Old Pathway The traditional stress/rest MPS pathway requires two imaging sessions either the same day or on two separate days according to the patient's Body Mass Index (BMI). For a 1 day protocol typically the injected activity is split in a 1:3 ratio between the first and second injections. For BMI <30, a total of 1125 MBq is administered. For BMI 30–35, a total of 1475 MBq is administered. For BMI>35, a total of 2000 MBq is administered 1:1. If the BMI is not provided by the requesting clinician then a letter is sent to the patient asking their height and weight.

New Pathway From September 2014 through December 2014, we introduced selective stress only imaging. A single high dose MIBI stress dose of of 800 MBq, was authorised for all patients, regardless of BMI. After stress imaging, a decision was made regarding the necessity of rest imaging.

Results Patients were referred from 10 hospitals. 407 patients were scanned. 55% were male and 45% female. Their average age was 66 years (+/-12.9). Their average BMI was 29.4 kg/m^2 (+/-6.4).

Of the total, 35% percent were normal at stress imaging and did not have rest imaging. Of the 65% who were abnormal, 60% of the combined rest/stress scans were interpreted as abnormal. As a result of this, the average time from request to appointment date for the stress scan reduced from 27 to 14 days.

The average time between stress scan and rest scan was 2.5 (range: 1–21 days). The average report turn around time from request to report for MPS was 5 days, but 97% of normal stress only scans were reported the same day. Furthermore, the administered radiation dose was lower for 67% of all patients.

In additional to scanning parameters being assessed, patients preferences were also assessed via a questionnaire. 49% expressed a preference for the test to be split across 2 days. 7% said either 1 day or 2 days. 20% of patients had to take a day off work to attend the hospital.

Conclusion Stress only MPS reduces radiation exposure, increases efficiency and thereby reduces cost.

123 THE DEVELOPMENT OF A DIETARY PREPARATION PROTOCOL FOR OPTIMAL INFLAMMATORY IMAGING USING 18F-FDG PET

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10.1136/heartjnl-2016-309890.123

Background Positron emission tomography (PET) performed utilising the glucose analogue and radiotracer 18F-FDG (FDG) to detect active inflammation, offers enormous potential for detecting disease presence and activity in myocarditis. However, the clinical utility of PET imaging is limited by the unpredictable nature of physiological FDG uptake within cardiac muscle. Methods to minimise physiological FDG uptake are necessary to depict active inflammation and to avoid avoid false negative and positive findings. Studies have suggested the use of fasting conditions to shift myocardial metabolism to primary free fatty acid (FFA) utilisation for energy and oxygen consumption to suppress physiological FDG uptake by normal myocardium. Specific dietary preparation regimens using a very high-fat content and low-carbohydrate diet prior to scanning have also been proposed, as has the administration of heparin to reduce FDG uptake through the activation of lipoprotein lipase (LPL) and hepatic lipase (HL) which enhances plasma lipolytic activity and leads to preferential free fatty acid consumption.

This study was therefore performed to develop an optimal dietary preparation protocol for imaging in PET/CT. We sought to establish whether patient preparation using heparin in addition to a high fat, low carbohydrate diet and prolonged fast prior to scanning ncreases the diagnostic capability of PET scanning in myocarditis.

Methods All patients referred for PET scans for the assessment of myocarditis were enrolled. We prospectively examined three preparation rationales to determine the optimal rationale for minimising physiological FDG-uptake. All subjects underwent strict preparation prior to scanning using one of the following protocols to enable comparison:

1. A 6-hour fast only, no dietary preparation.
2. Dietary preparation for 24 h prior to the scan with a high-fat content, low carbohydrate diet, followed by a prolonged fast for 12 h.
3. Dietary preparation for 24 h prior to the scan with a high-fat content, low carbohydrate diet, followed by a prolonged fast for 12 h and the administration of 50IU/kg of heparin prior to scanning.

The imaging findings were analysed by two independent and blinded clinicians. FDG uptake was categorised as none (complete suppression, no uptake = 0), focal (localised uptake, = 1), focal on diffuse (2) and diffuse (poor, failed suppression, = 4).

Results A total of 280 PET scans were performed. 36 patients underwent preparation using a 6-hour fast only, 128 underwent dietary preparation and a 12-hour fast, and 118 underwent dietary preparation followed by a 12-hour fast and the administration of heparin prior to scanning.

A 6-hour fast alone was associated with a 44% failure to suppress physiological metabolism – equivalent to almost one in every two scans being suboptimal or un-interpretable. In contrast, restriction of carbohydrate intake with high fat intake alongside a prolonged 12-hour fast was successful in supressing normal myocardial metabolism in 89% of subjects. Similarly, of those who also received heparin, there was a failure to suppress normal myocardial metabolism in 11% of subjects.

Conclusion Strict patient preparation prior to PET scanning is key to ensuring an optimal diagnostic disease assessment in myocarditis. A short duration of fasting of 6-hours or less alone is suboptimal in suppressing physiological uptake. In contrast, strict dietary preparation (using a high fat, low carbohydrate diet) and prolonged fast of > 12 h increases the diagnostic capability by 75%. Interestingly, the addition of heparin to this regimen provided no additive benefit suggesting that the addition of heparin adds little to the diagnostic quality of scans and is therefore unnecessary.

Valve disease/pericardial disease/ cardiomyopathy

124 THE USE OF NEXT GENERATION SEQUENCING TO DETERMINE GENOTYPE-PHENOTYPE CORRELATIONS IN DILATED CARDIOMYOPATHY

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10.1136/heartjnl-2016-309890.124

Background Dilated cardiomyopathy (DCM) is the leading cause of non-ischaemic heart failure. It causes approximately 10,000 deaths in Europe per year. Studies suggest that up to 50% of patients with DCM have a genetic predisposition and that mutations within one of 40 genes coding for structural and functional cardiac proteins accounts for up to 40% of familial disease. The relevance of genotype to clinical phenotype, treatment and prognosis is poorly understood. This study aimed to clinically and genetically characterise probands with familial DCM using next-generation sequencing technology (NGS) to determine specific genotype-phenotype correlations in DCM.

Methods 72 probands with DCM were consecutively recruited into this study within our centre. All received pre-test genetic counselling and provided written informed consent. All patients were clinically characterised using a standard phenotyping protocol. Genomic data isolated from peripheral blood lymphocytes using standardised protocols, was collected and screened for all major genes involved in cardiomyopathies and selected candidate genes using NGS. Eighty-six genes with 1528 exons representing 500kb of coding sequence, were studied using target enrichment methods (Agilent SureSelect System) followed by sequencing on the Illumina HiSeq 2000 platform. Clinical parameters were correlated with genotype findings.

Results Variant calling from 72 probands (male 54%, age 41 years (mean 40.7, range 14 to 68 years) generated 1415 exonic and splice-site calls. After filtering, 420 distinct candidate variants were reported, 253 of which were published pathogenic mutations, 112 of which were frameshift insertion/deletion or splice-site variants predicted to cause loss of function (thus likely to be pathogenic). A further 55 novel variants were considered potentially pathogenic on the basis of preliminary *in silico* analysis. Each proband, on average, carried 3 published pathogenic variants which included known mutations associated with DCM and other forms of cardiomyopathy. Up to two further variants predicted to be pathogenic were identified per DCM proband illustrating genetic heterogeneity. However, there was no simple correlation between a specific mutation or the number of mutations and the clinical phenotype. Moreover, there was no evidence of a 'poly-hit' theory of multiple mutations contributing to a worsened phenotype nor of specific variant demonstrating causality of phenotype suggesting that genotype-phenotype correlations in DCM are complex and may be influenced by both genetic and epigenetic factors.

Conclusion This study demonstrates the exciting potential for utilisation of next generation sequencing in routine clinical practice. However it highlights the genetic heterogeneity and high frequency of novel variants with uncertain effects on gene function in DCM. This presents considerable challenges for clinical interpretation. Large-scale phenotyping is therefore required to fully understand genotype-phenotype relations in DCM.

Imaging

125 DETERIORATION OF RIGHT VENTRICULAR FUNCTION ON EXERCISE DETECTED BY EXERCISE CARDIAC MAGNETIC RESONANCE IMAGING IN PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION

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10.1136/heartjnl-2016-309890.125

Introduction Right ventricular (RV) function is a prognostic factor in patients with idiopathic PAH (iPAH). However at diagnosis, no factors differentiate those who will progress to RV dysfunction at 1 year. RV dysfunction in disease becomes more manifest on exercise and there is interest in the capacity of the RV to augment during exercise and its relationship with outcomes. Cardiac magnetic resonance imaging (CMR) is an accurate method of evaluating cardiac function during exercise. We aimed to study the ability of CMR to detect changes in RV and left ventricular (LV) function during exercise in healthy controls (HC) and patients with iPAH and hypothesised that RV dysfunction in iPAH becomes more apparent on exercise. Methods: Resting and exercise CMR was carried out on 10 HC (6 women, average age 36 ± 12) and 10 patients with iPAH (8 women, average age 37 ± 9) using a Philips 1.5T Achieva (Best, Netherlands) with a 32-channel cardiac coil. Supine exercise was performed using an MR compatible ergometer (Lode, Netherlands) at 40% of the watts achieved on cardiopulmonary exercise testing. Free breathing, real time LV short axis stack images were acquired during rest and exercise. LV and RV volumes were analysed using cvi42 (Circle Cardiovascular Imaging, Canada) using conventional endocardial surface segmentation. The Wilcoxon signed rank test was used to compare within groups and the Mann Whitney U test for between group analysis. Results: Results are summarised in Table 1. HCs exercised at a mean of 81 ± 25W and patients at a mean of 44 ± 11W. In HCs, RV and LV stroke volume (SV) increased on exercise, driven by a decrease in RV and LV end systolic volume (ESV). LV and RV

Abstract 125 Table 1 Resting and Exercise cardiac parameters, values are expressed as mean ± standared deviation

	Healthy controls			iPAH			Mann Whitney U value	
	Rest	Exercise	p value	Rest	Exercise	p value	Rest	Exercise
Heart rate	60±9	120±12	0.005	63±13	114±18	0.005	0.791	0.427
LVEDV	139±23	146±24	0.059	116±20	110±23	0.093	0.041	0.003
LVESV	48±7	38±10	0.013	39±10	26±8	0.005	0.028	0.016
LVSV	90±16	109±16	0.005	77±13	83±17	0.047	0.07	0.005
LVEF	65±2	74±4	0.005	66±5	76±5	0.005	0.545	0.257
RVEDV	152±34	151±23	0.575	146±22	158±23	0.022	0.762	0.364
RVESV	68±19	50±17	0.007	66±14	77±18	0.059	1	0.004
RVSV	85±16	101±10	0.007	79±11	81±19	0.386	0.496	0.023
RVEF	56±4	68 ± 6	0.005	55±4	51±10	0.333	0.545	0.001