

Coronary Arteries: what you need to know

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Clinical Background

Despite progress in prevention and early diagnosis, coronary artery disease (CAD) remains the leading cause of mortality in the world, accounting for 13% of all deaths worldwide. For many years, invasive x-ray coronary angiography has been the standard of reference for the diagnosis of significant CAD (defined as more than 50% stenosis of the coronary artery lumen). Although several non-invasive tests are available to help discriminate patients with and without significant angiographic disease, studies demonstrated that up to 40% of patients referred for elective x-ray coronary angiography have no clinically significant stenoses. Despite the absence of significant narrowing, these patients remain subjected to the potential risks of x-ray angiography. Therefore, development of a clinically useful non-invasive technique is desirable.

Of the non-invasive techniques, multidetector row computed tomography (MDCT) and magnetic resonance imaging (MRI) are currently most promising for non-invasive imaging of the coronary arteries. MDCT is a rapidly evolving technique with the main advantage of isotropic high spatial resolution. MRI, however, is the established standard of reference technique for studying cardiac anatomic structure and function and does not involve exposure to ionizing radiation. Furthermore, MRI contrast agents are much safer than the iodinated contrast agents as evidenced by the much lower rate of allergic reactions and the absence of clinically detectable nephrotoxicity.

This review will focus on coronary anatomy, coronary artery disease and the technical foundations and the current status of clinical coronary MRI. It will conclude by identifying areas where improvements are needed. Since the coronary arteries were first depicted with MRI in the mid 1980's, many technical developments have been implemented that have led to major improvements in image quality. Nowadays it is possible to routinely visualize the proximal and middle parts of the coronary arteries and their branches with high accuracy. In addition, development of vessel wall imaging techniques is promising for detection of wall abnormalities in the absence of significant CAD.

I. Coronary Artery Anatomy

The coronary arteries are conductive vessels running through the epicardial surface of the heart, embedded in adipose tissue, and showing short segments of mild penetration into the myocardial tissue, called *myocardial bridging*. Coronary arteries are distributed over the heart as a crown-shape network, showing anastomotic communications between its different branches, particularly at the level of the base and the apex of the left ventricle.

The coronary arteries are variable in terms of diameter, with the left artery being larger than in the right in more than half of individuals, with the opposite occurring in nearly 20%. Coronary arteries emerge from the aorta through the coronary *ostia*, located at the right (or anterior) and the left (or left posterior) sinuses of Valsalva. The coronary *ostia* are situated at the level of the *sinotubular junction* or slightly below it (56% of cases), followed by a high left orifice and a low right orifice or at the level of the junction (30% of individuals). Although usually two coronary arteries (right and left) are seen emerging from the aorta, three or even four independent origins have been described. In these cases, frequently (36% of individuals) the right conal artery is the one with an independent origin. Also, it is not rare to find separate origins for both main branches of the left artery. On the other hand, the origin of both arteries from a single coronary sinus has been described, either from a single ostium or from separate orifices at the same sinus.

Left Coronary Artery System

The left coronary artery is a large artery with an approximate diameter of 5 mm at its origin, that supplies an extensive portion of the walls of the left chambers of the heart, including most of the interventricular septal mass. The initial segment of the left coronary artery – called *left main* (LM) – is variable in length, is embedded in adipose tissue, and courses between the main pulmonary artery and the left atrial appendage. Rarely (<1% of individuals), the LM is absent, with independent origins of its main branches from the left coronary sinus. At the level of the left atrioventricular groove, the LM gives two or three branches: 1) the *left anterior descending* (LAD) artery; 2) the *left circumflex* artery (LCx) and, occasionally, 3) the *intermediate* artery (also called ramus intermedius).

Left anterior descending (LAD)

The LAD is a large vessel—4–5 mm in diameter at its proximal portion—that occupies the anterior interventricular groove, running in parallel with the great cardiac vein. It usually extends to the apical region of the left ventricle and, in two thirds of individuals, it reaches the distal or even the middle portion of the posterior interventricular groove. In these cases, the LAD frequently shows anastomotic connections with the left posterior descending artery (PDA). The LAD gives some branches along its course^{2–4,6,8–12}:

- Left conal artery: with an origin in the proximal LAD, it communicates with the right conal artery, with which it constitutes the “arterial ring of Vieussens,” along with the vasa vasorum of the aorta and pulmonary artery.
- Right anterior ventricular branches: usually irrelevant in number and diameter, as the right ventricle is almost exclusively irrigated through the right coronary artery.

- Left anterior ventricular branches (diagonal arteries): 13–15: Variable in number, these branches distribute diagonally over the anterior aspect of the left ventricle. The origin of the first diagonal artery is used as the anatomical point dividing the middle and distal segments of the LAD.
- Anterior septal branches: variable in number, these branches arise orthogonally from the LAD and distribute into the anterior two thirds of the interventricular septum. The first septal branch is usually a well-developed vessel, its origin being considered as the reference point dividing the proximal and middle portions of the LAD. Rarely, this first septal branch courses closely parallel to the LAD.

Left circumflex (LCx)

The LCx is also a large vessel, similar in diameter to the LAD, although more variable in terms of length and anatomical distribution. The proximal portion of the vessel lies beneath the left atrial appendage and, from there its course follows the anterior aspect of the left atrioventricular groove, ending at the obtuse margin of the heart. In cases of anatomical dominance of the left coronary system, the LCx goes beyond this region and gives off the posterior descending artery (PDA). The LCx gives origin to different branches during its course:

- Anterior or anterolateral ventricular branches: when present, these small vessels arise proximally and course parallel to the first diagonal artery. When this artery is absent, it is substituted by these branches.
- Sinusal or sinoatrial branch: although usually arising from the right artery, the sinusal branch emerges from the proximal segment of the LCx in 30–35% of individuals, courses around the left atrium, and reaches the sinus node region at the superior vena cava drainage.
- Atrial arteries: these small vessels are usually located beneath the base of the left atrial appendage or at the posterior aspect of the left atrium.
- Obtuse marginal branches: usually one or two, their origin is used as a reference dividing the proximal and medial segments of the LCx. These branches are well developed vessels emerging orthogonally from the LCx and coursing along the left margin of the heart until they reach the apex, where they can communicate with vessels from the LAD.
- Posterior ventricular branches: although the posterior wall of the left ventricle is mostly irrigated by branches from the right PDA, when this vessel is absent, a variable number of these posterior ventricular branches—together with a number of interventricular branches of the LCx—are responsible for the blood supply to this region.
- Atrioventricular nodal branch: it arises from the LCx in up to 20% of subjects, particularly in cases of left dominance.

Intermediate coronary artery

In 25-40% of individuals, the LM divides into three branches; in addition to the LAD and the LCx, a third vessel is found, known as median or intermediate artery or ramus intermedius. The intermediate artery runs over the antero-lateral aspect of the left ventricle, giving septal anterior branches and it supplies the anterior papillary muscle.

Right Coronary Artery (RCA) System

The RCA supplies the blood flow for the right atria and ventricle and, when dominant (see below), also for a variable extension of the posterior aspect of the left ventricle. Originating from the right coronary sinus, the proximal segment of the RCA courses closely to the right atrial appendage and is then located on the anterior aspect of the right atrioventricular groove, where it is embedded in adipose tissue. At its medial segment, the RCA rounds the right acute margin of the heart and through the posterior aspect of the right atrioventricular groove it reaches the region of the crux cordis. There are variants of this anatomical distribution: in 10% of individuals the RCA ends at the level of the acute margin of the heart, or between this region and the crux cordis; in 60% the RCA extends beyond the crux cordis and reaches the inferior wall of the left ventricle where it shows connections with the distal LCx artery; finally, in 20% of subjects the vessel arrives to the left cardiac margin, irrigating the area corresponding to the LCx. The RCA gives different branches along its course:

- Right conal branch: as mentioned earlier, in up to 36% of individuals, this vessel shows an origin in the anterior aspect of the right coronary sinus, independent from the one of the RCA.
- Sinus node branch: this vessel originates from the RCA in more than 50% of individuals, usually arising from the most proximal portion of the RCA or, rarely, from its middle or even distal segment. The sinus node branch courses over the base of the right atrial appendage, ending at the drainage of the superior vena cava into the right atrium.
- Atrial branches: variable in number and size, these branches are distributed over the anterolateral aspect of the right atrium, although a posterior branch also does exist, supplying both atria or the posterior left atrium exclusively.
- Acute marginal branch: this anterior right ventricular branch is usually a well-developed vessel, coursing over the right ventricular free wall, near the right acute margin of the heart, and reaching the region of the apex in most individuals.
- Posterior right ventricular branches: these are small vessels—not always present—arising from the distal RCA, and irrigating the inferior aspect of the right ventricle. Their degree of development is inverse to that of the acute marginal branch, which occasionally distributes over the same region.
- Interventricular posterior branch (or right *posterior descending artery* [PDA]): this artery is a branch of the RCA in up to 90% of individuals, arising from the *crux cordis*, the region where both posterior atrioventricular grooves meet with the posterior interventricular groove. In nearly 70% of subjects, the right PDA is a single branch coursing along the posterior interventricular groove, ending next to the most distal recurrent branch of the LAD, in the region of the apex. In the remaining 30% of cases, 2 or 3 smaller branches are present, coursing in parallel at both sides of the interventricular groove. The PDA irrigates the posterior aspect of both ventricles.
- Right posterobasal or posterolateral arteries: widely variable in number, size, and distribution pattern, these vessels usually course over the inferior aspect of the left ventricle.

Coronary artery dominance

The term “dominant” refers to the coronary artery that reaches the *crux cordis* and gives origin to the PDA. Three patterns of anatomical coronary artery dominance can be distinguished:

- Type I (~77% of individuals): in this case, the RCA gives origin to the PDA.
- Type II (~8%): in this case, the PDA is a branch of the LCx, appearing either as a single or set of vessels.
- Type III (“balanced” circulation; ~15%): in this case there are two PDA, one provided by each vessel (RCA and LCx), coursing in parallel to the posterior interventricular groove.

Anomalies of the Coronary Arteries

Anomalous origin of a coronary artery is seen relatively infrequently (in less than 3% of all congenital heart diseases, and less than 1% of the general population). Mostly they are incidental findings in coronary angiographies performed due to symptoms not related with these abnormalities. Due to their 3D imaging capabilities both MRI and MDCT are well suited for the exact characterization of the coronary artery anomalies. However, because congenital anomalies of the coronary arteries may be the cause of myocardial ischemia and even sudden death it is clinically important to recognize two different types of these anomalies: 1) anomalies that may induce myocardial ischemia, and 2) anomalies not leading to myocardial ischemia

- Anomalies leading to myocardial ischemia:

In most of these anomalies, a segment of a coronary artery is seen to course abnormally between the wall of the aortic root and that of the main pulmonary artery (*‘malignant course’*). This situation may cause myocardial ischemia due to enlargement of the great vessels, secondary to transiently increased flow, for instance during exercise. Also, in cases of abnormal coronary artery course around the anterior aspect of the pulmonary artery, ischemia may be present in those situations of enlargement of the right ventricular outflow tract, as in pulmonary hypertension. Abnormal origin of coronary arteries from the pulmonary artery, either the LCA (Bland-White-Garland syndrome) or the RCA, is also an obvious cause of myocardial ischemia.

- Anomalies not leading to myocardial ischemia

Some of the congenital coronary anomalies are not related to myocardial ischemia and are detected incidentally. However, their knowledge is important for two reasons, particularly when non-coronary cardiac surgery is planned to prevent inadvertent damage of the aberrant coronary vessel, and to appropriately guide the coronary artery cannulation. These coronary anomalies have been detected in 0.5–1% of invasive coronary angiographies, although it is not always feasible to describe at angiography the exact course of the abnormal artery in relation to the great vessels. The most frequently found types of coronary anomalies are the following:

- Origin of the LCx from the right coronary sinus or RCA: It is the most frequent non-ischemic anomaly in this group (0.67% of diagnostic invasive coronary angiograms). The LCx is seen emerging posteriorly to the RCA, and then following a course inferior and posterior to the aortic root, reaching the left atrioventricular groove. Very rare in this case is a course of the LCx between the walls of the great vessels.

- Origin of the LAD in the RCA: This anomaly is present in 4–5% of patients with tetralogy of Fallot or pulmonary atresia with ventricular septal defect. The LAD frequently shows a course anterior to the infundibular portion of the right ventricle, usually not leading to myocardial ischemia, which has potential implications in the case of surgical correction.

II. Atherosclerotic Coronary Artery Disease

Atherosclerosis is considered to be a chronic inflammatory disease of the large arteries. The disease already starts at an early age, and remains clinically silent for decades. The initiation of atherosclerosis is characterized by upregulation of leukocyte adhesion molecules, subsequent adhesion and migration of monocytes into the arterial neointima and differentiation of these monocytes in macrophages. Once macrophages are present in the arterial intima, they are able to ingest unlimited amounts of modified lipoproteins. Accumulations of these lipid filled macrophages are the major component of the first macroscopically visible, but clinically silent, atherosclerotic plaque: the *intimal xanthoma* or *fatty streak*.

When atherosclerotic lesions progress, more and more (inflammatory) cells enter the lesion. Besides accumulations of lipid filled macrophages, increased amounts of T-lymphocytes, smooth muscle cells and (myo)fibroblasts enter the atherosclerotic plaque and form a '*pathological intimal thickening*'. When this process advances, (myo)fibroblasts and vascular smooth muscle cells migrate further into the atherosclerotic lesion, deposit extracellular matrix, and form a *fibrous cap* that overlies the atherosclerotic plaque. In the mean time, the center of the plaque is deprived of oxygen, and a *necrotic core* develops. The necrotic core contains extracellular lipids, cholesterol crystal esters, and sometimes calcification. *Apoptosis* or programmed cell death of vascular smooth muscle cells may be a key factor in this process. Moreover, activation of angiogenic pathways results in an increased number of neo-vessels in the plaque. Atherosclerotic lesions containing a fibrous cap and/or a lipid core are called *atheromata*. With the progression of atherosclerosis, the arterial wall reshapes.

When atherosclerotic plaques encroach the lumen, the circumference of the artery increases, thereby partially or totally maintaining the original luminal diameter, a process called *outward or positive remodeling*. Therefore, luminal arteriography often masks the severity of atherosclerosis. Although there are multiple modalities which are capable of assessing plaque morphology, MRI is at present the sole non-invasive modality with the potential to detect outward remodeling in asymptomatic subjects. Depending on their composition, atheromata are subdivided in 'fibrous cap atheromata' and 'thin fibrous cap atheromata' (fibrous cap thickness < 65 μm). The latter are characterized by large lipid accumulations, large lipid cores, increased amounts of inflammatory cells and intra-plaque capillaries and thin fibrous caps. These atherosclerotic lesions are designated 'vulnerable plaques' and are considered precursors for the lesions that are responsible for the majority of cardiovascular complications of atherosclerosis: the ruptured atherosclerotic plaque.

When atherosclerotic plaques rupture, thrombogenic components of the atherosclerotic plaques come into contact with the blood and a thrombus develops that

can occlude the artery, thereby resulting in myocardial infarction. Morphologically, plaque rupture is defined as an area of fibrous cap disruption whereby the overlying thrombus is in continuity with the lipid core.

Factors correlated with plaque vulnerability and plaque rupture are increased activity of inflammatory (e.g. leukocyte adhesion molecules, chemokines and cytokines) and proteolytic pathways (e.g. matrix-metalloproteinases and cathepsins). Other factors associated with plaque vulnerability and plaque rupture are increased thrombogenicity and an increased angiogenesis.

It is important to realize that plaque rupture does not always imply a fatal event. In patients who died of non-cardiovascular causes, plaque rupture was present in 10% of atherosclerotic lesions. Non-fatal lesions can contain areas of (repeated) plaque rupture and thrombosis. If a thrombus remains mural rather than occlusive and its lysis is incomplete, re-endothelialization followed by fibrous thrombus organization results in accelerated plaque growth. Besides true plaque ruptures, intra-plaque haemorrhage is described as a phenomenon in lesions that are not necessarily associated with plaque rupture.

Recently, it has been stated that plaque rupture does not occur as an isolated phenomenon, but rather as a systemic disease. In this view, it is preferred to refer to the 'vulnerable patient' instead of a patient with a localized vulnerable atherosclerotic plaque. 'Vulnerable patients' often present with multiple ruptured plaques. In an angiography study of patients with an acute coronary syndrome (ACS), 39.5% of the patients had multiple complex plaques that were associated with an increased incidence of recurrent ACS. In another study using intravascular ultrasonography (IVUS), 79% of the patients presenting with an ACS had multiple ruptured plaques at other sites than the culprit lesion that caused the clinical symptoms. It can therefore be postulated that the occluding thrombus at the culprit lesion determines the clinical presentation, but is only a focal manifestation of an underlying systemic disease process that includes several rupture prone or vulnerable lesions. Systemic factors that are correlated with plaque rupture are altered blood rheology, increased coagulability, increased systemic inflammation and recurrent infections.

III. Technical aspects of coronary magnetic resonance imaging

Equipment considerations

State-of-the-art coronary MRI is performed on 1.5 T MR-systems using dedicated cardiac phased-array radiofrequency coils applied to the chest wall. Technological advances in MR system architecture now enable accelerated data acquisition by simultaneously using 16- or 32 receiver channels.

Appropriate cardiac receiver coils are required to meet the high in-plane spatial resolution requirements for coronary MRI while maintaining a sufficient signal-to-noise ratio (SNR) in comparison with use of the standard system built-in body coil. The use of these coils should be standard for all coronary MRI examinations. Because the SNR decreases with the distance from the surface receiver coil, cardiac specific coils have been optimized for the size of the heart and the distance of the heart from the chest wall. The right, left main and left anterior descending (LAD) coronary arteries are located

relatively close to the anterior chest wall, and therefore can be visualised with good image quality. However, the more distal parts of the circumflex artery are more difficult to depict.

With phased-array coils, parallel imaging techniques such as sensitivity encoding (SENSE), Simultaneous Acquisition of Spatial Harmonics (SMASH), and generalized autocalibrating partially parallel acquisitions (GRAPPA) can be used to accelerate image acquisition by using the locally differing sensitivities of the separate receiver coil elements. The acceleration factor is depending on the coil type, and is typically 2-3 fold. With the newest generation of 16- and 32- receiver channel systems even higher factors can be reached. However, a trade-off must be made between image quality and scan duration since the use of high acceleration factors will lead to decreased SNR and contrast-to-noise ratio (CNR).

In recent years, 3.0T high field MRI systems have become more widely available and are starting to be used for coronary imaging. The potential doubling of the SNR at 3.0 T could lead to further progress in cardiac applications, including coronary MRI. However, increased susceptibility effects and specific absorption rate (SAR)-limitations due to altered penetration of radio-frequency (RF) pulses are potentially disadvantageous. Nevertheless, first results of cardiac and coronary MRI at 3T are promising and show increased SNR and CNR compared to the results at 1.5T. However, the theoretically predicted twofold gain in SNR has not yet been achieved and current techniques do not result in significantly improved image quality and diagnostic accuracy compared with the quality and accuracy at 1.5T. On the other hand, Huber et al. found the added SNR at 3T to be sufficient to use parallel imaging with a reduction factor of two. This resulted in a 50% reduction in scan duration, with largely preserved image quality despite inevitable SNR loss relative to conventional full acquisition at 3T.

Spatial resolution requirements

Coronary MRI data acquisition is technically challenging because of the tortuosity and small calibre – on average 1.5-5.5 mm - of the coronary arteries. In this context, it is important to realize that accurate assessment of the degree of stenosis demands at least three pixels across the normal vascular lumen. This constraint imposes a submillimeter in-plane resolution on coronary artery MRI protocols, especially in protocols used for stenosis detection. The use of modern MR systems in combination with dedicated surface coils and optimized pulse sequences readily enables submillimeter in-plane resolution with current best pixel sizes of around 0.7 x 0.7 mm and slice thickness in the order of 1 mm. Despite satisfying the resolution requirement, the spatial resolution of coronary MRI is currently lower compared to the resolution of x-ray angiography (in the order of 0.2-0.3 mm), and MDCT (pixel sizes of around 0.5 mm). A drawback of coronary MRI is that higher resolution images increase acquisition time, and lead to lower SNR when all other parameters are kept constant.

Motion compensation

Because the MR data acquisition process is inherently motion sensitive, techniques must be applied to compensate for cardiac and respiratory motion.

During the cardiac cycle, in-plane coronary artery displacement can be up to 5 mm. The right coronary artery is more motion sensitive than the left coronary system.

The data points needed to reconstruct an image of the heart and coronary arteries are generally obtained over multiple consecutive cardiac cycles. This strategy is also known as ‘segmented’ k-space sampling and demands accurate four-lead vector ECG registration²⁵ and a regular cardiac rhythm. Studies have demonstrated that coronary artery motion is minimal during middiastolic diastasis, the cardiac rest period, and that best image quality is obtained when the acquisition window is optimized by using a subject specific trigger delay after detection of the R-wave. The length of both the end systolic and middiastolic rest periods depends on heart rate and can be individually determined from high temporal resolution cine-images. To obtain good image quality, an individually tailored acquisition window, preferably less than 120 ms, is advised. An additional important source of motion artefacts are beat-to-beat variations in heart rate and premature heartbeats. The possibility to exactly tailor acquisition duration and to correct for variations in heart rate is a major strength of MR imaging.

In addition to taking into account cardiac contraction, respiratory motion has to be compensated for as well. At present, two techniques are used for respiratory motion compensation: 1) *breath holding* and 2) *navigator gating*. Breath holding is highly dependent on patient cooperation. In addition, patients with pulmonary disease or heart failure are often not able to hold their breath long enough. Furthermore, slow cranial shifting of the diaphragm (drift) during breath hold can still cause motion of the heart and the coronary arteries during acquisition.

The navigator gating technique on the other hand, uses a 2D pencil beam that is placed on an interface that reflects respiratory motion, e.g. the lung-liver or lung-myocardium interface. The navigator monitors the motion of this interface during free breathing. Data are accepted only when the selected interface falls within a user-defined window positioned around the end-expiratory level of the interface. With this technique, there is less patient cooperation involved. However, diaphragmatic drift can also occur during free breathing. Therefore, drift correction of the navigator window during the scan is essential to maintain sufficient efficiency and reasonable scan duration.

In terms of image quality and diagnostic accuracy, the free-breathing navigator technique is superior to breath hold coronary MRI. Because of longer scan duration (not limited by breath hold) better SNR or spatial resolution can be achieved.

Pulse sequences and vessel-to-background contrast

Both bright blood and black blood techniques have been evaluated in two-dimensional (2D) and three-dimensional (3D) pulse sequences. Bright blood sequences tend to overestimate atherosclerotic lesions because of artificial darkening caused by focal turbulent flow. The vessel luminal diameter may therefore be underestimated in comparison with conventional x-ray angiography. On the other hand, the signal intensity of thrombus, vessel wall and various components of plaque may appear high on bright blood coronary MRI, thereby obscuring focal stenoses. Despite these drawbacks, the majority of sequences for coronary artery lumen imaging are bright blood approaches with 2D or 3D gradient echo sequences with Cartesian segmented k-space sampling. 2D breath-hold coronary MRI has been shown to be a promising and valuable method for assessment of the native coronary arteries. With 3D methods scan duration is longer, but these methods also have inherent advantages, including minimizing bulk cardiac motion, superior SNR, the acquisition of thin contiguous sections and the ability to postprocess

and reformat the data set. Recently, bright blood balanced steady state free precession (bSSFP) sequences have gained considerable interest. SSFP imaging is a very promising technique for coronary MRI at 1.5 T with high SNR and CNR. Nevertheless, this sequence is more sensitive to magnetic field inhomogeneities. The increased main field inhomogeneity and B1 field variations at 3T can potentially be problematic for SSFP imaging.

In contrast, black blood coronary imaging is performed using spin echo sequences. With this technique, there is a potential for enhancing CNR in comparison with gradient echo approaches. In addition, black blood sequences appear to be particularly advantageous for patients with implants such as vascular clips or sternal wires because spin echo techniques are less sensitive to the susceptibility artefacts from metallic implants.

The coronary arteries can be imaged using either a double-oblique, targeted approach or a whole heart scan. With the former technique, a 3D slab is acquired in a user-defined orientation around the coronary arteries. The vessel of interest can be covered with high spatial resolution and scan duration is shorter compared to whole heart MRI. Although extensive parts of the coronary arteries can be depicted with this technique multiple scans are required since not all coronary arteries can be covered with one scan. The whole heart technique is a magnetization prepared bSSFP sequence using an acquisition volume covering the whole heart. The major advantages of the technique are that positioning of the imaging volume is relatively simple, it facilitates high-quality coronary MRI of the complete coronary artery tree in a single measurement and it allows post-processing and display of datasets similar to MDCT. A drawback at present is the relatively long acquisition duration, although this will change with more widespread use of parallel imaging techniques.

An important aspect of coronary imaging is the contrast between coronary arteries and surrounding epicardial fat and myocardium. In comparison with MDCT, exogenous contrast media are not necessary to achieve coronary artery enhancement. Several techniques have been developed to increase CNR between the blood and myocardium. Perivascular fat can be suppressed by applying fat saturation or spectral presaturation with inversion recovery (SPIR) prepulses. Suppression of the myocardium can be achieved by the use of a T2-preparatory prepulse, a technique in which a dedicated prepulse is used to achieve a decreased signal from myocardium while maintaining the signal from blood, leading to an improved CNR between blood and myocardium and better vessel definition.

The role of exogenous contrast agents remains to be established, as the use of contrast medium can actually reduce vessel-to-background contrast. For instance, commonly used extracellular agents extravasate into the myocardium and perivascular fat, thereby decreasing the contrast between the coronaries and the myocardium. The use of intravascular contrast agents could be an alternative since these agents exhibit prolonged intravascular retention and have a longer plasma half life and shorter T1, resulting in higher signal intensity. This allows imaging over a longer period of time so navigator techniques can be used to obtain images of high quality with high blood/muscle contrast and better vessel delineation. In addition, contrast agents may become an important adjunct to coronary imaging at 3T as these agents ameliorate some of the imaging artefacts encountered at higher field strengths.

Clinical Indications for coronary MRI

1) Detection of anomalous coronary arteries

Projection x-ray angiography used to be the imaging test of choice for the diagnosis and characterization of these anomalies. However, detection of anomalies may be difficult and the exact anatomical course can be difficult to determine. One of the main advantages of using MRI instead of x-ray angiography is the visualization of the coronary arteries in relation to other mediastinal structures such as the right ventricular outflow tract. 3D coronary MRI is well suited for the depiction of anomalous coronary artery origins and is now the method of choice in young patients in whom coronary artery anomaly is suspected or needs to be further clarified, or if the patient has another cardiac anomaly associated with coronary anomalies.

2) Kawasaki disease and follow up of coronary artery aneurysms

Kawasaki disease, an acute vasculitis of unknown origin, is the leading cause of acquired coronary artery disease in children in developing countries and is now reported as a potential risk factor for adult ischemic heart disease and sudden death in early adulthood. There is a 25% chance of serious cardiovascular damage if treatment is not initiated early in the course of the disease. Coronary damage, including dilatation, aneurysms (defined as coronary diameter > 4 mm) and giant aneurysms (coronary diameter > 8 mm), develop in up to 5% of timely treated patients. In addition to aneurysm development in infants and children, this syndrome may eventually lead to thrombotic occlusion, premature atherosclerosis and progression to ischemic heart disease. Serial evaluation of coronary aneurysms is important, and regression of these aneurysms has been reported in approximately 50% of the patients. In young children, transthoracic echocardiography is usually adequate for detecting and following these aneurysms, but this technique becomes often inadequate as children grow. An alternative method for follow up is MRI, which is considered equivalent to coronary angiography.

3) Detection of stenoses in native coronary arteries

The most important potential clinical application of coronary MRI is detection of stenoses in native coronary arteries. For this purpose bSSFP bright-blood techniques are mostly used where areas of focal stenosis produce signal voids of varying severity related to the angiographic degree of stenosis. However, gradient echo coronary MRI may sometimes overestimate the degree of stenosis as blood flow alterations in stenotic segments can cause signal loss in the segment distal to the lesion. Similarly, the presence of heavy calcification in the coronary vessel wall may lead to signal voids that artificially suggest the presence of significant stenoses. On the other hand, adequate collateral blood flow is readily detected as it results in signal in the lumen distal to an occlusion.

An infrequent cause of chest pain is myocardial bridging, a condition in which part of a coronary artery is situated in the myocardium and compressed during systole. Long tunneled segments of coronary arteries, more severe systolic diameter narrowing of the tunneled segment and tachycardia may result in myocardial ischemia. MRI using

systolic and diastolic acquisition windows can be used to non-invasively detect and follow patients with myocardial bridging.

4) Assessment of Coronary Artery Bypass Grafts (CABG)

Occlusion or stenosis of grafts can occur after coronary artery bypass grafting and this incidence increases over time. Because of their fixed position and large lumen size, bypass grafts are relatively easy to image despite possible artefacts due to the presence of sternal wires or metal clips.

IV. Areas for Improvement

To further improve coronary MR imaging we need:

- Faster acquisitions (preferably in breath-hold or by improved navigator gating). Parallel imaging will play an important role here.
- Higher spatial resolution to better characterize stenoses.
- Integration of stenosis imaging with perfusion imaging (which stenoses are really significantly flow-limiting?)
- Better and faster plaque imaging and characterization (which plaques are dangerous?)
- better shimming and more homogeneous RF penetration at 3T
- to investigate how to best use contrast agents to depict the coronary arteries and vessel wall.

V. Further Reading

Articles

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