INTRODUCTION
Some cardiac abnormalities contain fat. Identification of this and its precise location can permit an accurate diagnosis to be made and appropriate patient management to be recommended. Cardiac MR (CMR) provides anatomical, functional and accurate tissue characterisation of these fat containing pathologies. Some of these abnormalities are discrete lesions such as lipomas. Other pathologies show a different form of fat distribution, such as Intracardial lipid accumulation in Fabry’s disease, and recently epicardial fat has been proposed as an indicator of risk for adverse cardiac events.

LIPOMAS
Accounting for approximately 10% of benign cardiac tumours, cardiac lipomas are encapsulated mature adipose cells. They are usually asymptomatic and are found incidentally at imaging and can be intra-cavitary, intra-myocardial or intra-epicardial. Due to their characteristic homogeneous mature lipid composition, they have characteristic MR appearances with fat signal identified on all sequences. This will predominantly result in uniform high signal on T1 (figure 2) and T2 imaging (figure 1). If there is intermediate signal intensity on T2 weighted images, confirmation of fat signal over sub-acute haemorrhage can be demonstrated through uniform signal suppression on fat saturated images (figure 3). No first pass or late gadolinium enhancement will be present.

LIPOMATOUS HYPERTROPHY OF THE INTERATRIAL SEPTUM
The lipomatous hypertrophy is not a true neoplasm, rather hypertrophy of pre-existing fat resulting in an increase in the transverse diameter of the inter-atrial septum exceeding 2cm. The cardiac MR signal will once again reflect the lipid composition, high signal on SSFP cine imaging (figure 4) and HASTE (figure 5). However this is not encapsulated, and will spare the fossa ovalis resulting in the typical “dumb-bell” shape within the inter-atrial septum. Once again, no first pass or late gadolinium enhancement should be present.

FATTY REPLACEMENT OF INFARCTS
Although the pathophysiology is not fully understood, as a result of either healing or remodelling following collagen deposition, myocytes differentiate into adipocytes with lipomatous metaplasia seen on histology. The myocardial fat related to infarct will often demonstrate thin linear or curvilinear high signal on T1 imaging (figure 6) with high signal also seen on the SSFP cine imaging with a characteristic “black boundary artefact” which indicates fat. The capability of MR to determine a specific mural location can be used with sub-endocardial most common although, mid-wall or sub-epicardial is often visible on MR where it is not seen on CT. The diagnosis is confirmed by the other features of myocardial infarction apparent on MR with myocardial thinning, regional wall motion abnormality and matched sub-endocardial or full thickness late gadolinium enhancement within a defined coronary territory. (Figure 7 T2W imaging, Figure 8, Same patient with 2T1W fat saturation).

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY
ARVC causes a combination of structural and functional abnormality of the right ventricle due to fibro-fatty replacement of normal myocytes. This is believed to be due to an abnormality of desmosomes, intercellular adhesion molecules. This structural disruption of the electrical pathways can lead to arrhythmias and may cause sudden cardiac death. This is a progressive disease with initial involvement of the right ventricular inflow tract, outflow tract or apex. This progresses to diffuse RV and LV changes over time resembling dilated cardiomyopathy. Initial diagnosis of ARVC is difficult with modified Major and Minor criteria based on clinical, ECG and imaging appearances developed to aid diagnosis. Although regional RV hypokinosis and RV dysfunction with preserved LVSF are included in the criteria, tissue characterisation on MR is not currently included, though this can help to make this diagnosis in early or subtle cases. MR appearances of fibro-fatty replacement can be seen on MR with high signal on T1W images (figure 9) demonstrated in clear contrast to the low signal myocardium and this process classically extends from the epicardium toward the endocardium. However, image quality must be high as differentiation from adjacent epicardial fat is extremely challenging. Accurate diagnosis needs to correlate with the presence of focal RV akinesia, aneurysm formation, RV dilatation with specific reductions in the calculation of the RV end-diastolic volume index and RV ejection fraction.

FABRY’S DISEASE
Fabry’s Disease is a glycolipid storage disease resulting in progressive intracellular accumulation of glycosphingolipids. Early diagnosis can change the course of the disease slowing the progression of left ventricular hypertrophy and subsequent heart failure. Routine CMR techniques will demonstrate myocardial hypertrophy which can be concentric (figure 10 black blood) or mimic the morphological subtypes of hypertrophic cardiomyopathy. In addition, there is a predilection for mid-myocardial late gadolinium enhancement in the basal inferolateral wall (figure 10). These standard imaging techniques may fail to detect lipid in the myocardium in Fabry’s Disease. However T1 mapping (figure 12) has been shown to demonstrate significantly shortened myocardial T1 values (normal range 950-1050 msec) in patients with intracellular fat deposition and genetically proven Fabry disease.

CONCLUSION
The full range of CMR imaging sequences, including the recently developed sequences of T1 mapping, permits an accurate tissue characterisation of these abnormalities which in cases can reduce the need for invasive diagnostic procedures.

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Cardiac MR Assessment of Lipid Containing Pathologies
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