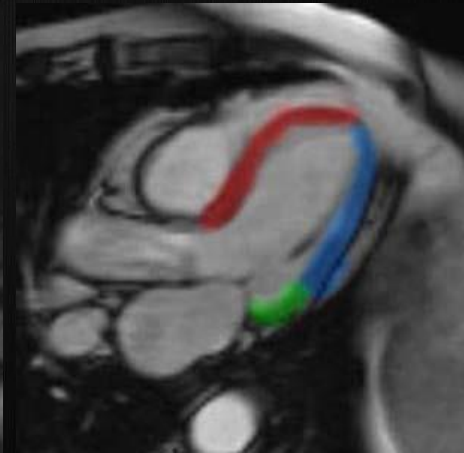
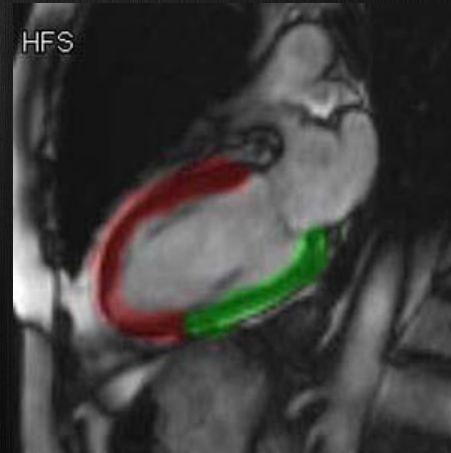
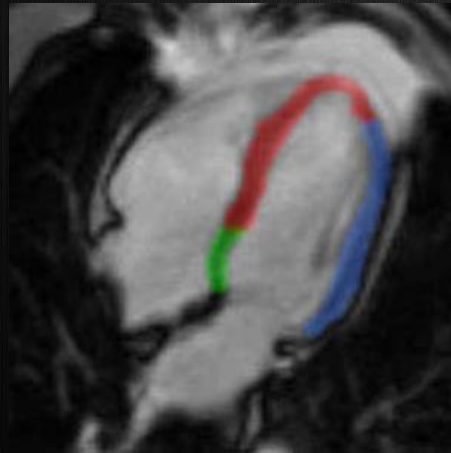
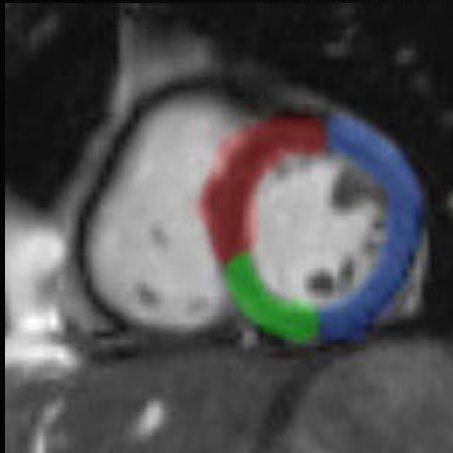


# Ischemia

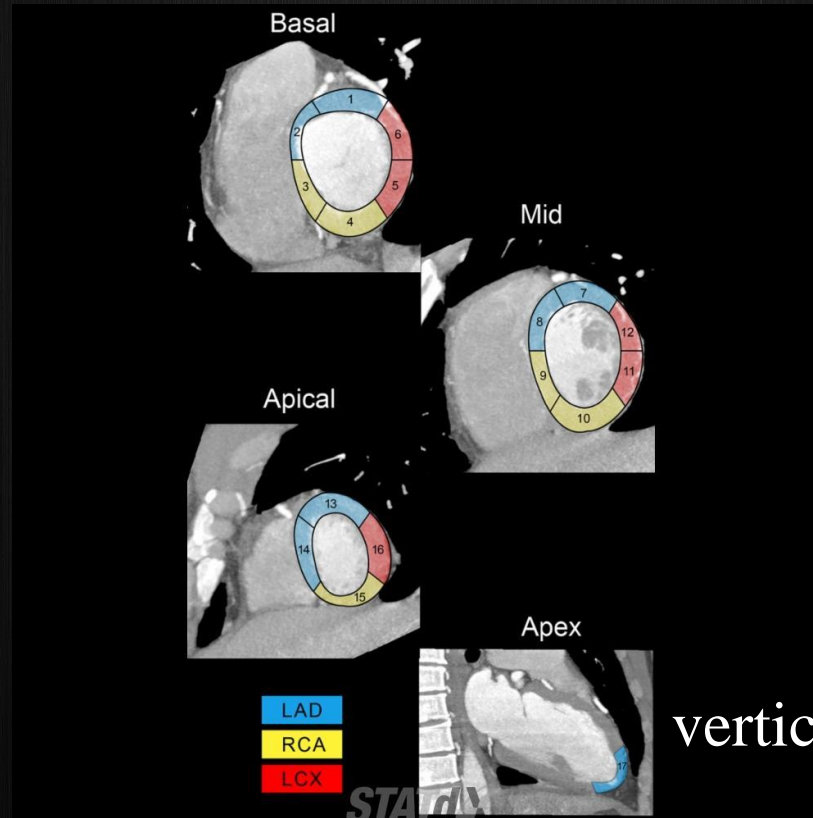
- At core of infarct, may see microvascular obstruction surrounded by infarct. Gad cannot reach it. Worse prognosis.
- Ischemia: bright T2, low perfusion, hypokinesia, normal DE
- Infarct: bright DE, acute if T2edema and normal wall thickness

# Coronary Artery Perfusion Territories

- Left anterior descending artery (LAD)
- Left circumflex artery (LCx)
- Right coronary artery (RCA)



# Enhancement patterns



- It should be emphasized, however, that great variation in the distribution of coronary blood flow is observed in clinical practice.

SHORT AXIS



Apical



X = 16

LAD

RCA

LCX

- Graphic illustrations of the left ventricular myocardial segments and the distribution of coronary blood flow to these segments.

- Segmentation of left ventricular myocardium is used to assess myocardial perfusion, left ventricular function and coronary anatomy.

- Myocardial perfusion and function can be assessed using nuclear medicine cardiac SPECT and cardiac MR

VERTICAL  
LONG AXIS



STAT

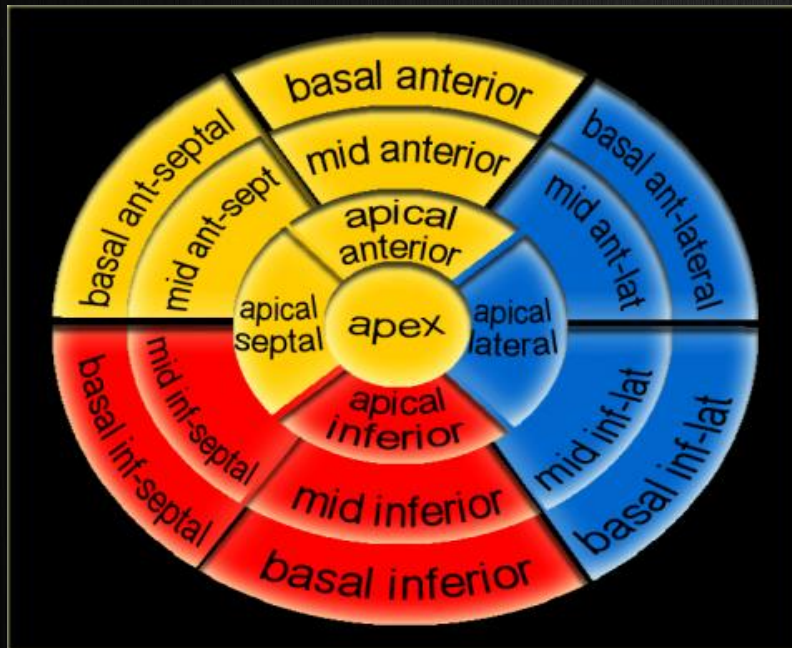




## 17 segments model

Myocardial segments with abnormal enhancement or wall motion disturbances are named and localized according to the 17 segments model of the American

Heart Association (37). Individual myocardial segments can be assigned to the 3 major coronary arteries with the recognition that there is anatomic variability.



## 17 segments model

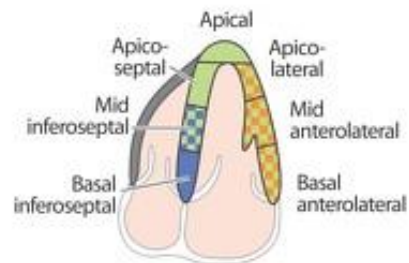
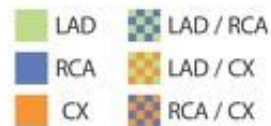
1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral

- mid anterior
- mid anteroseptal
- mid inferoseptal
- mid inferior
- mid inferolateral
- mid anterolateral

- apical anterior
- apical septal
- apical inferior
- apical lateral

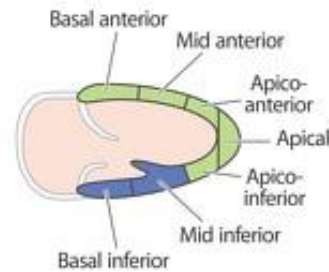
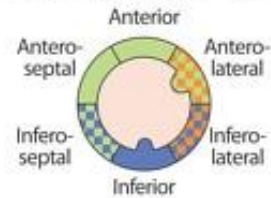
- apex

## Coronary vessel plot



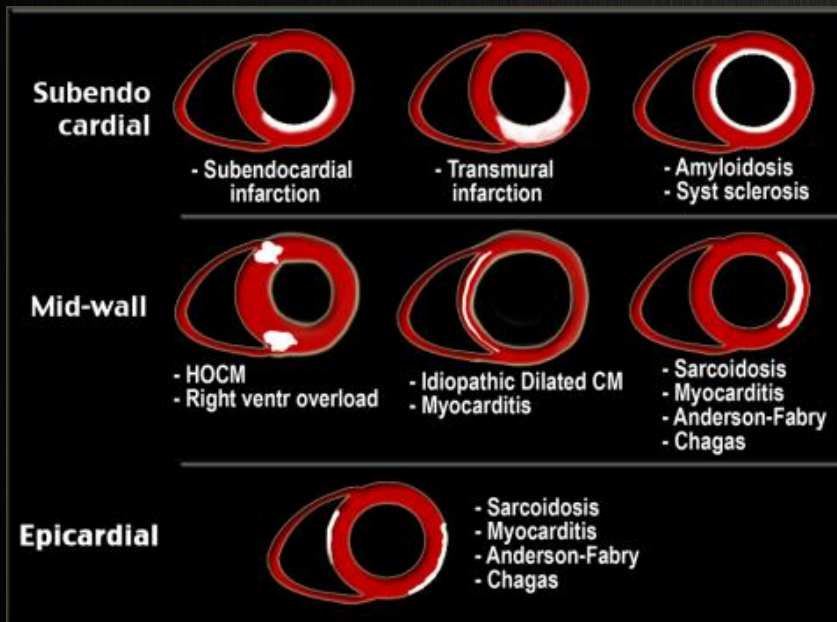
**Horizontal  
long axis (HLA)**

## Short axis (SA)



**Vertical  
long axis (VLA)**

# Enhancement patterns



In normal myocardium there will be early wash out of contrast.

In injured myocardium the wash out is very slow resulting in delayed enhancement after 10 - 15 minutes compared to the normal myocardium.

Delayed enhancement of myocardial tissue is seen in many pathophysiologic scenarios:

- Retention of contrast material by fibrous tissue
- Increased extravascular space
- Inflammation
- Tumor neovasculature in primary and secondary tumors



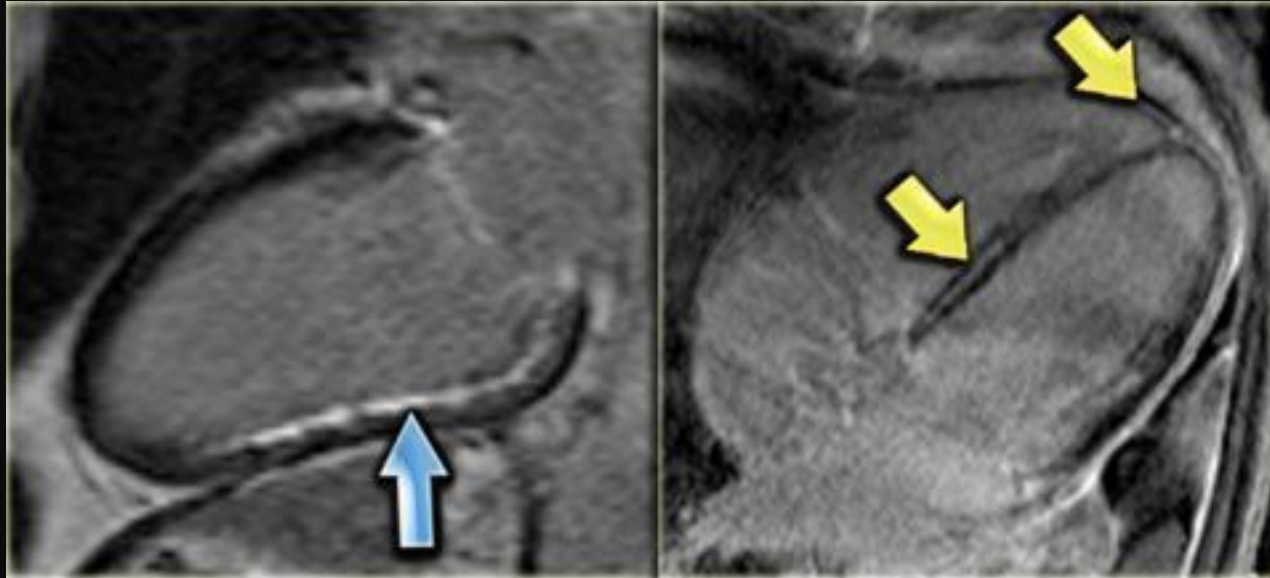
# Ischemic versus non-ischemic

## ■ *Ischemic CM*

- is defined as dysfunction of the left ventricle as a result of a chronic lack of oxygen due to coronary artery disease.
- Delayed enhancement MR images will show fibrosis, which appears as high signal intensity in an area of coronary artery distribution.
- Since all infarctions start subendocardially and may progress to transmural, the subendocardial region is always involved.

## ■ *Non-ischemic CM*

- has a variable etiology, i.e. genetic, toxic, metabolic, infectious and idiopathic.
- In nonischemic myocardial disease the delayed enhancement usually does not occur in a coronary artery distribution and is often midwall or epicardial rather than subendocardial or transmural.



Long axis late enhancement image in a patient with an inferior wall infarction with subendocardial enhancement in the territory of the right coronary artery RIGHT:

4-chamber late enhancement image in a patient with idiopathic dilated cardiomyopathy with midmyocardial enhancement

# Infarction and delayed enhancement

- Infarcted myocardium is bright on late-enhancement images. All patients with ischemic cardiomyopathy demonstrate delayed enhancement in a typical 'CAD' pattern, one in which the subendocardium is always involved.
- When a coronary artery is occluded the infarction always starts subendocardially and progresses towards the epicardium depending on the duration of the occlusion [6].
- Both acute and chronic infarctions enhance. In acute infarctions the contrast enters the damaged myocardial cells due to myocyte membrane disruption.
- In chronic infarctions the late enhancement is a result of retention of contrast material in the large interstitial space between the collagen fibers in the fibrotic tissue [7].

# No reflow phenomenon

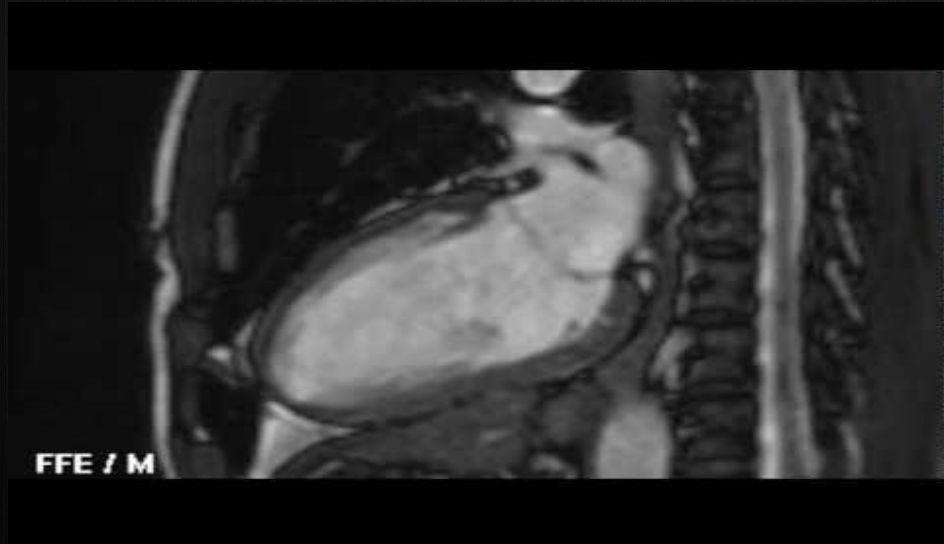
- No reflow phenomenon is the failure of blood to reperfuse an ischemic area after the physical obstruction has been removed or bypassed.
- No reflow zones are identified on late-enhancement images as a dark core surrounded by an enhancing rim.
- This finding indicates the presence of damaged microvasculature in the core of an area of infarction
- The presence of a 'no reflow' zone is associated with worse functional outcome, larger infarcts and adverse clinical outcome [8,9].
- Both acute and chronic infarctions demonstrate delayed-enhancement, but an acute infarction can often be distinguished by the presence of a 'no reflow' zone and high signal on T2 weighted images.



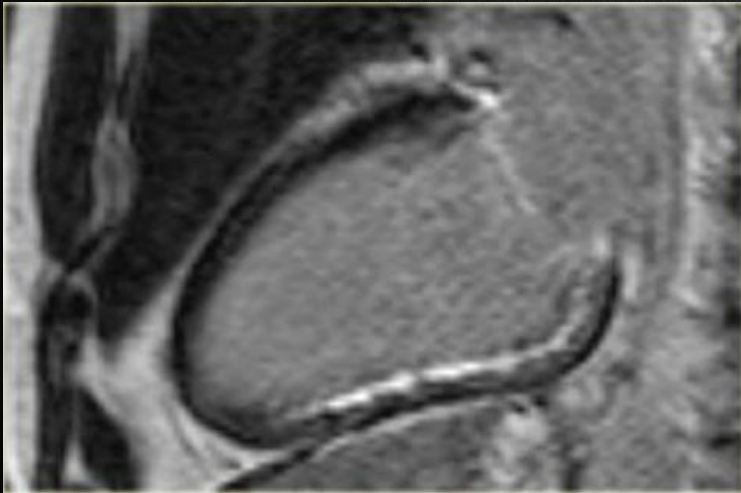
# Stunning

- Cine imaging in combination with delayed-enhancement MR allows identification of:
- *Myocardial stunning* following acute myocardial infarction and
- *Hibernating myocardium* in the setting of chronic ischemic heart disease.
- Stunning is defined as postischemic myocardial dysfunction that persists despite restoration of normal blood flow.  
Over time there can be a gradual return of contractile function depending on the transmural extent of the ischemia [10].
- If the degree of transmural extent as seen on the delayed enhancement images is less than 50%, the myocardial function is likely to recover [11].
- On the left a long axis cine 6 days after revascularization of an acute inferior wall infarction.  
First study the video and then continue reading.
- There is good contraction of the normal anterior wall
- Despite the revascularization there is hypokinesia of the inferior wall.
- Continue with the delayed enhancement image.

# Stunning



# Stunning



Long axis late enhancement image 6 days after revascularized acute inferior wall infarction

On the left the long axis delayed enhancement image of the same patient. There is less than 50% enhancement of the myocardium.

This is a good prognostic sign and we can expect a restoration of some of the contractile function.

Continue with the cine-view four months later.

# Hibernation

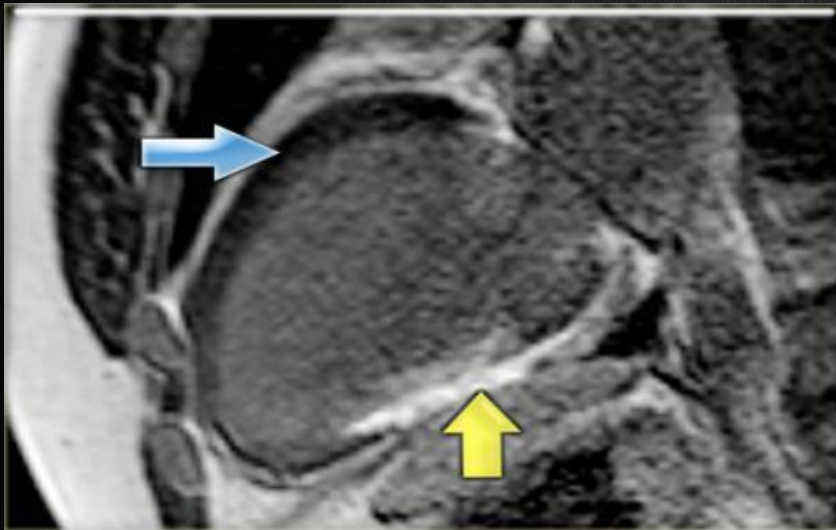
- Hibernation is a state in which some segments of the myocardium exhibit abnormalities of contractile function at rest [10].
- This phenomenon is highly significant clinically because it usually manifests itself in the setting of chronic ischemia, that is potentially reversible by revascularization. The reduced coronary blood flow causes the myocytes to enter a low-energy 'sleep mode' to conserve energy.
- There is an inverse relationship between the transmural extent of hyperenhancement, and the likelihood of wall motion recovery following revascularization.  
If the transmural extent of late enhancement is less than 50% the function is likely to improve after revascularization [12].
- On the left long axis cine-images of a patient with a severe stenosis of the LAD.  
First study the video and then continue reading.

The cine images show:

- Hypokinesia of the anterior wall.
- Hypokinesia of the inferior wall.
- Poor ejection fraction: 17%.  
Continue with the late enhancement image.



# Hibernation

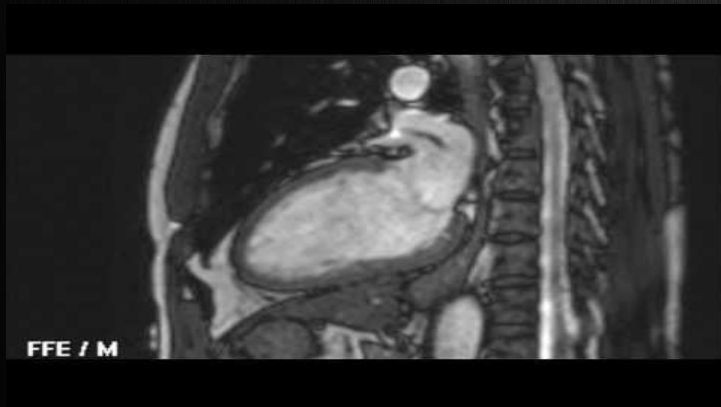


Hibernation of the anterior wall (blue arrow) and old transmurular inferior wall infarction (yellow arrow).

O

- On the left the long axis late enhancement image in the same patient. Notice the following:
- Transmurular enhancement of the inferior wall, which can be diagnosed as an old infarction (yellow arrow)
- No enhancement of the hypokinetic anterior wall (blue arrow). So it can be concluded, that this is probably the result of hibernation.

# Hibernation



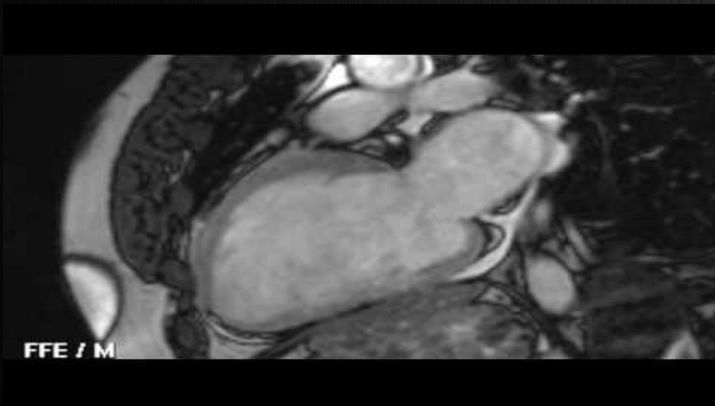
On the left the same patient four months after the inferior infarction and revascularization. First study the video and then continue reading.

The long axis cine shows improved function of the inferior wall.

Now it can be concluded that the hypokinesia was due to stunning.

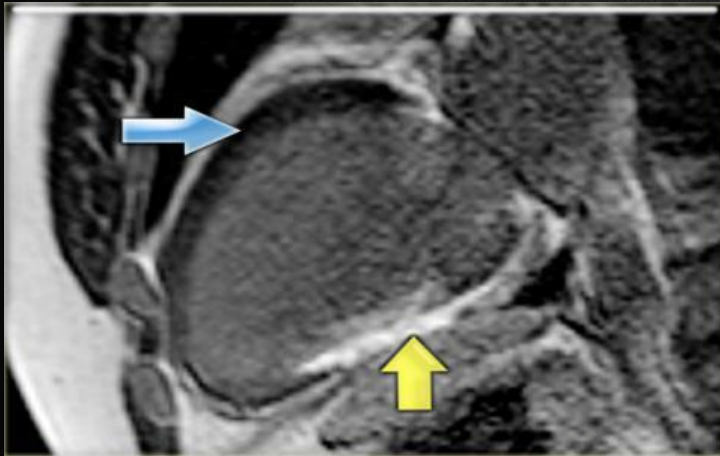
Myocardial regions that demonstrate little or no evidence of hyperenhancement (i.e. infarction)

# Hibernation



- Hibernation is a state in which some segments of the myocardium exhibit abnormalities of contractile function at rest [10].
  - This phenomenon is highly significant clinically because it usually manifests itself in the setting of chronic ischemia, that is potentially reversible by revascularization.
  - The reduced coronary blood flow causes the myocytes to enter a low-energy 'sleep mode' to conserve energy.
  - There is an inverse relationship between the transmural extent of hyperenhancement, and the likelihood of wall motion recovery following revascularization.
  - If the transmural extent of late enhancement is less than 50% the function is likely to improve after revascularization [12].
  - On the left long axis cine-images of a patient with a severe stenosis of the LAD.  
First study the video and then continue reading.
- The cine images show:
- Hypokinesia of the anterior wall.
  - Hypokinesia of the inferior wall.
  - Poor ejection fraction: 17%.  
Continue with the late enhancement image.

# Hibernation



Hibernation of the anterior wall (blue arrow) and old transmural inferior wall infarction (yellow arrow).

On the left the long axis late enhancement image in the same patient.

Noice the following:

- Transmural enhancement of the inferior wall, which can be diagnosed as an old infarction (yellow arrow)
- No enhancement of the hypokinetic anterior wall (blue arrow).

So it can be concluded, that this is probably the result of hibernation.