

# Overview of Delayed Myocardial Enhancement in Cardiac MRI

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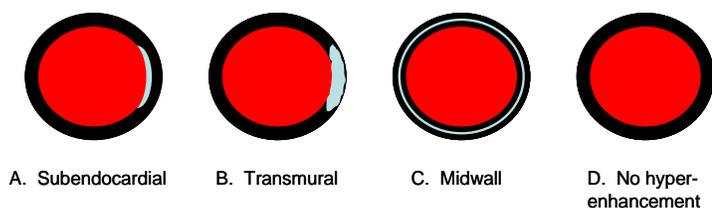
## Background:

Although MRI has long been used for cardiac imaging, significant improvements in MRI scanners for cardiac specific pulse sequences have only been commercially available since the late 1990's. Cardiac MRI is a complex and difficult examination that needs not only state-of-the-art MRI scanners, but also staff that is experienced with performing cardiac MRI studies. The main advantages of cardiac MRI are that it can provide information on myocardial morphology, function, blood flow, myocardial perfusion, and myocardial injury or infiltrative/fibrotic changes in a non-invasive examination during a single session [1]. Delayed myocardial enhancement (MDE) imaging takes advantage of the properties of the Gadolinium (Gd)-chelates to help define certain pathologies. This abstract will address the main points to be considered when performing a contrast enhanced cardiac MR study with MDE.

## Teaching Points:

Gd-chelates can be used for first-pass perfusion of the myocardium, and they can also be used for delayed enhancement evaluation. Starting with the former, Gd-chelate perfusion MRI can be performed at rest, or with stress. Pharmacological stress using adenosine is the most commonly performed cardiac MRI perfusion analysis procedure [2]. The patient is stressed with a two to three-minute adenosine infusion at 140 ug/kg/min and then a short axis fast steady state gradient echo cine sequence is acquired in a multi-phase fashion for over one minute during a dynamic injection of 1 mmol/kg Gd-chelate, infused at 3 to 5 mL/sec. Perfusion defects will appear as dark areas within the normally enhancing myocardium because areas where the myocardium is damaged will take up the contrast agent more slowly. At approximately 8 to 10 minutes after the Gd-chelate injection, a higher resolution, using a segmented inversion recovery technique is then acquired in two planes, usually the short axis and the long axis of the heart. Hyperintense areas will appear in the myocardium where necrosis, scar, fibrosis, or infections occur because they retain the contrast agent longer than healthy myocardium [3-8]. The combination of perfusion imaging and MDE can help to evaluate which areas of the myocardium are still viable.

Hyper-enhancement patterns can be found with different types of pathology. Ischemic heart disease typically manifests as a subendocardial region of hyper-enhancement, which if extensive, may become transmural, but it is specific to vascular distribution [5]. Figure 1 depicts a few of the patterns often seen on MDE. The finding of hyper-enhancement within the wall of the myocardium can be significant and have an impact on the course of a patient's care. Technologists need to make sure that the MDE images are the best possible quality by educating the patient on the cardiac MDE procedure and through understanding the MDE protocol.



**Figure 1.** Myocardial delayed enhancement patterns vary by type of pathology. Ischemic heart disease is commonly associated with subendocardial (A) and transmural (B) types of enhancement patterns. Mid-wall or central enhancement (C) is often seen in late stages of heart failure or dilated cardiomyopathy. No hyper-enhancement (D) is seen in both healthy tissue and diffuse fibrosis.

## Summary:

Cardiac MRI with MDE is technique that can provide important information about the state of a patient's myocardium. The administration of Gd-chelates can help detect perfusion in rest or stress, and then also provide delayed hyper-enhancement information after a short waiting period. The patterns of delayed hyper-enhancement help discern the type of pathology that may help contribute to optimal patient care.

## References:

- 1). Kramer CM. *Cardiol Clin* 1998;16(2):267-276.
- 2). Gudmundsson P, Winter R, Dencker M, et al. *Clin Physiol Funct Imaging* 2006;26(1):32-38.
- 3). Stork A, Muellerleile K, Bansmann PM, et al. *Eur Radiol* 2007;17(3):610-617.
- 4). Knopp MV, Schoenberg SO, Rehm C, et al. *Invest Radiol* 2002;37(12):706-715.
- 5). McCrohon JA, Moon JC, Prasad SK, et al. *Circulation* 2003;108(1):54-59.
- 6). Vohringer M, Mahrholdt H, Yilmaz A, Sechtem U. *Herz* 2007;32(2):129-137.
- 7). Mahrholdt H, Wagner A, Judd RM, Sechtem U, Kim RJ. *Eur Heart J* 2005;26(15):1461-1474.
- 8). Weinsaft JW, Klem I, Judd RM. *Cardiol Clin* 2007;25(1):35-56, v.