tachyarrhythmias in HCM with regard to DE on contrast-enhanced CMR.

Methods: A total of 177 HCM patients (age 41 ± 16 years; 95% asymptomatic or mildly symptomatic) were evaluated by 24-hour ambulatory Holter ECG and contrast-enhanced CMR.

Results: A total of 72 patients (41%) had DE (transmural in 39/72), occupying 8.5±7.8 percent of the LV wall. Presence of premature ventricular contractions (PVCs), couplets and nonsustained ventricular tachycardia (NSVT) were more common in patients with DE than those without DE (PVCs: 89% *vs.* 72%; couplets: 40% *vs.* 17%; NSVT: 28% *vs.* 4%; p < 0.0001-0.007). Patients with DE also had greater numbers of PVCs (202 ± 655 *vs.* 116 ± 435), couplets (1.9 ± 5 *vs.* 1.2 ± 10), and NSVT runs (0.4 ± 0.8 *vs.* 0.06 ± 0.4) than did non-DE patients (all p<0.0001); DE was an independent predictor of NSVT (relative risk 7.3, 95% CI 2.6 - 20.4; p<0.0001). Of note, however, extent (%) of DE was similar in patients with and without PVCs (8.2% *vs.* 9.1%; p=0.93), couplets (8.5% *vs.* 8.4%; p=0.99) and NSVT (8.3% *vs.* 8.5%; p=0.35).

Conclusions: In this large HCM cohort with no or only mild symptoms, myocardial fibrosis detected by CMR was associated with greater likelihood and increased frequency of ventricular tachyarrhythmias (including NSVT) on ambulatory Holter ECG. Therefore, contrast-enhanced CMR identifies HCM patients having increased susceptibility to ventricular tachyarrhythmias with implications for sudden death risk stratification.

In this large HCM cohort, myocardial fibrosis detected by CMR was associated with greater likelihood of ventricular tachyarrhythmias on ambulatory Holter ECG. Therefore, CMR identifies HCM patients having increased susceptibility to ventricular tachyarrhythmias with implications for sudden death risk stratification.

### 234. A NOVEL IN VIVO MARKER FOR ISCHEMIC TISSUE INJURY EARLY AFTER CORONARY OCCLUSION

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Background: Early identification of acute myocardial ischemia is a diagnostic challenge. We aimed at identifying the earliest time point at which T2-weighted cardiovascular magnetic resonance imaging could visually identify acute ischemia.

Methods: We studied seven dogs with serial T2-weighted and cine imaging at baseline, during and early after transient coronary occlusion (25-35 minutes) in a 1.5 T MRI system. Late gadolinium enhancement was used to assess irreversible injury.

Results: 28±4 minutes after experimental coronary artery occlusion, we observed a transmural area of high T2 signal intensity (contrast to noise ratio to remote myocardium 11.0±10; p<0.0001), matching areas with new onset regional wall motion abnormalities. Late enhancement imaging performed after reperfusion did not show irreversible injury in any of the dogs.

Conclusion: We provide the first preliminary evidence that T2-weighted CMR imaging represents a novel in vivo marker for ischemic tissue injury likely before the onset of irreversible injury. T2-weighted CMR may offer a novel potential means of identifying acute ischemia in acute coronary syndromes.
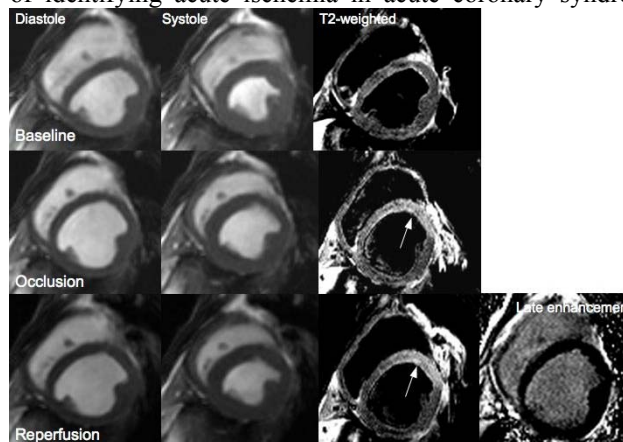
We aimed at identifying the earliest time point at which T2-weighted imaging visually identifies acute ischemia in a dog model(n=7).T2 imaging detected ischemia 28±4 minutes after coronary occlusion before the onset of irreversible damage as identified by late gadolinium enhancement

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235. SIMULTANEOUS MYOCARDIAL FIRST-PASS PERFUSION AND STRAIN IMAGING WITH DENSE

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Introduction: We describe a new sequence capable of measuring both perfusion and myocardial strain using DENSE(1) in a single scan. The images possess dark-blood

contrast and upon infusion of Gd-DTPA, ischemic areas remain bright while normal areas become dark. The phase maps of the same images provide displacement and strain maps, which facilitates the correlation of function and perfusion. This technique should enhance the diagnostic value of time-limited stress tests.

Methods: Scans were performed on a 1.5T Siemens Avanto scanner. In anesthetized Yorkshire pigs (n = 2) the diagonal branch of the LAD was ligated for 90 minutes to induce ischemia, and then released for reperfusion. Perfusion-strain scans were performed before and during ischemia and after reperfusion with 3 separate IV infusions of 0.1 mmole/kg Gd-DTPA (Magnevist) at 3.0 ml/sec. A multi-slice, single-shot DENSE sequence with true-FISP readout was used to acquire 4 short-axis slices in a heartbeat (Fig.1). The most basal slice was acquired first without refocusing the encoding moments, and provided a saturation-recovery type of LV cavity blood signal for estimating the contrast input function(2,3).Gradually ramped flip-angles through the readout train equalized the echo amplitudes. Imaging parameters were true-FISP echo spacing of 2.5 ms, matrix size of 128×40, FOV of 512×120mm with restricted excitation(4) (equivalent to a matrix size of 128×96 and 3/4 phase-encode FOV), slice thickness of 6-8mm. A data set contained 360 images of 4(slices)×3(encoding directions)×30(repetitions). The 3 encoding directions combined in-plane encoding of 4.0 mm/radian in-plane and through-slice of 1.0mm/radian. Each direction was acquired in a separate heartbeat. The interval between scanned heartbeats was spaced over 2.0 sec to allow spin recovery.

The scan lasted 3 - 4 minutes during which the contrast was given. Due to T_1 relaxation between displacement-encoding and slice acquisition (200 to 400ms for the 3 perfusion slices), normal myocardium became dark upon Gd infusion while the ischemic region remained bright. Perfusion and strain were measured in the other 3 slices. All images were registered with deformable registration to remove respiratory motion. Pixel-wise perfusion maps were obtained with Marquardt-Levenberg fitting using the Fermi transfer function(5). Circumferential strain maps were obtained from each set of 3 encoding directions(4), and then averaged over the 30 sets.

Results: The myocardial time-intensity curves of a normal (anterior) and an ischemic (inferolateral) segment are shown in Fig.2 during occlusion, with representative images. The quantitative perfusion (in 0 - 2 ml/g/min scale) and strain (in 0 - 0.2 scale) of the 3 slices of a pig before, during ischemia and after reperfusion are shown in Fig.3. Reperfusion resulted in partial recovery of flow, and recovery of function in the periphery of the ischemic zones.

Discussion: To our knowledge this approach is unique because of the positive contrast unlike conventional saturation-recovery imaging, and therefore may not have

the associated artifacts such as dark rims. A limitation is the lower temporal resolution needed for spin recovery. However, at 3ml/sec injection rate this did not affect the perfusion measurements in this study. Future work will include comparison with microsphere flow measurements.

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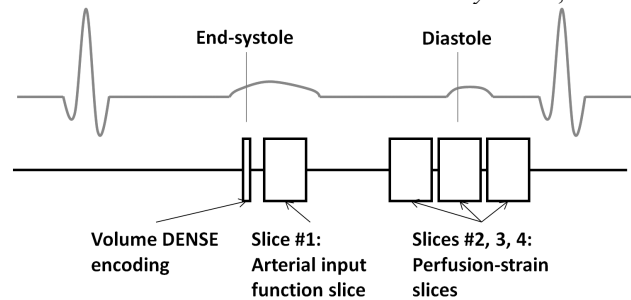
Figure 1 Timing diagram of the acquisition of the 4 slices in a heartbeat.
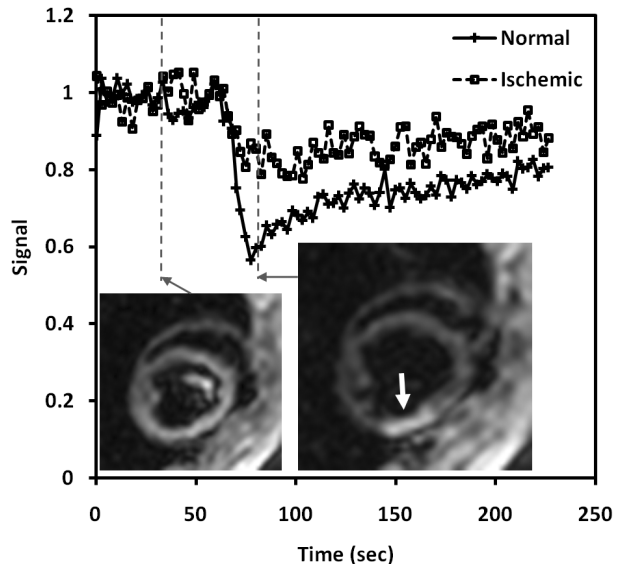
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Figure 2 Time-intensity curves of normal and ischemic segments during occlusion and images before and after Gd infusion.