surgery were referred to our centre. Twelve patients were excluded because of co-morbidities and were considered clinically inappropriate MitraClip (n=5; 42%) and/or because of unsuitable valve morphology (n=7; 58%). Of 18 patients who underwent the procedure, the mean age was 73 (range 58–85) years and 14 were men. MR was functional in 15 (83%), due to valve prolapse in 2 and degenerative valve disease in one. Two clips were deployed in four patients. The median procedure time was 227 min (123–380 min). Procedures longer than 2 h involved multiple clip application as well as coronary angioplasty. Mean hospital stay was 6 days (1–18 days) and 78% of patients were discharged within 1 week. Complications included failure to remove the catheter after the MitraClip application in one patient with severe pre-existing heart failure who later died of heart failure, bleeding from the groin and septicaemia. One patient who had been hospitalised for 5 months with severe heart failure died from pericardial tamponade most likely due to atrial wall damage. Among surviving patients, MR severity improved to grade I/II in 84%, to grade III in 16%. Of patients initially in NYHA IV, one died, one improved to NYHA I, one to NYHA III and one did not improve. Of 14 patients initially in NYHA III, Nine (62%) improved to NYHA I/II, one died and four did not improve substantially.
Conclusion In high-risk patients who have been declined mitral valve surgery, MV repair using the MitraClip procedure can be done with a moderate risk and with a high procedural success rate. Advances in expertise and technology will improve efficacy and patient safety.

080 SAFETY OF LMWH FOR BRIDGING ANTICOAGULATION BEFORE FULL WARFARINISATION IN MECHANICAL VALVES

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Objective Early following mechanical heart valve replacement, patients are at high risk of valve thrombosis and bleeding complications. There are no published guidelines on the use of specific bridging therapies until full warfarinisation is achieved. Use of unfractionated heparin (UFH) in these patients is widespread. We set out to examine the rate of thrombosis shortly after surgery until full anticoagulation was achieved in patients receiving low molecular weight heparin (LMWH).
Methods Prospectively collected data on 765 patients who had mechanical aortic or mitral valve replacement from January 2002 to September 2011 was reviewed. Of these patients 182 received intravenous or subcutaneous UFH due to co-morbidities and were excluded from our analysis. The remaining patients received 5000 units of Dalteparin (Pfizer) twice daily from the first postoperative day. Post operative echocardiography results were examined for valve thrombosis, and evidence of gastrointestinal or intracranial bleeds was sought. Anticoagulation was deemed therapeutic when the INR was >2.0.
Results 583 patients who had mechanical aortic or mitral valve replacement at our unit from January 2002 to September 2011 were included. These consisted of 451 aortic and 173 mitral valves. 59 patients had multiple valves replaced, including concomitant tricuspid replacement. 385 (66%) patients were male, the mean age was 56 years. There were 4 in-hospital deaths (0.69%). Mean time to reach therapeutic INR was 3.4 days. 18 (3.1%) patients had resternotomy for bleeding. No patients had evidence of valve thrombosis. There were no intracranial bleeds.
Conclusion Use of LMWH compares favourably with published series of bridging with UFH. There was a low incidence of postoperative bleeding complications and no evidence of valve thrombosis. Use of standardised dose of LMWH simplifies administration and there is no need for monitoring of the activated partial thromboplastin time.

081 ACUTE DERANGEMENT OF CARDIAC ENERGY METABOLISM AND OXYGENATION DURING STRESS IN HYPERTROPHIC CARDIOMYOPATHY—A POTENTIAL MECHANISM FOR SUDDEN CARDIAC DEATH

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Introduction Hypertrophic cardiomyopathy (HCM) is the commonest cause of sudden cardiac death in the young. The sarcomere mutations increase the energy cost of contraction and impaired resting energetics (phosphocreatine/adenosine triphosphate, PCr/ATP, measured by 31Phosphorus MR Spectroscopy, 31P MRS) has been shown. In addition, abnormal perfusion in HCM is an independent predictor of clinical deterioration and death. However, whether microvascular dysfunction is severe enough to result in deoxygenation has not been explored. We hypothesise: (1) Cardiac energetics are further impaired acutely during exercise in HCM, but not in normals or athletes (physiological hypertrophy); (2) This energetic abnormality contributes to diastolic impairment, is independent of the degree of hypertrophy or patchy fibrosis; (3) Tissue oxygenation during stress is impaired in HCM.
Methods 31P MRS (3T) was performed in 35 age and gender matched HCM patients, 12 athletes and 20 normal controls at rest and during 8 min of steady leg exercise lying prone with 2.5 kg weights. BOLD signal intensity change (SIÄ) and myocardial perfusion reserve index (MPRI) were measured with adenosine stress.
Results There was no difference in resting PCr/ATP between normals (2.14±0.36) and athletes (2.04±0.32, p=0.36). Resting PCr/ATP was significantly reduced in HCM (1.71±0.35, p<0.0001). During exercise there was a further reduction in PCr/ATP in HCM (1.56±0.31, p<0.05) but not in normals (2.13±0.34, p=0.98), or athletes (2.09±0.50, p=0.63). There was no correlation between cardiac mass, average wall thickness and rest or exercise energetics. Peak filling rates (PFR) were significantly reduced in HCM (rest:

HCM 572±176, normal 745±138 p=0.01; exercise HCM 648±191, normal 845±160 p=0.02). There was a significant correlation between PFR and PCr/ATP at both rest (rs=0.78, p=0.001) and exercise (rs=0.54, p=0.039). There was significantly reduced BOLD SIΔ response in HCM (10±11% vs normal, 18±14% and athletes 17±10%, p<0.0001) as well as MPRI (normal: 1.8±0.6; athletes: 2.0±0.9, HCM 1.3±0.6, p=0.001). There was a weak but significant correlation between BOLD SIΔ and MPRI (R=0.27, p<0.0001) and between BOLD SIΔ and end diastolic wall thickness (R=0.24, p<0.001). MPRI (β 0.2, p<0.001) and wall thickness (β −0.2, <0.001) are independent predictors of BOLD SIΔ. For β myosin heavy chain mutation cohort (n=12), there was a significant relationship between change in PCr/ATP and either BOLD SIΔ (R=0.48, p=0.05).

Conclusion During exercise, the pre-existing energetic deficit in HCM is further exacerbated, independent of hypertrophy. Additionally, oxygenation is blunted during stress. This may lead to acute derangement of energy dependent ion homeostasis during acute stress, resulting in ventricular arrhythmias. We offer a possible explanation for the high incidence of exercise related death in HCM and suggest that treatments that optimise energetics may be protective.

082 RIGHT VENTRICULAR HYPERTROPHY AND THE ATHLETE'S HEART: UTILITY OF THE ECG AS A SCREENING TOOL

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Introduction Right ventricular hypertrophy (RVH) is a manifestation of various congenital and acquired cardiopulmonary disorders which may lead to premature morbidity and mortality. Physiological RVH is also reported among healthy athletes. European (ESC) guidelines define ECG markers of RVH in young athletes as "uncommon and training-unrelated," warranting further investigation to exclude "pathological RV dilatation or hypertrophy." Conversely, recent American guidelines state that evidence is lacking to support such a strategy. There have been no studies to correlate ECG markers of RVH with imaging data in young athletes.

Methods 214 asymptomatic, elite athletes underwent ECG and transthoracic echocardiography. Sensitivity and specificity, as well as positive and negative predictive values (PPV and NPV) of published ECG criteria for RVH were assessed against echo findings (see Abstract 082 table 1). RV free wall thickness (RVWT) was measured in the subcostal plane as per ESC recommendations. RV end-diastolic area (RVEDA) was also calculated in each case.

Abstract 082 Table 1

	Prevalence (%)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
R:S(V1) >1	3.7	0.0	95.1	0.0	96.6
R:S(V5/V6) <1	1.4	0.0	98.6	0.0	96.7
R(V1) >7 mm	8.9	14.3	91.9	5.3	97.1
R(V1) + S(V5/V6) >10.5 mm	14.5	28.6	86.0	6.5	97.3
R′(V1) >10 mm	0.5	0.0	99.5	0.0	96.7
qR(V1)	0.0	0.0	100.0	0.0	96.7
Right axis deviation (>110°)	1.9	0.0	98.1	0.0	96.7
Right atrial enlargement (P-wave >2.5 mm)	0.9	0.0	99.0	0.0	96.7

Results Mean age was 21.4 years, 76.7% male. Mean RVWT was 3.8 mm (range 2–6 mm). Only 7/214 (3.3%) of athletes, all male, demonstrated RVH on echo (RVWT ≥6 mm). Inter- and intra-observer variability for RVWT measurements were 10% and 14% respectively. All ECG criteria for RVH had low sensitivity and PPV for echocardiographic RVH, although specificity and NPV were high. The Sokolow-Lyon voltage criterion for RVH (R(V1) + S(V5/6) >10.5 mm), which is specifically mentioned in the ESC guidelines, was seen in 14% of athletes. Mean RVEDA did not differ between athletes with RVH on ECG and those without (both groups 27.3 cm^2, p=1.0).

Conclusions Published ECG criteria have an unacceptably low correlation with echo evidence of RVH, which is rare in athletes. Adherence to current ESC guidelines would result in a large number of additional investigations, with the potential for undue distress, disruption to training, and inappropriate resource utilisation. Our data support American guidance that RVH voltage criteria violations should not prompt further investigation, which may have significant implications for the burden of testing required after ECG screening of British athletes.

083 ANNULO-APICAL ANGLES AND TAPSE TO RAPIDLY ASSESS RIGHT VENTRICULAR SYSTOLIC FUNCTION: A CARDIAC MAGNETIC RESONANCE STUDY

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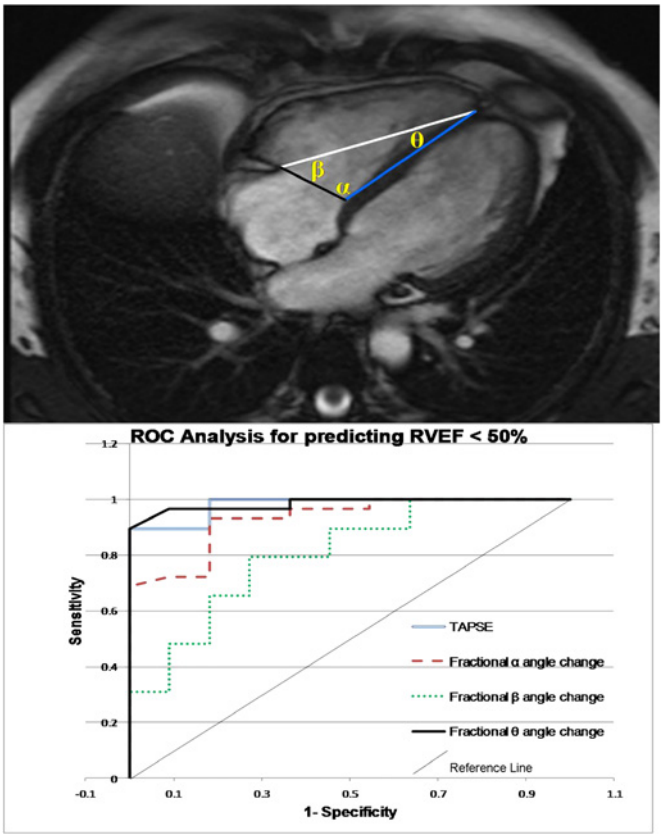
Background Volumetric assessment of the right ventricle (RV) by Cardiac Magnetic Resonance (CMR), albeit time-consuming, provides accurate and reproducible measurement of RV ejection fraction (RVEF). Tricuspid annulus peak systolic excursion (TAPSE)



Abstract 083 Figure 1 Top: AAAs in ED on a 4 chamber view. Bottom: ROC curve analysis.