MRI for Technologists

4712-301 Cardiac MRI

PROGRAM INFORMATION

MRI for Technologists is a training program designed to meet the needs of radiologic technologists entering or working in the field of magnetic resonance imaging (MRI). These units are designed to augment classroom instruction and on-site training for radiologic technology students and professionals planning to take the review board examinations, as well as to provide a review for those looking to refresh their knowledge base in MR imaging.

All movies contained within this content can be viewed by clicking on the link provided or by going to www.YouTube.com/ICPMEducation

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Review Date: **June 2018**
Expiration Date: **May 1, 2020**

This material will be reviewed for continued accuracy and relevance. Please go to www.icpme.us for up-to-date information regarding current expiration dates.

OVERVIEW

Cardiac MRI introduces the learner to the anatomy and function of the heart and the mechanism by which blood is circulated through the heart. This unit will discuss timing methods that allow images to be acquired with the patient's cardiac rhythm. Primary pulse sequences and planes of acquisition are reviewed. Common acquisition protocols are provided at the end of the material.

After completing this educational material, the reader should be able to:

- Explain the unique challenges of imaging the heart
- Identify the basic anatomy of the heart and great vessels
- Explain cardiac gating principles and patient safety and setup
- Discuss the principles of the primary pulse sequences and image contrast
- Describe the principles of bright-blood and black-blood pulse sequences
- Demonstrate proper planes of acquisition for obtaining standard cardiac views
- Explain common applications of cardiac MR imaging
- Apply common acquisition protocols

Thomas Schrack, BS, ARMRIT
Manager, MR Education and Technical Development
Fairfax Radiological Consultants
Fairfax, VA

Currently serving as Manager of MR Education and Technical Development at Fairfax Radiological Consultants in Fairfax, VA, Thomas Schrack served as Adjunct Faculty Instructor for Northern Virginia Community College from more than 10 years, teaching MR physics and clinical procedures. He serves on the Board of Examiners of the American Registry of Magnetic Resonance Imaging Technologists (ARMRIT) and in 2013 was elected to the Board of Directors. Mr. Schrack is also the Co-Founder and Program Director of the Tesla Institute of MRI Technology, a school offering certification in MRI for radiologic technologists and others interested in entering the field of MRI.

Mr. Schrack is the author of *Echo Planar Imaging: An Applications Guide,* GE Healthcare, 1996, and contributing author, *Magnetic Resonance Imaging in Orthopaedics & Sports Medicine* with David Stroller, MD, 1997. Working with International Center for Postgraduate Medical Education, Mr. Schrack has authored or co-authored several units of the *MRI for Technologists* series, including *MRI Systems and Coil Technology, MR Image Postprocessing and Artifacts, Patient and Facility Safety in MRI, MRI Contrast Agent Safety, Advanced MRI Neurological Applications, MRI of the Brain and Spine, Musculoskeletal MRI, Clinical Magnetic Resonance Angiography, MRI of the Body, and Cardiac MRI.*

Mr. Schrack is a graduate of The Pittsburgh NMR Institute, James Madison University, and Northern Virginia Community College.

EDUCATIONAL CREDIT

This program has been approved by the American Society of Radiologic Technologists (ASRT) for 3.5 hours ARRT Category A continuing education credit.

HOW TO RECEIVE CREDIT

Estimated time to complete this activity is 3.5 hours. The posttest and evaluation are required to receive credit and must be completed online.

- In order to access the posttest and evaluation, enroll in the online course at icpme.us
- Read the entire activity either online or download and print the pdf.
- Log in to your account at icpme.us to complete the posttest and evaluation, accessible through the course link in your account.
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FDA Drug Safety Communication: FDA warns that gadolinium-based contrast agents (GBCAs) are retained in the body; requires new class warnings

https://www.fda.gov/Drugs/DrugSafety/ucm589213.htm Accessed June 14, 2018.

05-16-2018 Update

In addition to approving the updated prescribing information concerning the gadolinium retention safety issues described in the Drug Safety Communication below, FDA has also approved new patient Medication Guides for all GBCAs.

Health care professionals and patients can access the patient Medication Guides according to the GBCA drug name* on the Medication Guides webpage, or the latest prescribing information by searching in Drugs@FDA.

All MRI centers should provide a Medication Guide the first time an outpatient receives a GBCA injection or when the information is substantially changed. In general, hospital inpatients are not required to receive a Medication Guide unless the patient or caregiver requests it. A health care professional who determines that it is not in a patient's best interest to receive a Medication Guide because of significant concerns about its effects may direct that it not be provided to that patient; however, the Medication Guide should be provided to any patient who requests the information.†

*The brand names of the GBCAs can be found in Table 1 below.
†For more information on distribution of Medication Guides, see the Guidance Document, the Drug Info Rounds Video, or the Code of Federal Regulations at 21 CFR 208.26.

This is an update to the FDA Drug Safety Communication: FDA identifies no harmful effects to date with brain retention of gadolinium-based contrast agents for MRIs; review to continue issued on May 22, 2017.

12-19-2017 Safety Announcement

The U.S. Food and Drug Administration (FDA) is requiring a new class warning and other safety measures for all gadolinium-based contrast agents (GBCAs) for magnetic resonance imaging (MRI) concerning gadolinium remaining in patients' bodies, including the brain, for months to years after receiving these drugs. Gadolinium retention has not been directly linked to adverse health effects in patients with normal kidney function, and we have concluded that the benefit of all approved GBCAs continues to outweigh any potential risks.

However, after additional review and consultation with the Medical Imaging Drugs Advisory Committee, we are requiring several actions to alert health care professionals and patients about gadolinium retention after an MRI using a GBCA, and actions that can help minimize problems. These include requiring a new patient Medication Guide*, providing educational information that every patient will be asked to read before receiving a GBCA. We are also requiring manufacturers of GBCAs to conduct human and animal studies to further assess the safety of these contrast agents.

GBCAs are used with medical imaging devices called MRI scanners to examine the body for problems such as cancer, infections, or bleeding. GBCAs contain gadolinium, a heavy metal. These contrast agents are injected into a vein to improve visualization of internal organs, blood vessels, and tissues during an MRI, which helps health care professionals diagnose medical conditions. After being administered, GBCAs are mostly eliminated from the body through the kidneys. However, trace amounts of gadolinium may stay in the body long-term. Many GBCAs have been on the market for more than a decade.

Health care professionals should consider the retention characteristics of each agent when choosing a GBCA for patients who may be at higher risk for gadolinium retention (see Table 1 listing GBCAs). These patients include those requiring multiple lifetime doses, pregnant women, children, and patients with

inflammatory conditions. Minimize repeated GBCA imaging studies when possible, particularly closely spaced MRI studies. However, do not avoid or defer necessary GBCA MRI scans.

Patients, parents, and caregivers should carefully read the new patient Medication Guide* that will be given to you before receiving a GBCA. The Medication Guide explains the risks associated with GBCAs. Also tell your health care professional about all your medical conditions, including:

- If you are pregnant or think you might be pregnant
- The date of your last MRI with gadolinium and if you have had repeat scans with gadolinium
- If you have kidney problems

There are two types of GBCAs based on their chemical structures: linear and macrocyclic (see Table 1 below). Linear GBCAs result in more retention and retention for a longer time than macrocyclic GBCAs. Gadolinium levels remaining in the body are higher after administration of Omniscan (gadodiamide) or OptiMARK (gadoversetamide) than after Eovist (gadoxetate disodium), Magnevist (gadopentetate dimeglumine), or MultiHance (gadobenate dimeglumine). Gadolinium levels in the body are lowest after administration of Dotarem (gadoterate meglumine), Gadavist (gadobutrol), and ProHance (gadoteridol); the gadolinium levels are also similar across these agents.

*The Medication Guide will be posted once it is approved.

Table 1. FDA-Approved GBCAs*

Brand name	Generic name	Chemical Structure
Dotarem†	gadoterate meglumine	Macrocyclic
Eovist	gadoxetate disodium	Linear
Gadavist†	gadobutrol	Macrocyclic
Magnevist	gadopentetate dimeglumine	Linear
MultiHance	gadobenate dimeglumine	Linear
Omniscan‡	gadodiamide	Linear
OptiMARK‡	gadoversetamide	Linear
ProHance†	gadoteridol	Macrocyclic

*Linear GBCAs result in more gadolinium retention in the body than macrocyclic GBCAs.
†Gadolinium levels remaining in the body are LOWEST and similar after use of these agents.
‡Gadolinium levels remaining in the body are HIGHEST after use of these agents.

To date, the only known adverse health effect related to gadolinium retention is a rare condition called nephrogenic systemic fibrosis (NSF) that occurs in a small subgroup of patients with pre-existing kidney failure. We have also received reports of adverse events involving multiple organ systems in patients with normal kidney function. A causal association between these adverse events and gadolinium retention could not be established.

We are continuing to assess the health effects of gadolinium retention in the body and will update the public when new information becomes available. We are requiring the following specific changes to the labeling of all GBCAs:

- A *Warning and Precaution*
- Changes related to gadolinium retention in the *Adverse Reactions, Pregnancy, Clinical Pharmacology*, and *Patient Instructions* sections

We urge patients and health care professionals to report side effects involving GBCAs or other medicines to the FDA MedWatch program.

4712-301

Cardiac MRI

*Please note: items in **bold** can be found in the glossary.*

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INTRODUCTION

Heart disease remains a leading cause of death throughout the world and is the number one cause of death in the United States, crossing ethnicities, cultures, and genders[1]. In addition to the value of magnetic resonance imaging (MRI) for the head, body and extremities, cardiac MRI also offers tremendous value for the diagnosis of heart disease and congenital anomalies.

MRI of the heart presents a set of challenges unlike any other anatomical area of the body. A superficial review of the heart reveals an organ well-suited for MR imaging: the heart is a relatively large organ made up of fat, muscle, and blood, all components well-visualized with MRI. However, MRI of the heart presents numerous complex imaging challenges.

CHALLENGES TO CARDIAC MRI

The heart is a complex organ and presents several hurdles to effective imaging:

- The heart is *always* moving. While the normal heart has a regular beat cycle, many cardiac pathologies that lead to the need for an MR examination cause an irregular heartbeat or **arrhythmia**. Capturing an image free of motion-related blur is more difficult to obtain in the setting of an arrhythmia.
- While the heart is relatively symmetrical, it does not sit symmetrically within the chest but rather is situated just left-of-center in the mediastinum. Moreover, the inferior apical portion of the heart is more anterior in the chest, while the superior portions are more posterior. Proper visualization of the heart, then, requires complex **oblique** slice orientations.
- The heart is not one pump but four, and the four cardiac chambers do not contract and relax in a uniform way. Instead of "squeezing" and relaxing uniformly as if squeezing juice from an orange, the heart beats in a twisting motion, much like wringing water from a wet towel. The complicated motion of the beating heart requires precise image acquisition timing.
- Blood is continually flowing in and out of the heart in complex patterns. Flowing blood anywhere in the body can cause flow-related artifacts and ghosting. The heart's large blood pools flowing in various directions and velocities pose a daunting challenge.
- The heart sits between the lungs. With air-filled lungs, the risk of susceptibility artifacts increases with higher magnetic field strengths.

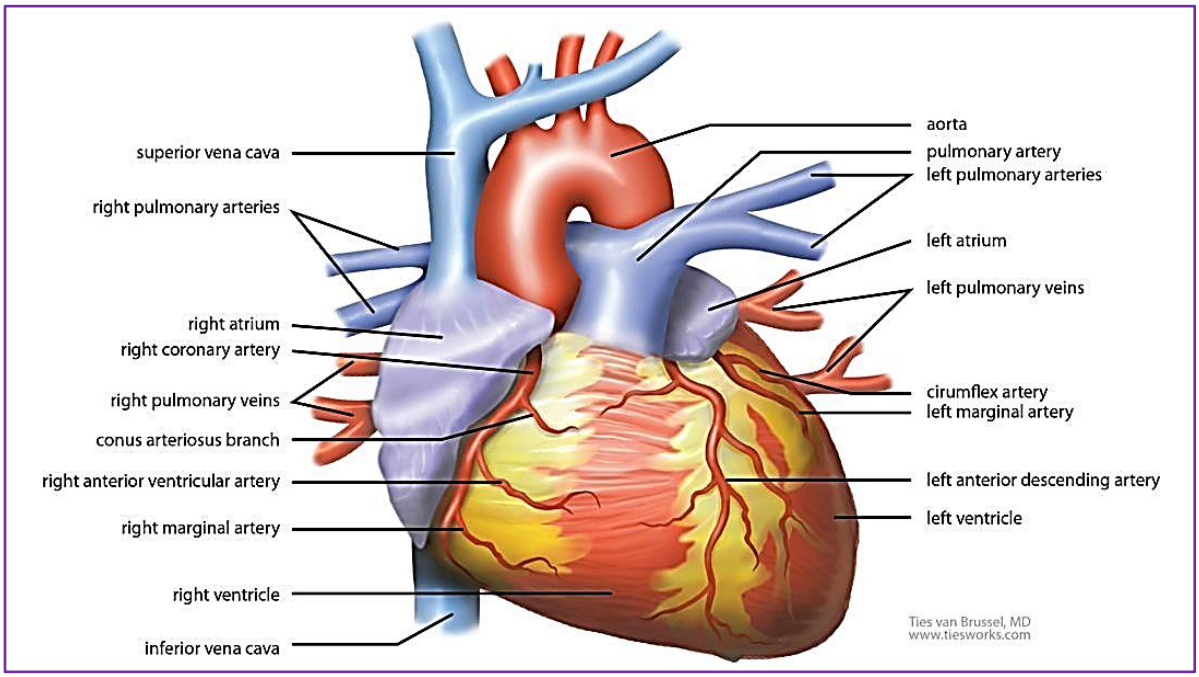

Figure 1. Anatomy of the heart.

Available at Wikimedia.

BASIC CARDIAC ANATOMY

The human heart is a spectacular piece of engineering. It is the fastest growing organ in a developing fetus and begins beating in as little as four weeks. At eight weeks, the heart has developed such that it is a miniature version of an adult heart. In an adult, the heart is generally the size of one's fist and weighs approximately 11 ounces (312 grams). The normal heart beats approximately 100,000 times a day, moving 2,000 gallons (~9,000 liters) of blood daily.

Four Chambers

The four chambers of the heart are divided into right and left halves. The two upper chambers, the left **atrium** and right atrium, receive blood and then pump it into their corresponding left and right **ventricles** which then pump blood out of the heart. The two sides of the heart are separated by the cardiac **septum (Figures 1, 2, and 3).**

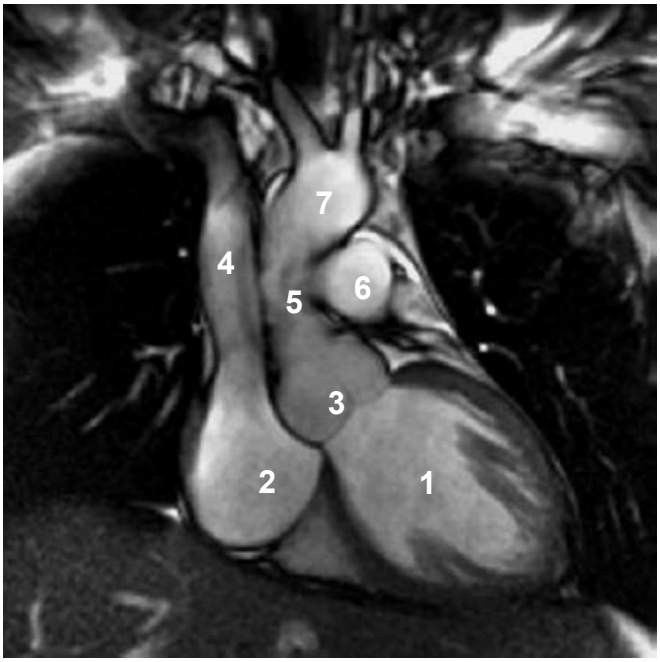

Figure 2. MRI of the heart. Anatomy of the heart from an oblique front view.

1. Left Ventricle
2. Right Atrium
3. Aortic Valve
4. Superior Vena Cava
5. Ascending Aorta
6. Left Pulmonary Artery
7. Aortic Arch

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

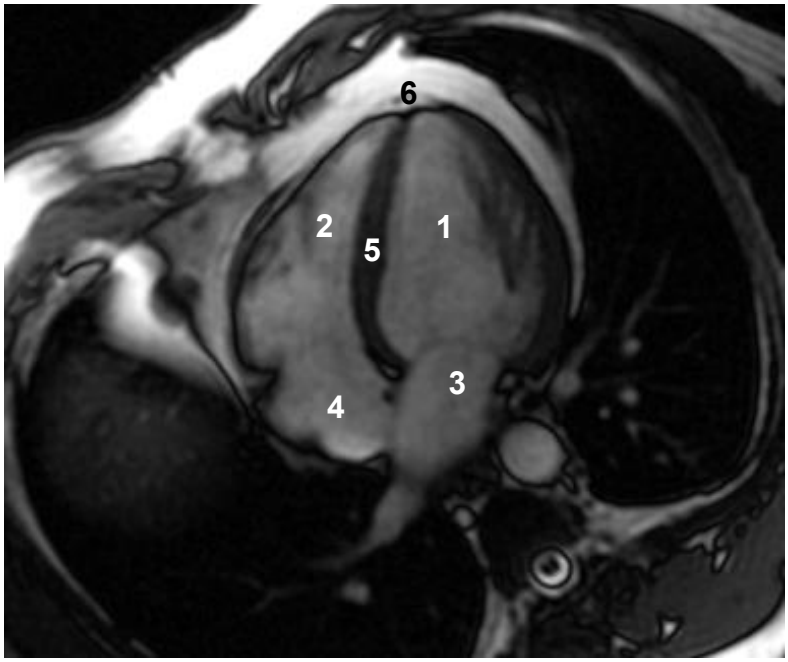

Figure 3. MRI of the heart. Anatomy of the heart from a horizontal long axis 4-chamber view.

1. Left Ventricle
2. Right Ventricle
3. Left Atrium
4. Right Atrium
5. Interventricular Septum
6. Apex

The right atrium receives deoxygenated blood from the body via the superior and inferior vena cava, the largest vein in the body. The body's lowest measurable blood pressure is inside the right atrium when it is fully relaxed. When the right atrium contracts, blood is forced through the tricuspid **valve** and into the right ventricle. Once filled, the right ventricle contracts, forcing the tricuspid valve to close, and pushes the blood through the pulmonary valve into the pulmonary artery en route to the lungs. The blood is oxygenated in the lungs and returned to the heart via the pulmonary vein, which empties in the left atrium. When full, the left atrium contracts, pushing the blood through the mitral valve and into the left ventricle. Once the left ventricle reaches capacity, it contracts, forcing the mitral valve to close, and pushes blood from the ventricle through the aortic valve into the aorta, the largest artery of the body. When in contraction, the left ventricle produces the body's highest blood pressure. Inside each ventricle are webs of cardiac muscle called **papillary muscles** that aid in "pulling" the ventricles inward during contraction (**Figure 4**).

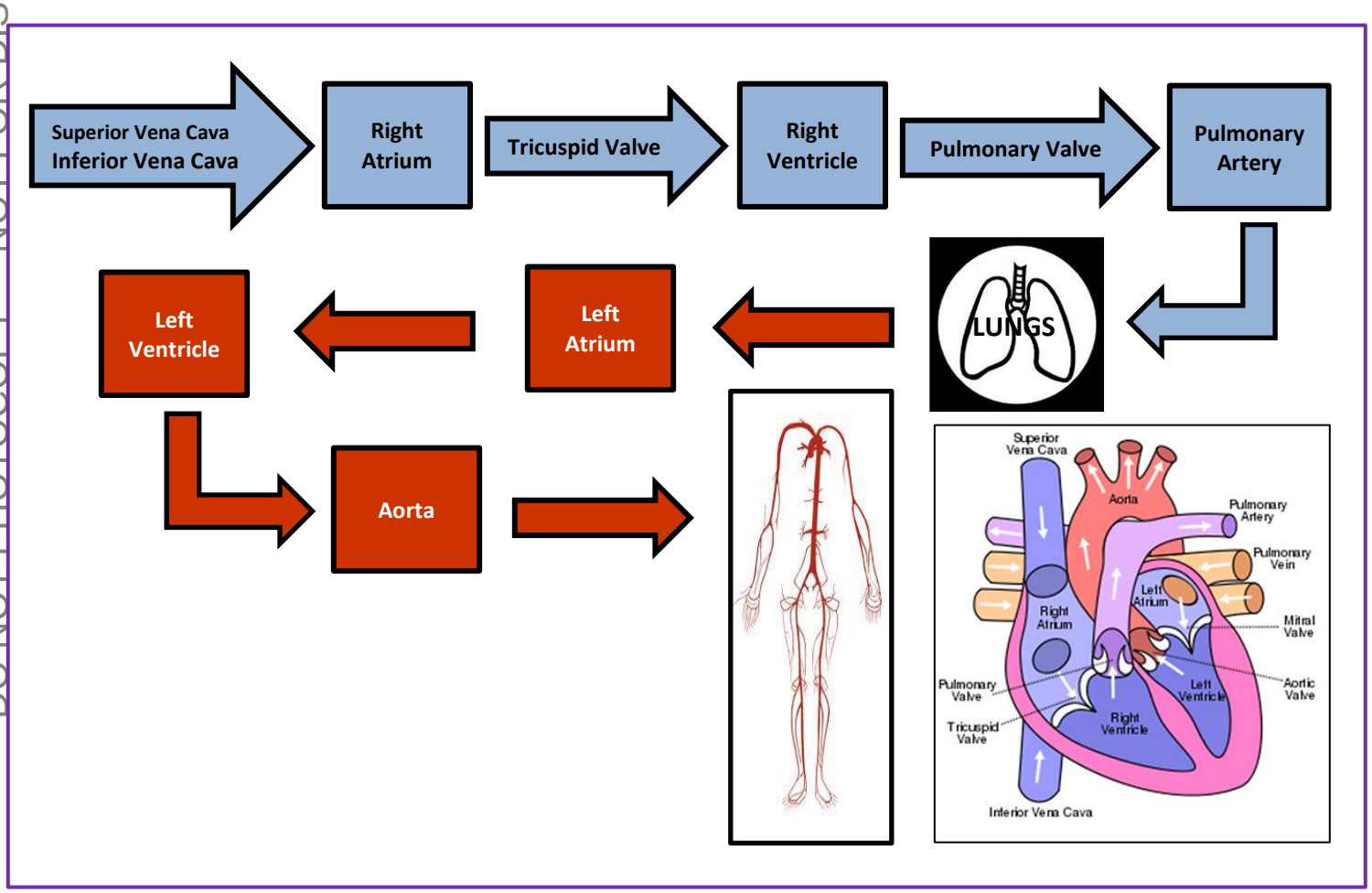

Figure 4. Cardiac circulation.

Available at: Wikimedia Commons.

The pressure of the contracting left and right ventricles forces the tricuspid and mitral valves to close. However, it is not uncommon for a small amount of blood to be forced back into the atrium. These sounds are easily detected by **auscultation** of the heart through a stethoscope and are known as heart **murmurs**. Most murmurs are benign and do not require regular follow-up. However, a more serious concern is when a valve is weak and swings in the opposite direction like a barn door, allowing more blood to be pushed back into the atrium resulting in reduced blood supply to the heart. This condition is referred to as a **prolapse.** A valvular prolapse can be mild, requiring no intervention, to severe, requiring valve replacement.

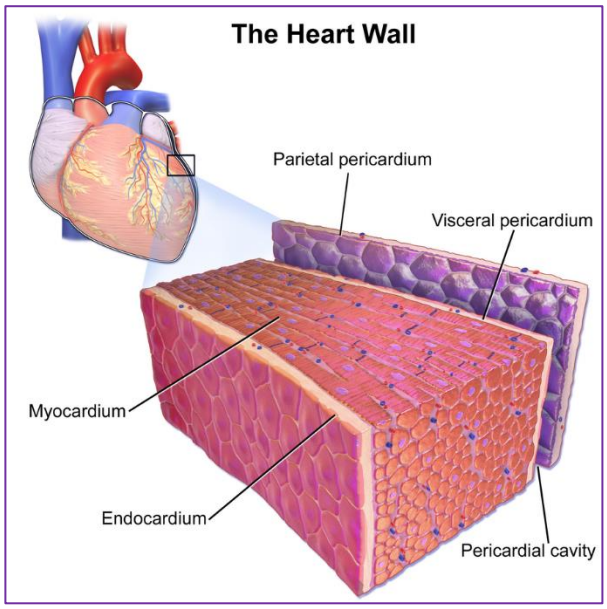

Figure 5. Layers within the lining of the heart.
Courtesy Bruce Blausen. Available at Wikimedia.

Outside the Heart

The lining of the heart is made up of four distinct layers (**Figure 5**). The outermost layer, the **pericardium,** is a thin protective coating around the heart. Just beneath the pericardium is the **epicardium.** Between these two layers is a small amount of **pericardial fluid** that allows the heart to expand and contract without friction. Below the epicardium resides a thick layer of powerful heart muscle called the **myocardium**, the primary contracting force of the heart. It twists and swells with each contraction to vigorously force blood out of the ventricles to the body. If the blood supply to the myocardium is severely restricted, large segments of the myocardium will die from lack of oxygen. The result is a myocardial infarction (MI) or, more commonly, “heart attack.”

Coronary Arteries

The heart of course needs a blood supply. Arising just distal to the aortic valve at the very base of the aorta, the right and left **coronary arteries** wrap themselves around the heart and provide a constant source of oxygenated blood. The heart is the first organ to receive blood as the blood flows from the aorta to the coronary arteries (**Figures 6, 7, and 8**).

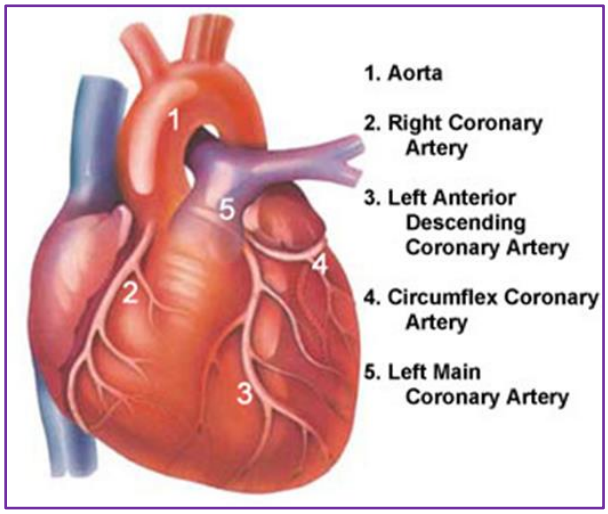

Figure 6. Illustration of the coronary arteries.
Image courtesy of David Darling.

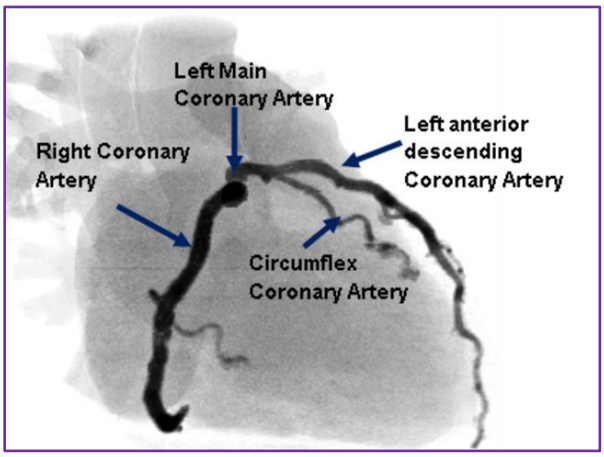

Figure 7. CT angiogram of coronary arteries.
Courtesy of Vital Images, Toshiba Medical Systems.

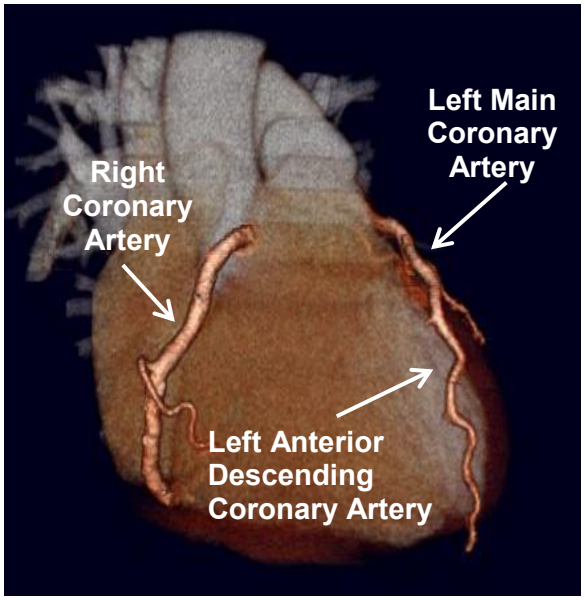

Figure 8. CT angiogram of coronary arteries.
Courtesy of Vital Images, Toshiba Medical Systems.

The left coronary artery comes off the aorta and slides around the pulmonary artery and the center of the heart, branching off to form the circumflex artery. The right coronary artery follows the right atrium and right ventricle. Since the myocardium requires a constant and unceasing supply of oxygen, many of the smaller branches of the coronary arteries contain interconnections known as **anastomoses** that provide alternate flow pathways to the myocardial cells. Interestingly, most cells of the body reach their maximum blood flow during ventricular contractions. This is not true for myocardial cells. During ventricular contraction, the blood flow to myocardial cells is severely restricted due to the "squeezing" of the muscle. It is during peak relaxation that myocardial cells are able to receive peak blood flow.

After coronary artery flow passes through the capillary beds, the blood enters the cardiac veins that run parallel to the coronary arteries. The cardiac veins converge posteriorly at the **coronary sinus**, which returns blood to the right atrium.

The Great Vessels

The major arteries arising from the heart are the **aorta** and the **pulmonary artery**.

AORTA

The aorta is the largest artery in the body, 12-18" long depending on the person's height. The ascending aorta, the part of the aorta immediately coming off the left ventricle, quickly gives way to the aortic arch.

Three arteries arise from the aortic arch: the left

subclavian artery, the left common carotid artery, and the brachiocephalic artery (also referred to as the innominate artery). The brachiocephalic artery quickly bifurcates into the right common carotid artery and the right subclavian artery**.** Distal to the aortic arch, the aorta traverses downward as the thoracic aorta. After passing through the diaphragm, the aorta becomes the abdominal aorta until it bifurcates into the left and right iliac arteries.

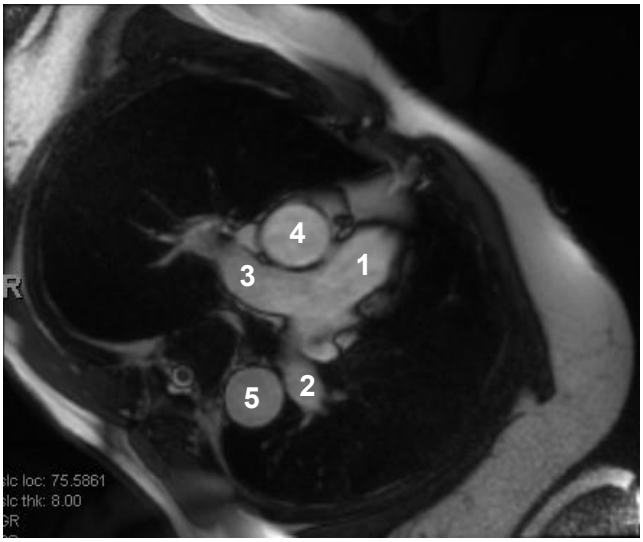

Figure 9. MRI of the pulmonary arteries and aorta.

1. Main Pulmonary Artery
2. Left Pulmonary Artery
3. Right Pulmonary Artery
4. Ascending Aorta
5. Descending Aorta

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Figure 10. **Movie.** MRA of the pulmonary arteries.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Vena Cava

The **vena cava** is the largest vein of the body, running virtually parallel to the aorta. The superior vena cava collects the venous return for all veins *above* the heart and empties into the right atrium. The inferior vena cava collects the venous supply from all areas of the body *below* the heart and also empties into the right atrium.

Pulmonary Vein

The right and left **pulmonary veins** carry oxygenated blood from the lungs back to the heart, emptying the blood into the left atrium.

Pulmonary Artery

The pulmonary artery arises directly from the right ventricle. The artery quickly bifurcates into the left and right pulmonary arteries (**Figures 9 and 10)**. These branches carry blood to the lungs where the blood is oxygenated. Carbon dioxide is exchanged for oxygen throughout **capillary** networks located within the **alveolar sacs**.

The major vessels of the heart are unique in that the pulmonary arteries transfer *de-oxygenated* blood, while the pulmonary veins carry *oxygenated* blood, contrary to the function of the rest of the body's vasculature.

THE ELECTROCARDIOGRAM WAVEFORM

In order to capture the heart free of motion or in perfect motion, the pulse sequence timing must be exact and in unison with the cardiac cycle. The process of timing the pulse sequence to the cardiac cycle is accomplished using a technique called **cardiac gating**. To understand the mechanism of cardiac gating and to learn to use it effectively, one must have a basic understanding of the electrical activity of the heart.

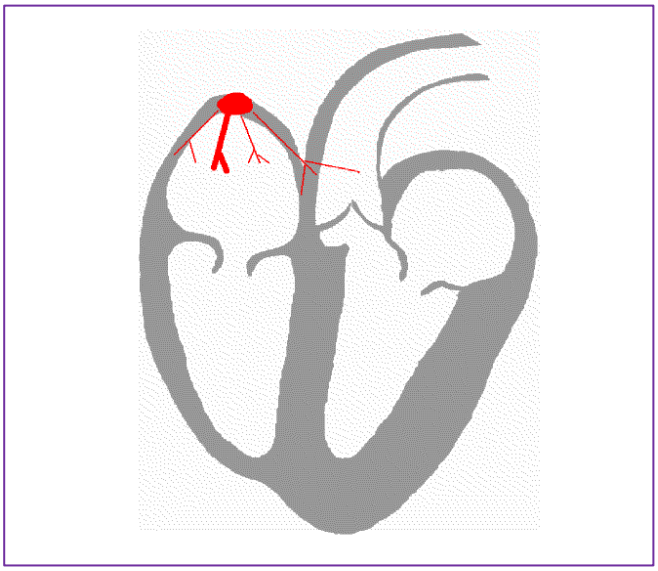

Figure 11. Electrical pathways of the heart.
Courtesy of Kalumet. MOVIE available at: Wikimedia.

Electrical conduction of the heart occurs as electrical impulses pass through the right atrium and are distributed by two specialized nerve bundles. The **sinoatrial node** (S-A node), located superiorly in the right atrium, initiates the electrical pulse in a very rhythmic manner, causing the right and left atria to contract simultaneously. The electrical impulse next travels to the **atrioventricular node** (A-V node), where the electrical impulse spreads out to the ventricles, causing the cells of the myocardium to contrast and twist, squeezing the blood from the two lower chambers (**Figure 11**).

The electrical activity of the heart is strong enough to be recorded by measuring the voltage potential between two areas of the heart. The changes in electrical potential are caused by the release of energy during a contraction, called **depolarization,** and the re-energizing of the atria and ventricles, called **repolarization**. The recording and displaying of the heart's electrical activity is called an **electrocardiogram** or **ECG.** An ECG translates the heart's electrical activity into line tracings on paper (**Figure 12**).

The **P-wave** indicates peak *atrial* contraction (depolarization), while the **R-wave** indicates the peak *ventricular* contraction. The **QRS complex** indicates ventricular depolarization. The **T-wave** indicates ventricular relaxation (repolarization); atrial repolarization produces a wave too small to be seen on the ECG waveform.

SINGLE VS MULTIPHASE IMAGING

The complex motion of the heart — the contracting and expanding of four chambers and the twisting motion of the myocardium — leads to significant motion and blood flow artifacts that degrade image quality. To minimize or eliminate these artifacts, the image sequence must be synchronized with the patient's cardiac rhythm, accomplished by using cardiac gating.

The ECG waveform displays the contraction of the heart muscle, or **systole,** and its subsequent relaxation, **diastole.** When the pulse sequence is timed to the ECG waveform, the data collected are arranged such that the heart appears frozen in a moment in time, called a **single-phase image**.

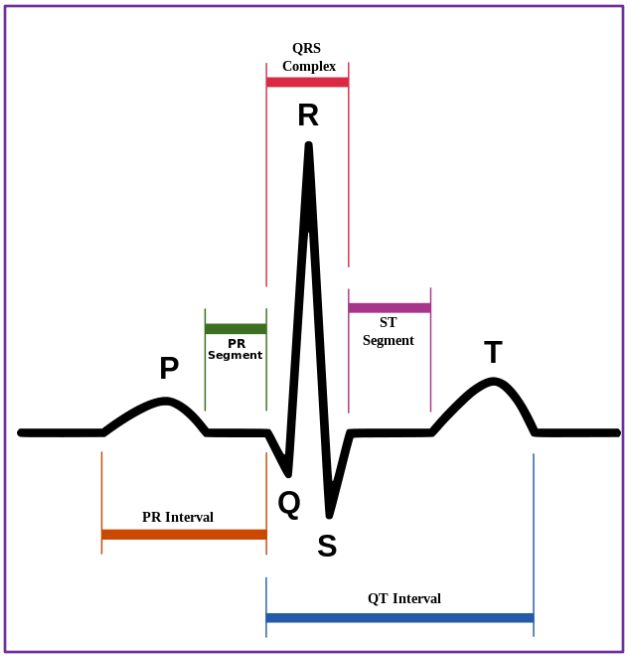

Figure 12. ECG waveform of a normal heart.

Available at: Wikipedia.

It is also possible to image so quickly through the cardiac cycle that images are displayed in motion, allowing observation of the heart while beating. A **cine** loop displays the heart in motion as a **multiphase** data set.

As the heart beats, there is a cycle to its rhythm, and this cycle has a beginning and an end and can be divided into segments. Each segment is referred to as a phase of the cardiac cycle. As discussed, a single-phase view of the heart is one in which a single slice location is displayed as a static image (**Figure 13).** A multiphase view is one in which a single slice location of the heart is displayed in a cine loop. Much like a cartoon is drawn in several individual frames and then played back as a moving picture, a multiphase view of the heart is done in the same way. In this view, a particular slice location is imaged several times throughout the cardiac cycle; the more cardiac phases acquired, the more "real time" the multiphase view appears. In cardiac MRI, 16 phases is typically the minimum number of phases collected in order to accurately view the heart motion in real time (**Figure 14**).

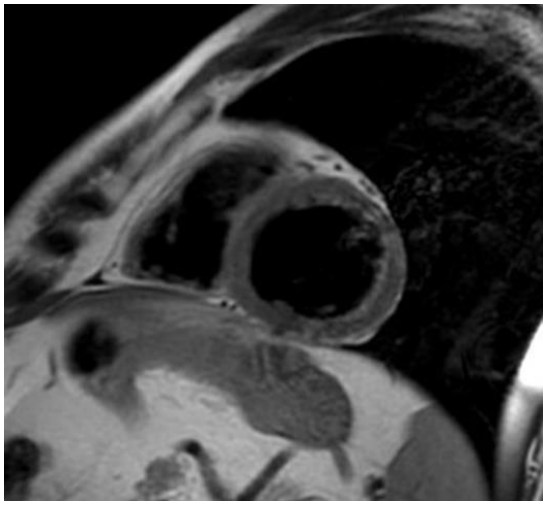

Figure 13. Single-phase short axis view of the right and left ventricles and myocardium.

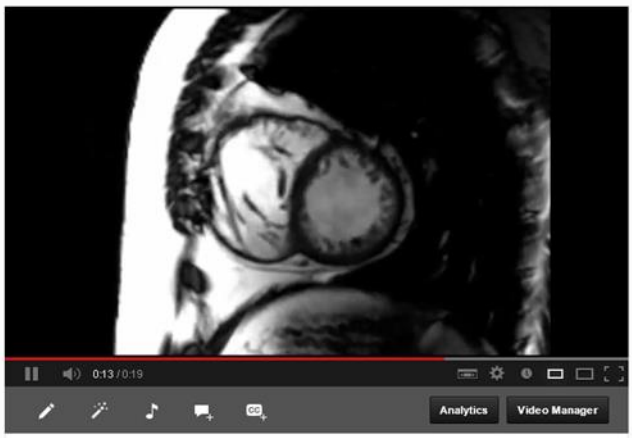

Figure 14. Movie. Multiphase, multi-location short axis view of the left ventricle.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

CARDIAC GATING

The objective of cardiac gating is to time the pulse sequences to the patient's cardiac rhythm in order to reduce or even eliminate motion and flow artifacts. Gating can be accomplished in two ways: through the placement of cardiac electrodes on the chest, called **ECG gating**, or through the use of a pulse sensor placed on the finger or toe, called **peripheral gating**.

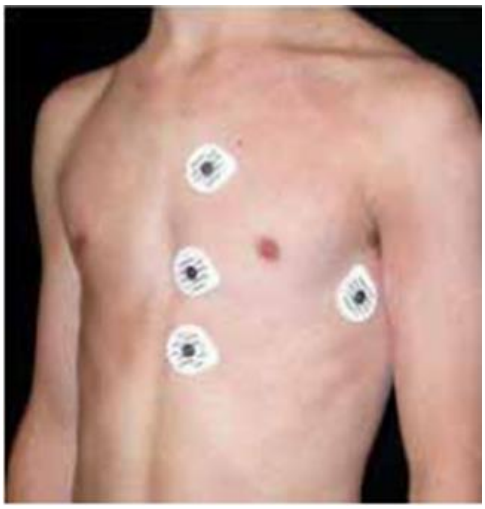

Figure 15. Correct ECG electrode placement for cardiac triggering.

Courtesy GE Healthcare.

ECG Gating

ECG electrodes must be certified as MR-compatible, and the placement of MR-compatible electrodes must follow the manufacturer's recommendations. Manufacturers provide color-coding or other labeling to ensure correct electrode placement.

The objective of electrode placement is *not* to obtain a diagnostic ECG waveform but instead to achieve the highest R-wave for launching the pulse sequence, called **triggering**. The voltage potential between two electrodes is referred to as a **lead.** (The electrodes are commonly referred to as the "leads," but this is technically incorrect.)

With a typical 4-electrode placement on the patient's chest, the voltage potential between the pairs of electrodes is known as Lead 1 and Lead 2. The further the distance between the two electrodes, the higher the voltage potential and therefore the higher the peak of the R-wave trigger. However, greater distance between the electrodes introduces electronic noise, which increases the risk of false triggering. Conversely, there is a decrease in electronic noise with less distance between electrodes, but a smaller peak for the R-wave trigger. The correct distance between electrodes is dictated by both the recommendations of the manufacturer and the quality of the displayed ECG waveform (**Figure 15**.)

For ECG placement, preparation of the chest area may be required. For example, if the patient's chest is particularly hairy, areas of the chest may need to be shaved. Since electrodes require excellent conductive contact with the skin, it is recommended that rubbing alcohol or soaps not be used in chest preparation as these products remove oils from the skin that aid in electrode contact. Patients with metal sternal wires or with large breasts may require alternate electrode placement. Because most systems rely on high, positive R-waves, it is critical that the ECG waveform be checked before the start of the study. If the R-wave is small or inverted, then lead polarity may need to be adjusted. Typically this can be done at the console. Alternatively, the electrode positions can be readjusted (**Figure 16**).

Figure 16. Movie. ECG waveform on the MR operator console.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

THE ECG GATING PROCESS

ECG gating uses the peak of the R-wave to trigger the start of a cardiac pulse sequence. The time between one R-wave and the next is referred to as the **R-to-R interval** and is the determining factor for calculating the effective TR (time to recovery), as well as the amount of time allotted for data acquisition. Data acquisition occurs between R-waves and is clearly a finite amount of time. If there is not enough time within an R-to-R interval to acquire the necessary data, the operator can increase that time by "skipping" an R-wave. Depending on the patient's heart rate, the pulse sequence used, the number of slice locations to be acquired, and the required spatial resolution, the operator decides how many R-waves to skip before initiating the next pulse sequence.

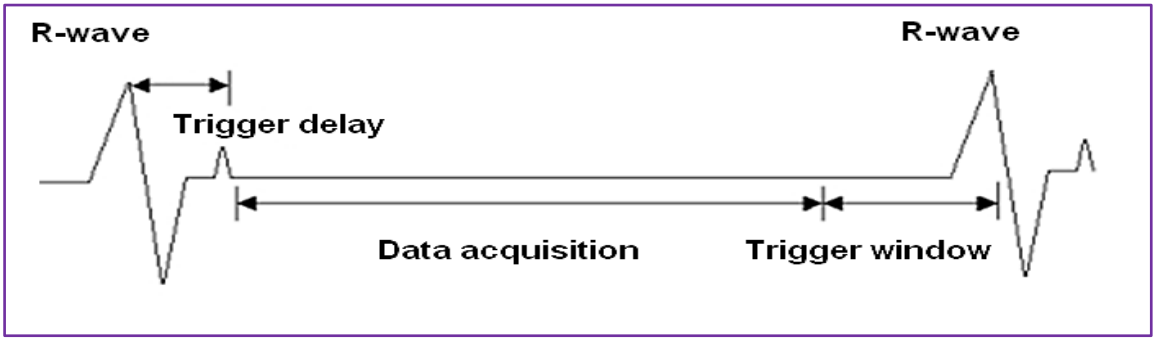

Figure 17. ECG gating. In ECG gating, the patient's cardiac cycle is monitored. Once reliable monitoring by the MR system is established based on the consistency of the heart rate, the timing of the pulse sequence can be synchronized to the cardiac cycle, greatly reducing artifacts from cardiac motion. In a typical gating sequence, the R-wave is detected and data acquisition begins after a time delay of a few milliseconds and continues for a predetermined amount of time. Once data acquisition is complete, the trigger window opens and the system begins sensing for the next R-wave, and the cycle begins again.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

As an example, if the R-to-R interval is 1 second (1000msec) and each pulse sequence is initiated at the peak of each R-wave, the effective TR is 1000msec. However, if that TR period is not effective for obtaining the required tissue contrast or the required amount of image data, the operator can opt for skipping every other R-wave by starting a new sequence every second R-wave or "2xRR." Here the effective TR is now 2000msec. If 3xRR is selected, then the effective TR is 3000msec and so forth. The correct R-to-R interval is determined by the required imaging parameters balanced with the patient's breath-holding abilities. The general rule is the greater the R-to-R interval selected, the more data that can be acquired — but the longer the patient's breath-hold time.

TRIGGER DELAY

Other timing parameters require operator input, one of which is **trigger delay**. Trigger delay is the time from detection of the R-wave to the beginning of the data acquisition, usually the minimal time determined by the MR system.

TRIGGER WINDOW

Another timing parameter requiring operator input is the **trigger window**, which is the time from the last data acquisition until the next R-wave is sensed by the MR system. The trigger window is selected by the user and allows the MR system to stop data acquisition and begin sensing for the next R-wave. A large trigger window (typically more than 30% of the R-to-R interval time) decreases the risk of a "skipped" R-wave but also reduces the time for data acquisition. The MR system will recommend a minimum trigger window based on the *consistency* of the patient's heart rate.

Figure 18. Pulse sensor or photoplethysmograph.

Courtesy of Fairfax Radiological Consultants.

Figure 19. Movie. Example of a peripherally-gated waveform.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

If R-waves are skipped due to erratic heart rates or narrow trigger windows, breath-hold times increase significantly and image degradation occurs. In single-phase acquisition, typically no data collection occurs during the QRS complex. For multiphase imaging sequences, data acquisition is continuous throughout the entire cardiac cycle (**Figure 17**).

Peripheral Gating

ECG gating is not possible in some clinical situations, eg, post-acute myocardial infarction (MI) or patients with a severe arrhythmia. For these patients, peripheral gating can be used.

Peripheral gating uses the peak reflection of the capillary blood as a substitute for the R-wave. A peripheral pulse sensor is used, usually placed on the fingertip, although toes can be used. Pulse sensors used in MRI are often mistakenly referred to as “pulse oximeters.” However, pulse sensors in MRI do not measure the oxygen level in the blood but rather are a reflection of blood filling and emptying in the capillary beds. The formal name for this type of sensor is **photoplethysmograph** (**Figure 18**).

Because of the delay between the time of the contraction of the heart — the R-wave — and the time required for the blood to travel from the heart to the finger, peripherally-gated sequences tend to begin in diastole, the period of relaxation and repolarization. If systolic imaging is desired, retrospective gating can be used.

Regardless of how the information is obtained, ECG gating is usually preferred to peripheral gating when imaging for any cardiac **morphology.** Peripheral gating is useful when a stable cardiac-gated waveform cannot be obtained or when cardiac morphology is not being sought, for example, when imaging the aortic arch (**Figure 19**).

Retrospective and Prospective Gating

In **prospective gating**, the R-wave triggers the beginning of the pulse sequence, and the R-to-R interval serves as the TR period. Each line of ***k*-space** is acquired within the same cardiac cycle from R-wave to R-wave. Prospective gating is usually used in single-phase imaging.

In **retrospective gating**, data are acquired continuously and not triggered by the R-waves. However, the ECG pattern or peripheral pulse pattern is also recorded at the same time. The MR data then are partitioned according to the time in the heart cycle when the signal was collected to ensure that cardiac motion is reduced. In this way, the heart can be imaged throughout systole *and* diastole. Multiphase imaging almost always employs a retrospective gating technique.

Safety Considerations of Cardiac Gating

Regardless of the method used, both ECG and peripheral gating require the placement of conductive cables while the patient is inside the magnet bore. As with any cabling in the bore, care must be taken to reduce the risk of burning the patient. *Always carefully follow the manufacturer's recommendations to ensure reliable and safe gating.*

In general, the following conditions apply:

- Never allow any cable to loop onto itself. The loop creates a closed circuit that greatly increases the potential for a hot spot at the loop intersection.
- Minimize the length of cable in the bore. In many cases, this means placing the patient feet first in the magnet and running the ECG cables out of the bore with the least amount of cabling actually in the bore itself.
- Do not allow multiple points of contact between the cable and the patient. After the initial connection to the patient, be sure that the cable does not come into contact with the patient again. If indicated, place MR-compatible pads between any portion of the cable and the patient.
- Never use cabling that is cracked or frayed.
- Always follow the manufacturer's recommendations.

CARDIAC PULSE SEQUENCES AND IMAGE CONTRAST

Image contrast is simply distinguishing between two different tissue types. As with all MRI, optimizing image contrast in cardiac imaging is of paramount importance for visualizing anatomy and pathology.

Image contrast in cardiac MRI can typically be grouped into four categories:

- Bright-blood imaging
- Black-blood imaging
- Phase contrast imaging
- Gadolinium contrast-enhanced imaging

A comprehensive cardiac MR study usually, but not always, employs these four methods of image contrast. Which imaging contrast protocols to use are determined by the diagnostic question: what are we looking for? Ultimately it is the radiologist in consultation with the referring physician who determines the optimal imaging protocol.

Bright-blood Imaging

Bright-blood imaging is a contrast-enhanced technique that visualizes blood in the heart and the great vessels as bright against the darker myocardial background. This differentiation is achieved through a type of pulse sequence usually referred to as “steady-state” imaging (GE: FIESTA™, Siemens: FISP™, Philips: balanced FFE™) (**Figure 20**). All of these pulse sequences use a gradient echo-based (GRE) sequence. As with any gradient echo sequence, the use of short TR periods results in **residual transverse magnetization**.

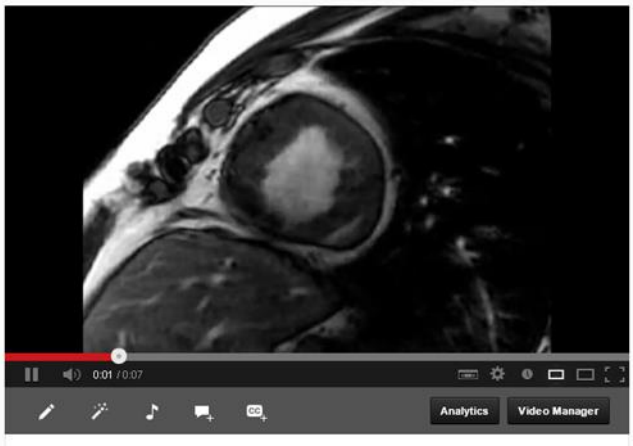

Figure 20. Movie. Multiphase, short axis bright-blood FIESTA image of the left ventricle.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Residual Transverse Magnetization

To understand residual transverse magnetization, recall that some tissues, like cerebral spinal fluid, have long T2 times, that is, they remain in the transverse magnetization plane longer than short T2 tissues, like muscle.

If the T2 time of a tissue is longer than the selected TR, the transverse magnetization of that tissue never fully decays. Within just a few TR periods, the residual transverse magnetization reaches equilibrium where the amount of residual transverse magnetization remains the same for each TR period throughout the pulse sequence. In other words, it has reached a “steady state.”

With typical GRE sequences, residual transverse magnetization can become problematic as it will result in an image that is a mix of T1 and T2* (T2-star) contrast. Recall that T2* contrast is a combination of the intrinsic T2 of a tissue and T2' (T2-prime), which is dephasing due to susceptibility effects.

To rid the images of the contrast mixture of T1 and T2*, a gradient or radiofrequency (RF) pulse is typically applied at the end of each TR period to eliminate or "spoil" the residual transverse magnetization. These GRE sequences are typically referred to as "spoiled" GRE sequences.

In cardiac imaging, however, the residual transverse magnetization plays an important role in tissue contrast. The steady-state sequences in cardiac imaging are either 2D or 3D cardiac-gated sequences that balance the gradients to preserve the residual transverse magnetization, as well as the ultra-short TR periods that reduce the effects of susceptibility in any GRE-based sequence. The resultant contrast is based on the T2:T1 ratio of the tissues. Those tissues that have a long T2 and short T1 (large residual transverse magnetization) such as blood have a high T2:T1 ratio and therefore appear bright (**hyperintense**), ie, a bright-blood image. Tissues with a low T2:T1 ratio (small residual transverse magnetization) like myocardial muscle appear very dark (**hypointense**).

An added benefit of the ultra-short TR periods used in bright-blood steady-state imaging is that data acquisition can occur within one breath-hold. In multiphase bright-blood imaging, breath-hold acquisition times are further accomplished through the use of rapid *k*-space filling, called views-per-segment. Recall that in multiphase imaging, the R-to-R interval is segmented with each partition designated as a cardiac phase from systole through diastole. Within each partition, lines of *k*-space or views (each line of *k*-space corresponds to a phase-encoding step) are quickly filled to create an image of the heart at a particular location and in a particular cardiac phase.

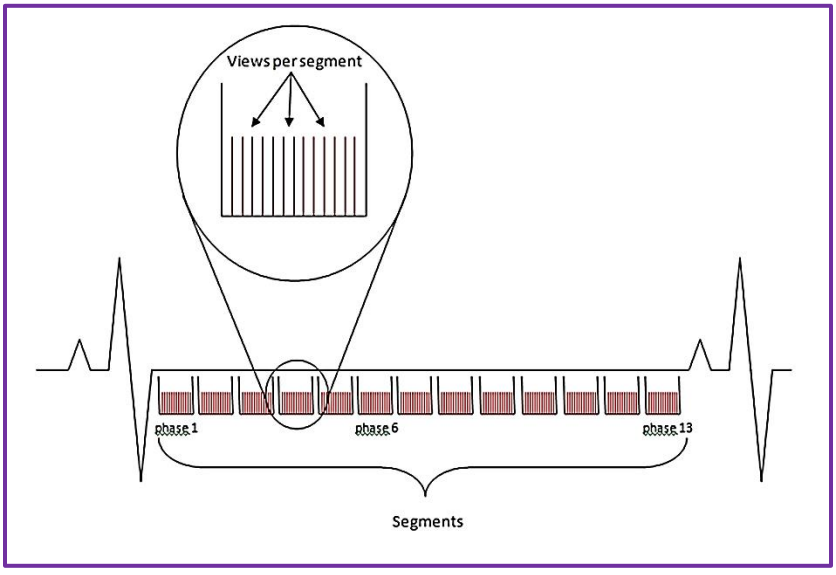

Figure 21. Segmented waveform. R-to-R interval shows different partitioning and views per segment.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

Each "view" is synonymous with a line of *k*-space, just as an RF echo is synonymous with a line of *k*-space in a fast/turbo (FSE/TSE) spin echo sequence. The more views that can be obtained within a given cardiac phase, the shorter the breath-hold time for the patient. However, if too many views per segment are acquired per cardiac phase, the number of phases obtained may be fewer. Conversely, if many cardiac phases are required, fewer views-per-segment can be acquired, making breath-hold times longer (**Figure 21**).

The key to obtaining high-quality multiphase cardiac images is balancing the desired number of cardiac phases with the patient's breath-holding ability. As with all advanced imaging techniques, higher performance gradients make this balance attainable. High-performance gradient MR systems are able to acquire 20 cardiac phases within a 10-20 second breath-hold for a patient with an average heart rate of 60 – 70 bpm.

For example, if 256 lines of *k*-space are desired to create a cardiac image at any given cardiac phase and 20 views-per-segment are selected, then only 13 heart beats are required to collect the needed data. At a heart rate of 70 beats per minute, the breath-hold time is approximately 15 seconds.

TEMPORAL AND SPATIAL RESOLUTION

The *number* of cardiac phases obtained within a specified R-to-R time interval in a cardiac multiphase series is its **temporal resolution.** The *size and number of the voxels* in the image is its **spatial resolution.**

These imaging characteristics are often diametrically opposed. If the temporal resolution is set too low, for example, at six cardiac phases, then assessing heart motion becomes difficult if not impossible. However, obtaining high temporal resolution may require reducing spatial resolution. High spatial resolution is required to see detail in the heart, including assessment of valve function, but increasing spatial resolution often sacrifices temporal resolution. In other words, high temporal resolution and high spatial resolution are equally desirable for achieving proper diagnostic value. Therefore careful adjustment of both temporal and spatial resolution parameters is required to allow adequate amounts of each. Sample protocols are provided at the conclusion of this material to aid in determining that balance.

Black-blood Imaging

In order to assess the health and viability of the myocardium, it is useful to place the myocardium against the backdrop of a dark blood pool. Black-blood cardiac imaging, as one might surmise, provides the opposite of bright-blood imaging in that the cardiac blood pool and the blood in the great vessels are hypointense against a background of myocardium that is not as dark.

To be clear, the appearance of the myocardium using this contrast method is not hyperintense as the blood pool in the bright-blood steady-state sequences. Rather, the myocardium simply does not appear as dark as the blood pool, which can be considered black in its gray scale. In fact, one type of black-blood imaging – also called dark-blood imaging – is designed to render the intensity of the myocardium as *hypo*intense. Black-blood contrast is achieved through the use of a fast/turbo spin echo sequence and inversion-recovery (IR) preparation pulses.

Double Inversion Recovery Black-Blood Imaging

In double IR black-blood imaging, a non-slice selective inversion pulse is applied over the entire body to invert all water spins from the +Z axis of the longitudinal magnetization to the –Z axis. Thereafter, a slice-selective IR pulse is applied to the particular location of the heart being imaged, re-inverting all the spins in the slice back to the +Z axis and in essence placing the spins in the slice back to their original state. A time delay — TI or "inversion time" — is set from the application of the first non-selective pulse until data acquisition of the slice of interest. That time delay is set to the **null point** of blood, the time from inversion of the longitudinal magnetization to –Z until the longitudinal magnetization of *a particular tissue* crosses the midpoint between –Z and +Z where no **net magnetization** exists. For example, the null point for fat at 1.5 Tesla is ~ 150msec, and the null point for cerebral spinal fluid is ~ 2200msec. When data acquisition for the cardiac slice of interest is acquired, non-inverted blood in the ventricles washes out and is replaced with "nulled" blood that is extremely hypointense (**Figures 22 and 23**).

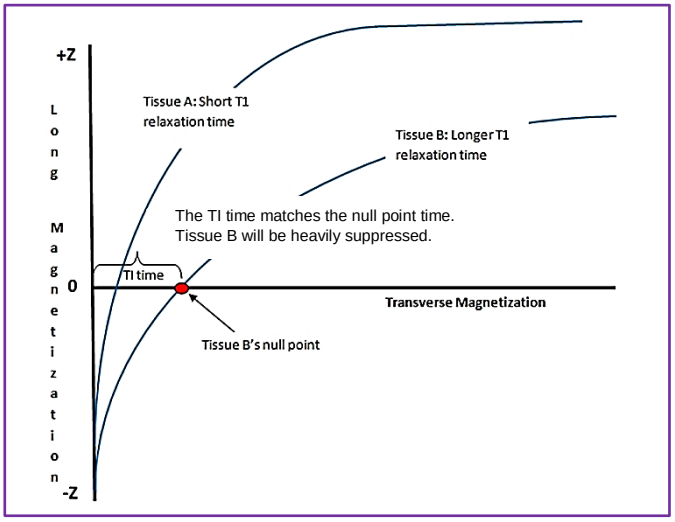

Figure 22. T1 inversion recovery relaxation curve demonstrating tissue null points and resulting in maximum signal suppression.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

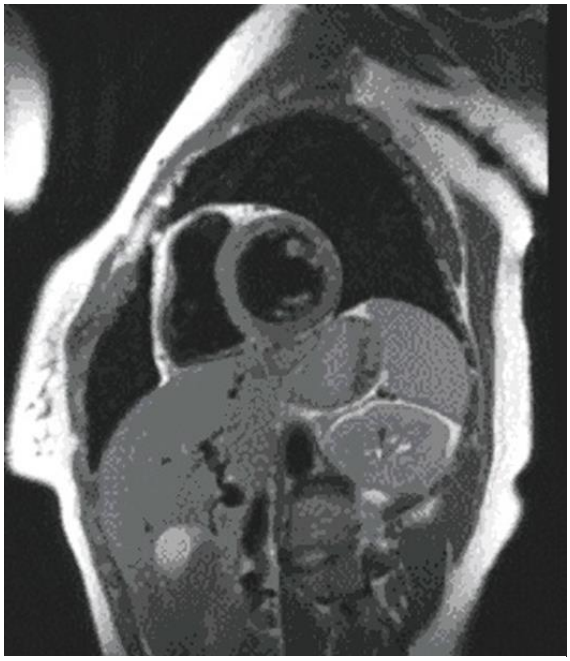

Figure 23. Double IR short axis black-blood image of the left ventricle.

Courtesy GE Healthcare.

TRIPLE IR BLACK-BLOOD OR DOUBLE IR BLACK-BLOOD WITH FAT SUPPRESSION

Fat is normally present to some degree around, and interlaced with, the myocardium. To better assess myocardial health, fat-suppressed black-blood imaging is often used to differentiate fat from myocardial muscle. To achieve this contrast, two imaging sequences are available: triple IR black-blood imaging and double-IR black-blood imaging using a fat saturation RF pulse.

Triple IR black-blood imaging is obtained in the same way as double IR black-blood imaging except the root pulse is a fast/turbo spin echo IR (FSE-IR or TSE-IR) instead of FSE/TSE alone. The addition of a time to inversion (TI) delay set to the null point of fat (~150msec at 1.5T) is applied to the slice selective pulse. The result is a black-blood image with fat-suppressed myocardium (**Figure 24**).

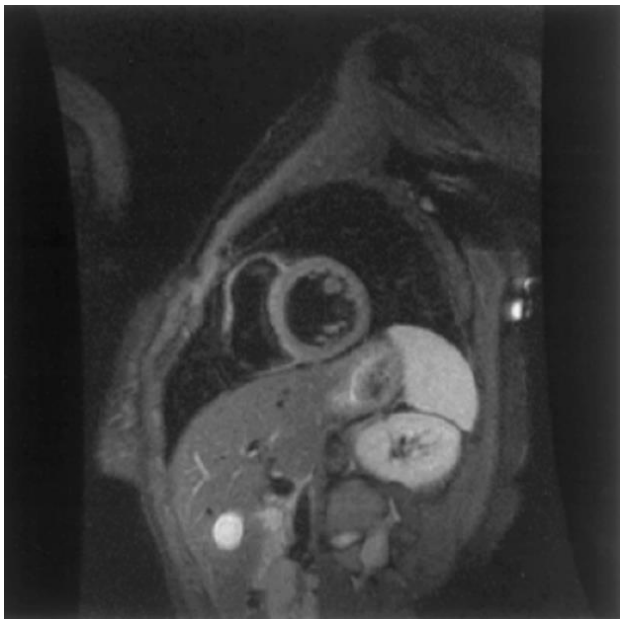

Figure 24. Triple IR short axis black-blood image showing fat-suppressed (hypointense) myocardium.

Courtesy GE Healthcare.

An alternative to triple IR is to employ a double IR black-blood sequence with a fat-saturating RF pulse directed to the cardiac slice of interest, resulting in a black-blood image with fat-saturated myocardium (**Figure 25**). Determining the best method is left to the individual imaging facility with guidance from the manufacturer.

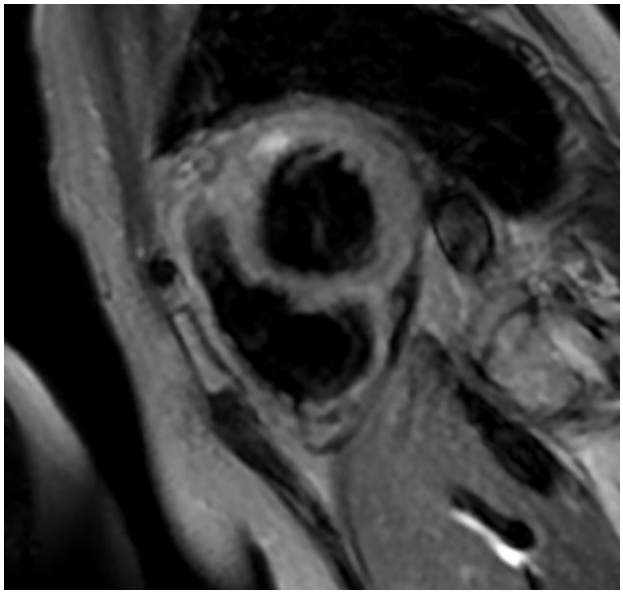

Figure 25. Double IR short axis inversion recovery sequence showing a fat-saturated myocardium.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Phase Contrast Imaging

Phase contrast imaging (PC) is an MR angiography (MRA) pulse sequence useful for evaluating cardiac function, particularly in assessing valvular and aortic and pulmonary great vessel function. Recall that angiography is a study of the vasculature, usually involving administration of a gadolinium-based contrast agent. It is important to note that the plane of acquisition must be perpendicular to the vessel of interest, dissecting it into a circle and not an oval to make quantifiable measures.

As an example, patients born with **Tetralogy of Fallot** often require surgical repair and restructuring of the pulmonary arteries. To assess the status of the patient, phase contrast imaging has the unique ability to provide *quantitative* flow data. While phase contrast can be used as either a 2D or 3D imaging technique, 2D PC imaging is typically used in cardiac imaging.

Phase contrast imaging relies on gradient manipulation to provide flow data. PC imaging manipulates the accumulated phase shifts of the spins in flowing blood by applying **bipolar gradient** pulses – gradient pulses applied in equal but opposite magnitudes – to produce angiographic images against a dark background of stationary tissue. The advantage of PC cardiac imaging is the ability to quantify both flow *direction* and *velocity*.

To understand the mechanism behind PC imaging, recall that spins are placed in the transverse magnetization plane x, y following the initial excitation pulse. While stationary spins do not accumulate phase shifts, moving spins do accumulate phase shifts as the spins move from one local gradient field to the next. The application of controlled gradient fields takes advantage of the movement of flowing spins, as well as the non-movement of stationary spins. This is accomplished by the application of bipolar gradients.

When subjected to a gradient field of a given magnitude and direction, spins undergo a phase shift in accordance with the Larmor Equation:

$$\acute{\omega} = \gamma \beta_0$$

where $\acute{\omega}$ is the **Larmor frequency**, γ is the gyromagnetic ratio, and β_0 is the strength of the local magnetic field. The phase shift is proportional to the magnitude and direction of the gradient field. As an arbitrary reference point, the phase shift can be given a value of +1. Assuming the spin is stationary**,** subjecting it to a second gradient field equal in magnitude but the opposite direction, the phase shift becomes -1. When the phase shifts of the spin are combined, the net phase shift for the stationary spin is 0 and yields no signal (**Figures 26 and 27**).

It is the combined image that displays signal from flowing blood, but in the individual acquisitions, information can be obtained and displayed that yields directional flow. For example, if the flow-encoding gradient used is the x-gradient, the flow that is encoded is left/right flow. Using the y- and z-gradients subsequently yields anterior/posterior and superior/inferior flow directions. In clinical practice, when evaluating complex multi-directional flow, eg, intracranial vasculature, all three gradient directions are used.

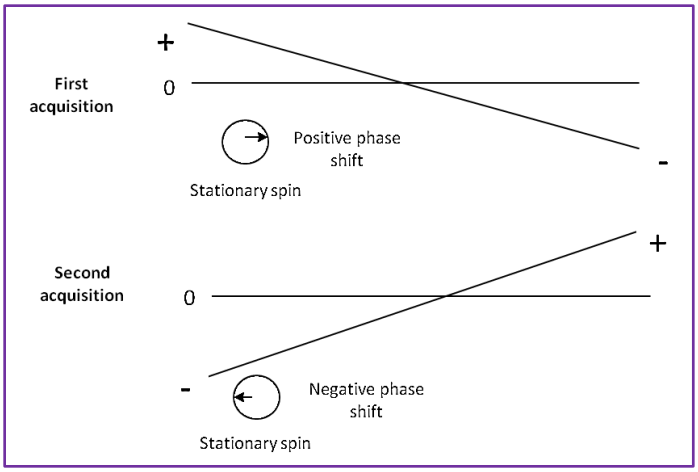

Figure 26. The first acquisition uses a bipolar gradient field to induce a *positive* phase shift on a stationary spin. The second acquisition is an equal but reversed gradient field to induce a negative phase shift. When the two acquisitions are combined, the new phase shift equals 0, yielding no signal.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

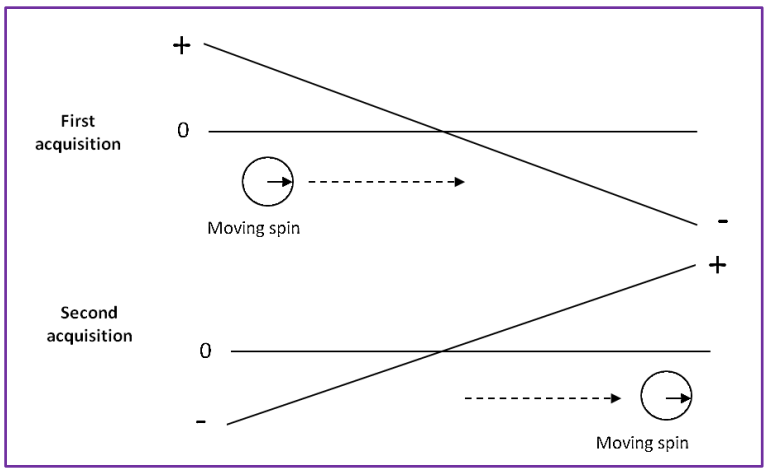

Figure 27. However, the results are different when dealing with a *moving* spin. In the case of flowing blood, the spin accumulates a phase shift from the first gradient acquisition to the second. When the two acquisitions are combined, the phase shifts of the flowing spin have a positive net phase shift in the transverse magnetization plane. When the data sets of the first and second acquisitions are combined, the only signal produced is that of the flowing blood.

Again, spins of flowing blood accumulate a phase shift when bipolar flow-encoding gradients are applied. The amount of phase shift produced is proportional to the velocity of the flowing blood. In order to correctly encode spins from flowing blood to produce the highest signal, the amplitude of the flow encoding must be optimally set. This is accomplished via the velocity encoding value or VENC.

The VENC is set to the highest estimated flow velocity of the vasculature being evaluated. For example, the normal maximum velocity for the ascending aorta is 100cm/sec in an adult. Therefore the proper VENC for gaining maximum signal in a phase contrast MRA of the ascending aorta is greater than 100cm/sec, typically 200cm/sec. If the VENC is set too low, for example, at 50cm/sec for carotid artery evaluation, flow that is greater than 50cm/sec will be **aliased** as slower flow. Further, if the VENC is set too low, flowing spins can accumulate a phase shift that is greater than 180^0. As an example, a phase shift of $+200^0$ is the same as a phase shift of -160^0. Not only will this signal appear as slower flow but will also appear to flow in the *opposite* direction.

See **Table 1** for examples of great vessel velocities.

Vessel	Flow Velocity
Ascending aorta	100cm/sec
Descending aorta	100cm/sec
Main pulmonary artery	80cm/sec
Left and right pulmonary artery branches	60cm/sec

Table 1. Great vessel velocities.

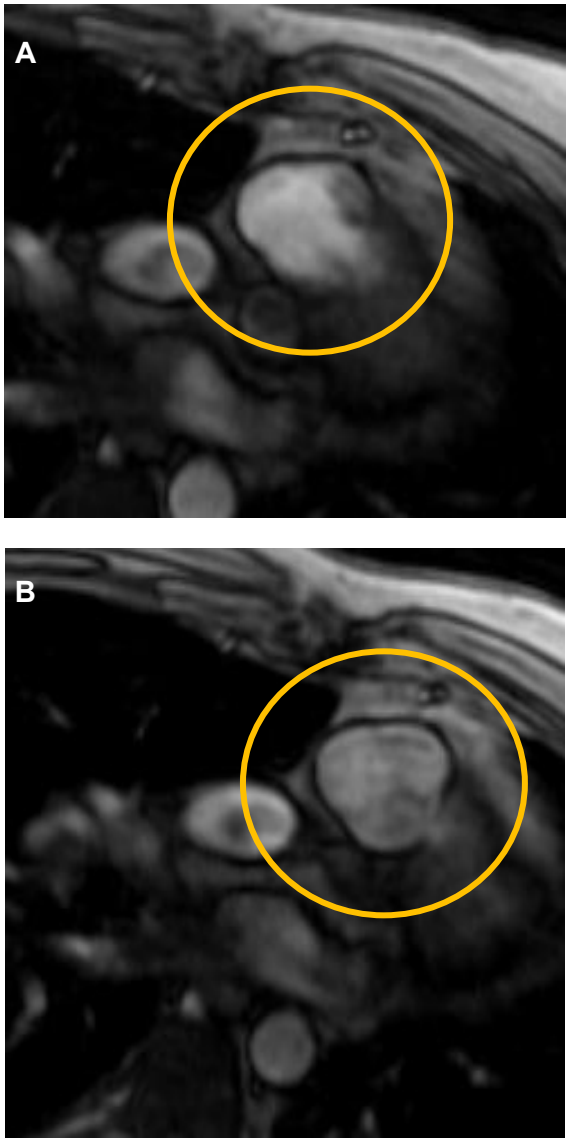

Figure 28. Multiphase phase contrast of the pulmonary valve. (A) Open valve. (B) Closed valve.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

As mentioned previously, phase contrast imaging can characterize not only flow *direction* but also flow *velocity*. The brightness of the flowing blood signal is directly proportional to the amplitude of the flow-encoding gradient. When the optimal VENC is used, flow velocity that most closely matches the amplitude of the flow-encoding gradient exhibits the brightest **voxel** signal. Slower flow is displayed as less bright, and retrograde flow is displayed as dark voxels. Since the brightness of the signal is proportional to the VENC used, it is possible to quantify phase contrast MRA flow velocity (**Figures 28 and 29**).

To measure pressure gradients across a **stenosis**, phase contrast MRI can be used to estimate the maximum blood velocity going through a stenotic vessel, referred to as **vmax**. If vmax is measured in units of meters per second, then the pressure gradient across the stenosis (in units of mmHg) can be estimated to be 4x vmax $(m/sec)^2$.

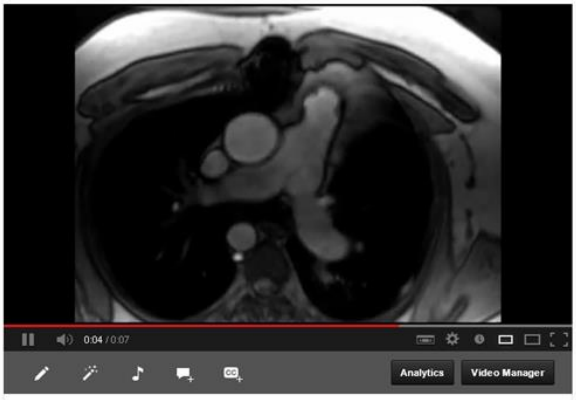

Figure 29. **Movie.** Multiphase phase contrast sequence demonstrating flow through the aortic valve.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Gadolinium Contrast-enhanced Cardiac Imaging

Gadolinium-based contrast agents (GBCAs) play a key role in assessing myocardial health in two important ways: to assess myocardial blood perfusion and to assess myocardial muscle viability using delayed enhancement. Both techniques require the use of a GBCA but employ different imaging methods.

CARDIAC PERFUSION

For the evaluation of myocardial tissue at risk of becoming **ischemic**, an intravenous drug is administered that causes the heart to increase its pumping action, simulating an exercise state. Two such drugs are dobutamine, an agent that increases regional blood flow, and adenosine, a **vasodilator**. After injection of the stress agent, the patient is closely monitored. When sufficient cardiac stress has been achieved, a gadolinium-based contrast agent is injected intravenously. The uptake pattern of the contrast agent to the myocardium can indicate areas of myocardial muscle that are at risk of becoming ischemic.

When acquiring and reconstructing the images rapidly, ischemia-induced abnormalities can be diagnosed with MRI in a manner similar to what is routinely performed using stress echocardiography.

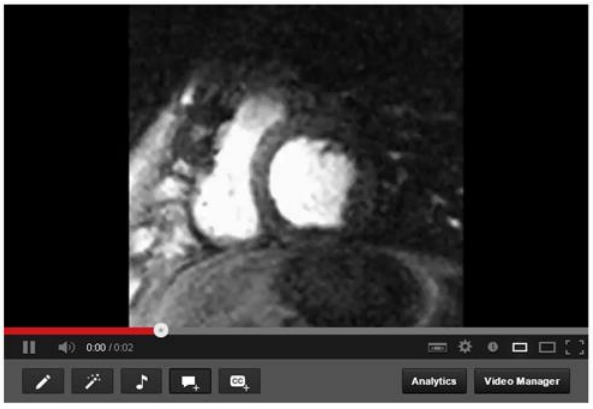

Figure 30. Movie. Perfusion study of the myocardium after GBCA administration. Note areas of perfusion deficit in the left ventricle indicated by the thin dark bands at 4 o'clock and 8 o'clock.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy GE Healthcare.

Perfusion studies examine *variations* in the distribution of the GBCA to identify blood flow patterns and areas of decreased myocardial perfusion. Normally-perfused myocardial tissue accepts a greater volume of contrast than ischemic areas; therefore, normal tissue enhances to a greater degree after contrast agent delivery than ischemic areas (**Figure 30**).

Cardiac perfusion has not only demonstrated the ability to detect myocardial ischemia but also shows potential for quantification of disease. In the clinical setting, cardiac perfusion imaging is usually performed in an inpatient setting where emergency personnel and equipment are readily available in the event of a cardiac incident.

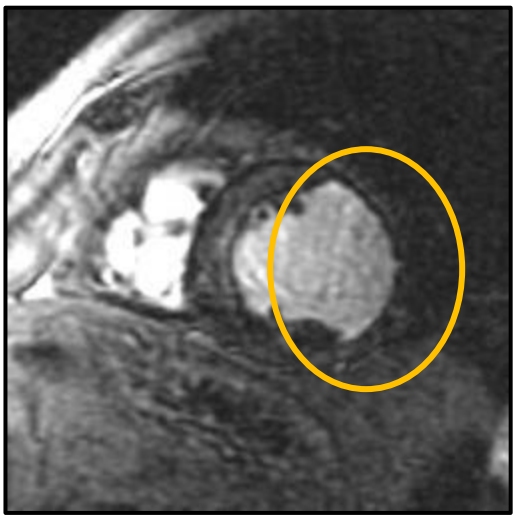

Figure 31. Myocardial delayed enhancement demonstrating area of complete ischemia of left ventricle.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

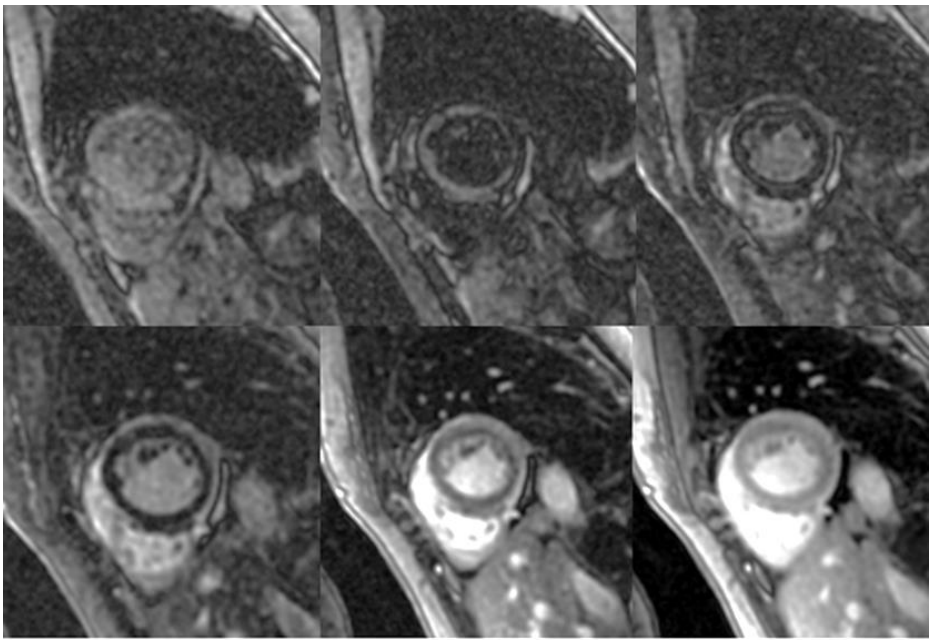

Figure 32. FSE/TSE inversion recovery sequence of the same location using varying TI delay times. This sequence is used to determine the optimal TI time to contrast dark myocardium against a bright-blood pool. In this example, the bottom left image demonstrates the best contrast, with a TI time of 250msec.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

MYOCARDIAL DELAYED ENHANCEMENT

Myocardial delayed enhancement (MDE) is a cardiac imaging method that, like perfusion imaging, uses a GBCA to assess myocardial health. Since a stress agent is not used in MDE, it is a safer technique. Imaging is delayed after contrast injection and is a useful method for assessing myocardial dysfunction and infarction. MDE is used for patients not only with infarction but for patients with other diseases like sarcoidosis that can weaken and damage the heart muscle.

Healthy myocardium is extremely vascular, and the blood flowing through it exhibits a rapid "wash-in/wash-out" effect. Tissue enhancement of the myocardium using a GBCA is rapid and short-lived, that is, the myocardium enhances quickly and also returns to signal equilibrium quickly (**Figure 31**).

Necrotic myocardium or myocardial tissue at risk of becoming ischemic reveals a different enhancement pattern. By definition, ischemic or at-risk tissue has poor blood perfusion. As the contrast agent reaches areas of ischemic or near-ischemic tissue, there will be no rapid wash-out effect. Rather, the contrast agent "seeps" into the ischemic tissue over time. A delay from time of injection to optimal contrast enhancement of the ischemic myocardium is approximately 10 minutes.

To further highlight tissue contrast between healthy and ischemic myocardium, a fast/turbo spin IR sequence is used to nullify healthy myocardium, thereby contrasting diseased myocardium (hyperintense) against healthy myocardium (hypointense).

The most important parameter of the delayed enhancement sequence is the TI time, typically 200-300msec for optimal myocardial suppression. The TI time can vary considerably depending on the length of delay from the time of injection to the delayed-enhancement imaging, optimally 10 minutes. Manufacturers offer methods for quickly determining the optimal TI time for any given patient (**Figure 32**).

PLANES OF ACQUISITION

Although the heart is a complex organ, it is somewhat symmetrical, with two smaller chambers sitting atop two larger chambers separated by a thick strip of muscle, the septum. Using this perspective, localizing for optimal evaluation of the cardiac anatomy might seem relatively simple. However, the heart is not conveniently shaped in **orthogonal** angles.

The heart rests in two different **oblique** planes in the chest. Moreover, the heart is cone-shaped, with the point (the apex) inferior to the base (**Figures 33 and 34)**. Indeed, localizing for optimal slice obliquity can be challenging. Fortunately there are well established guidelines for aiding in localizing the planes of acquisition in cardiac MRI.

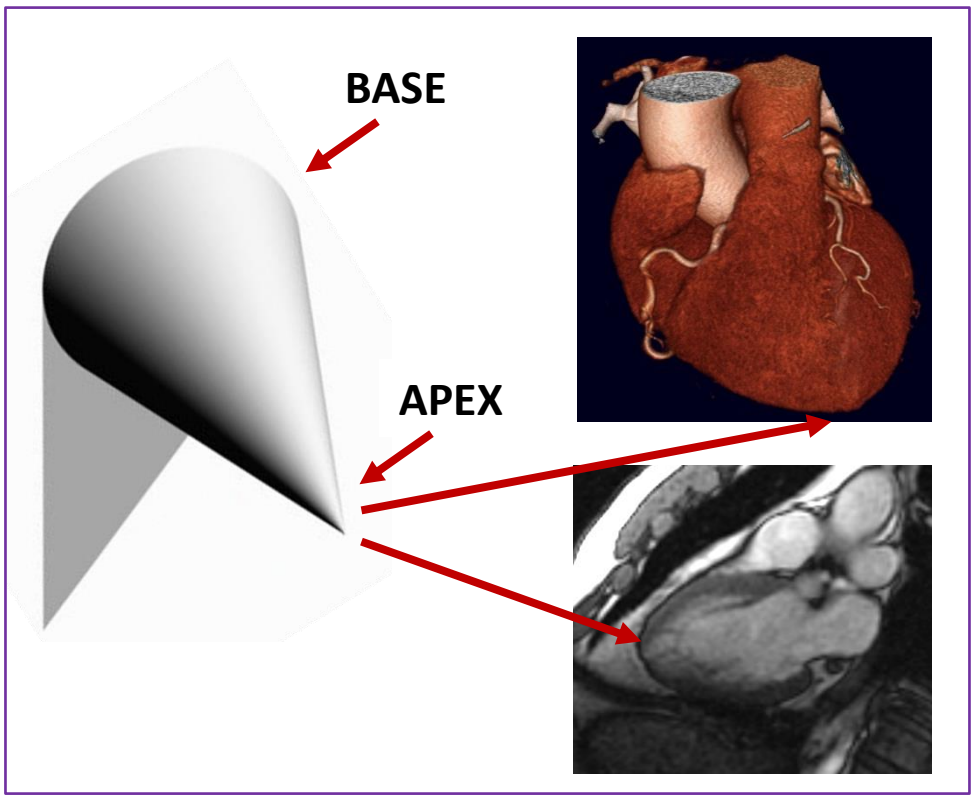

Figure 33. Illustration of the heart. The apex is inferior to the base.

Top right: 3D volume-rendered image of the heart compiled from CT data.

Image courtesy of Vital Images. Inc., Toshiba Medical Systems.

Bottom right: 2-chamber bright-blood image of the heart displaying the left ventricle and atrium.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Cardiac imaging generally employs several standard views of the heart and its structures, with specialized views for particular applications. All cardiac imaging begins with several fast, low-resolution localizers acquired first.

The general views of the heart used in almost all cardiac applications consist of:

- Short Axis View
- Horizontal Long Axis 4-chamber View
- Vertical Long Axis 2-chamber View

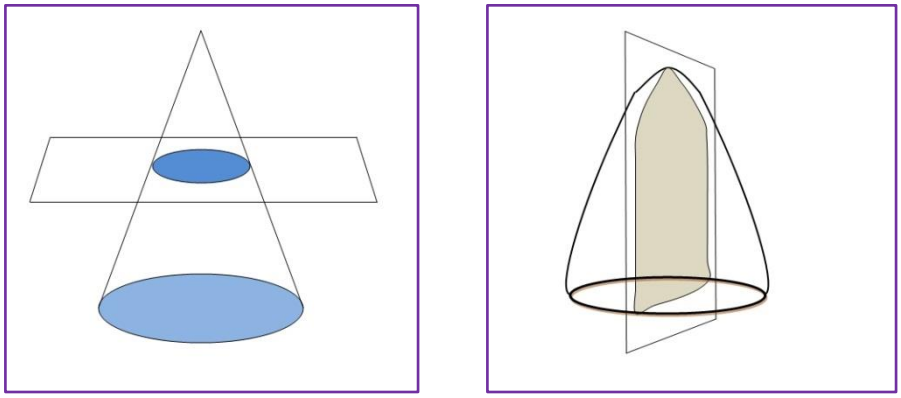

Figure 34. When imaging the heart, the heart is bisected in the same way a cone is bisected into parallel and perpendicular angles as shown above.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

NOTES

Short Axis View

The short axis (SA) view of the heart acquires slices that run perpendicular to the long plane of the left ventricle (**Figure 35**).

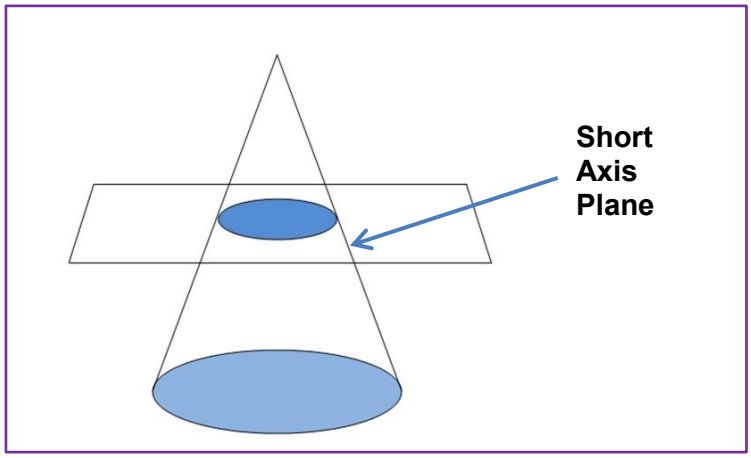

Figure 35. Short axis view.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

Begin by obtaining a 3-plane localization series of the chest (**Figure 36**). Locate an axial view that displays the left ventricle. This scan will run 10-15 seconds. Using ECG-gating, prescribe a single oblique slice through **the center of the ventricle**. This scan is accomplished in a single breath-hold of 10-15 seconds (**Figure 37**). The resultant image is a 2-chamber localizer image of the left ventricle and the left atrium (**Figure 38**).

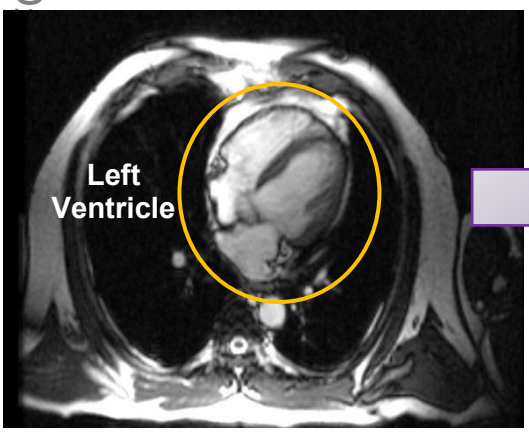

Figure 36. Axial view obtained from a 3-plane localizer displaying the left ventricle.

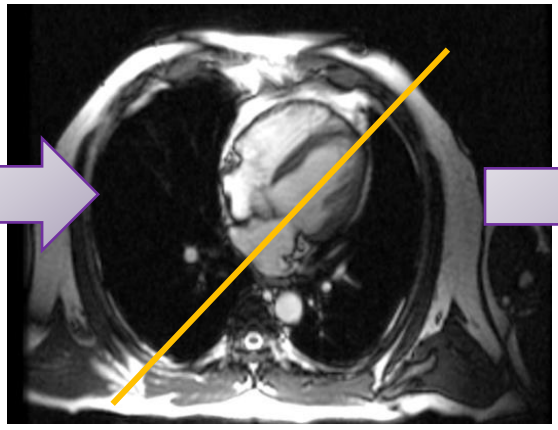

Figure 37. Single oblique slice through the center of the ventricle.

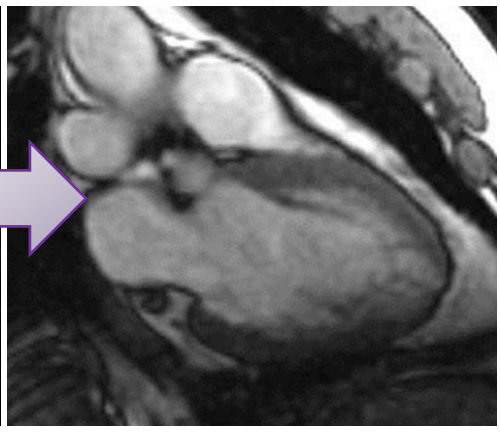

Figure 38. 2-chamber localizer image of the left ventricle and the left atrium.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

From the 2-chamber localizer image, prescribe one ECG-gated slice through the center of the left ventricle perpendicular to the long axis. This scan is a breath-hold of 10-15 seconds (**Figure 39**). The resultant image is a low-resolution short axis view of the right and left ventricles. Note that this view is **not** the higher-resolution short axis used for diagnostic purposes (**Figure 40**).

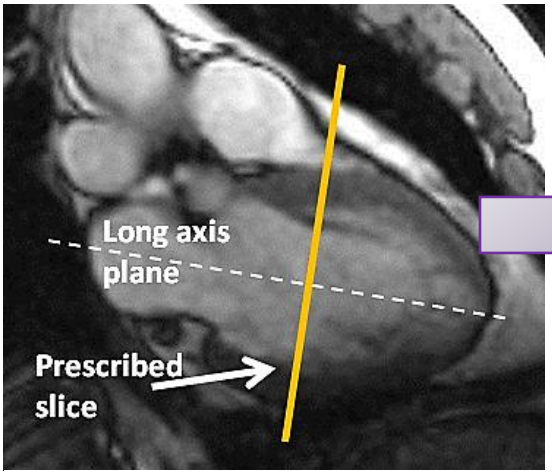

Figure 39. From the 2-chamber localizer image, prescribe a slice through the center of the left ventricle perpendicular to the long axis of the left ventricle.

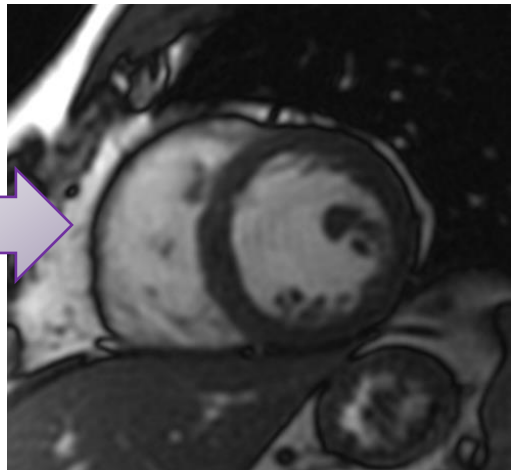

Figure 40. Low-resolution short axis view of the right and left ventricles.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

From the short axis localizer scan, prescribe a single ECG-gated slice through the apex through the center of the left ventricle. This is a 10-15 second breath-hold (**Figure 41**). The resultant image is a low-resolution horizontal long axis view of all four chambers of the heart. Note that this is **not** the long axis view used for diagnosis (**Figure 42**).

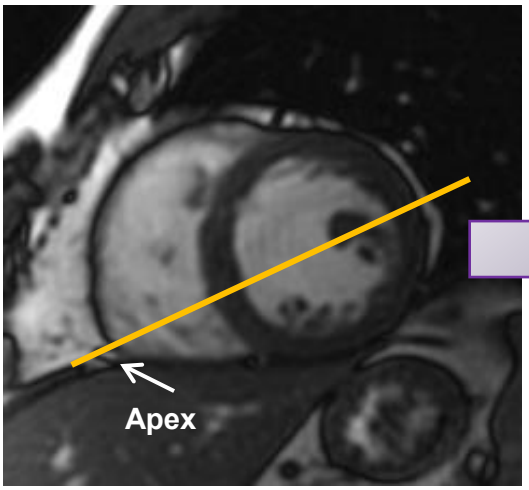

Figure 41. Slice through the apex and center of the left ventricle.

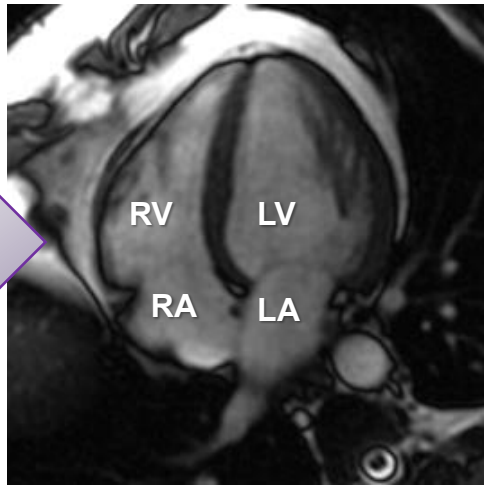

Figure 42. Low-resolution horizontal long axis view of all four chambers of the heart.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Prescribe the higher-resolution diagnostic short axis series perpendicular to the long plane of the left ventricle from the apex through the mitral valve. This view is used for single-phase and multiphase steady-state imaging (FIESTA, FISP, balanced FFE) as well as for black-blood imaging (**Figure 43**). The result is a stack of high-quality short axis images through the ventricles from the apex through the mitral valve. These are bright-blood images using steady-state imaging (**Figures 44 and 45**).

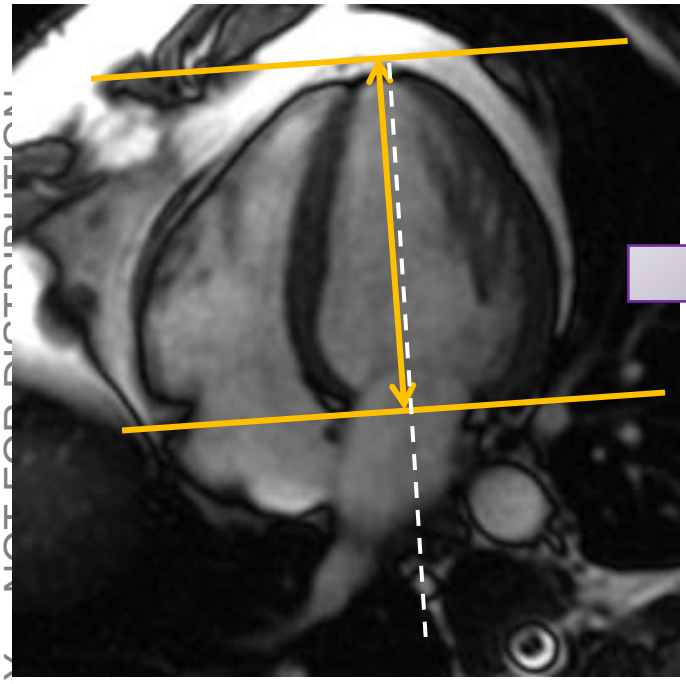

Figure 43. Short axis series prescribed perpendicular to the long plane of the left ventricle from the apex through the mitral valve.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

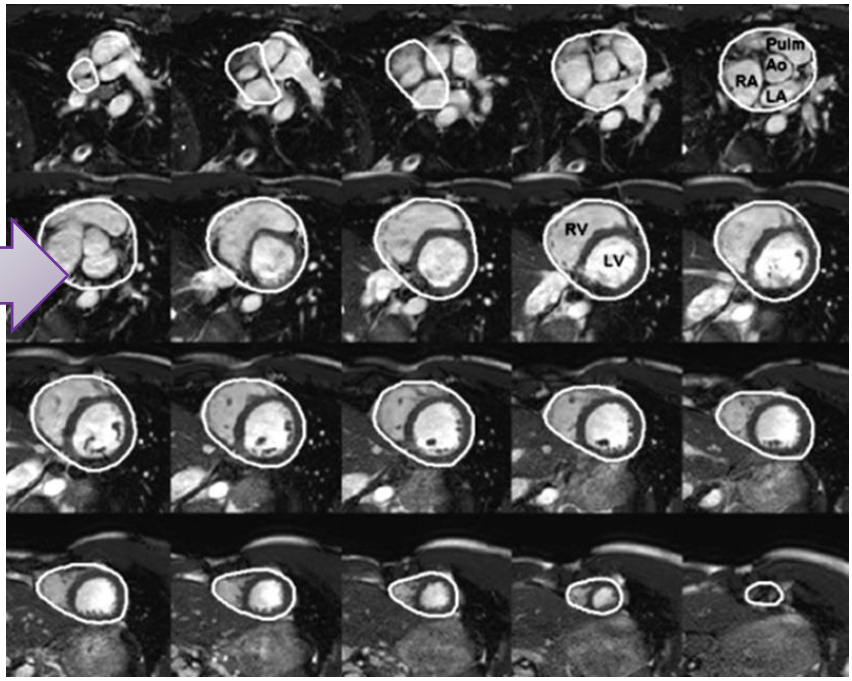

Figure 44. Stack of high-quality short axis images through the ventricles from the apex through the mitral valve.

Courtesy GE Healthcare.

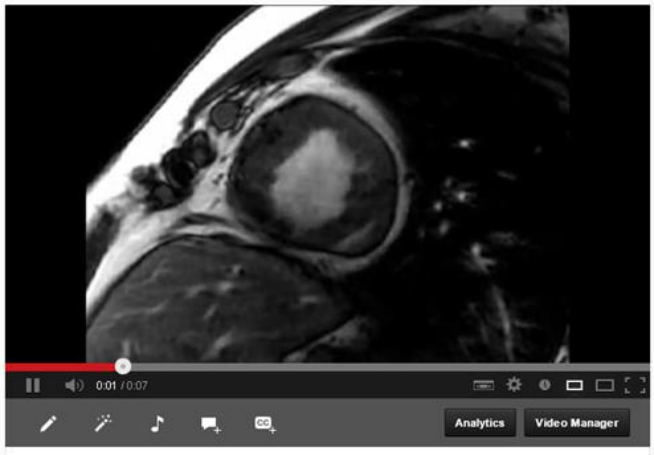

Figure 45. **Movie.** Short axis bright-blood steady-state FIESTA of the left ventricle.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Horizontal Long Axis 4-Chamber and Vertical Long Axis 2-Chamber Views

The long axis (LA) view of the heart acquires slices that run parallel to the long plane of the left ventricle (**Figure 46**).

To obtain high-quality long axis views of the heart, begin with the same localizers that have already been acquired for the short axis views *(refer to Figure 36)*.

Using a 2-chamber view, prescribe the short axis. It will be from this short axis view that the 4-chamber long axis view is prescribed (**Figure 47**).

Long axis views, whether horizontal or vertical, are always prescribed off the short axis view. The operator should determine whether to use the SA localizer or the diagnostic SA series. Choose whichever SA view most clearly displays the cardiac anatomy.

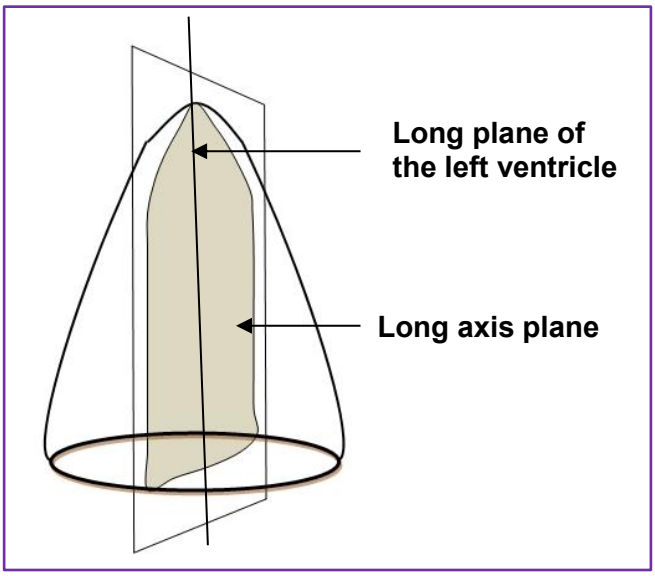

Figure 46. Long axis view of the heart.

Courtesy of Tom Schrack, ARMRIT, Fairfax Radiological Consultants.

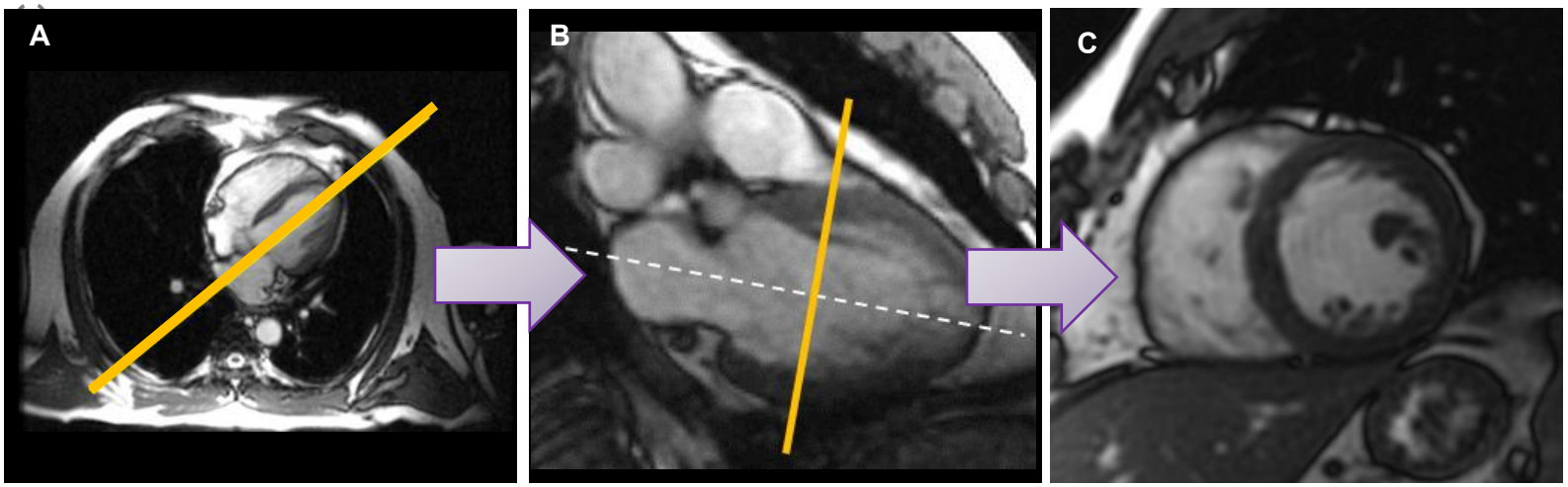

Figure 47. (A) SA axial 3-plane localizer. (B) 2-chamber view. (Ct) Short axis view.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

PRESCRIBING THE HORIZONTAL LONG AXIS 4-CHAMBER VIEW

Prescribe the horizontal long axis view (HLA) in the using the SA view *(refer to Figure 47C)*. Angle the prescription from the apex through the center of the left ventricle and scan through heart as shown (**Figure 48**). The resultant image is a horizontal long axis 4-chamber view displaying the left and right ventricles and the left and right atria (**Figures 49 and 50**).

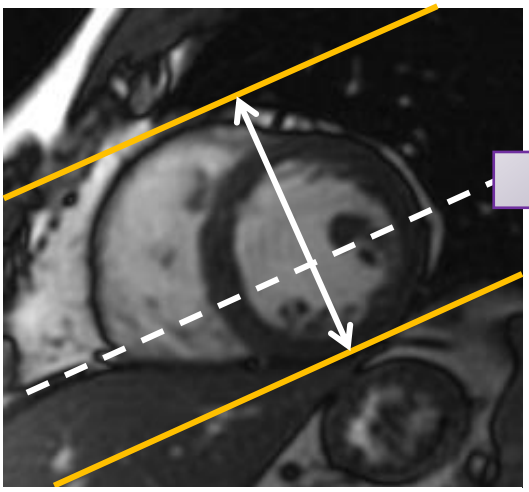

Figure 48. Angle the prescription from the apex through the center of the left ventricle and scan through heart as shown (yellow lines).

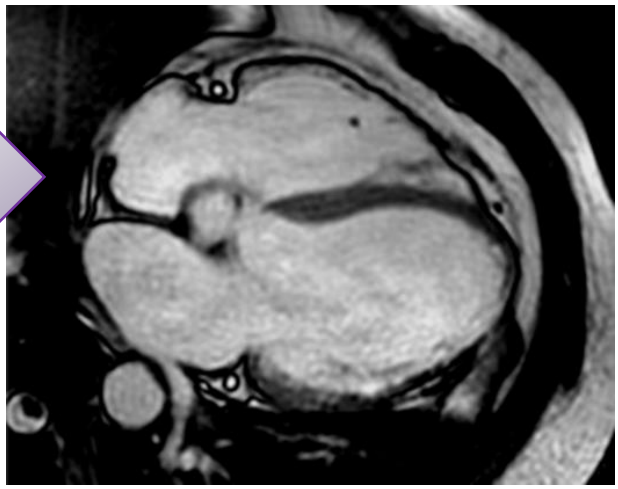

Figure 49. Horizontal long axis 4-chamber view of the heart displaying the left and right ventricles and the left and right atria.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

PRESCRIBING THE VERTICAL LONG AXIS 2-CHAMBER VIEW

To prescribe the vertical long axis view (VLA) of the left ventricle, an SA view is used *(refer to Figure 47C)*. This prescription differs from the HLA in that the slice angle is vertically prescribed as shown (**Figure 51**). The resultant image is the vertical long axis 2-chamber view of the left ventricle and left atrium. Note the mitral valve is also visible (**Figures 52 and 53**).

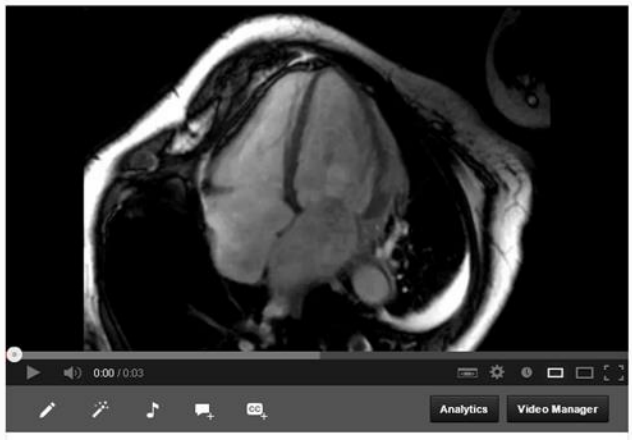

Figure 50. Movie. Horizontal long axis view demonstrating left and right ventricles and left and right atria.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

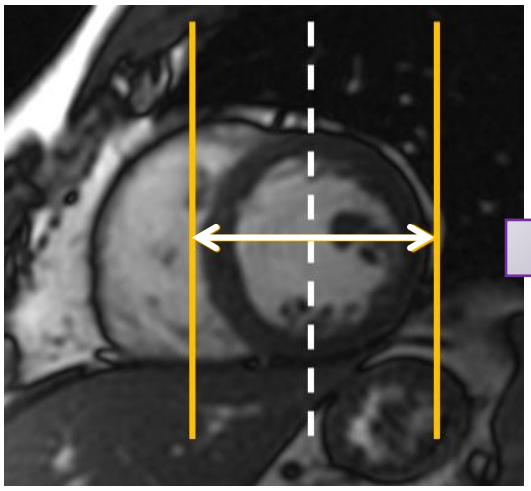

Figure 51. Short axis view displaying the prescription for the vertical long axis 2-chamber view.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

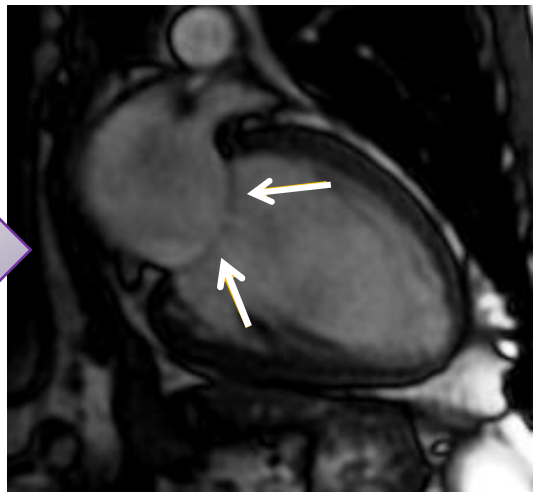

Figure 52. Vertical long axis 2-chamber view of the mitral valve (arrows), left ventricle, and left atrium.

LEFT VENTRICULAR OUTFLOW TRACT VIEW

When evaluating pathologies of the ascending aorta such as an aortic coarctation (a "kink"), aortic stenosis, or aortic valve insufficiency, it is routine to obtain a view of the left ventricular outflow tract (LVOT). The LVOT is the pathway of flowing blood from the left ventricle through the aortic valve into the ascending aorta. Obtaining this view is done with an ECG-gated multiphase steady-state technique (FIESTA, FISP, balanced FFE) that is angled to demonstrate this pathway during the cardiac cycle.

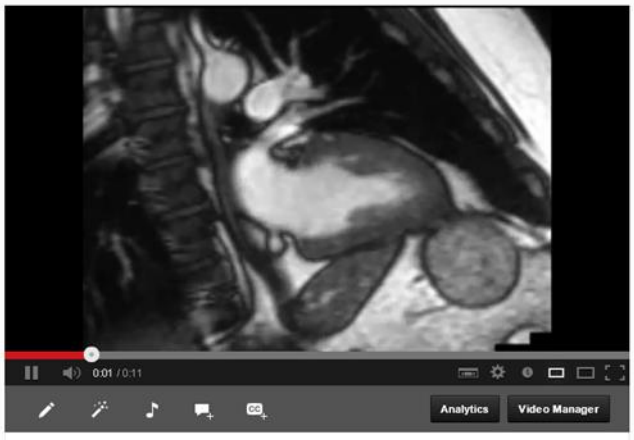

Figure 53. Movie. Vertical long axis bright-blood FIESTA cine of the left ventricle and left atrium.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

To obtain the LVOT view, begin with the SA view that demonstrates the most superior portion of the left ventricle as well as the inferior view of the left atrium. This view is typically referred to as the "snowman" view due its resemblance to a snowman (**Figure 54**). The snowman view reveals the superior aspect of the left ventricle and the inferior aspect of the left atrium. Prescribe a single slice bisecting the snowman view (**Figure 55**). The resultant view displays the atrial inflow tract of the left atrium, mitral valve, and left ventricle (**Figure 56**).

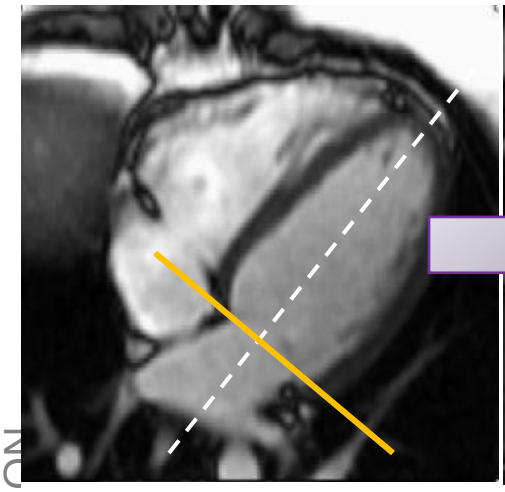

Figure 54. SA view location where the "snowman" view is demonstrated (yellow line).

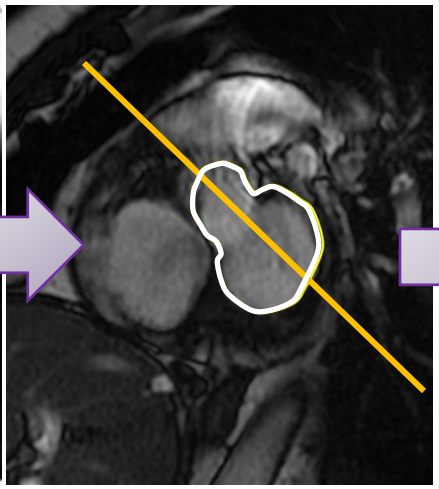

Figure 55. Snowman view revealing the superior aspect of the left ventricle and the inferior aspect of the left atrium.

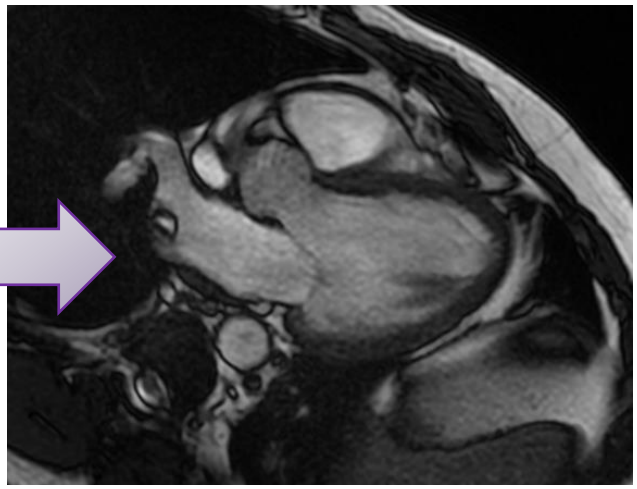

Figure 56. Atrial inflow tract of the left atrium, mitral valve, and left ventricle.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Prescribe a single-slice ECG-gated multiphase steady-state image through the left ventricle extending into center of the ascending aorta (**Figure 57**). The resultant image displays the left ventricular outflow tract demonstrating the ascending aorta, aortic valve, and left ventricle (**Figure 58 and 59**).

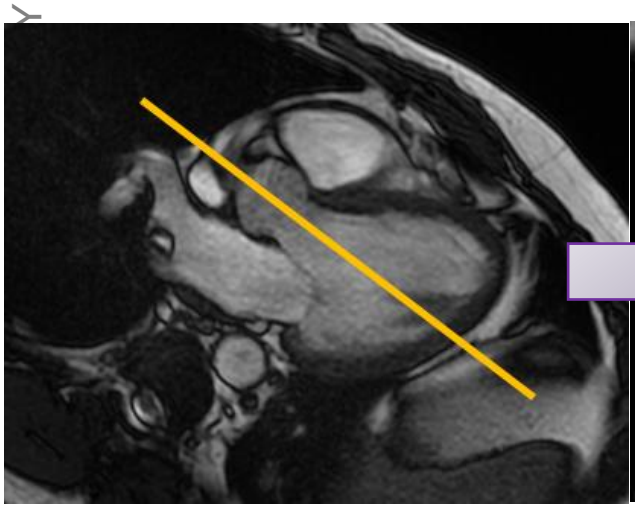

Figure 57. Prescribe a single-slice ECG-gated multiphase steady-state image through the left ventricle extending into the center of the ascending aorta.

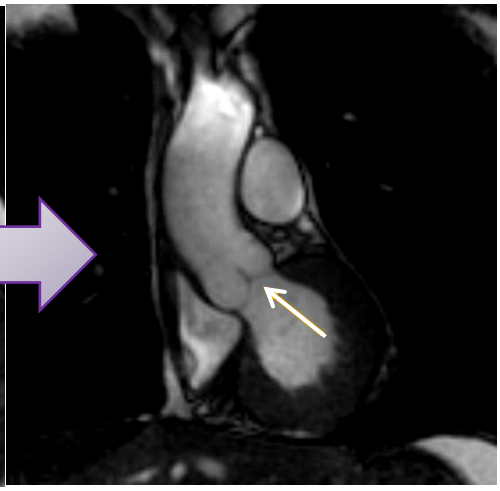

Figure 58. Left ventricular outflow tract view demonstrating the ascending aorta, aortic valve (arrow), and left ventricle.

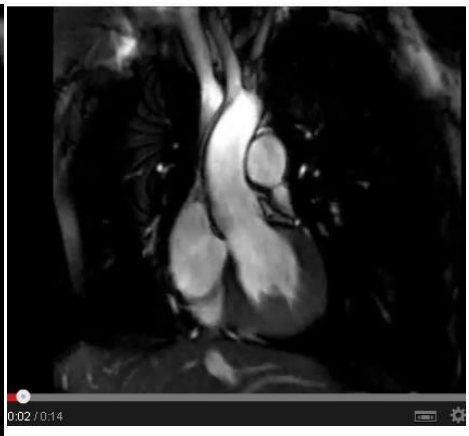

Figure 59. Movie. Left ventricular outflow tract view.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

LOCALIZING FOR ASCENDING AORTA PHASE CONTRAST IMAGING

When aortic valve insufficiency is suspected or aortic stenosis is present, quantifying the blood flow through the aortic valve is often required. To obtain reliable quantified data that reports flow velocity and direction, remember that 2D phase contrast imaging is used. Correct localization of the ascending aorta just distal to the aortic valve is essential and fortunately is fairly straightforward.

Begin with an axial localizer that displays the pulmonary artery and the bifurcation into the left and right pulmonary arteries and demonstrates the best cross-sectional view and location of the ascending aorta (**Figure 60**). Note the location on the image from the annotated RAS coordinates which are automatically displayed on all images: R= right/left; A= anterior/superior; S= superior/inferior. For example: “S30” means superior 30mm from the landmark at the beginning of the exam. Using the slice location, eg, S30, prescribe an axial ECG-gated phase contrast slice at the S30 location. Use the optimized shimming capabilities of your MR system for any PC imaging (**Figure 61**). The resultant image is a multiphase phase contrast image set that yields quantitative blood flow information through the ascending aorta just distal to the aortic valve (**Figure 62**).

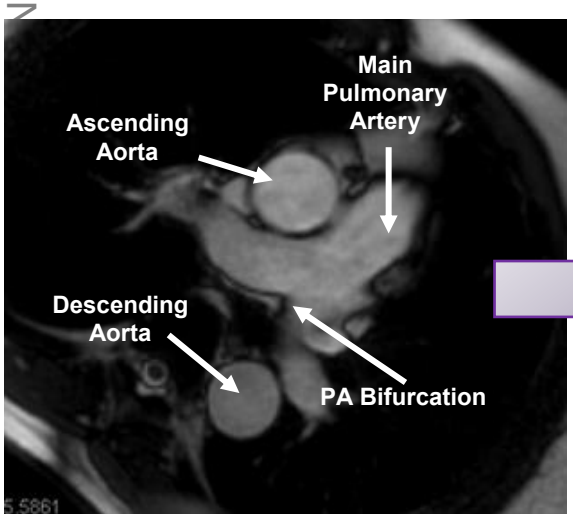

Figure 60. Axial localizer. Bright-blood image (FIESTA) demonstrating the ascending and descending aorta at the level of the main pulmonary artery and its bifurcation.

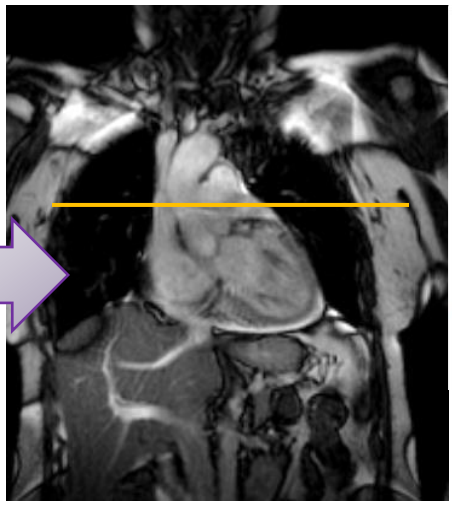

Figure 61. Using the slice location noted from the localizer image, prescribe an axial ECG-gated phase contrast slice.

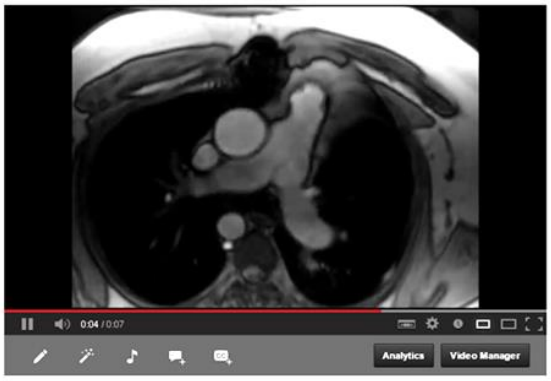

Figure 62. Movie. Multiphase phase contrast image set yielding quantitative blood flow information through the ascending aorta just distal to the aortic valve.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Localizing for Pulmonary Artery and Pulmonary Valve Phase Contrast Imaging

Phase contrast imaging of the pulmonary valve and pulmonary artery is an essential component of cardiac MRI applications such as for Tetralogy of Fallot. The quantification of blood flow through the valve and arteries is a key knowledge point required for complete diagnosis and treatment decisions.

To properly localize for the pulmonary artery and valve, first acquire an oblique sagittal view of the pulmonary artery. This can be easily accomplished by prescribing one slice off the axial view of the heart that demonstrates the pulmonary bifurcation (**Figure 63**). The resultant localizer image demonstrates the main pulmonary artery in profile (**Figure 64**). From the localizer image, prescribe the phase contrast image at the valve plane that runs perpendicular to the long plane of the pulmonary artery (**Figure 65**). The resultant image is a phase contrast image of the pulmonary valve (**Figure 66**).

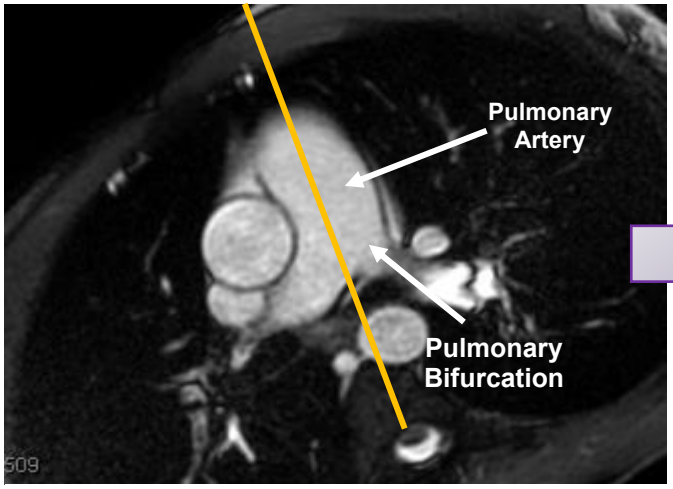

Figure 63. Axial slice demonstrating the pulmonary artery and bifurcation (arrows). Prescribe a slice that is parallel with the long plane of the artery (line).

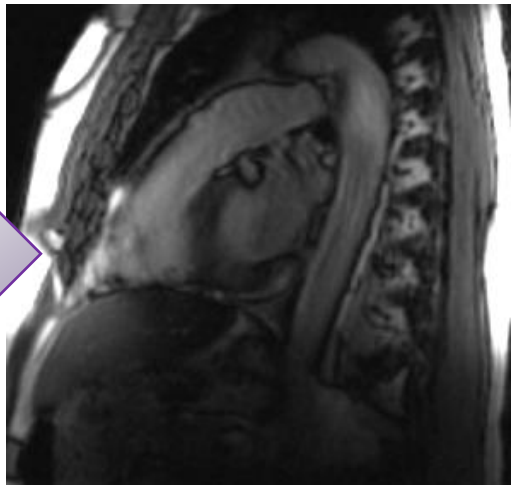

Figure 64. Main pulmonary artery (in profile).

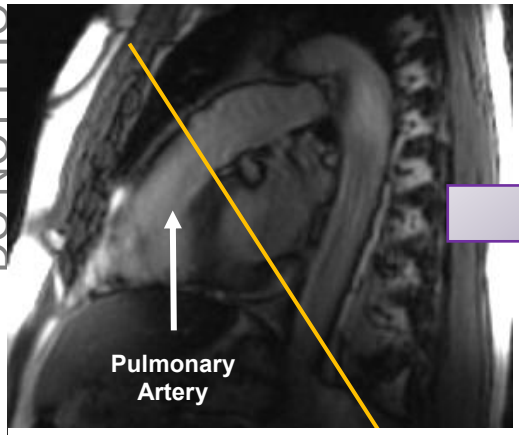

Figure 65. Prescribe the phase contrast image at the valve plane that runs perpendicular to the long plane of the pulmonary artery (line).

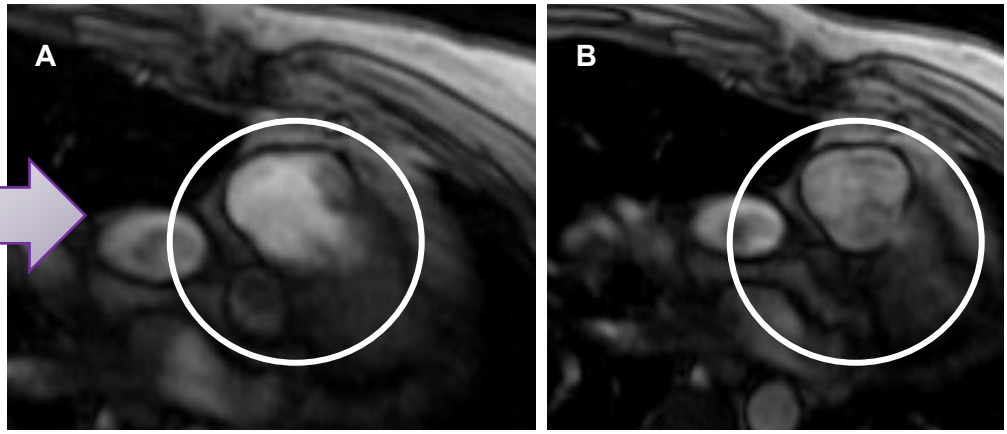

Figure 66. Multiphase phase contrast of the pulmonary valve. (A) Open valve. (B) Closed valve.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

PULMONARY ARTERY BRANCHES PHASE CONTRAST IMAGING

To prescribe phase contrast images of the left and right pulmonary artery branches, use the same axial localizer used to prescribe the sagittal pulmonary artery localizer *(refer to Figure 60)*. Prescribe phase contrast images through the left and right branches to provide cross-sectional views of each branch as shown below (**Figure 67 and Figure 68**).

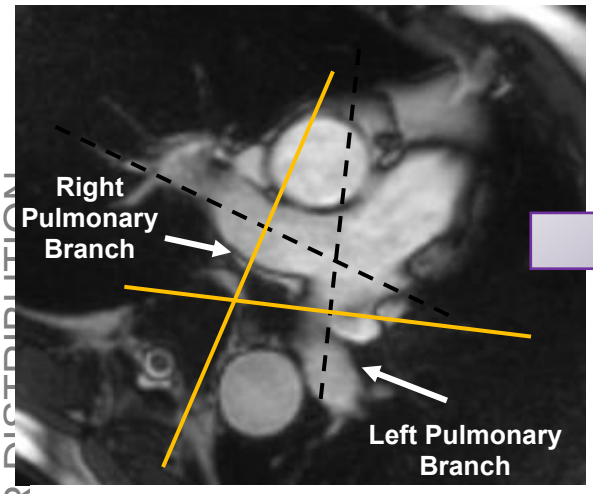

Figure 67. Prescription lines for cross-sectional views of the left and right pulmonary arteries.

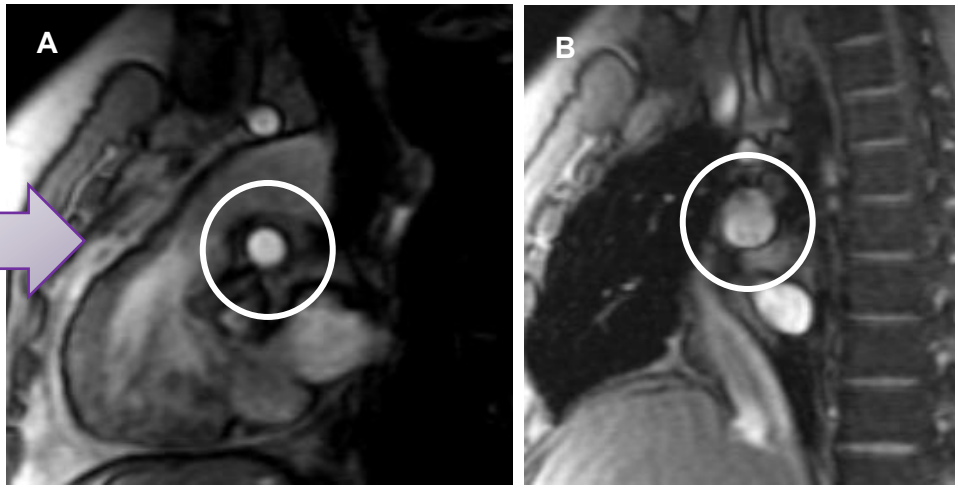

Figure 68. Phase contrast image of the (A) left pulmonary branch and (B) right pulmonary branch

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

APPLICATIONS FOR CARDIAC MRI

Applications for cardiac imaging are as varied as the pathologies that arise in the heart itself. To be sure, there are numerous imaging modalities that provide anatomical and functional information about the heart. As examples, stress echo ultrasound is commonly used to assess valvular pathologies as well as myocardial function; CT angiography (CTA) of the coronary arteries yields excellent information about soft and hard plaque in and around the **lumen** of the coronary arteries; and stress perfusion nuclear medicine demonstrates myocardial dysfunction while the heart is under stress. With the possible exception of CTA of the coronary arteries, magnetic resonance imaging of the heart can provide much of, if not all, the same information as these other modalities.

This section describes some the major applications for cardiac MRI and what specific cardiac sequence is best-suited for answering the clinical question.

Left Ventricular Function

The left ventricle is responsible for pumping oxygenated blood from the heart to the rest of the body. In order to accomplish this monumental task, the left ventricle contains far more myocardial muscle and mass than the right ventricle and both atria. Accessing the health of the left ventricle is essential for determining the overall health of the heart. Optimal left ventricular (LV) function can be undermined by numerous conditions and pathologies. Age, obesity, hypertension, and coronary artery disease are common causes of diminished LV function. Infiltrative processes such as amyloidosis and sarcoidosis, though rare, lead to markedly impaired LV function[2].

Assessing LV function is done both subjectively and objectively. Multiphase bright-blood steady-state imaging provides excellent direct visual evidence of the pumping action of the left ventricle in real time. More objectively, it is possible — and quite simple — to assign a numerical value to the efficiency of the left ventricle. This value is commonly known as the left ventricular ejection fraction (LVEF) (**Figure 69)**.

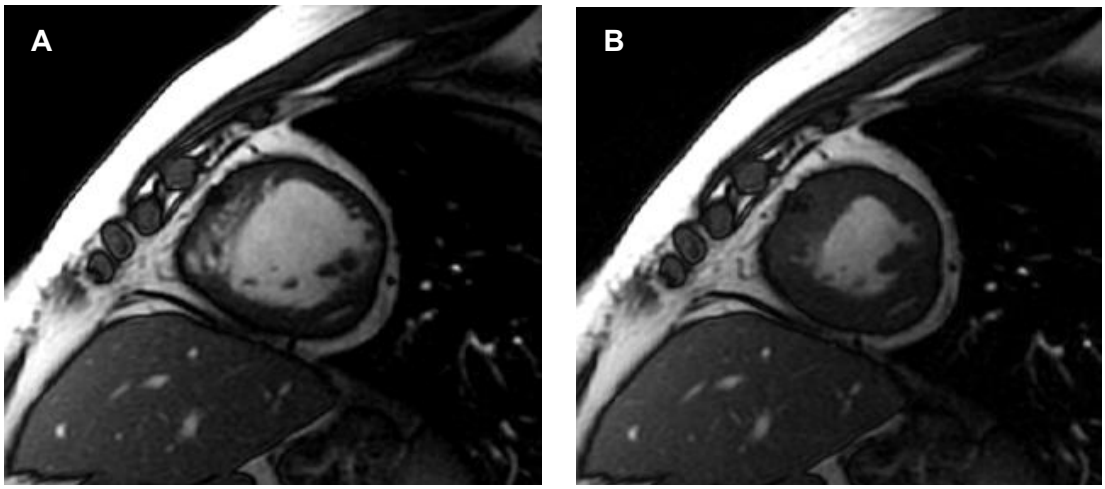

Figure 69. Short axis bright-blood steady-state images demonstrating end diastole and end systole. (A) LV at end (peak) diastole when the LV is filled with blood to capacity. (B) LV at end (peak) systole when the LV is at its peak constriction pushing (ejecting) blood from the ventricle through the aortic valve to the body via the aorta.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

EJECTION FRACTION

Ejection Fraction Measurement	What it Means
55-70%	Normal
40-55%	Below Normal
Less than 40%	May confirm diagnosis of heart failure
<35%	Patient may be at risk of life-threatening irregular heartbeats

Table 2. Normal-to-abnormal LV ejection fractions [3].

The ejection fraction (EF) is defined as the volume of blood in the ventricle at end diastole, when the ventricle has the maximum amount of blood in the chamber, compared to end systole, when the ventricle has the least amount of blood in the chamber. The difference, or ratio, between the two volumes yields the ejection fraction, represented as a percentage (**Table 2**).

Most MR manufacturers and numerous analysis software vendors provide automated programs for segmenting the ventricle to calculate the ejection fraction for any patient. Typically the ventricle is imaged from apex through the aortic valve using a bright-blood steady-state technique with 16-22 cardiac phases for each location (**Figures 70 and 71**).

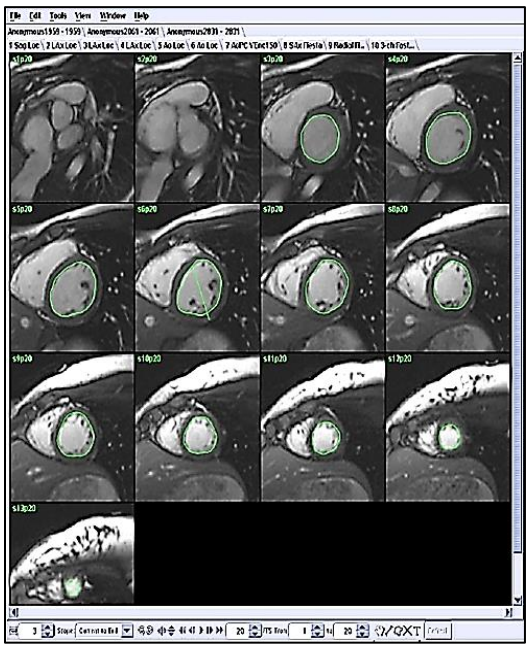

Figure 70. Multiple locations of the left ventricle during a single cardiac phase as displayed on a postprocessing work station. Cardiac processing software greatly aids in the automated edge detection (green circles) of the pericardium and endocardium of the LV.

Courtesy of GE Healthcare.

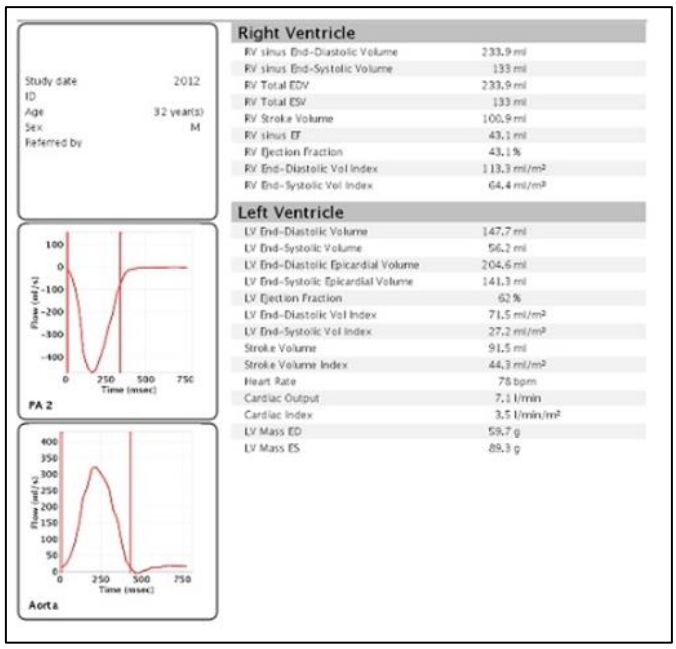

Figure 71. Example of an automatically derived cardiac function report. While automated segmentation is more time efficient, manual correction and editing of ventricle boundaries is critical for accurate reporting.

Courtesy of GE Healthcare.

Tetralogy of Fallot

Tetralogy of Fallot (TOF) is a rare congenital defect of the heart that typically encompasses the following four abnormalities (**Figure 72**).

1. Moderate-to-severe narrowing of the pulmonary valve and outflow tract
2. Displacement of the ascending aorta and aortic valve toward midline into the cardiac septum
3. Abnormal thickening of the right ventricle
4. An ventricular septal defect (an opening in the cardiac septum that separates the right and left ventricles)

These defects result in poor oxygenation of the blood to the body to the point where the infant experiences dyspnea (shortness of breath) and cyanosis (bluish coloration of the finger and toenail beds, as well as around the mouth). Surgical repair in the first years of life dramatically improves symptoms. Progress of the patient is followed through adolescence and the teen years in the event additional surgery is required. With corrective surgery, the prognosis is excellent, with 90% of infants reaching adulthood and leading normal, active lives. Without corrective surgery, death typically occurs by the age 20[4].

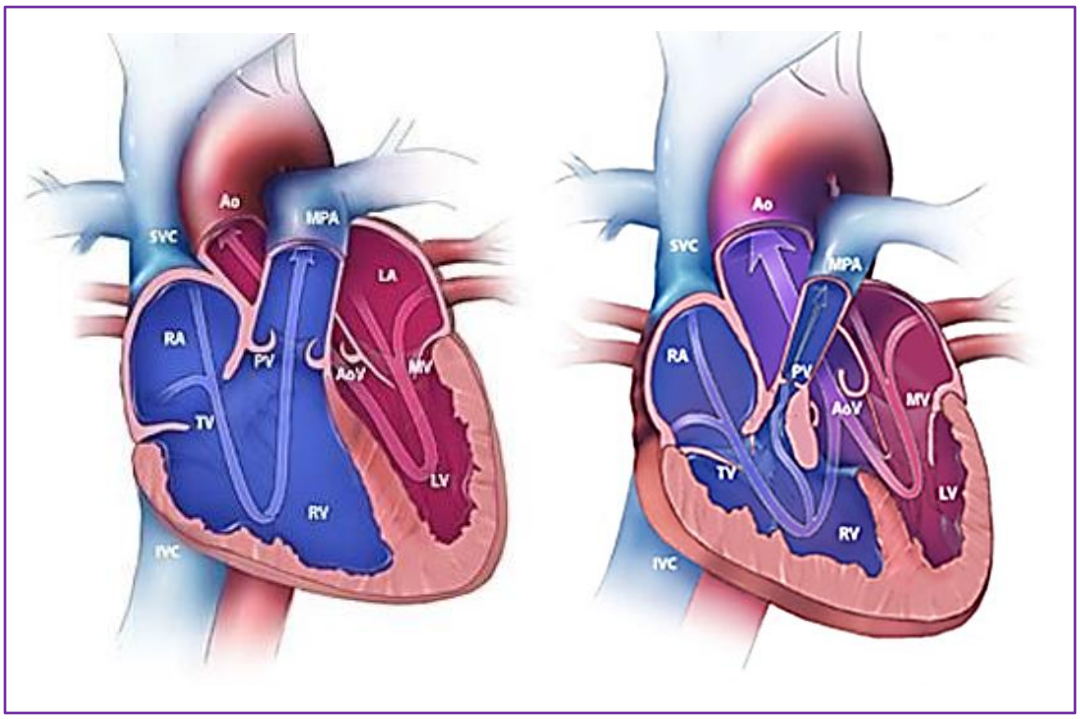

Figure 72. (Left) Normal heart anatomy. (Right) Tetralogy of Fallot.
Available at Centers for Disease Control and Prevention.

Cardiac MRI of Tetralogy of Fallot typically includes bright-blood short axis and long axis views; phase contrast imaging of the pulmonary valve, the left and right pulmonary branches, and the aortic valve; and MR angiography of the pulmonary artery and its branches (**Figures 73 and 74**).

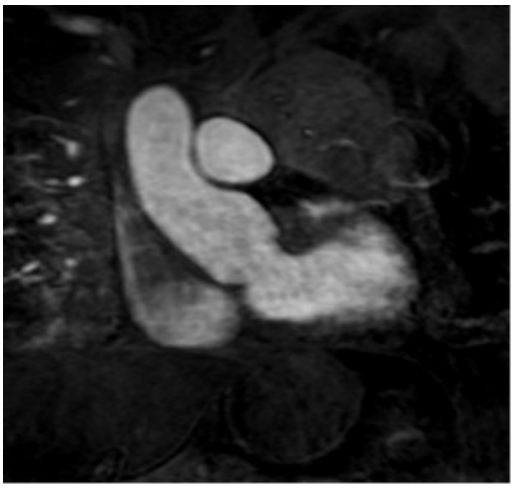

Figure 73. MRA of the pulmonary artery and ascending aorta of a 33-year-old male with corrected TOF. Though the aorta measures prominently at 42mm, it is stable compared to previous examinations. The pulmonary artery also remains stable at 37mm.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

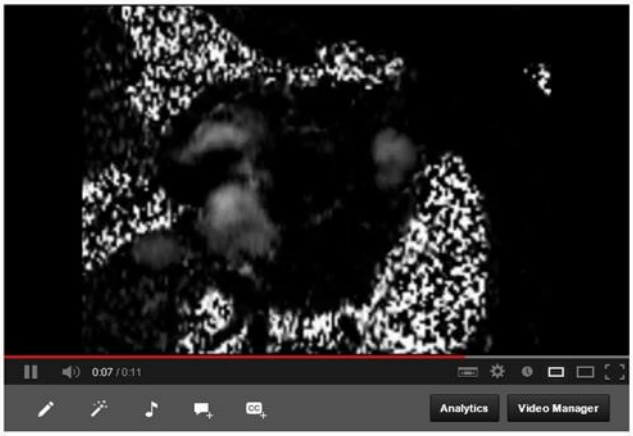

Figure 74. Movie. Same patient. Phase contrast imaging of the main pulmonary artery. Quantitative analysis revealed blood flow velocity of 130cc/sec. Ejection fraction of the left ventricle was measured at 62%, well within the normal range.

Click here to view this **movie** on the ICPMEducation channel.

Myocardial Infarction

Infarction of the coronary arteries occurs when blood flow through an artery becomes blocked, preventing oxygen from reaching the myocardial muscle (**Figure 75**). As with any tissue, muscle dies from lack of oxygen. A mild myocardial infarction can actually result in no permanent damage to the myocardium. A moderate MI results in partial ischemia, with the remaining healthy myocardium continuing to adequately pump blood to the body. In the event of a severe MI, a majority of the myocardium dies, resulting in the cessation of all cardiac pumping action and death.

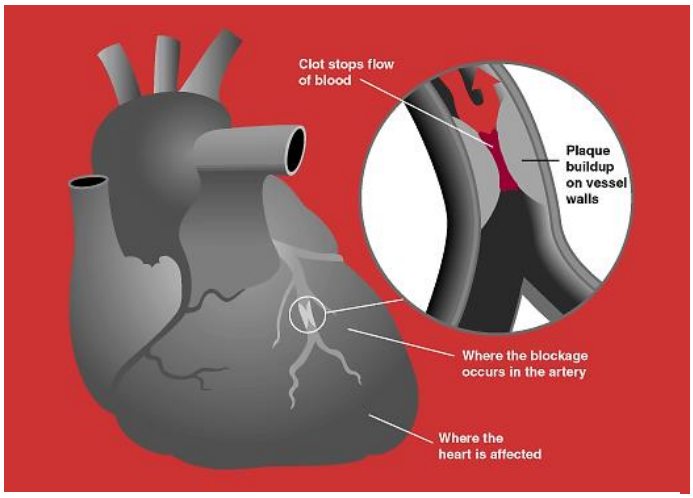

Figure 75. Anatomy of a myocardial infarction.

Available at Wikimedia.

MRI offers great value for accessing the extent of myocardial damage in a post-MI patient. Using bright-blood imaging, the motion of the ventricles indicates location and extent of the heart's ability to continue pumping blood. Black-blood imaging aids in accessing the thickness of the ventricles, which can indicate abnormal stress on the heart. Myocardial delayed enhancement imaging is critical for evaluating the location and extent of myocardial muscle ischemia. Stress perfusion MRI aids in the assessment of myocardial tissue *at risk* of becoming ischemic (**Figures 76, 77, and 78**).

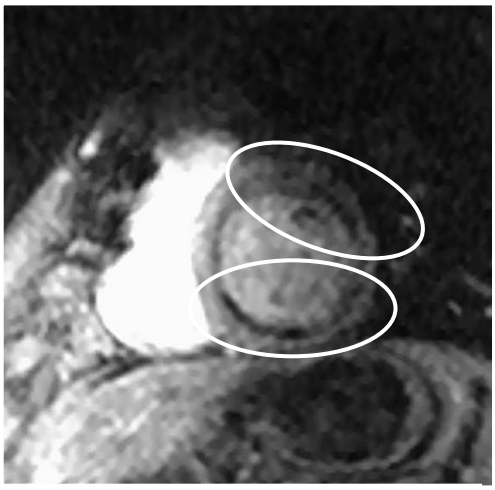

Figure 76. Multiphase cardiac perfusion after GBCA injection following the administration of the pharmacological stress agent dobutamine. Note dark areas of hypointensity demonstrating perfusion deficit.

Courtesy GE Healthcare

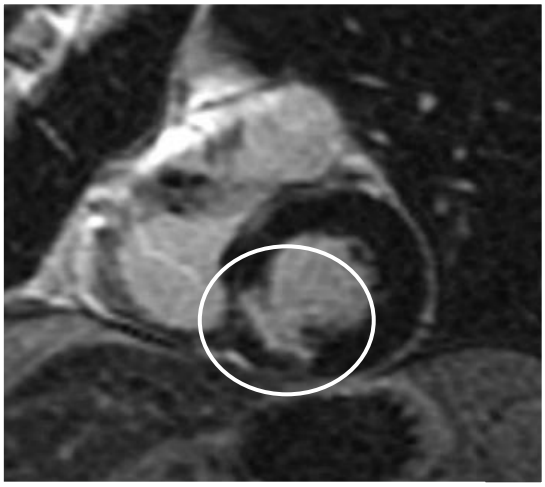

Figure 77. Myocardial delayed enhancement demonstrating ischemic myocardium.

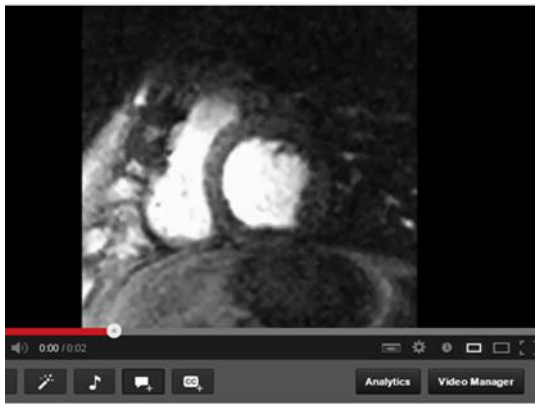

Figure 78. Movie. Multiphase short axis cardiac perfusion cine after injection of a pharmacologic stress agent that demonstrates myocardial muscle at risk of ischemia.

Click here to view this **movie** on the ICPMEducation channel.

Cardiac Amyloidosis

Amyloidosis is an abnormal build-up of amyloid protein that can occur anywhere in the body, including the myocardium. This protein build-up in the myocardial tissue gradually replaces healthy myocardial muscle, dramatically reducing the heart's ability to contract properly. Cardiac amyloidosis is often referred to as "stiff heart syndrome." Symptoms of cardiac amyloidosis include shortness of breath, heart **palpitations**, difficulty breathing when reclined, and frequent urination at night. Treatment may include a low-salt diet and the use of diuretics and calcium blockers. In the most severe cases, heart transplant may be considered. Regardless of treatment, cardiac amyloidosis is a chronic and deadly disease. The average life span from time of diagnosis to death is one year[5].

On MR imaging, the ventricles appear enlarged with a thickened myocardium. Delayed enhancement imaging demonstrates marked enhancement (**Figure 79**).

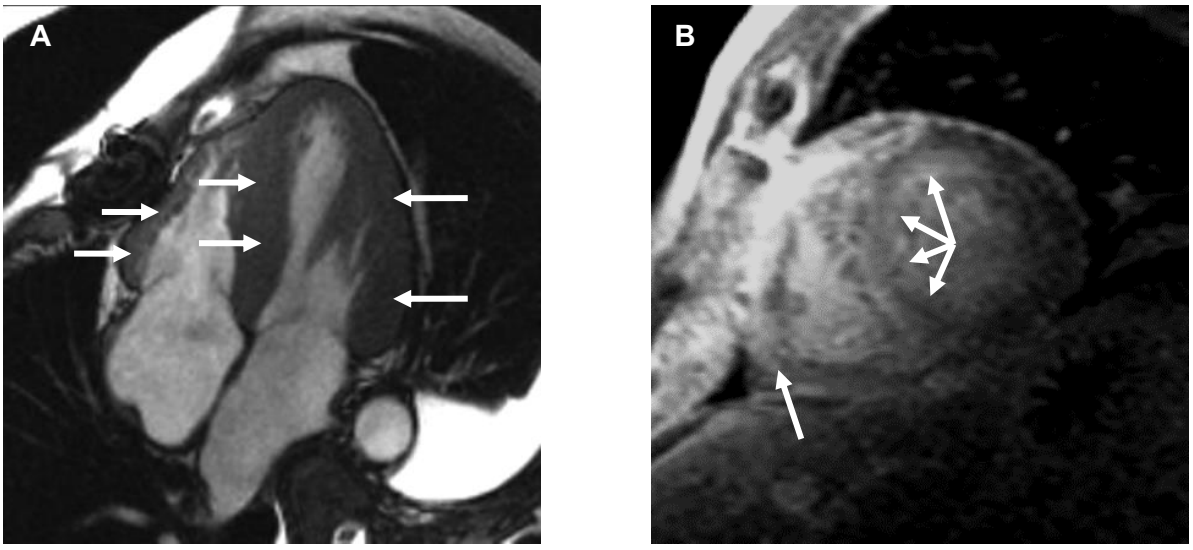

Figure 79. A 61-year-old male with cardiac amyloidosis. (A) Horizontal long axis 4-chamber bright-blood view. Note the markedly thickened myocardium. (B) Multiphase delayed enhancement. The myocardium of both the left and right ventricles demonstrates marked diffuse enhancement.

Courtesy of RI vanden Driesen.
vanden Driesen RI, Slaughter RE, Strugnell WE. MR Findings in Cardiac Amyloidosis. AJR *Am J Roentgenol*. 2006;186:1682-1685.

Cardiac Sarcoidosis

Sarcoidosis is a rare disease of unknown origin. Like amyloidosis, sarcoidosis can affect any organ in the body and is marked by increasing deposits of granulomas, inflammatory cells that are formed in organs. While there is no definitive cause of sarcoidosis, current evidence points to genetic variables along with environmental factors. African Americans are by far the most common ethnic group affected by sarcoidosis. Sarcoidosis can be a benign or life-threatening condition. Recent studies have shown that the mortality rate for patients with cardiac sarcoidosis is greater than 50%[6].

Twenty-five percent of all sarcoidosis cases involve the heart. Symptoms include impaired cardiac function, **syncope**, fluid build-up in the pericardium, and sudden death. Treatments are based on severity of disease and include the use of corticosteroids and other anti-inflammatory medications[6,7].

Findings of cardiac sarcoidosis on MRI include focal areas of hyperintensity on T2-weighted imaging as well as diffuse enhancement on postcontrast delayed enhancement imaging. Myocardial thickening and contraction abnormalities are also common and best visualized using a bright-blood steady-state imaging technique (**Figure 80**).

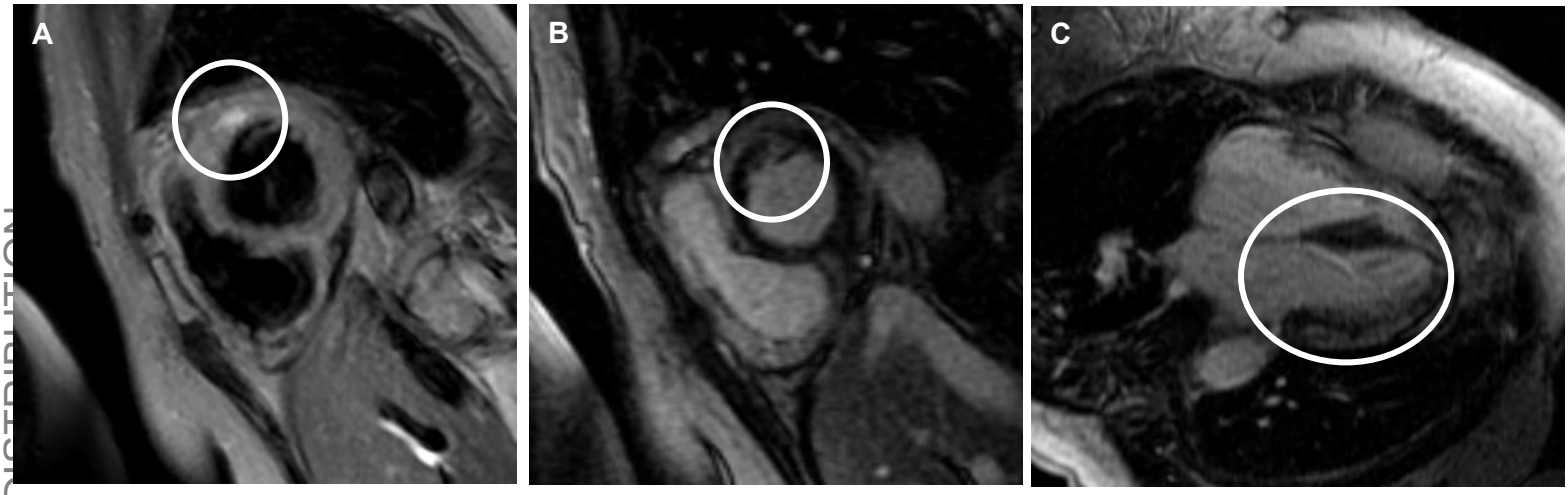

Figure 80. A 52-year-old female with cardiac sarcoidosis. (A) T2-weighted, double IR black-blood sequence with fat saturation. Note the focal area of hyperintensity in the anterior left ventricle myocardium. (B) MDE sequence with a 10-minute delay from injection. Note the same focal area of abnormal enhancement. (C) Horizontal long axis MDE 15 minute postcontrast delay. Areas of focal enhancement are visible in the myocardium of the left ventricle.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

Arrhythmogenic Right Ventricular Dysplasia

Arrhythmogenic right ventricular dysplasia (ARVD) is a genetic **cardiomyopathy** of the portion of the myocardium known as **desmosomes,** cell structures associated with cell adhesion between myocardial cells. Cellular defects occur primarily in the right ventricle and result in reduced ventricular mobility or **hypokinesis.** The healthy myocardium is gradually replaced with fatty and/or fibrotic deposits, resulting in arrhythmias which disrupt blood flow through the heart and cause syncope and dizziness. In the most serious cases, the first symptom of ARVD is sudden cardiac death in an otherwise healthy individual[8].

MR findings of cardiac imaging demonstrate hyperintensity related to rapid T1 relaxation times of fatty tissue on double IR black-blood imaging. Triple IR black-blood imaging (or double IR with fat saturation) provides confirmation of fatty deposits when the hyperintense fat signal converts to hypointensity. Multiphase bright-blood steady-state imaging demonstrates reduced contraction of the right ventricle wall with ARVD (**Figure 81**).

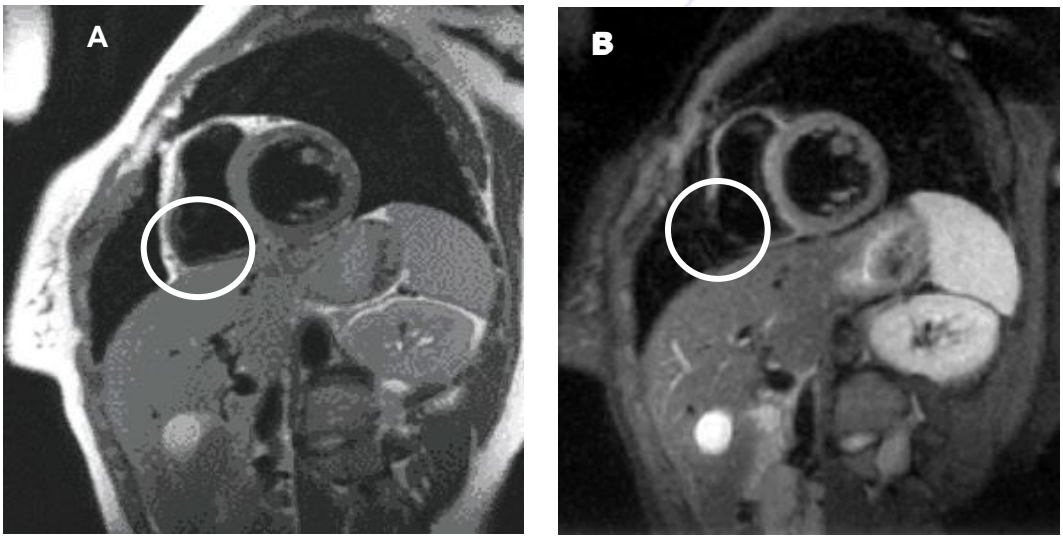

Figure 81. Examples of right ventricular dysplasia. (A) Short axis view using double IR black-blood imaging demonstrates normal left ventricle and myocardium. The right ventricular myocardium shows some evidence of increased fatty deposit and decreased myocardial muscle at the apex (circle). (B) Same view and location using triple IR black-blood imaging with fat suppression. The lack of fat signal as well as the absence of the myocardium at the right ventricular apex clearly confirm the presence of RV dysplasia (circle).

Courtesy of GE Healthcare.

Cardiac Mass/Neoplasm

Tumors, cancerous growths, or any neoplasm can occur anywhere in the body. Though cardiac neoplasm is rare, the heart is no exception. Primary cardiac tumors, that is, tumors that originate in cardiac tissue, may be benign or malignant, but even benign heart tumors are serious and can be life threatening.

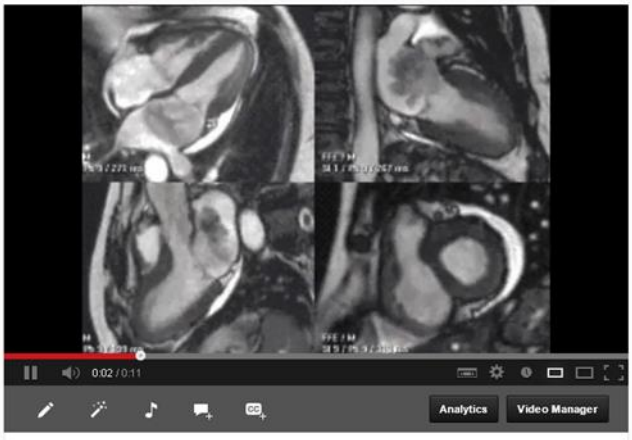

Figure 82. **Movie.** Example of a myxoma in the left atrium using bright-blood steady-state multiphase imaging. Note the movement of the myxoma into the left ventricle during diastole.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of Jccmoon at Wikipedia.

Types of benign cardiac tumors include myxomas, tumors of the connective tissue most often found in the left atrium and which make up approximately 50% of all primary heart tumors (**Figure 82**). Rhabdomyomas invade the ventricles and usually occur in children; tetromas are located around the pulmonary artery and aorta and are sometimes associated with cases of Tetralogy of Fallot in children. Fibromas occur in the myocardium and endocardium and affect the valves, and lipomas, which are fatty growths usually found in the epicardium, may occur anywhere in the heart[9,10].

Most malignant primary cardiac tumors are usually varying types of sarcomas such as angiosarcomas, liposarcomas, and fibrosarcomas. Malignant cardiac tumors can occur anywhere in the myocardium and epicardium[9].

Diagnosis of cardiac tumor is difficult based on symptoms alone as the symptoms may mimic other cardiac conditions: shortness of breath, chest pain, and arrhythmia. Since cardiac tumors affect heart function, imaging becomes an important differentiator between cardiac tumor and cardiac disease.

Visualization of a cardiac tumor is usually straightforward, using standard cardiac sequences such as pre- and postcontrast black-blood IR imaging and bright-blood steady-state multiphase imaging. Moreover, not only are tumors visualized, but their effect on cardiac *function* is often simple to assess using these same imaging techniques coupled with a phase contrast imaging sequence that visualizes valve function and blood flow dynamics (**Figures 83 and 84)**.

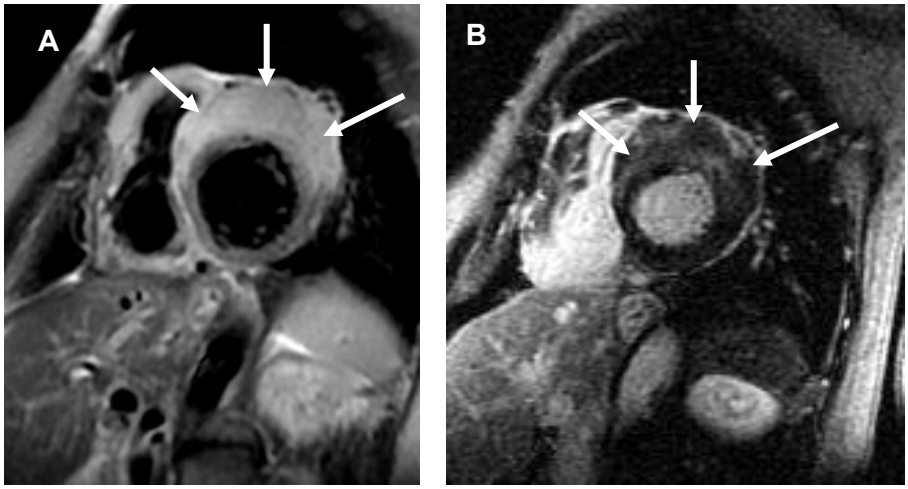

Figure 83. Example of a cardiac sarcoma involving the left ventricle.
(A) Pre-contrast short axis T2-weighted double IR with fat saturation.
(B) MDE following a 10-minute delay postcontrast. Note the large sarcoma invading the left ventricular myocardium.

Courtesy of Fairfax Radiological Consultants, Fairfax, VA.

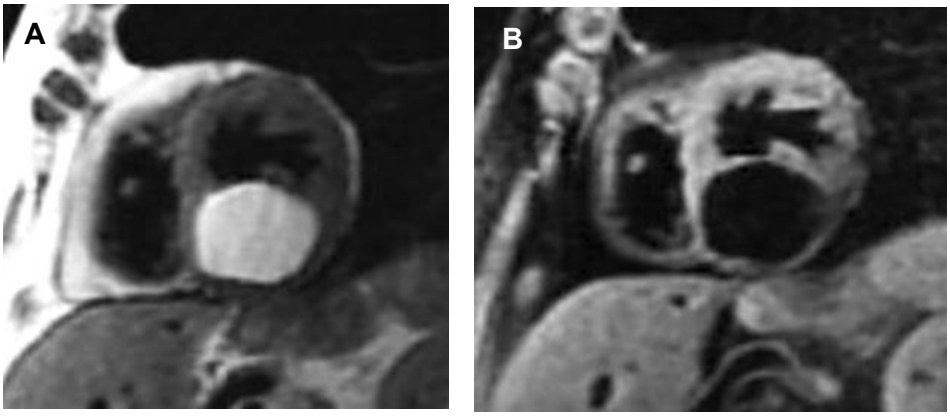

Figure 84. Example of a cardiac lipoma involving the inferior wall of the left ventricle. (A) Short axis black-blood double IR demonstrating hyperintense signal of a large lipoma. (B) Triple IR black-blood image. This fat suppression technique confirms the presence of a lipoma.

Source: Magnetic resonance imaging diagnosing a left ventricular lipoma in a patient with T wave changes on ECG, A Azarine, Heart 2005;91:873 doi:10.1136/hrt.2004.052050

Atrial and Ventricular Septal Defects

Atrial and ventricular septal defects (ASD/VSD) are malformations of the cardiac septum that result in an opening between the left and right sides of the heart. Atrial septal defects are more common than ventricular defects, comprising 6-14% of all congenital heart anomalies. ASD or VSD can be mild to moderate to severe and are most closely associated with the size of the opening in the septal wall. Typical symptoms of larger ASD/VSDs include left ventricular failure, as well as atrial arrhythmia, particularly atrial fibrillation. These defects may close spontaneously during infancy or can be treated medically if sufficiently small. If closure is required, repair of an ASD/VSD may be able to be performed percutaneously, although an open heart procedure may be the only alternative[11].

Septal defects are not difficult to visualize and diagnose on MR. Horizontal long axis multiphase steady-state imaging is the sequence of choice for visualizing the exact location of the defect as well as its extent. Quantitative phase contrast imaging is also essential for assessing flow dynamics across the defect (**Figures 85, 86, and 87**).

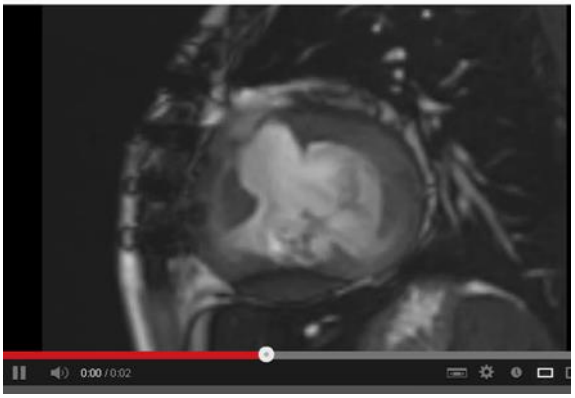

Figure 85. **Movie.** Short axis bright-blood view of a combined atrial and ventricular septal defect demonstrating both atrial leaflets.

Click here to view this **movie** on the ICPMEducation channel.

Courtesy of CS Broberg and WJ Woodward, Oregon Health Sciences University.

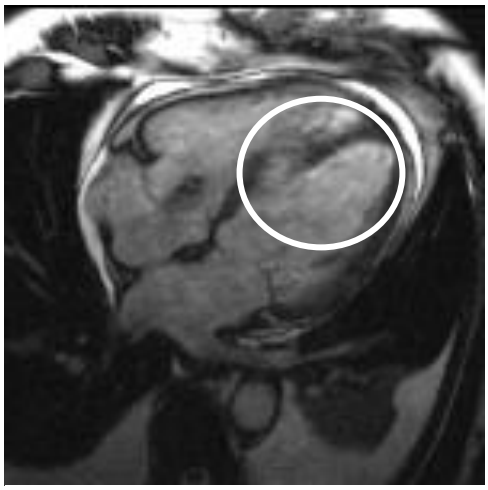

Figure 86. Bright-blood steady-state image of an adult with a large ventricular septal rupture

Courtesy of Robert J. Russo, MD. Available at: YouTube.

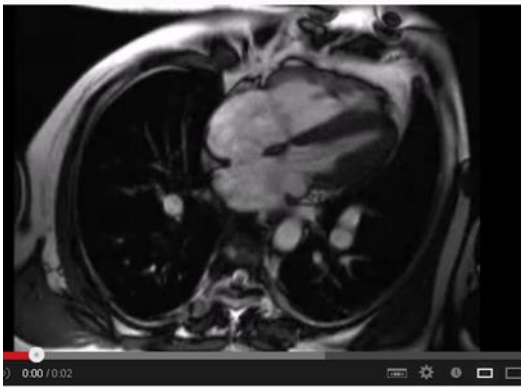

Figure 87. Movie. Multiphase bright-blood steady-state horizontal long axis view of the heart demonstrating large ASD in an adult. Note the flow dynamics between the right and left atria and the enlarged right ventricle.

Courtesy of Robert J. Russo, MD. Available at: YouTube

SUMMARY

MRI has long held great promise in cardiac imaging. Indeed, MRI ECG-gating techniques have been widely available for more than 20 years. Hardware and software development have greatly aided in the evolution and expansion of cardiac MRI applications, as with all MRI applications in the body.

Cardiac MRI has proven itself as extremely useful for diagnosing and staging cardiac disease. Today, cardiac MRI provides virtually all of the same information as any other imaging modality in one imaging session. This, then, begs the question: So why has cardiac MRI not proliferated as rapidly as other applications? The answers are varied, complicated, and subject to individual and institutional opinion.

Other imaging modalities, such as ultrasound stress echo, are more patient-friendly and less expensive yet limited in diagnostic scope. CT angiography of coronary arteries is fast, reliable, and not patient-dependent to the degree that MRI is, but CTA is unable to assess myocardial viability and exposes the patient to radiation. MR, while extremely well-suited as a cardiac diagnostic tool, suffers disadvantages such as expense, availability, patient dependence, and long exam times.

As cardiac MRI finds its place among the current arsenal of diagnostic imaging tools, radiologists will become more familiar with and confident about the tremendous value that cardiac MRI brings to the MR suite.

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PROTOCOLS

Regardless of application, cardiac MRI typically usually includes a standard set of imaging sequences and planes. Below is an example of a standard set of cardiac sequences used in the majority of all cardiac applications.

General Cardiac Protocol

SERIES	COMMENTS
Localizing Series	
Localizer (Loc)	Gated 3-plane
Gated 2-chamber Loc	Single slice prescribed off axial slice to obtain horizontal 2-chamber view
Gated Short Axis (SA) Loc	Single slice prescribed off 2-chamber view to obtain a SA view
Gated Horizontal Long Axis Loc (HLA)	Single slice prescribed off SA loc to obtain optimal HLA view
Diagnostic Series	
Diagnostic Bright-blood SA	High resolution bright-blood steady-state SA through the entire LV
Diagnostic 2xIR Black-blood SA	Double IR through the entire LV
Diagnostic 3xIR Black-blood SA	Triple IR (or 2xIR with fat saturation) through the entire LV
Diagnostic Bright-blood HLA	High resolution bright-blood steady-state HLA through the entire LV

Tetralogy of Fallot Protocol

SERIES	COMMENTS
General Cardiac Protocol (above) plus:	
Phase Contrast (PC) Main Pulmonary Artery (MPA)	PC cross-section through the MPA valve plane VENC: 150-200
PC Left/Right Pulmonary Branches	PC cross-section through the left and right PA branches VENC: 100-150
PC Aortic Valve	PC cross-section through aortic valve plane VENC: 250-300
Pulmonary MRA	Contrast-enhanced pulmonary artery MRA

Cardiac Morphology Protocol (MI, Amyloidosis, Sarcoidosis)

SERIES	COMMENTS
General Cardiac Protocol (above) plus:	
SA 10-min Delay Myocardial Delayed Enhancement (MDE)	SA MDE through the entire LV. TI selected for optimal LV suppression
HLA MDE	HLA through the entire LV. TI selected for optimal LV suppression

Right Ventricular Dysplasia Protocol

SERIES	COMMENTS
General Cardiac Protocol (above) plus:	
SA 10-min Delay MDE	SA MDE through the entire LV. TI selected for optimal RV suppression
HLA MDE	HLA through the entire LV. TI selected for optimal RV suppression

Cardiac Mass

SERIES	COMMENTS
General Cardiac Protocol (above) plus:	
SA 10-min Delay MDE	SA MDE through the entire LV. TI selected for optimal RV suppression
HLA MDE	HLA through the entire LV. TI selected for optimal RV suppression
PC Gated Volume	Gated postcontrast series through mass

Atrial/Ventricular Septal Defect Protocol

SERIES	COMMENTS
General Cardiac Protocol (above) plus:	
PC Septum	PC parallel to the plane of the atrial or ventricular septum (perpendicular to the ASD/VSD) VENC: 150-200
PC MPA	PC cross-section through the MPA valve plane VENC: 150-200

GLOSSARY OF ABBREVIATIONS

A-V node	atrioventricular node
ARVD	arrhythmogenic right ventricular dysplasia
ASD	atrial septal defect
CTA	computed tomography angiography
ECG/ EKG	electrocardiogram
EF	ejection fraction
FSE / TSE	fast spin echo/turbo spin echo
GBCA	gadolinium-based contrast agent
GRE	gradient echo
HLA	horizontal long axis
IR	inversion recovery
LA	long axis
LV	left ventricular
LVEF	left ventricular ejection fraction
LVOT	left ventricular outflow tract
MDE	myocardial delayed enhancement
MI	myocardial infarction
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
PC	phase contrast
RF	radiofrequency
S-A node	sinoatrial node
SA	short axis
T2*	T2-star
T2'	T2-prime
TI	time to inversion
TR	time to recovery, recovery time, repetition time
VLA	vertical long axis
VENC	velocity encoding value
VSD	ventricular septal defect

GLOSSARY OF TERMS

aliasing
a common artifact caused when the field-of-view selected is smaller than the area of tissue excited; also known as “wrap-around”

alveolar sacs
located at the end of the respiratory tree, consisting of clusters of alveoli wrapped in blood vessels. Deoxygenated blood from the heart is pumped through the pulmonary artery to the lungs, where oxygen is exchanged for carbon dioxide via the aveoli.

anastomoses
in medicine, the collateral connections that provide alternate blood flow pathways to the cells of the myocardium (as opposed to surgical anastomoses)

aorta
the largest artery in the body; consisting of the ascending aorta, aortic arch, descending aorta, thoracic aorta, and abdominal aorta; 12-18” long depending on the person’s height and about an inch in diameter at its widest point

arrhythmia
also dysrhythmia or irregular heartbeat; an arrhythmia is a result of abnormal electrical activity in the heart

arrhythmogenic
capable of inducing or promoting arrhythmias

atria/atrium
the two upper chambers of the heart. The right atrium receives deoxygenated blood from the superior vena cava, inferior vena cava, and coronary sinus. The left atrium receives oxygenated blood from the left and right pulmonary veins.

atrioventricular node (A-V node)
located between the atria and ventricles, the A-V node receives electrical impulses from the sinoatrial node and sends them to the ventricles causing the ventricles to contract; a natural pacemaker

auscultation
listening to the internal sounds of the body, usually through a stethoscope

bifurcation
the splitting of a main body into two parts, eg, the point at which the main pulmonary artery splits into the right and left pulmonary arteries or where the abdominal aorta bifurcates into the left and right common iliac arteries

bipolar gradients
gradient pulses applied in equal but opposite magnitudes

black-blood MRI
pulse sequences designed to null the signal of flowing blood so vessels can be assessed without interference from a bright-blood signal; usually an inversion recovery technique

bright-blood MRI
pulse sequences designed to show blood as hyperintense compared to other tissues; usually steady-state imaging techniques

capillaries
the smallest blood vessels in the body that allow exchange of water, oxygen, and carbon dioxide between blood and surrounding tissues

cardiomyopathy
heart disease that is typically inherited, not curable, but can be successfully treated. The three main types of cardiomyopathy are hypertrophic, dilated, and arrhythmogenic right ventricular.

cine
in MRI, the recording of images in such a rapid fashion that the images can be displayed in a movie loop to demonstrate real-time movement or function

coronary arteries
the vessels that deliver oxygen to the heart muscle, the myocardium. The primary arteries are the right and left coronary arteries.

coronary sinus
the point at which the coronary veins converge; the sinus then returns the blood to the right atrium

deoxygenation
the process by which oxygen in the blood is used by the body and replaced with carbon dioxide; venous blood is deoxygenated

depolarization
in the heart, the release of energy during contraction

desmosome
cell structures associated with cell adhesion between myocardial cells

diastole
the period of time when the atria and ventricles relax and refill after the left ventricle contracts (systole)

ECG gating
a process of timing the pulse sequence to the cardiac cycle by placing cardiac electrodes on the chest

electrocardiogram (ECG or EKG)
the recording and displaying of the heart's electric activity

epicardium
layer of tissue that covers the myocardium

gating
in cardiac MR imaging, a process of timing the pulse sequence to the cardiac cycle

great vessels
the large vessels that bring blood to and from the heart: the aorta, superior vena cava, inferior vena cava, pulmonary arteries, and pulmonary veins

hyper/hypointense
in MRI, a hyperintense image shows the appearance of bright tissue against a dark background, as contrasted to hypointense, the appearance of dark tissue against a lighter background

hypokinesis
as relates to cardiac imaging, reduced ventricular mobility, primarily in the right ventricle

infarct/infarction
a necrotic area of tissue caused by loss of blood flow

ischemia
restriction of the blood supply

***k*-space**
the domain in which the information from the phase-encoding step is placed during a pulse sequence. Each line of *k*-space corresponds to each phase-encoding step; once the required amount of *k*-space is filled, image reconstruction with a Fourier transform can begin.

Larmor frequency
the frequency at which magnetic resonance is produced in a sample of hydrogen nuclei or other types of nuclei used in MRI; the frequency at which the hydrogen nuclei precess when disturbed from their alignment in the B_0 magnetic field. Named for Irish physicist and mathematician, Joseph Larmor (1857-1942).

lead
the voltage potential between pairs of electrodes when acquiring an ECG

lumen
the inside of an artery or vein; does not pertain to the vessel wall itself

morphology
the form or shape of an organism

multiphase imaging
view of the heart in which a single slice location is displayed in motion in a cine loop

murmur
a sound heard on stethoscope that is the result of turbulent blood flow. Murmurs can be benign or functional due to physiologic conditions outside the heart. Pathologic murmurs can result from narrowed or leaking heart valves or other various problems and should be evaluated by a cardiologist

myocardium
the heart muscle itself and the primary contracting force

net magnetization
the sum of magnetic vectors of individual hydrogen spins that have been excited through resonant RF transmission. Z direction net magnetization is referred to longitudinal magnetization, and x,y direction net magnetization is referred to transverse magnetization.

null point
the time from inversion of the longitudinal magnetization to –Z until the longitudinal magnetization of a particular tissue crosses the midpoint between –Z and +Z where no net magnetization exists

obliquity/oblique
neither perpendicular nor parallel in relation to a given line or surface

orthogonal
pertaining to or involving right angles (90°)

palpitation
an unusually or abnormal rapid heart beat

papillary muscles
web-like muscles located inside both the right and left ventricles that aid in contraction by "pulling in" the chambers and thereby preventing prolapse of the mitral and tricuspid valves

pericardial fluid
fluid that resides between the epicardium and pericardium that allows the heart to expand and contract without friction

pericardium
the outermost layer of the heart comprised of a thin protective coating

peripheral gating
a process of timing the pulse sequence to the cardiac cycle by using the peak reflection of capillary blood as a substitute for the R-wave; useful when a stable ECG-gated waveform cannot be obtained or when cardiac morphology is not being sought, eg, imaging the aortic arch

photoplethysmograph
a pulse sensor placed on the finger or toe that measures blood filling and emptying of the capillary beds; not to be confused with pulse oximeter, which measures oxygen level in the blood

prolapse (of a valve)
in the heart, blood normally flows in one direction through the four heart valves. A prolapsed valve allows blood to squirt in a reverse direction, called regurgitation. A prolapsed valve can be the result of a congenital anomaly or a disease process.

prospective gating
gating process in which the R-wave triggers the beginning of the pulse sequence, and the R-to-R interval serves as the TR period. Each line of *k*-space is acquired at the same cardiac cycle from R-wave to R-wave; usually used in single-phase imaging

pulmonary vein
the right and left pulmonary veins carry oxygenated blood from the lungs back to the heart, emptying the blood into the left atrium and completing the pulmonary cycle

QRS complex
the most obvious portion of an ECG tracing that corresponds to the depolarization of the right and left ventricles; the R-wave represents the central point at which the ventricles are contracting

R-to-R interval
The time interval from one R-wave to the next

repolarization
in the heart, the re-energizing of the atria and ventricles after a contraction

residual transverse magnetization
when the T2 time of a tissue is longer than the selected TR, the transverse magnetization of that tissue never fully decays. This residual transverse magnetization will reach equilibrium when the amount of transverse magnetization stays the same for each TR period throughout the rest of the pulse sequence, reaching a "steady state"

retrospective gating
a gating process of timing the pulse sequence to the cardiac cycle by continuously acquiring data not triggered by the R-waves, allowing data acquisition throughout both systole and diastole; usually used in multiphase imaging

septum
in the heart, the walls that separate the left atrium and ventricle from the right atrium and ventricle

single-phase imaging
view of the heart in which a single slice location is displayed as a static image

sinoatrial node (S-A node)
located in the right atrium, the S-A node initiates the electrical pulse that is the normal sinus rhythm; a natural pacemaker

spatial resolution
the size and number of the voxels in an image

stenosis
abnormal narrowing of a vessel

syncope
loss of consciousness, fainting due to low blood flow to the brain and usually caused by low blood pressure

systole
contraction of the left ventricle that forces blood out of the heart to the body

T1
amount of time for 63% of a tissue's net magnetization to recover to longitudinal magnetization

T1-weighting
generation of MR images under conditions that highlight T1 differences between tissues

T2
amount of time for 63% of a tissue's net magnetization to decay in the transverse plane

T2-weighting
generation of MR images under conditions that highlight T2 differences between tissues

T2* (T2-star)
a combination of the intrinsic T2 of a tissue and T2'

T2' (T2-prime)
dephasing due to susceptibility effects

TI (time to inversion)
the time allowed from the initial 180-degree excitation pulse until the 90-degree pulse in an inversion recovery pulse sequence; TI determines the amount of T1 recovery time for a given tissue

TR (time to recovery, repetition time)
the amount of time allowed for T1 recovery of the longitudinal magnetization; also the time from the beginning of one pulse sequence to the beginning of the next

T-wave
represents repolarization of the ventricles on an ECG tracing

temporal resolution
in cardiac MRI, the number of cardiac phases obtained in a cardiac multiphase series

tetralogy of Fallot
a congenital heart defect that involves four anatomical abnormalities and requires corrective surgery. Named for French physician, Étienne-Louis Arthur Fallot who, although not the first, described the condition in 1888.

trigger delay
the time from detection of the R-wave to the beginning of the data acquisition; set by the operator

trigger window
the time from the end of data acquisition until the next R-wave is sensed by the MR system; set by the operator

triggering
the placing of electrodes to obtain the highest R-wave for launching a pulse sequence

valves
the valves of the heart open and close to move blood through the heart. The mitral and tricuspid valves are located between the atria and ventricles; the pulmonary and aortic valves are located in the arteries leaving the heart

vasodilation
the widening or expanding of blood vessels; the opposite of vasoconstriction, the narrowing of blood vessels

vena cava
the largest vein in the body; the inferior vena cava runs virtually parallel to the aorta

ventricles
the pumping chambers of the heart located beneath the atria that force blood to the body

vmax
the maximum blood velocity going through a stenotic vessel

voxel
a pixel that also displays depth as a 3rd dimension; from "volume element"