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Task Force Report

The clinical role of magnetic resonance in cardiovascular disease

Task Force of the European Society of Cardiology, in Collaboration with the Association of European Paediatric Cardiologists

Introduction

Magnetic resonance is helpful in the diagnosis and assessment of cardiovascular disease. However, there are no published guidelines on the clinical role of the technique, although with the growing interest of clinicians in cardiovascular magnetic resonance their need is now pressing.

The board of the European Society of Cardiology has created a Task Force to establish the current clinical role of magnetic resonance imaging and spectroscopy in the diagnosis and assessment of diseases of the heart and great vessels. Because magnetic resonance is a multidisciplinary technique, the task force was composed of cardiologists, paediatric cardiologists and radiologists and these guidelines are jointly supported by the European Society of Cardiology and the Association of European Paediatric Cardiology. Task Force recommendations are based on evidence from published literature and from the clinical experience of its members. Whenever there is little published evidence for a recommendation this is indicated, but usually no recommendation is made in this circumstance. To strengthen the clinical value of the guidelines, the role of magnetic resonance is described alongside that of other imaging techniques, with emphasis on the relative role with respect to echocardiography. The rapid development of magnetic resonance means that its indications are expanding. Thus, these guidelines will require updating with time.

The value of imaging techniques in individual circumstances is often defined as appropriate, acceptable, rarely justified, and not indicated. Consequently, the classification of the ACC/AHA Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures[1] has been adapted:

Class I — provides clinically relevant information and is usually appropriate; may be used as a first line imaging technique
Class II — provides clinically relevant information and is frequently useful, but similar information may be provided by other imaging techniques
Class III — may provide clinically relevant information but is infrequently used because information from other imaging techniques is usually adequate
Class IV — does not provide clinically useful information
Inv — potentially useful, but still under investigation

Technical aspects

Although an understanding of the physics of magnetic resonance is important in appreciating the capabilities of the technique, a full description is beyond the scope of this report. A brief description of the technical aspects of magnetic resonance is included to aid understanding of the terms used.

Nuclear magnetization

The hydrogen nucleus is abundant in water and it is therefore the most clinically useful of the nuclei exhibiting the phenomenon of magnetic resonance. The proton, which forms the hydrogen nucleus, behaves as a small magnet. If placed in a magnetic field, the nucleus will align with the field and it will precess in the same way that a spinning top precesses in a gravitational field. The frequency of precession depends on the strength of the

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Task Force Members are listed in the Appendix.

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magnetic field and the type of nucleus. For hydrogen, this precessional or resonance frequency is approximately 21 MHz for a field of 0·5 Tesla and 63 MHz for a field of 1·5 Tesla. The nuclei align parallel or antiparallel to the field, and only a small net magnetization vector arises from tissue containing many hydrogen nuclei, since almost equal numbers align in opposite directions at body temperature. The small excess of nuclei in the parallel state leads to a net magnetization vector in the direction of the applied field, but at equilibrium there is no magnetization in the plane perpendicular to this field because the precession of individual protons is uncoordinated.

The magnetic resonance signal

If the nuclei are exposed to radiowaves at the resonant frequency, the net magnetization vector is rotated at an angle to the applied field depending upon the amount of energy applied, and the angle is termed the flip angle. After such a displacement, the net magnetization vector relaxes back to its former position tracing out a spiral. Until it reaches this equilibrium position, a component of magnetization exists perpendicular to the applied magnetic field. This component can induce a radio signal at the frequency of precession in an aerial and such a signal is called the free induction decay. The net magnetization vector as it returns to equilibrium can be described by two components. The component parallel to the main field returns to equilibrium by interacting with surrounding molecules. The component perpendicular to the field returns to zero by interaction of the nuclei with each other and also because of local field inhomogeneities, and this is more rapid. Both processes are exponential with time constants T1 (longitudinal relaxation) and T2 (transverse relaxation). Depending upon the sequence of radiofrequency pulses used to disturb the net magnetization vector and the time at which the magnetic resonance signal is recorded, the magnitude of signal and hence the contrast in the image can be made to reflect proton density, T1, T2 or mixtures of each. If the contrast mainly reflects T2, then these images are 'T2 weighted', etc.

Image formation

Magnetic resonance imaging is the process of localizing the radio signal from relaxing nuclei and displaying the signal as an image. Localization is accomplished by additional magnetic fields applied as gradients superimposed upon the externally applied main field. The gradients vary the field strength along their direction which means that the nuclei are excited or irradiate at frequencies depending upon their position.

Images are reconstructed from a number of lines of data in 'frequency' or 'k' space as opposed to 'image' space. In conventional cardiac magnetic resonance imaging, each line is acquired from the same part of successive cardiac cycles and so ECG triggering of the acquisition is essential. The number of lines determines the resolution of the image but also directly influences the duration of the acquisition. Typically, 256 cardiac cycles are used in conventional imaging.

Pulse sequences

A pulse sequence is a combination of radiofrequency pulses and magnetic field gradients. The pulse sequences most commonly used for the heart are the spin echo and gradient echo sequences. In the spin echo sequence, the magnetization vector is tilted by 90° and after a brief time by another 180°. This second tilt leads to coherence of the spins and hence to an echo of the free induction decay signal at a time after the 90° pulse called the echo time (TE). In the images formed from a spin echo sequence, moving blood gives no signal and appears black because it does not experience both radiofrequency pulses. Other tissues give signal which will depend on proton density, T1 or T2 according to the timing of the sequence. Echo times ranging from 20–40 ms are used most commonly. The longer echo times have greater contrast between moving blood and other tissues but lower signal and more motion artefact than the shorter echo times. Spin echo sequences are routinely used for multi-slice anatomical imaging.

The gradient echo sequence consists of an initial radiofrequency pulse tilting the magnetization by a variable angle usually between 10° and 30°. This is followed by switching magnetic field gradients, which dephase and rephase precession of the protons to form an echo. An advantage of this sequence is images of the same plane can be acquired at multiple points of the cardiac cycle, producing a cine acquisition. Because this sequence can be very short, moving blood gives a high signal and appears white unless it is turbulent, when signal may be lost. Conventional gradient echo imaging acquires one line of the image each cardiac cycle, but if a number of lines is acquired in rapid succession overall imaging time is reduced at the expense of a decrease in temporal resolution. For instance, imaging time can be reduced to 15–30 s and hence within a single breath-hold.

Gradient echo sequences have relatively low soft tissue contrast and they are mainly used for studies of ventricular or valve function. However, they can also be adapted to provide high signal from moving blood and low signal from static tissue, allowing two dimensional or three dimensional magnetic resonance angiography. Turbulent flow leads to loss of signal in the gradient echo sequence and the technique can be used to locate assess abnormal jets through valves and other structures.

Modifications of any sequence allow a grid of lines of magnetic saturation to be applied to an image, and this has been termed myocardial tagging. This provides a tool unique to magnetic resonance and it

allows three dimensional tracking of myocardial motion and sophisticated analysis of myocardial strains.

With the development of stronger magnetic gradient systems, several methods of rapid imaging can now be implemented. Echo planar imaging involves acquisition of all image lines (in k-space) after a single excitation. Variants of the technique involve coverage of k-space in a spiral rather than rectilinear fashion (spiral echo planar), and segmental coverage of k-space with several (perhaps eight) excitations to form an image. The echo planar technique is applicable to both spin echo and gradient echo sequences and so there is considerable flexibility in its application. It is however technically demanding and it is not yet used routinely in cardiac imaging.

Velocity mapping is a technique that encodes velocity in a chosen direction in the phase of the magnetic resonance signal. A quantitative image of phase can be formed and the pixel values are velocity in $mm \cdot s^{-1}$. The technique is accurate and flexible with a wide dynamic range allowing encoding of velocities from several $mm \cdot s^{-1}$ to at least $10\ m \cdot s^{-1}$. Integration of these velocities over a vessel area and over the cardiac cycle allows flow to be measured, and pressure gradients formed by discrete jets can be estimated using the modified Bernoulli equation. The method can be applied to any sequence but most commonly a cine gradient echo sequence is used. Appropriate selection of echo time is important since signal is lost in turbulent jets with long echo times. Left sided jets in aortic stenosis and coarctation may require echo times down to 3 ms for accurate visualization of the jet.

Contrast agents

Specific contrast agents are available for magnetic resonance and these are mainly gadolinium complexes with similar physiological properties to the iodinated contrast media used in general radiology. Although natural contrast is inherent to the magnetic resonance technique, the additional flexibility of extrinsic contrast can be valuable in certain circumstances. These agents act by shortening T1 or T2 or both. The main uses in cardiac imaging are to increase contrast between blood and soft tissue for cine functional imaging or angiography, and to assess signal within masses such as tumours and cysts. Bolus injections of contrast can also be combined with rapid gradient imaging to assess the passage of the contrast through the myocardium and hence myocardial perfusion. This technique is still under investigation.

The scanner

A magnetic resonance scanner consists of five major components. The magnet, which is usually superconducting, produces the static magnetic field, which has to be homogeneous and stable with time, and yet large enough to contain a human body. Resistive gradient coils within the bore of the magnet produce the gradients superimposed upon the main field, and the currents within these coils are driven by the gradient amplifiers. The performance of the gradient system determines the speed of acquisition. A radiofrequency aerial, often referred to as a transmitter coil, is coupled to a radiofrequency amplifier to irradiate the patient with the pulses needed to disturb the magnetization. The same or different aerials can be used to receive the radio signal, and surface coils designed to be applied closely to the part of the body to be imaged are frequently used.

Safety

Magnetic resonance imaging is safe and no long-term ill effects have been demonstrated. Claustrophobia occurs in approximately 2% of patients. Very rapidly changing gradients may induce muscle twitching or effects on the retina, but clinical systems operate below the threshold for such effects. Metallic implants such as hip prostheses, mechanical heart valves and sternal sutures present no hazard since the materials used are not ferromagnetic. Patients with electrical stimulators such a pacemakers or cardioverter-defibrillators should not be studied because of the risk of causing arrhythmias from potentials induced in the endocardial wire and the rapid rhythms that some pulse generators develop in a rapidly changing magnetic environment. With appropriate safeguards however patients with pacemakers have been scanned on occasion.

Congenital heart disease

General aspects

Evaluation of congenital heart disease is one of the main strengths of magnetic resonance (Table 1), but its value depends upon the age and clinical condition of the patient. In general, magnetic resonance is more valuable in the older patient with complex anatomy. Sedation is required in small children and physiological monitoring may be difficult in the confines of the magnet in critically ill infants. Thus, magnetic resonance imaging is usually performed after transthoracic echocardiography in neonates and infants. In contrast, body habitus and interposition of lungs become an increasing problem for echocardiography in adolescents or adults, or at any age after surgery. Although some of these problems can be overcome by using the transoesophageal approach, expertise in magnetic resonance imaging is desirable in centres specializing in the care of patients with grown-up congenital heart disease[2,3].

Cardiac catheterization will not be replaced in the near future by magnetic resonance imaging, but the need for diagnostic catheterization and the length and risks of the examination can be minimized by its use[2,4].

Table 1 Indications for magnetic imaging in patients with known or suspected congenital heart disease

Indication	Class
General	
Evaluation of anatomy and/or function if questions remain after echocardiography or X-ray angiocardiography	I
Prior to cardiac catheterization in complex malformations	I
Follow-up studies when echocardiography does not provide complete information and cardiac catheterization is not desirable because information about pressures and vascular resistance is not the primary concern	I
Specific	
Viscero-atrial situs	
Isolated anomalies	II
Anomalies associated with complex congenital disease	I
Atria and venous return	
Atrial septal defect (secundum and primum)	II
Anomalous pulmonary venous return, especially in complex anomalies and cor triatriatum	I
Anomalous systemic venous return	I
baffle repair or correction of anomalous pulmonary venous return	I
Atrioventricular valves	
Anatomical assessment of mitral and tricuspid valves	III
Valve function	III
Ebstein's anomaly	III
Atrioventricular septal defect	III
The ventricles	
Isolated ventricular septal defect	III
Ventricular septal defect with other complex anomalies	I
Ventricular aneurysms and diverticula	II
Evaluation of right and left ventricular function	I
The semilunar valves	
Isolated pulmonary valve stenosis and dysplasia	III
Supravalvular pulmonary stenosis	II
Pulmonary regurgitation	I
Isolated aortic valve stenosis	III
Subaortic stenosis	III
Supravalvular aortic stenosis	I
The arteries	
Malposition of the great arteries	II
Postoperative follow-up of shunts	I
Aortic (sinus Valsalvae) aneurysm	I
Aortic coarctation	I
Vascular rings	I
Patient ductus arteriosus	III
Aorto-pulmonary window	I
Coronary artery anomalies in infants	Inv
Anomalous origin of coronary arteries in adults	I
Pulmonary atresia	I
Central pulmonary stenosis	I
Peripheral pulmonary stenosis	Inv

As catheter-based interventions are used increasingly in paediatric cardiology, diagnosis should be as complete as possible before catheterization which then should become a one stage procedure with completion of diagnosis (if necessary) and intervention being performed at the same time.

Magnetic resonance imaging is generally less operator dependent than echocardiography, but a thorough understanding of the anatomical and functional principles of congenital disease is required for a reliable study. All parts of the cardiovascular system can be imaged, a feature which makes magnetic resonance imaging especially useful in complex cases. For a complete magnetic resonance examination, both spin echo and gradient echo series should be performed. Preferably, spin echo images in the transverse and one

additional orthogonal plane (sagittal or coronal depending on the case) should be available. For answering specific clinical questions, additional oblique sections may be required. Functional information is provided by gradient echo sequences, which have to be tailored to the clinical question. When clinically indicated, the examination should use velocity mapping to assess flow.

Viscero-atrial situs

The viscero-atrial situs (situs solitus, situs inversus, situs ambiguus) and malposition of the heart (dextrocardia, levocardia) are easily identified by conventional diagnostic tools (ECG, X-ray, echocardiography, abdominal ultrasound). However, in the presence of additional lesions such as atrio-ventricular discordance, ventriculo-arterial discordance, anomalous pulmonary or systemic venous connections, difficulties may arise in defining the topographic relationship of the major cardiac segments. Magnetic resonance provides images of the heart which are easily related to surrounding structures[4] and thus it provides reliable and highly accurate diagnoses[5]. In patients with complex anomalies, especially in older ones, magnetic resonance imaging is usually complementary to echocardiography indicating that it should be used routinely to maximize non-invasive information before catheterization.

The veins and atria

Magnetic resonance imaging is potentially valuable in the detection and quantification of atrial septal defects. In older patients, the best technique for imaging the inter-atrial septum is transoesophageal echocardiography and magnetic resonance imaging will provide additional information in only a few patients. In infants, however, magnetic resonance imaging may be used instead of transoesophagel echocardiography following a transthoracic study. Pulmonary and systemic flow can be measured more accurately than by any other technique and so magnetic resonance has a clinical role when such measurements are important[6].

One limitation of echocardiography is the difficulty of evaluating anomalous pulmonary venous return, but magnetic resonance imaging appears to be the best non-invasive technique for this[7–9]. In particular in complex cases, magnetic resonance imaging is superior to conventional imaging techniques since complete demonstration of the pulmonary veins may not be achieved by echocardiography or by X-ray angiography (sensitivity of magnetic resonance imaging 95%, X-ray angiography 69%, echocardiography 38%[8]). Magnetic resonance imaging may also be indicated to identify partial anomalous venous return in patients with atrial septal defects.

When gradient echo sequences are used, magnetic resonance imaging successfully reveals the presence and severity of systemic or pulmonary venous obstruction, which may occur pre- or post-operatively and may be difficult to diagnose by echocardiography. Therefore, a major indication for magnetic resonance imaging is the postoperative evaluation of patients after intra-atrial baffle repair in transposition of the great arteries or correction of anomalous pulmonary venous return[8,10,11]. Moreover, systemic venous anomalies (bi-lateral superior caval vein, interrupted inferior caval vein) are correctly identified by magnetic resonance imaging[5,12].

In cor triatriatum, magnetic resonance imaging may be used if echocardiography is unclear, since the membrane within the atrium is well demonstrated as well as anomalous pulmonary venous return if present[8,13].

The atrioventricular valves

In general, real-time echocardiography, especially by the transoesophageal route, is better suited to show the anatomy and motion of the atrioventricular valves[14]. Magnetic resonance imaging may provide additional information in selected patients[15], especially in infants with an atrioventricular canal defect, where the attachment of the superior and inferior bridging leaflets to the crest of the interventricular septum may be defined more accurately than by transthoracic echocardiography[16].

Anatomy of the tricuspid valve in Ebstein's anomaly is best seen by echocardiography, in adults by the transoesophageal route. However, in cases of doubt the three dimensional arrangement of the malformation and the resulting tricuspid incompetence is well depicted by magnetic resonance imaging[17,18].

The ventricles

Magnetic resonance imaging is sensitive and specific for the detection of ventricular septal defects (sensitivity 84%–100%, specificity 95%)[5,12,14,16,19–21]. Difficulties may arise with small peri-membranous or muscular defects if spin echo images are used alone, but cine gradient echo imaging improves sensitivity by virtue of its ability to detect and localize abnormal jets[22]. As with an isolated atrial septal defect, magnetic resonance imaging usually does not add much information in isolated ventricular septal defect when the diagnosis is already established by echocardiography. Magnetic resonance velocity mapping, however, allows the shunt size to be determined accurately[6], and this may become increasingly important with the move to rely solely on information obtained non-invasively before referral for surgery.

Magnetic resonance imaging has an important role in demonstrating ventricular anatomy in complex anomalies such as in tetralogy of Fallot, double outlet ventricle, pulmonary atresia, tricuspid atresia, and univentricular hearts[5,7,21,23,24], but discrepancies with

X-ray angiography arise in only a few cases (3–8%)[5,15]. Thus, magnetic resonance imaging is often complementary to echocardiography and both techniques should be used before catheterization. Follow-up studies by magnetic resonance imaging and echocardiography may reduce the need for invasive investigation in these cases.

Because of its limited field of view, echocardiography has limitations in the evaluation of congenital ventricular aneurysms, particularly if it is necessary to define the site, size and relationships of congenital diverticulae[25,26]. Thus, magnetic resonance imaging is indicated whenever such anomalies are suspected.

Right ventricular function is an important determinant of outcome in may forms of congenital heart disease, and magnetic resonance imaging has the ability to measure both right and left ventricular volume and mass accurately and reproducibly[27–31]. This may be particularly helpful in patients after the Mustard procedure. The consequences of pulmonary regurgitation on right and left ventricular function can be evaluated by combining measurements of ventricular volume and diastolic function[32,33]. Such functional measurements are likely to have important prognostic and therapeutic implications for the post-operative management of patients with congenital heart disease.

The semilunar valves

Congenital stenoses of the semilunar valves are easily identified by echocardiography, and maximal and mean gradients can be determined by Doppler ultrasound. Although cine gradient echo imaging with velocity mapping can also detect and quantify stenoses[34,35], Doppler echocardiography is usually adequate. Magnetic resonance imaging may be particularly useful in localizing stenoses below, at, or above the valve if echocardiography fails to give complete information[36].

Patients with previous pulmonary valve surgery often have important pulmonary regurgitation, and this may be difficult to assess by echocardiography. In such cases, magnetic resonance imaging can aid the decision on replacement of the valve[37].

The arteries

Magnetic resonance imaging is an excellent method of demonstrating the size, position and connections of the central great arteries[23,38,39]. Similarly, abnormalities of the thoracic and abdominal aorta can be demonstrated, including aneurysm, dilation, stenosis, duplication, and vascular rings[36,40–42].

Two-dimensional-echocardiography and Doppler ultrasound are usually adequate for the diagnosis and assessment of coarctation, but difficulties may be encountered in older children or adults when magnetic resonance imaging has a clear role, particularly for diffuse narrowings of the arch. Velocity mapping may be helpful for assessing the severity of coarctation either from the pressure gradient[43] or from measurements of collateral flow[44]. For follow-up after surgical repair or angioplasty magnetic resonance imaging is probably the technique of choice[45–47].

Visualization of a patent arterial duct is normally achieved in infants by echocardiography, but magnetic resonance imaging has a role in older patients where it is often better than echocardiography[48]. In patients with a suspected aorto-pulmonary window, magnetic resonance imaging may be helpful to establish the differential diagnosis or to demonstrate the additional defect[7].

Magnetic resonance imaging can demonstrate congenital anomalies or inflammatory changes of the coronary arteries as in Bland-White-Garland syndrome[49] or Kawasaki disease[50]. Recent reports indicate that congenital anomalies of the proximal coronary arteries can be shown reliably in adults[51,52], and the technique may even have advantages over X-ray angiography, since the relationship of the arteries to the aorta and pulmonary artery is an important determinant of risk in these patients, and this is not always clear following invasive angiography.

Visualization of the pulmonary artery and its main branches is essential in the surgical management of patients with diminished pulmonary blood flow. Echocardiography is of limited value because of reflection of the sound beam by the chest wall and lungs. Similarly, X-ray angiocardiography has its problems; since it can be dangerous in cyanotic children, small pulmonary arteries may be difficult to catheterize, and non-confluent vessels may not be seen even with pulmonary venous wedge contrast injection. Therefore this group of patients is particularly suited to magnetic resonance imaging. Studies of pulmonary atresia, tricuspid atresia, tetralogy of Fallot and unilateral pulmonary artery anomalies have shown very few discrepancies with X-ray angiocardiography in defining the anatomy of the central and hilar pulmonary arteries as well as in determining the sources of pulmonary blood flow[24,53–57]. In addition, magnetic resonance imaging can reveal undetected pulmonary and collateral vessels. A further major impact of magnetic resonance imaging is in serial follow-up examinations, where it can show the shape and size of the pulmonary arteries, the patency of systemic-to-pulmonary shunts, and extra-cardiac conduits[57–60], as well as measure pulmonary flow[61,62]. Extra-cardiac ventriculo-pulmonary shunts often degenerate in the long term, but this is not easy to assess by echocardiography because of the retro-sternal position of the graft. Magnetic resonance imaging provides accurate anatomical and functional information and is the technique of choice for following these patients[63].

Acquired disease of the great vessels

In addition to congenital heart disease, the clinical value of magnetic resonance imaging is well documented in

Table 2 Indications for magnetic resonance imaging in acquired disease of the great arteries

Indication	Class
Diagnosis of thoracic aortic aneurysm	I
Diagnosis and follow-up in Marfan disease	I
Aortic dissection	
Diagnosis of acute aortic dissection	II
Diagnosis of chronic aortic dissection	I
Diagnosis of aortic intramural haemorrhage	I
Diagnosis of penetrating atheromatous ulcers of the aorta	I
Follow-up of acquired aortic disease	I
Pulmonary artery anatomy	I
Pulmonary emboli	
Diagnosis of central pulmonary emboli	III
Diagnosis of peripheral pulmonary emboli	Inv
Assessment of pulmonary flow and pulmonary hypertension	III
Assessment of thoracic veins	I

patients with disease of the great vessels, more specifically with acquired disease of the aorta (Table 2). The large field of view and the flexibility of imaging planes provides a clear description of anatomy and of relationships to neighbouring structures. Rapid flow leads to high contrast between blood and the vessel wall, and velocity mapping can be used to assess flow both qualitatively and quantitatively.

Thoracic aortic aneurysm

Spin echo magnetic resonance imaging depicts the extent and diameter of fusiform or saccular aneurysms of whole thoracic aorta, regardless of aetiology[64–66]. In conditions such as mycotic pseudo-aneurysm and perivalvular pseudo-aneurysm complicating bacterial endocarditis, magnetic resonance imaging is more valuable than transthoracic echocardiography[67,68]. Gradient echo imaging can be used to detect and characterize mural thrombus[69–71], and velocity mapping provides a measure of associated aortic regurgitation[72,73]. Thin slice imaging (5 mm) or segmented k-space breath-hold angiography can be used to identify the involvement of aortic branches. Three dimensional angiography provides attractive and easily interpretable images, but its additional value over simpler techniques is not yet well defined[74].

Marfan's syndrome

The cardiovascular complications of Marfan's syndrome include aortic dilation and dissection, and this is the main cause of death. X-ray angiography[75], echocardiography[76], computed X- ray tomography[77], and magnetic resonance imaging[78–80] have all been used to image the aorta in Marfan patients, with excellent agreement between echocardiography and magnetic resonance imaging for measuring the size of the aortic root[78]. Aortic distensibility can be measured in both children[81] and adults[82], although the clinical importance of such measurements is still to be defined. The technique is valuable for follow-up since aneurysm expansion and dissection are clearly defined and are often detected in asymptomatic patients[83,84]. Thus the lack of ionising radiation and the ability to visualize the whole thoracic aorta make magnetic resonance a first line imaging technique in these patients.

Aortic dissection

There is substantial evidence that magnetic resonance imaging has the highest accuracy for the detection of aortic dissection of all imaging techniques[85–87]. It not only identifies the intimal flap and the site of tear, but it can also demonstrate associated abnormalities such as thrombus, aortic regurgitation, and pericardial effusion[87]. Although transoesophageal echocardiography is also sensitive, it has several pitfalls that reduce specificity[87]. Computed X-ray tomography is also accurate, but it cannot evaluate functional aspects such as blood flow, entry jets into the false lumen, and aortic regurgitation. Spiral computed X-ray tomography shortens imaging time without loss of accuracy[49], but there are not yet sufficient comparisons with magnetic resonance imaging to assess their relative clinical roles.

In populations with an intermediate likelihood of dissection (disease prevalence 10%), the positive predictive value of magnetic resonance imaging, computed X-ray tomography and transoesophageal echocardiography is higher (>90%) than that of X-ray angiography (65%)[88]. Invasive angiography should therefore no longer be used as a primary investigation, although it remains valuable for assessment of the coronary arteries if this is required. Practical aspects such as local expertise and availability of techniques will often determine

the best order of investigations. The problems of imaging haemodynamically unstable patients by magnetic resonance favour transoesophageal echocardiography in this situation, with magnetic resonance imaging or computed X-ray tomography reserved for equivocal cases or for a more complete assessment once the patient is stable. In subacute situations or for chronic follow-up, magnetic resonance imaging is the technique of choice[89].

Although aortic intramural haemorrhage was once considered a separate entity, it is now thought to be a precursor of dissection. Magnetic resonance imaging can detect such haemorrhage[80,90], and it has been used to show that the clinical profile and outcome of the condition is similar to classic dissection, and hence requires a similar therapeutic approach[91] The recognition of intramural haemorrhage relies on visualizing wall thickening (>7 mm) with a smooth surface and possibly containing areas of high signal. The high signal is the result of methaemoglobin which develops after several days and persists for several months, although it may be absent in the acute phase[80]. Both computed X-ray tomography and transoesophageal echocardiography are alternatives to magnetic resonance imaging[90,91] but X-ray angiography is not useful. Because intramural haemorrhage of the thoracic aorta may be associated with true dissection of the abdominal aorta, the latter should also be evaluated.

Penetrating ulcers and atherosclerotic disease

Penetrating ulcers are almost always associated with extensive atherosclerosis of the aorta, primarily affecting the intima and only secondarily the media. The disease therefore differs from dissection or intramural haematoma which are diseases of the media, although symptoms of all three may be similar. Penetrating ulcers usually arise in the middle and distal third of the descending thoracic aorta[92]. Treatment may be surgical or conservative and close follow-up is required in order to detect complications. Magnetic resonance imaging is accurate in making the diagnosis[93], and typical features include a thickened and irregular wall, an ulcer-shaped irregularity at a typical site, and haematoma in the wall of the aorta with high signal intensity caused by methaemoglobin. Flexible imaging planes and the ability to image without contrast material are advantages over computed X-ray tomography, although X-ray tomography is able to show displacement of intimal calcification which can be a helpful diagnostic sign[94]. The role of transoesophageal echocardiography is currently unknown.

Atherosclerotic plaques thicker than 4 mm in the aortic arch predict cerebral infarction and other vascular events[95]. Although such plaques can be detected by magnetic resonance imaging[96], its role in estimating risk is unknown.

Postoperative follow-up

All of the above conditions require follow-up whether treatment is conservative or surgical. The accuracy of magnetic resonance imaging, its versatility, non-invasive nature, and lack of necessity for ionizing radiation or contrast media, make it the method of choice for this purpose[85,97,98].

Pulmonary artery anatomy

Most pulmonary artery pathology occurs in the setting of congenital anomalies, which are discussed above. Dilation, thrombo-embolic involvement, and rare complications such as dissection are well demonstrated by magnetic resonance imaging[99,100].

Pulmonary embolism

Early studies suggested a possible role for magnetic resonance imaging in detecting central pulmonary embolism[101–103], but it cannot currently be recommended for routine use and ventilation-perfusion scintigraphy supplemented by X-ray angiography remains the diagnostic standard. Recent successful studies using spiral X-ray tomography indicate that this technique is a useful diagnostic alternative. The value of this cross-sectional technique, however, suggest that newer magnetic resonance techniques may find a role once more experience has been acquired. These techniques include magnetic resonance angiography using fast two dimensional time-of-flight gradient echo sequences combined with maximum intensity projections, and this appears to be sensitive (92–100%), but only moderate specific (62%)[104]. Better results have been obtained using phased-array coils and three dimensional angiography[105,106], or contrast-enhanced angiography[107]. Tagging can also be helpful to differentiate thromboembolism from flow related signal[108].

Pulmonary hypertension

Flow can be measured accurately by magnetic resonance velocity mapping in the main and left and right pulmonary arteries[109]. The cyclical pattern of flow is altered by pulmonary hypertension which leads to a lower peak velocity and greater retrograde flow at the end of systole[110]. Although the significance of these observations is not fully understood, it may be related to pulmonary vascular resistance and hence serve as a method of measuring resistance.

Thoracic veins

The superior and inferior caval veins and their relationship to the right atrium can be visualized by magnetic

Table 3 Indications for magnetic resonance imaging in patients with valvular heart disease

Indication	Class
Valve morphology	IV
Associated cardiac chamber morphology	III
Associated intracardiac thrombus	III
Associated ventricular or atrial function	III
Detection and quantification of regurgitation	II
Detection and quantification of stenosis	III
Detection of infective endocarditis and vegetations	IV
Detection of paravalvular abscess	II
Assessment of prosthetic valves	III

resonance imaging, and this has been useful for assessing thrombus and invasion or compression by tumour[111]. In addition, flow measurements using velocity mapping have shown altered patterns in pulmonary hypertension, pericardial constriction, and tricuspid regurgitation[112,113]. Pulmonary venous flow has been used to assess diastolic left ventricular filling and the severity of mitral regurgitation[114].

Valvular heart disease

Normal heart valves are thin, rapidly moving structures and they are poorly seen in spin echo images. Abnormal valves are more easily seen because they are thicker and they may be less mobile. Occasionally it is possible to obtain diagnostic information from spin echo images but cine gradient echo imaging is more informative. Valve calcification may lead to loss of signal but the absence of this sign cannot rule it out.

Regurgitation

The measurement of regurgitation is an important but difficult aspect of the assessment of valvular disease. The need for intervention is determined partly by the severity of symptoms, but other measurements such as the severity of regurgitation and ventricular function are important. Echocardiography and radionuclide ventriculography are commonly used to assess left ventricular function, but quantification of the regurgitation is often felt to require invasive studies with left ventricular cine X-ray angiography. Magnetic resonance imaging has the potential to help in several ways (Table 3).

The signal intensity of flowing blood during cine gradient echo imaging depends upon the nature of the flow. In general, flowing blood generates uniform high signal because of continuous replacement of magnetically saturated blood by fresh blood. Turbulence leads to loss of signal and so the turbulent jet of mitral regurgitation can be seen in the left atrium[115]. The size of the signal void can be used as a semi-quantitative measure of regurgitation[116] but the signal void does not necessarily correspond to the area of turbulence, and that it will vary with imaging parameters such as echo time. This is similar to colour flow Doppler where technical factors such as gain adjustment and filter setting are important[117]. A more fundamental problem common to both is that the size of the regurgitant jet is influenced by many factors in addition to the severity of regurgitation, such as the shape and size of the regurgitant orifice and the size of the receiving chamber.

There is a correlation between the angiographic grading of mitral regurgitation and the length and area of the regurgitant jet imaged by Doppler echocadiography. A similar correlation exists between colour Doppler and cine magnetic resonance although magnetic resonance shows a smaller area of flow disturbance[118]. This may be the result of different imaging planes or the fact that the techniques use different physical principles. A potential advantage of magnetic resonance is that it is able to sample a complete three dimensional volume and so may provide a more accurate assessment of the volume occupied by the regurgitant jet.

If only a single valve is regurgitant, comparison of left and right ventricular stroke volumes allows the regurgitant fraction to be calculated as described above. If single valves on both sides of the heart are regurgitant, the method can be extended by comparing ventricular stroke volumes with great vessel flow measured by magnetic resonance velocity mapping. The regurgitant fraction then compares well with the regurgitant grade assessed by Doppler echocardiography[37]. The method still fails if both valves on one side of the heart are regurgitant, but flow studies in the proximal aorta (or pulmonary artery) can be used to measure aortic (or pulmonary) regurgitation alone from the amount of retrograde diastolic flow in the artery[72], and it is then possible to assess even the most complex cases.

Stenosis

As with regurgitant jets proximal to a valve, a turbulent distal jet can be used to detect potential stenosis. Although the size of the region of signal loss is related to the pressure gradient across a lesion, it has not proved simple to use this as a measure of stenosis because other factors can lead to turbulence and signal loss without

Table 4 Indications for magnetic resonance imaging in patients with pericardial disease, cardiac tumours, cardiomyopathies, and cardiac transplants

Indication	Class
Pericardial effusion	III
Constrictive pericarditis	II
Detection and characterization of cardiac tumours	I
Hypertrophic cardiomyopathy	II
Dilated cardiomyopathy	III
Arrhythmogenic right ventricular dysplasia	Inv
Restrictive cardiomyopathy	II
Post-cardiac transplantation	
Acute rejection	IV
Chronic rejection	III
Other complications	IV

stenosis. For instance, it is not uncommon to see turbulence distal to a rheumatic valve that is not stenosed because of the disturbed flow across it. The degree to which this signal loss is seen depends to a large extent upon the echo time of the gradient echo sequence used. Short echo times are less susceptible to signal loss. Despite these problems, the technique has been used successfully in the follow-up of patients after balloon mitral valvuloplasty[119]. The modified Bernoulli equation makes a number of assumptions which when valid allows the pressure gradient across a stenosis to be calculated from the peak velocity. The method is widely used in Doppler ultrasound and it can be applied in the same way to velocity measurements made by magnetic resonance. A sequence with a short echo time is important in order to avoid loss of signal from areas of turbulence. The technique is accurate both in vitro and in vivo[120] and a close agreement between magnetic resonance and Doppler measurements has been demonstrated in patients with mitral and aortic stenosis and with stenoses of conduits and great vessels[35,63].

An advantage of magnetic resonance over Doppler measurements of peak velocity is that the magnetic resonance technique allows velocities in any direction to be measured without the limitation of acoustic windows and the requirement to align a beam along the direction of a jet. Provided that an appropriate velocity sensitivity is used during the acquisition aliasing does not occur, although if the anticipated velocity is underestimated this can be a problem. The resolution allows unambiguous distinction between the jets of mitral stenosis and aortic regurgitation. A disadvantage of magnetic resonance is that it is not yet real time and so careful alignment of the imaging plane is required in order to obtain an accurate measurement.

Prosthetic valves

Patients with prosthetic valves are commonly encountered but prostheses are not visualized because the alloy from which they are made contains no mobile hydrogen atoms and so does not give a magnetic resonance signal. There is distortion of the applied magnetic field by the difference of susceptibility between prosthesis and biological tissue and by eddy currents induced in the valve, and this leads to loss of signal from tissues for a variable distance around the prosthesis. This distance is small for spin echo images and neighbouring structures are seen normally, but the defect in the image is much larger in gradient echo images making it difficult to assess turbulent jets in the region of the valve. Metal valves are not ferromagnetic, however, and at currently available magnetic field strengths there is no effect of the field upon the working of the valve[121,122].

Cardiomyopathies and transplantation

Cardiomyopathy describes a number of conditions with either primary or secondary myocardial involvement. Magnetic resonance imaging has the potential to identify and differentiate the cardiomyopathies and it is also useful in secondary myocardial hypertrophy (Table 4).

Hypertrophic cardiomyopathy

Two dimensional and Doppler echocardiography are common methods of diagnosing and assessing function in hypertrophic cardiomyopathy[123,124], but they do not provide complete information in all cases, particularly in cases with localized apical hypertrophy[125]. In contrast, spin echo magnetic resonance imaging provides reliable images of the whole myocardium and gives a three-dimensional assessment of regional hypertrophy. Cine gradient echo imaging and velocity mapping also provide functional measurements[126–128], including a measure of outflow tract gradient, if present, but magnetic resonance imaging has not been shown to be better than Doppler echocardiography for this parameter. Magnetic tagging demonstrates regional myocardial motion and strain in a unique fashion[129]. Thus, magnetic resonance imaging is a second line technique in hypertrophic cardiomyopathy if echocardiography does not provide complete information.

Secondary hypertrophy

Measurements of myocardial mass have been validated in animals[130–132] and in humans[133,134] and magnetic resonance imaging is now the standard against which other techniques are judged. Increased muscle volume (and hence mass) can be observed in athletes and patients with left ventricular hypertrophy, and the regression of hypertrophy following treatment of hypertension can be monitored[135].

Dilated cardiomyopathy

The anatomical and functional abnormalities of dilated cardiomyopathy are clearly demonstrated and measured by magnetic resonance imaging. The appearance is, however, non-specific and it is not possible to distinguish dilated cardiomyopathy from other causes of left ventricular dysfunction such as ischaemic heart disease. Advantages over echocardiography include better depiction of the right ventricle[136] and low variability of functional measurements[137], but echocardiography normally provides adequate clinical information, and the role of magnetic resonance is mainly in quantifying the effects of therapy[138]

Arrhythmogenic right ventricular dysplasia

Magnetic resonance imaging is probably the best imaging technique for demonstrating structural and functional abnormalities of the right ventricle. Global and regional abnormalities can be seen in arrhythmogenic right ventricular dysplasia and also in patients with right ventricular tachycardia who do not have abnormalities on other imaging techniques[139,140]. These abnormalities include regional thinning, failure of systolic thickening, ventricular dilation, impaired global systolic and diastolic function, and areas of high signal intensity within the myocardium which indicate fatty infiltration[141,142].

Restrictive cardiomyopathy

Magnetic resonance imaging can show anatomical and functional abnormalities associated with restrictive cardiomyopathy[143], although echocardiography remains the initial diagnostic tool in patients with unexplained symptoms of right and/or left ventricular failure. The main contribution of the technique is the straightforward and accurate differentiation of restrictive cardiomyopathy from constrictive pericarditis by virtue of visualization of the abnormal pericardium in the latter condition in spin echo images[144]. Cine gradient echo imaging to assess the functional consequences of both diseases may be helpful in patients with limited ultrasonic windows. Computed X-ray tomography (conventional, spiral or electron beam) may have similar accuracy in making this distinction.

Cardiac transplantation

Imaging may play a role in solving two common clinical problems in patients after heart transplantation. First, the early detection of acute episodes of rejection and second, the identification of accelerated coronary artery disease commonly considered as chronic rejection.

Acute rejection is characterized on spin echo images as an increase in myocardial mass[145] and areas of high myocardial signal[146], which indicate myocardial oedema and/or infiltration by mononuclear cells. Changes in relaxation times and regional functional abnormalities may also be observed, but none of these abnormalities is sensitive for detecting early acute rejection. Similarly, magnetic resonance imaging is not helpful in chronic rejection, although it may reveal other complications of transplantation such as pericardial disease or intracavitary masses[145].

Cardiac tumours

Echocardiography, especially using the transoesophageal approach, is well suited to detecting and localizing tumours and it may provide complete information on origin, extent, and resectability. Magnetic resonance imaging is, however, valuable as a secondary technique to confirm or exclude the diagnosis when echocardiography gives incomplete information[147] (Table 4). Important information provided includes the relationship and involvement of surrounding structures such as adjacent vessels and mediastinal structures[148]. Pericardial involvement is shown more clearly by magnetic resonance imaging than by echocardiography[149], and it may be particularly helpful in planning surgery[147].

Some features of the images allow limited tissue characterization but a definitive distinction between malignant or benign masses can only rarely be made. High signal intensity on T1 images may represent fatty tumours such as lipoma or liposarcoma, recent haemorrhage, cystic lesions with high protein content, or melanoma. Low signal intensity may represent a cyst with low protein content, signal loss in a vascular malformation, calcium, or air[150]. On T2 images, cysts have high signal intensity irrespective of protein content. Solid tumours increase in signal with following injection of contrast, but cysts are unchanged.

Pericardial disease

Both magnetic resonance imaging and computed X-ray tomography are well suited to imaging the pericardium,

Table 5 Indications for magnetic resonance imaging in patients with coronary artery disease

Indication	Class
Assessment of myocardial function	III
Detection of coronary artery disease	
Analysis of regional left ventricular function during stress	III
Assessment of myocardial perfusion	Inv
Coronary angiography	Inv
Bypass graft angiography	III
Assessment of coronary flow	Inv
Detection and quantification of acute myocardial infarcts	IV
Sequelae of myocardial infarction	
Myocardial viability	II
Ventricular septal defect	III
Mitral regurgitation	III
Intraventricular thrombus	II

but magnetic resonance is also able to quantify associated functional abnormalities[112] (Table 4). Both techniques have a much larger field of view than echocardiography, but echocardiography remains the usual initial technique because of its simplicity and availability.

Pericardial effusion

Gradient echo images normally show pericardial fluid with high signal because of motion related enhancement caused by cardiac pulsation. T1 spin echo images show pericardial fluid with variable signal depending upon the content and the motion of the fluid[151]. Transudates normally have relatively low signal intensity, whereas exudates and subacute and chronic haemorrhagic effusions have high signal. Imaging is particularly helpful in patients with loculated or complex configurations of effusions which may be difficult to localize by echocardiography. As in other conditions, magnetic resonance imaging can be helpful whenever there is doubt following echocardiography.

Constrictive pericarditis

The characteristic appearance of constrictive pericardial disease is pericardial thickening, an elongated and narrow right ventricle, a sigmoid shaped septum with abnormal motion, enlargement of the right atrium and inferior caval vein, and stagnant blood in the atria. Pericardial thickening is the hallmark of constriction, although rare cases without thickening have been described[144]. All of these changes are readily seen using spin echo imaging[144,152] and the positive predictive accuracy of the technique in one study was 100%[144]. Calcification is more easily identified by computed X-ray tomography, although large deposits of calcium can be seen by magnetic resonance as areas of low signal. Calcification is less commonly a diagnostic feature in recent years[153] and so this is not a major disadvantage. Magnetic resonance imaging and computed X-ray tomography are better than echocardiography in measuring pericardial thickness, but magnetic resonance has the advantage of permitting assessment of haemodynamic impairment. Velocity mapping may be helpful in assessing haemodynamics since there is attenuation of the diastolic peak of flow in the caval veins with any cause of impaired right ventricular filling[154]. Because computed X-ray tomography does not require ECG triggering and because imaging time is shorter, it may be preferred in sick patients or in those with arrhythmias.

Congenital pericardial anomalies

Pericardial cysts can be distinguished from other tumours by their characteristic low signal in T1 weighted images and high signal in T2 images. However, differentiation from a necrotic or cystic mediastinal tumour can be difficult.

Absence of the pericardium is associated with a leftward shift of the long axis of the left ventricle. Partial absence may lead to a localized protrusion, although the defect itself may be difficult to see[155].

Coronary artery disease

Magnetic resonance imaging can contribute to the assessment of patients with coronary artery disease in a number of ways, although coronary and myocardial perfusion imaging have not reached the stage where they can be useful in routine practice. The most useful area is in the assessment of myocardial thickness and thickening, particularly for assessing myocardial viability and hibernation (Table 5).

Ventricular function

Magnetic resonance imaging appears to be the most accurate technique for assessing left ventricular mass[130,131,156,157]. Although spin echo images depict the endocardial boundary more clearly, cine gradient echo imaging is faster and equally accurate in practice[158]. Because the technique makes few assumptions about ventricular geometry it is particulary useful in irregular ventricles such as after infarction[134]. Left and right ventricular volumes[159–161], left ventricular ejection fraction[162,163], and regional left[164–167] and right ventricular function[168,169] can all be assessed. Reproducibility is excellent[133,137,138,170,171]. Thus, magnetic resonance imaging should be considered whenever accurate measurements are required, such as in monitoring therapy. In clinical practice, regional and global function is often estimated visually and without quantification from the computer screen.

Myocardial tagging is a technique unique to magnetic resonance imaging[172,173] and it can be used to measure regional strain, motion and thickening with sufficient spatial resolution to distinguish endocardial and epicardial function[174–176]. It has proved a valuable research tool but it is not commonly used in routine practice.

Detection of coronary artery disease

Stress imaging of left ventricular function

Several approaches to detecting flow limiting coronary stenoses have been described. Cine gradient echo imaging can be used to assess regional ventricular function at rest and during a number of interventions. Dynamic exercise is difficult within the confines of a magnet and it leads to unacceptable motion artefact. As with echocardiography, dobutamine appears to be more appropriate than dipyridamole for pharmacological stress because it more consistently provokes ischaemia rather than perfusion inhomogeneity[177,178]. The diagnostic accuracy of this approach is between 75% and 90% depending on the number of diseased vessels[178–182], and this can be improved by quantification especially in patients with single vessel disease[183]. To date, there are no comparisons of stress magnetic resonance imaging and stress echocardiography but the techniques are similar in principle, if different in the quality of myocardial images obtained.

Myocardial perfusion imaging

Magnetic resonance is able to assess myocardial perfusion at rest and during pharmacological stress in a research setting[184]. Rapid imaging with acquisition once each cardiac cycle allows the myocardial transit of a bolus of contrast to be observed and parameters related to myocardial perfusion to be derived[185–187]. The typical pattern in a territory served by a diseased artery is delayed and attenuated appearance of contrast[185,188]. Comparisons with myocardial perfusion scintigraphy are, however, limited and it is not clear how robust the technique will prove in clinical practice. One important limitation that is being addressed is the limited coverage of the ventricle that can be obtained in a single study, but multi-slice perfusion studies may be possible.

Coronary angiography

Probably the most important approach to detecting coronary disease is magnetic resonance coronary arteriography, since a successful non-invasive technique would transform assessment of the patient with known or suspected coronary disease. Magnetic resonance angiography is already used routinely in many centres for the carotid and intracerebral arteries, for aortography and for the ileo-femoral system. Imaging of the coronary arteries is, however, much more demanding because of their size, complex three dimensional anatomy, and rapid motion.

Rapid gradient echo imaging within a single breath-hold was the first technique to provide promise[189], and sensitivities for proximal stenoses range from 63%[190] to 90%[191], with best results for the left main coronary artery[192]. Spatial resolution, signal loss from turbulence and other artefact, and the inability to evaluate the distal coronary tree currently limit clinical application to the assessment of anomalous coronary arteries and other special circumstances, but several developments may overcome these limitations. These include more powerful gradients for rapid imaging, better surface coils, and real time tracking of diaphragmatic motion in order to avoid the need for breath holding[193–195]. Similar principles can be used to image coronary bypass grafts. Using both spin echo[196,197] and gradient echo imaging[198,199] accuracies of around 90% for predicting graft patency have been reported. However, anastomotic stenoses or progression of native vessel disease are not easy to demonstrate reliably. Measurement of graft flow is possible and this obviously provides more important information than patency alone[200–202].

Further development of all of these techniques and comparative evaluation alongside computed X-ray tomography of the coronary arteries can be expected in the coming years.

Coronary flow

A further approach to detecting and characterising coronary stenosis is the measurement of coronary velocity and flow[203–206]. Using rapid gradient echo velocity mapping coronary flow velocities can be measured at rest and after maximal coronary dilation with adenosine, and good agreement has been demonstrated with invasive measurements in animals[207] and in humans[208]. In theory, it should be possible to measure coronary flow reserve and preliminary results are encouraging.

Acute myocardial ischaemia and infarction

Areas of acute ischaemia and infarction develop high signal intensity on T2 weighted spin echo images[209–212], and regional myocardial thinning is the most specific feature of acute necrosis[213–215]. Paramagnetic contrast agents may help in the detection and sizing of infarction[216–218]. However, the difficulties of imaging acutely ill patients means that these techniques have no role in assessment of acute infarction.

Magnetic resonance imaging may be helpful in detecting the short and long-term sequelae of acute infarction. For instance, regional thinning is indicative of infarction and in the long term severe thinning indicates transmural scar[219]. Assessment of myocardial thickness and thickening are therefore finding a role in the assessment of myocardial viability and hibernation. There is a correlation between these parameters and uptake of 18F-fluorodeoxyglucose assessed by positron emission tomography[220,221]. In addition, in akinetic regions, improvement in response to low dose dobutamine infusion also indicates viability and likely hibernation[221].

Complications of acute infarction such as aneurysm[25], ventricular septal defect[22], and mitral regurgitation are readily demonstrated. Because echocardiography can give misleading results when looking for ventricular thrombus after infarction[222], magnetic resonance imaging may be warranted when investigating embolic complications[150,223]. It also has advantages over other techniques in identifying pseudoaneurysms of the left ventricle[224].

Magnetic resonance spectroscopy

The signal source for magnetic resonance imaging is exclusively the ^{1}H nucleus, more specifically the abundant ^{1}H nuclei in water (H_2O) and fat (CH_2 and CH_3 groups). Magnetic resonance spectroscopy also involves other nuclei with a net nuclear spin such as ^{32}P. An important problem for spectroscopy is its low sensitivity, since all nuclei other than ^{1}H have a substantially lower magnetic resonance sensitivity and are present in concentrations orders of magnitude lower than ^{1}H in water and fat.

^{31}P spectroscopy allows high energy phosphate metabolism to be investigated and it therefore provides a non-invasive estimate of the energetic state of the heart. Six resonances can be identified in the myocardium: the three ^{31}P atoms of ATP (α, β, γ) phosphocreatine (PCr), inorganic phosphate (Pi) and monophosphate esters (MPE). Each of these resonates at a different frequency by virtue of their chemical environments and so each appears with a different 'chemical shift', expressed in parts per million relative to the main magnetic field. The area under each resonance is proportional to the amount of signal and hence to the amount of the nucleus. Absolute concentrations can be evaluated by acquiring spectra with a known external standard. In addition, intracellular pH can be measured from the chemical shift difference between PCr and Pi.

Clinical magnetic resonance spectroscopy faces a number of problems, such as long examination time, cardiac and respiratory motion, localization of signal, low sensitivity and hence a large voxel size (currently approximately 30 cc), and the difficulties of measuring absolute concentrations of metabolities. Higher fields strengths[225], improvements in hardware and software and the nuclear Overhauser effect may allow some of these obstacles to be overcome. If so, we can anticipate true three-dimensional metabolic imaging with voxel sizes of less than 5 cc.

Almost without exception, human cardiac spectroscopy has been confined to the ^{31}P nucleus. Although it has contributed to our understanding of cardiac patho-physiology it remains a research technique without a validated clinical role. At present, cardiac ^{31}P spectroscopy has been applied in three main areas[226].

Coronary artery disease

In patients with left anterior descending stenosis and a reversible thallium perfusion defect, the anterior myocardial PCr/ATP ratio is normal at rest[227], but it decreases during isometric exercise and returns towards normal after exercise[228]. In areas with fixed thallium defects PCr/ATP is reduced at rest and it does not decrease with exercise[229]. Measurement of absolute concentrations of PCr and ATP has recently been reported[230]. ATP at rest is reduced in patients with fixed thallium defects but unchanged in those with reversible defects. This suggests that these measurements may be useful for the assessment of myocardial metabolism and viability after myocardial infarction.

Heart failure

The PCr/ATP ratio is reduced in patients with advanced heart failure of ischaemic[231] and non-ischaemic origin[227], but it may be unchanged in the early stages[227,232]. The reduction correlates with the clinical severity of heart failure, and it improves with medical treatment[227]. Thus, ^{31}P-magnetic resonance may provide an objective index of the severity of heart failure and it may allow treatment and progression of disease to be monitored. The ratio may also be related to prognosis[233].

Valve disease

In patients with aortic and mitral valve disease the PCr/ATP ratio is unchanged in mild stenosis and incompetence, but it is reduced with the onset of

heart failure[233]. Further studies are required to assess whether this might be helpful in timing valve replacement.

References

[1] Guidelines for clinical use of cardiac radionuclide imaging. Report of the American College of Cardiology/American Heart Association Task Force of diagnostic and Therapeutic Cardiovascular Procedures (Committee on Radionuclide Imaging), developed in collaboration with the American Society of Nuclear Cardiology. J Am Coll Cardiol 1995; 25: 521–47.

[2] Hirsch R, Kilner PJ, Connelly MS, Redington AN, St John Sutton MG Somerville J. Diagnosis in adolescents and adults with congenital heart disease. Prospective assessment of individual and combined roles of magnetic resonance imaging and transesophageal echocardiography. Circulation 1994; 90: 2937–51.

[3] Hoppe U, Dederichs B, Deutsch HJ, Theissen P, Schicha H, Sechtem U. Congenital heart disease in adults and adolescents: comparative value of transthoracic and transesophageal echocardiography and MR imaging. Radiology 1996; 199: 669–77.

[4] Geva T, Vick GW, Wendt RE, Rokey R. Role of spin echo and cine magnetic resonance imaging in presurgical planning of heterotaxy syndrome. Comparison with echocardiography and catheterization. Circulation 1994; 90: 348–56.

[5] Kersting-Sommerhoff BA, Diethelm L, Stanger P *et al*. Evaluation of complex congenital ventricular anomalies with magnetic resonance imaging. Am Heart J 1990; 120: 133–42.

[6] Hundley WG, Li HF, Lange RA. *et al*. Assessment of left-to-right intracardiac shunting by velocity-encoded, phase-difference magnetic resonance imaging. A comparison with oximetric and indicator dilution techniques. Circulation 1995; 91: 2955–60.

[7] Sieverding L, Klose U, Apitz J. Morphological diagnosis of congenital and acquired heart disease by magnetic resonance imaging. Pediatr Radiol 1990; 20: 311–9.

[8] Masui T, Seelos KC, Kersting-Sommerhoff BA, Higgins CB. Abnormalities of the pulmonary veins: evaluation with MR imaging and comparison with cardiac angiography and echocardiography. Radiology 1991; 181: 645–9.

[9] Hsu YH, Chien CT, Hwang M, Chiu IS. Magnetic resonance imaging of total anomalous pulmonary venous drainage. Am Heart J 1991; 121: 1560–5.

[10] Chung KJ, Simpson IA, Glass RF, Sahn DJ, Hesselink JR. Cine magnetic resonance imaging after surgical repair in patients with transposition of the great arteries. Circulation 1988; 77: 104–9.

[11] Kaemmerer H, Theissen P, Kaulitz R *et al*. Magnetresonanztomographische Beurteilung von Anatomie und Ventrikelfunktion nach Mustard-Korrektur bei Transposition der grossen Arterien. Z Kardiol 1992; 81: 217–25.

[12] Kersting-Sommerhoff B, Diethelm L, Teitel DF. *et al*. Magnetic resonance imaging of congenital heart disease: sensitivity and specificity using receiver operating characteristic curve analysis. Am Heart J 1989; 118: 155–61.

[13] Rumancik WM, Hernanz-Schulman M, Rutkowski MM *et al*. Magnetic resonance imaging of cor triatriatum. Pediatr Cardiol 1988; 9: 149–51.

[14] Jacobstein MD, Fletcher BD, Goldstein S, Riemenschneider TA. Evaluation of atrioventricular septal defect by magnetic resonance imaging. Am J Cardiol 1985; 55: 1158–61.

[15] Didier D, Higgins CB, Fisher MR, Osaki L, Silverman NH, Cheitlin MD. Congenital heart disease: gated MR imaging in 72 patients. Radiology 1986; 158: 227–35.

[16] Parsons JM, Baker EJ, Anderson RH *et al*. Morphological evaluation of atrioventricular septal defects by magnetic resonance imaging. Br Heart J 1990; 64: 138–45.

[17] Kastler B, Livolsi A, Zhu H, Roy E, Zollner G, Dietemann JL. Potential role of MR imaging in the diagnostic management of Ebstein anomaly in a newborn. J Comput Assist Tomogr 1990; 14: 825–7.

[18] Link KM, Herrera MA, D'Souza VJ, Formanek AG. MR imaging of Ebstein anomaly: results in four cases. Am J Roentgenol 1988; 150: 363–7.

[19] Didier D, Higgins CB. Identification and localization of ventricular septal defect by gated magnetic resonance imaging. Am J Cardiol 1986; 57: 1363–8.

[20] Lowell DG, Turner DA, Smith SM *et al*. The detection of atrial and ventricular septal defects with electrocardiographically synchronized magnetic resonance imaging. Circulation 1986; 73: 89–94.

[21] Mirowitz SA, Gutierrez FR, Canter CE, Vannier MW. Tetralogy of Fallot MR findings. Radiology 1989; 171: 207–12.

[22] Sechtem U, Pflugfelder P, Cassidy MC, Holt W, Wolfe C, Higgins CB. Ventricular septal defect: visulization of shunt flow and determination of shunt size by cine MR imaging. Am J Roentgenol 1987; 149: 689–92.

[23] Mayo JR, Roberson D, Sommerhoff B, Higgins CB. MR imaging of double outlet right ventricle. J Comput Assist Tomogr 1990; 14: 336–9.

[24] Kersting-Sommerhoff B, Seelos KC, Hardy C, Kondo C, Higgins SS, Higgins CB. Evaluation of surgical procedures for cyanotic congenital heart disease by using MR imaging. Am J Roentgenol 1990; 155: 259–66.

[25] Ahmad M, Johnson RJ, Fawcett HD, Schreiber, MH. Left ventricular aneurysm in short axis: a comparison of magnetic resonance, ultrasound and thallium-201 SPECT images. Magn Reson Imaging 1987; 5: 293–300.

[26] Speechly-Dick ME, Oliver RM, Slapak GI. Congenital left ventricular diverticula: a rare cause of sudden cardiac death. Postgr Med J 1992; 68: 378–80.

[27] Pattynama PM, De Roos A, Van der Wall EE, Van Voorthuisen AE. Evaluation of cardiac function with magnetic resonance imaging. Am Heart J 1994; 128: 595–607.

[28] Theissen P, Kaemmerer H, Sechtem U *et al*. Magnetic resonance imaging of cardiac function and morphology in patients with transposition of the great arteries following Mustard procedure. Thorac Cardiovasc Surg 1991; 3: 221–4.

[29] Fellows KE, Weinberg PM, Baffa JM, Hoffman EA. Evaluation of congenital heart disease with MR imaging: current and coming attractions. Am J Roentgenol 1992; 159: 925–31.

[30] Helbing WA, Rebergen SA, Maliepaard C, Hansen B, Ottenkamp J, Reiber JH. Quantification of right ventricular function with magnetic resonance imaging in children with normal hearts and with congenital heart disease. Am Heart J 1995; 130: 828–37.

[31] Helbing WA, Bosch HG, Maliepaard C *et al*. Comparison of echocardiographic methods with magnetic resonance imaging for assessment of right ventricular function in children. Am J Cardiol 1995; 76: 589–94.

[32] Niezen RA, Helbing WA, van der Wall EE, van der Geest RJ, Rebergen SA, de Roos A. Biventricular systolic function and mass studied with MR imaging in children with pulmonary regurgitation after repair for tetralogy of Fallot. Radiology 1996; 201: 135–40.

[33] Helbing WA, Niezen RA, Le Cessie S, van der Geest RJ, Ottenkamp J, de Roos A. Right ventricular diastolic function in children with pulmonary regurgitation after repair of tetralogy of Fallot: volumetric evaluation by magnetic resonance velocity mapping. J Am Coll Cardiol 1996; 28: 1827–35.

[34] de Roos A, Reichek N, Axel L, Kressel HY. Cine MR imaging in aortic stenosis. J Comput Assist Tomogr 1989; 13: 421–5.

[35] Kilner PJ, Firmin DN, Rees RS. *et al*. Valve and great vessel stenosis: assessment with MR jet velocity mapping. Radiology 1991; 178: 229–35.

[36] Boxer RA, Fishman MC, LaCorte MA, Singh S, Parnell VJ. Diagnosis and postoperative evaluation of supravalvular aortic stenosis by magnetic resonance imaging. Am J Cardiol 1986; 58: 367–8.

[37] Rebergen SA, Chin JG, Ottenkamp J, van der Wall EE, de Roos A. Pulmonary regurgitation in the late postoperative follow-up of tetralogy of Fallot. Volumetric quantitation by nuclear magnetic resonance velocity mapping. Circulation 1993; 92: 1123–32.

[38] Fletcher BD, Jacobstein MD. MRI of congenital abnormalities of the great arteries. Am J Roentgenol 1986; 146: 941–8.

[39] Parsons JM, Baker EJ, Hayes A *et al*. Magnetic resonance imaging of the great arteries in infants. Int J Cardiol 1990; 28: 73–85.

[40] al Ali F, Higgins CB, Gooding CA. MRI of tracheal and esophageal compression following surgery for congenital heart disease. J Comput Assist Tomogr 1994; 18: 39–42.

[41] Kersting-Sommerhoff BA, Sechtem UP, Fisher MR, Higgins CB. MR imaging of congenital anomalies of the aortic arch. Am J Roentgenol 1987; 149: 9–13.

[42] Jaffe RB. Magnetic resonance imaging of vascular rings.. Semin Ultrasound CT MR 1990; 11: 206–20.

[43] Mohiaddin RH, Kilner PJ, Rees S, Longmore DB. Magnetic resonance volume flow and jet velocity mapping in aortic coarctation. J Am Coll Cardiol 1993; 22: 1515–21.

[44] Steffens JC, Bourne MW, Sakuma H, O'Sullivan M, Higgins CB. Quantification of collateral blood flow in coarctation of the aorta by velocity encoded cine magnetic resonance imaging. Circulation 1994; 90: 937–43.

[45] Boxer RA, LaCorte MA, Singh S *et al*. Nuclear magnetic resonance imaging in evaluation and follow-up of children treated for coarctation of the aorta. J Am Coll Cardiol 1986; 7: 1095–8.

[46] Rees S, Sommerville J, Ward C *et al*. Coarctation of the aorta: MR imaging in late postoperative assessment. Radiology 1989; 173: 499–502.

[47] Simpson IA, Chung KJ, Glass RF, Sahn DJ, Sherman FS, Hesselink J. Cine magnetic resonance imaging for evaluation of anatomy and flow relations in infants and children with coarctation of the aorta. Circulation 1988; 78: 142–8.

[48] Bijl M, Bronzwaer JG, van Rossum AC, Verheught FW. Angina pectoris due to left main coronary artery compression in Eisenmenger ductus arteriosus. Am Heart J 1993; 125: 1767–71.

[49] Sommer T, Fehske W, Holzknecht N *et al*. Aortic dissection: A comparative study of diagnosis with spiral CT, multiplanar transesophageal echocardiography, and MR imaging. Radiology 1996; 199: 347–52.

[50] Niwa K, Tashima K, Kawasoe Y. *et al*. Magnetic resonance imaging of myocardial infarction in Kawasaki disease. Am Heart J 1990; 119: 1293–302.

[51] Post JC, van Rossum AC, Bronzwaer JG *et al*. Magnetic resonance angiography of anomalous coronary arteries. A new gold standard for delineating the proximal course? Circulation 1995; 92: 3163–71.

[52] McConnell MV, Ganz P, Selwyn AP, Li W, Edelman RR, Manning WJ. Identification of anomalous coronary arteries and their anatomic course by magnetic resonance coronary angiography. Circulation 1995; 92: 3158–62.

[53] Kersting-Sommerhof BA, Sechtem UP, Higgins CB. Evaluation of pulmonary blood supply by nuclear magnetic resonance imaging in patients with pulmonary atresia. J Am Coll Cardiol 1988; 11: 166–71.

[54] Vick GW, Rokey R, Huhta JC, Mulvagh SL, Johnston DL. Nuclear magnetic resonance imaging of the pulmonary arteries, subpulmonary region, and aorticopulmonary shunts: a comparative study with two-dimensional echocardiography and angiography. Am Heart J 1990; 119: 1103–10.

[55] Julsrud PR, Ehmann RL, Hagler DJ, Ilstrup DM. Extracardiac vasculature in candidate for Fontan surgery: MR imaging. Radiology 1989; 173: 503–6.

[56] Lynch DA, Higgins CB. MR imaging of unilateral pulmonary artery anomalies. J Comput Assist Tomogr 1990; 14: 187–91.

[57] Gomes AS, Lois JF, Williams RG. Pulmonary arteries: MR imaging in patients with congenital obstruction of the right ventricular outflow tract. Radiology 1990; 174: 51–7.

[58] Jacobstein MD, Fletcher BD, Nelson AD, Clampitt M, Alfidi RJ, Riemenschneider TA. Magnetic resonance imaging: evaluation of palliative systemic-pulmonary artery shunts. Circulation 1984; 70: 650–6.

[59] Canter CE, Gutierrez FR, Mirowitz SA, Martin TC, Hartmann AFJ. Evaluation of pulmonary arterial morphology in cyanotic congenital heart disease by magnetic resonance imaging. Am Heart J 1989; 118: 347–54.

[60] Canter CE, Gutierrez FR, Molina HP, Hartmann, AF, Spray TL. Noninvasive diagnosis of right-sided extracardiac conduit construction by combined magnetic resonance imaging and continuous-wave Doppler echocardiography. J Thorac Cardiovasc Surg 1991; 101: 724–31.

[61] Rebergen SA, Ottenkamp J, Doornbos J, van der Wall EE, Chin JG, de Roos A. Postoperative pulmonary flow dynamics after Fontan surgery: assessment with nuclear magnetic resonance velocity mapping. J Am Coll Cardiol 1993; 21: 123–31.

[62] Sampson C, Martinez J, Rees S, Somerville J, Underwood R, Longmore D. Evaluation of Fontan's operation by magnetic resonance imaging. Am J Cardiol 1990; 65: 819–21.

[63] Martinez JE, Mohiaddin RH, Kilner PJ *et al*. Obstruction in extracardiac ventriculopulmonary conduits: value of nuclear magnetic resonance imaging with velocity mapping and Doppler echocardiography. J Am Coll Cardiol 1992; 20: 338–44.

[64] Dinsmore RE, Liberthson RR, Wismer GL, Miller SW, Liu P, Thompson R. Magnetic resonance imaging of thoracic aortic aneurysms: comparison with other imaging methods. Am J Roentgenol 1986; 146: 309–14.

[65] Schneider R, Schoerner W, Paeprer H, Langer M, Felix R. MR imaging of aortic aneurysms-first results. Fortschr Roentgenstr 1986; 144: 17–24.

[66] Moore EH, Webb WR, Verrier ED *et al*. MRI of chronic posttraumatic false aneurysms of the thoracic aorta. Am J Roentgenol 1984; 143; 1195–6.

[67] McCuskey WH, Loehr SP, Smidebush GC, Link KM. Detection of mycotic pseudoaneurysm of the ascending aorta using MRI. Magn Reson Imaging 1993; 11; 1223–6.

[68] Akins EW, Slone RM, Wiechmann BN, Browning M, Martin TD, Mayfield WR. Perivalvular pseudoaneurysm complicating bacterial endocarditis: MR detection in five cases. Am J Roentgenol 1991; 156: 1155–8.

[69] White EM, Edelman RR, Wedeen VJ, Brady TJ. Intravascular signal in MR imaging: use of phase display for differentiation of blood-flow signal from intraluminal disease. Radiology 1986; 161: 245–9.

[70] Von Schulthess GK, Augustiny N. Calculation of T2 values versus phase imaging for the distinction between flow and thrombus in MR imaging. Radiology 1987; 164: 549–54.

[71] Castrucci M, Mellone R, Vanzulli A *et al*. Mural thrombi in abdominal aortic aneurysms: MR imaging characterization — useful before endovascular treatment?. Radiology 1995; 197: 135–9.

[72] Dulce MC, Mostbeck GH, O'Sullivan M, Cheitlin M, Caputo GR, Higgins CB. Severity of aortic regurgitation: interstudy reproducibility of measurements with velocity-encoded cine MR imaging. Radiology 1992; 185: 235–40.

[73] Sondergaard L, Lindvig K, Hildebrandt P *et al*. Quantification of aortic regurgitation by magnetic resonance velocity mapping. Am Heart J 1993; 125: 1081–90.

[74] White RD, Obuchowski NA, VanDyke CW *et al*. Thoracic aortic disease: evaluation using a single MRA volume series. J Comput Assist Tomogr 1994; 18: 843–54.

[75] Detrano R, Moodie DS, Gill CC, Markovich D, Simpfendorfer C. Intravenous digital subtraction

aortography in the preoperative and postoperative evaluation of Marfan's aortic disease. Chest 1985; 88; 249–54.
[76] Hirata K, Triposkiadis F, Sparks E, Bowen J, Boudoulas H, Wooley CF. The Marfan syndrome: cardiovascular physical findings and diagnostic correlates. Am Heart J 1992; 123; 743–52.
[77] Soulen RL, Fishman EK, Pyeritz RE, Zerhouni EA, Pessar ML. Marfan syndrome: evaluation with MR imaging versus CT. Radiology 1987; 165: 697–701.
[78] Schaefer S, Peshock RM, Malloy CR, Katz J, Parkey RW, Willerson JT. Nuclear magnetic resonance imaging in Marfan's syndrome. J Am Coll Cardiol 1987; 9: 70–4.
[79] Kersting SB, Sechtem UP, Schiller NB, Lipton MJ, Higgins CB. MR imaging of the thoracic aorta in Marfan patients. J Comput Assist Tomogr 1987; 11: 633–9.
[80] Wolff KA, Herold CJ, Tempany CM, Parravano JG, Zerhouni EA. Aortic dissection: atypical patterns seen at MR imaging. Radiology 1991; 181: 489–95.
[81] Savolainen A, Keto P, Hekali P *et al*. Aortic distensibility in children with Marfan syndrome. Am J Cardiol 1992; 70: 691–3.
[82] Adams JN, Brooks M, Redpath TW *et al*. Aortic distensibility and stiffness index measured by magnetic resonance imaging in patients with Marfan's syndrome. Br Heart J 1995; 73: 265–9.
[83] Laden N, Baciewicz FA, Grubb B. Labetalol and MRI as initial medical and diagnostic modalities in a marfanoid patient with expanding ascending aortic aneurysm. Chest 1990; 98: 1290–2.
[84] Banki JH, Meiners LC, Barentsz JO, Witkamp TD. Detection of aortic dissection by magnetic resonance imaging in adults with Marfan's syndrome. Int J Cardiol 1992; 8: 249–54.
[85] Deutsch HJ, Sechtem U, Meyer H, Theissen P, Schicha H, Erdmann E. Chronic aortic dissection: comparison of MR imaging and transesophageal echocardiography. Radiology 1994; 192: 645–50.
[86] Laissy JP, Blanc F, Soyer P *et al*. Thoracic aortic dissection: diagnosis with transesophageal echocardiography versus MR imaging. Radiology 1995; 194; 331–6.
[87] Nienaber C, von Kodolitsch Y, Nicolas V *et al*. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. N Engl J Med 1993; 328; 1–9.
[88] Barbant SD, Eisenberg MJ, Schiller NB. The diagnostic value of imaging techniques for aortic dissection. Am Heart J 1992; 124: 541–3.
[89] Gaubert JY, Moulin G, Mesana T *et al*. Type A dissection of the thoracic aorta: use of MR imaging for long-term follow-up. Radiology 1995; 196: 363–9.
[90] Yamada T, Tada S, Harada J. Aortic dissection without intimal rupture: diagnosis with MR imaging and CT. Radiology 1988; 168: 347–52.
[91] Nienaber CA, von Kodolitsch Y, Petersen, B *et al*. Intramural hemorrhage of the thoracic aorta. Diagnostic and therapeutic implications. Circulation 1995; 92: 1465–72.
[92] Braverman AC. Penetrating atherosclerotic ulcers of the aorta. Curr Opin Cardiol 1994; 9: 591–7.
[93] Yucel EK, Steinberg FL, Egglin TK, Geller SC, Waltman AC, Athanasoulis CA. Penetrating aortic ulcers: diagnosis with MR imaging. Radiology 1990; 177: 779–81.
[94] Kazerooni EA, Bree RL, Williams DM. Penetrating atherosclerotic ulcers of the descending thoracic aorta: evaluation with CT and distinction from aortic dissection. Radiology 1992; 183: 759–65.
[95] Atherosclerotic disease of the aortic arch as a risk factor for recurrent ischemic stroke. The French Study of Aortic Plaques in Stroke Group. N Engl J Med 1996; 334: 1216–21.
[96] Seelos KC, Funari M, Higgins CB. Detection of aortic arch thrombus using MR imaging. J Comput Assist Tomogr 1991; 15: 244–7.
[97] Auffermann W, Olofsson P, Stoney R, Higgins CB. MR imaging of complications of aortic surgery. J Comput Assist Tomogr 1987; 11: 982–9.
[98] Auffermann W, Olofson PA, Rabahie GN, Tavares NJ, Stoney RJ, Higgins CB. Incorporation versus infection of retroperitoneal aortic grafts: MR imaging features. Radiology 1989; 172: 359–62.
[99] Giovagnoni A, Ercolani P, Misericordia M, Terilli F, De NE. Evaluation of the pulmonary artery by cine MRI. J Comput Assist Tomogr 1992; 16: 553–9.
[100] Stern EJ, Graham C, Gamsu G, Golden JA, Higgins CB. Pulmonary artery dissection: MR findings. J Comput Assist Tomogr 1992; 16: 481–3.
[101] Posteraro RH, Sostman HD, Spritzer CE, Herfkens RJ. Cine-gradient-refocused MR imaging of central pulmonary emboli. Am J Roentgenol 1989; 152: 465–8.
[102] Erdman WA, Peshock RM, Redman HC *et al*. Pulmonary embolism: comparison of MR images with radionuclide and angiographic studies. Radiology 1994; 190: 499–508.
[103] Szucs RA, Rehr RB, Tatum JL. Pulmonary artery thrombus detection by magnetic resonance imaging. Chest 1989; 95: 232–4.
[104] Grist TM, Sostman HD, MacFall JR *et al*. Pulmonary angiography with MR imaging: preliminary clinical experience. Radiology 1993; 189: 523–30.
[105] Wielopolsky PA, Haacke EM, Adler LP. Three-dimensional MR imaging of the pulmonary vasculature: preliminary experience. Radiology 1992; 183: 465–72.
[106] Foo TK, MacFall JR, Hayes CE, Sostman HD, Slayman BE. Pulmonary vasculature: single breath-hold MR imaging with phased- array coils. Radiology 1992; 183: 473–7.
[107] Loubeyre P, Revel D, Douek P *et al*. Dynamic contrast-enhanced MR angiography of pulmonary embolism: comparison with pulmonary angiography. Am J Roentgenol 1994; 162: 1035–9.
[108] Hatabu H, Gefter WB, Axel L. *et al*. MR imaging with spatial modulation of magnetization in the evaluation of chronic central pulmonary thromboemboli. Radiology 1994; 190: 791–6.
[109] Mohiaddin RH, Paz R, Theodoropoulos S, Firmin DN, Longmore, DB, Yacoub MH. Magnetic resonance characterization of pulmonary arterial blood flow after single lung transplantation. J Thorac Cardiovasc Surg 1991; 101: 1016–23.
[110] Kondo C, Caputo GR, Masui T *et al*. Pulmonary hypertension: pulmonary flow quantification and flow profile analysis with velocity-encoded cine MR imaging. Radiology 1992; 183: 751–8.
[111] Hansen ME, Spritzer CE, Sostman HD. Assessing the patency of mediastinal and thoracic inlet veins: value of MR imaging. Am J Roentgenol 1990; 155: 1177–82.
[112] Mohiaddin RH, Wann SL, Underwood R, Firmin DN, Rees S, Longmore DB. Vena caval flow: assessment with cine MR velocity mapping. Radiology 1990; 177: 537–41.
[113] van Rossum AC, Sprenger M, Visser FC, Peels KH, Valk J, Roos JP. An in vivo validation of quantitative blood flow imaging in arteries and veins using magnetic resonance phase-shift techniques. Eur Heart J 1991; 12: 117–26.
[114] Galjee MA, Van Rossum AC, Van Eenige MJ. *et al*. Magnetic resonance imaging of the pulmonary venous flow pattern in mitral regurgitation: independence of the investigated vein. Eur Heart J 1995; 16: 1675–85.
[115] Nishimura T, Yamada N, Itoh A, Miyatake K. Cine MR imaging in mitral regurgitation: comparison with colour Doppler flow imaging. Am J Radiol 1989; 153: 721.
[116] Schiebler M, Axel L, Reichek N *et al*. Correlation of cine MR imaging with two-dimensional pulsed Doppler echocardiography in valvular insufficiency. J Comput Assist Tomogr 1987; 11: 627–32.
[117] Wong M, Matsumura M, Suzuki K, Omoto R. Technical and biologic sources of variability in the mapping of aortic, mitral and tricuspid color flow jets. Am J Cardiol 1987; 60: 847–51.
[118] Aurigemma G, Reichek N, Schiebler M, Axel L. Evaluation of mitral regurgitation by cine magnetic resonance imaging. Am J Cardiol 1990; 66: 621–5.

[119] Park JH, Han MC, Im JG, Oh BH, Lee YW. Mitral stenosis: evaluation with MR imaging after percutaneous balloon valvuloplasty. Radiology 1990; 177: 533–6.
[120] Firmin DN, Nayler GL, Klipstein RH, Underwood SR, Rees RS, Longmore DB. In vivo validation of MR velocity imaging. J Comput Assist Tomogr 1987; 11: 751–6.
[121] Soulen R, Higgins CB, Budinger TF. Magnetic resonance imaging of prosthetic heart valves. Radiology 1985; 154: 705–9.
[122] Shellock FG. MR imaging of metallic implants and materials: a compilation of the literature. Am J Roentgenol 1988; 151: 811–4.
[123] Devereux RB, Alonso DR, Lutas EM. *et al.* Echocardiographic assessment of left ventricular hypertrophy. Am J Cardiol 1986; 57: 450–8.
[124] Gardin JM, Dabestani A, Glasgow GA, Butman S, Burn CS, Henry WL. Echocardiographic and doppler flow observations in obstructed and non obstructed hypertrophic cardiomyopathy. Am J Cardiol 1985; 56: 614–21.
[125] Sardanelli F, Molinari G, Petillo A *et al.* MRI in hypertrophic cardiomyopathy: a morphofunctional study. J Comput Assist Tomogr 1993; 17: 862–72.
[126] Higgins CB, Byrd BF, Stark D *et al.* Magnetic resonance imaging of hypertrophic cardiomyopathy. Am J Cardiol 1985; 55: 1121–6.
[127] Suzuki J, Watanabe F, Takenaka K *et al.* New subtype of apical hypertrophic cardiomyopathy identified with nuclear magnetic resonance imaging as an underlying cause of markedly inverted T waves. J Am Coll Cardiol 1993; 22: 1175–81.
[128] Arrive L, Assayag P, Russ G, Najmark D, Brochet E, Nahum H. MRI and cine MRI of asymmetric septal hypertrophic cardiomyopathy. J Comput Assist Tomogr 1994; 18: 376–82.
[129] Kramer CM, Reichek N, Ferrari VA, Theobald T, Dawson J, Axel L. Regional heterogeneity of function in hypertrophic cardiomyopathy. Circulation 1994; 90; 186–94.
[130] Florentine MS, Grosskreutz CL, Chang W *et al.* Measurement of left ventricular mass in vivo using gated nuclear magnetic resonance imaging. J Am Coll Cardiol 1986; 8; 107–12.
[131] Keller AM, Peshock RM, Malloy CR *et al.* In vivo measurement of myocardial mass using nuclear magnetic resonance imaging. J Am Coll Cardiol 1986; 8: 113–7.
[132] Caputo GR, Tscholakoff D, Sechtem U, Higgins CB. Measurement of canine left ventricular mass using MR imaging. Am J Roentgenol 1987; 148: 33–8.
[133] Katz J, Milliken MC, Stray GJ *et al.* Estimation of human myocardial mass with MR imaging. Radiology 1988; 169: 495–8.
[134] Shapiro EP, Rogers WJ, Beyar R *et al.* Determination of left ventricular mass by magnetic resonance imaging in hearts deformed by acute infarction. Circulation 1989; 79: 706–11.
[135] Eichstaedt H, Danne O, Langer M *et al.* Regression of left ventricular hypertrophy under ramipril treatment investigated by nuclear magnetic resonance imaging. J Cardiovasc Pharmacol 1989; 13: 75–80.
[136] Doherty NE, Fujita N, Caputo GR, Higgins CB. Measurement of right ventricular mass in normal and dilated cardiomyopathic ventricles using cine magnetic resonance imaging. Am J Cardiol 1992; 69: 1223–8.
[137] Semelka RC, Tomei E, Wagner S *et al.* Interstudy reproducibility of dimensional and functional measurements between cine magnetic resonance studies in the morphologically abnormal left ventricle. Am Heart J 1990; 119: 1367–73.
[138] Doherty NE, Seelos KC, Suzuki J *et al.* Application of cine nuclear magnetic resonance imaging for sequential evaluation of response to angiotensin-converting enzyme inhibitor therapy in dilated cardiomyopathy. J Am Coll Cardiol 1992; 19: 1294–302.
[139] Auffermann W, Wichter T, Breithardt G, Joachimsen K, Peters PE. Arrhythmogenic right ventricular disease: MR imaging vs angiography. Am J Roentgenol 1993; 161; 549–55.
[140] Carlson MD, White RD, Trohman RG *et al.* Right ventricular outflow tract ventricular tachycardia: detection of previously unrecognized anatomic abnormalities using cine magnetic resonance imaging. J Am Coll Cardiol 1994; 24; 720–7.
[141] Casolo GC, Poggesi L, Boddi M *et al.* ECG-gated magnetic resonance imaging in right ventricular dysplasia. Am Heart J 1987; 113: 1245–8.
[142] Blake LM, Scheinman MM, Higgins CB. MR features of arrhythmogenic right ventricular dysplasia. Am J Roentgenol 1994; 162: 809–12.
[143] Sechtem U, Higgins CB, Sommerhoff BA, Lipton MJ, Huycke EC. Magnetic resonance imaging of restrictive cardiomyopathy. Am J Cardiol 1987; 59: 480–2.
[144] Masui T, Finck S, Higgins CB. Constrictive pericarditis and restrictive cardiomyopathy: evaluation with MR imaging. Radiology 1992; 182: 369–73.
[145] Revel D, Chapelon C, Mathieu D *et al.* Magnetic resonance imaging of human orthotopic heart transplantation: correlation with endomyocardial biopsy. J Heart Transplant 1989; 8: 139–46.
[146] Kurland RJ, West J, Kelley S *et al.* Magnetic resonance imaging to detect heart transplant rejection: sensitivity and specificity. Transplant Proc 1989; 21: 2537–43.
[147] Lund JT, Ehman RL, Julsrud PR, Sinak LJ, Tajik AJ. Cardiac masses: assessment by MR imaging. Am J Roentgenol 1989; 152: 469–73.
[148] Freedberg RS, Kronzon I, Rumancik WM, Liebeskind D. The contribution of magnetic resonance imaging to the evaluation of intracardiac tumours diagnosed by echocardiography. Circulation 1988; 77: 96–103.
[149] Montalescot G, Chapelon C, Drobinski G, Thomas D, Godeau P, Grosgogeat Y. Diagnosis of primary cardiac sarcoma. Report of 4 cases and review of the literature. Int J Cardiol 1988; 20; 209–19.
[150] Semelka RC, Shoenut JP, Wilson ME, Pellech AE, Patton JN. Cardiac masses: signal intensity features on spin-echo, gradient-echo, gadolinium-enhanced spin-echo, and Turbo-FLASH images. J Magn Reson Imaging 1992; 2; 415–20.
[151] Tscholakoff D, Sechtem U, du Geer G, Schmidt H, Higgins CB. Evaluation of pleural and pericardial effusions by magnetic resonance imaging. Eur J Radiol 1987; 7: 169–74.
[152] Sechtem U, Tscholakoff D, Higgins CB. MRI of the abnormal pericardium. Am J Roentgenol 1986; 147: 239–44.
[153] Vaitkus PT, Kussmaul WG. Constrictive pericarditis versus restrictive cardiomyopathy: A reappraisal and update of diagnostic criteria. Am Heart J 1991; 122: 1431–41.
[154] Kaemmerer H, Theissen P, Sechtem U, de Vivie ER. Follow-up using magnetic resonance imaging in adult patients after surgery for aortic coarctation. Thorac Cardiovasc Surg 1993; 41: 107–11.
[155] Schiavone WA, O'Donnell JK. Congenital absence of the left portion of parietal pericardium demonstrated by nuclear magnetic resonance imaging. Am J Cardiol 1985; 55: 1439–40.
[156] Allison JD, Flickinger FW, Wright JC *et al.* Measurement of left ventricular mass in hypertrophic cardiomyopathy using MRI: comparison with echocardiography. Magn Reson Imaging 1993; 11: 329–34.
[157] Yamaoka O, Yabe T, Okada M *et al.* Evaluation of left ventricular mass: comparison of ultrafast computed tomography magnetic resonance imaging, and contrast left ventriculography. Am Heart J 1993; 126: 1372–9.
[158] McDonald KM, Parrish T, Wennberg P *et al.* Rapid, accurate and simultaneous noninvasive assessment of right and left ventricular mass with nuclear magnetic resonance imaging using the snapshot gradient method [see comments]. J Am Coll Cardiol 1992; 19: 1601–7.
[159] Caputo GR, Suzuki J, Kondo C *et al.* Determination of left ventricular volume and mass with use of biphasic spin-echo MR imaging: comparison with cine MR. Radiology 1990; 177: 773–7.

[160] Mogelvang J, Thomsen C, Mehlsen J, Brackle G, Stubgaard M, Henriksen O. Evaluation of left ventricular volumes measured by magnetic resonance imaging. Eur Heart J 1986; 7: 1016–21.

[161] Mogelvang J, Stubgaard M, Thomsen C, Henriksen O. Evaluation of right ventricular volumes measured by magnetic resonance imaging. Eur Heart J 1988; 9: 529–33.

[162] Debatin JF, Nadel SN, Paolini JF *et al*. Cardiac ejection fraction: phantom study comparing cine MR imaging, radionuclide blood pool imaging, and ventriculography. J Magn Reson Imaging 1992; 2: 135–42.

[163] van Rossum AC, Visser FC, van Eenige MJ, Valk J, Roos JP. Magnetic resonance imaging of the heart for determination of ejection fraction. Int J Cardiol 1988; 18: 53–63.

[164] Sechtem U, Sommerhoff BA, Markiewicz W, White RD, Cheitlin MD, Higgins CB. Regional left ventricular wall thickening by magnetic resonance imaging: evaluation of normal persons and patients with global and regional dysfunction. Am J Cardiol 1987; 59: 145–51.

[165] Underwood SR, Rees RSO, Savage PE *et al*. Assessment of regional left ventricular function by magnetic resonance. Br Heart J 1986; 56: 334–40.

[166] Peshock RM, Rokey R, Malloy GM. *et al*. Assessment of myocardial systolic wall thickening using nuclear magnetic resonance imaging. J Am Coll Cardiol 1989; 14: 653–9.

[167] Azhari H, Sideman S, Weiss JL *et al*. Three-dimensional mapping of acute ischemic regions using MRI: wall thickening versus motion analysis. Am J Physiol 1990; 259: H1492–503.

[168] Johnson RA, Rubin LJ. Noninvasive evaluation of right ventricular function. Clin Chest Med 1987; 8: 65–80.

[169] Suzuki J, Caputo GR, Masui T, Chang JM, O'Sullivan M, Higgins CB. Assessment of right ventricular diastolic and systolic function in patients with dilated cardiomyopathy using cine magnetic resonance imaging. Am Heart J 1991; 122: 1035–40.

[170] Semelka RC, Tomei E, Wagner S *et al*. Normal left ventricular dimensions and function: interstudy reproducibility of measurements with cine MR imaging. Radiology 1990; 174: 763–8.

[171] Mogelvang J, Lindvig K, Sondergaard L, Saunamaki K, Henriksen O. Reproducibility of cardiac volume measurements including left ventricular mass determined by MRI. Clin Physiol 1993; 13: 587–97.

[172] Bolster BJ, McVeigh ER, Zerhouni EA. Myocardial tagging in polar coordinates with use of striped tags. Radiology 1990; 177: 769–72.

[173] Zerhouni EA, Parish DM, Rogers WJ, Yang A, Shapiro EP. Human heart: tagging with MR imaging — a method for noninvasive assessment of myocardial motion. Radiology 1988; 169: 59–63.

[174] Buchalter MB, Weiss JL, Rogers WJ *et al*. Noninvasive quantification of left ventricular rotational deformation in normal humans using magnetic resonance imaging myocardial tagging. Circulation 1990; 81: 1236–44.

[175] Lima JA, Ferrari VA, Reichek N *et al*. Segmental motion and deformation of transmurally infarcted myocardium in acute postinfarct period. Am J Physiol 1995; 268: H1304–12.

[176] Lima JA, Jeremy R, Guier W *et al*. Accurate systolic wall thickening by nuclear magnetic resonance imaging with tissue tagging: correlation with sonomicrometers in normal and ischemic myocardium. J Am Coll Cardiol 1993; 21: 1741–51.

[177] Pennell DJ, Underwood SR, Ell PJ, Swanton RH, Walker JM, Longmore DB. Dipyridamole magnetic resonance imaging: a comparison with thallium-201 emission tomography. Br Heart J 1990; 64: 362–9.

[178] Pennell DJ, Underwood SR, Manzara CC. *et al*. Magnetic resonance imaging during dobutamine stress in coronary artery disease. Am J Cardiol 1992; 70: 34–40.

[179] Baer FM, Voth E, Theissen P, Schneider CA, Schicha H, Sechtem U. Coronary artery disease: findings with GRE MR imaging and Tc-99m-methoxyisobutyl-isonitrile SPECT during simultaneous dobutamines stress. Radiology 1994; 193: 203–9.

[180] Baer FM, Voth E, Theissen P, Schicha H, Sechtem U. Gradient-echo magnetic resonance imaging during incremental dobutamine infusion for the localization of coronary artery stenoses. Eur Heart J 1994; 15: 218–25.

[181] Pennell DJ Magnetic resonance imaging during pharmacologic stress. Coron Artery Dis 1993; 4: 345–53.

[182] van Rugge FP, van der Wall EE, de Roos A, Bruschke AV. Dobutamine stress magnetic resonance imaging for detection of coronary artery disease. J Am Coll Cardiol 1993; 22: 431–9.

[183] van Rugge FP, van der Wall EE, Spanjersberg SJ *et al*. Magnetic resonance imaging during dobutamine stress for detection and localization of coronary artery disease. Quantitative wall motion analysis using a modification of the centerline method.. Circulation 1994; 90: 127–38.

[184] Wilke N, Simm C, Zhang J *et al*. Contrast-enhanced first pass myocardial perfusion: correlation between myocardial blood flow in dogs at rest and during hyperemia. Mag Reson Med 1993; 29: 485–97.

[185] Manning WJ, Atkinson DJ, Grossman W, Paulin S, Edelman RR. First-pass nuclear magnetic resonance imaging studies using gadolinium-DTPA in patients with coronary artery disease. J Am Coll Cardiol 1991; 18: 959–65.

[186] Wendland MF, Saeed M, Masui T, Derugin N, Moseley ME, Higgins CB. Echo-planar MR imaging of normal and ischemic myocardium with gadodiamide injection. Radiology 1993; 186: 535–42.

[187] Saeed M, Wendland MF, YU KK *et al*. Identification of myocardial reperfusion with echo planar magnetic resonance imaging. Discrimination between occlusive and reperfused infarctions. Circulation 1994; 90: 1492–501.

[188] Schaefer S, van Tyen R, Saloner D. Evaluation of myocardial perfusion abnormalities with gadolinium-enhanced snapshot MR imaging in humans. Radiology 1992; 185: 795–801.

[189] Manning WJ, Li W, Boyle NG, Edelman RR. Fat-suppressed breath-hold magnetic resonance coronary angiography. Circulation 1993; 87; 94–104.

[190] Duerinckx AJ, Urman MK. Two-dimensional coronary MR angiography: analysis of initial clinical results. Radiology 1994; 193; 731–8.

[191] Manning WJ, Li W, Edelman RR. A preliminary report comparing magnetic resonance coronary angiography with conventional angiography. N Engl J Med 1993; 328: 828–32.

[192] Pennell DJ, Bogren HG, Keegan J, Firmin DN, Underwood SR. Assessment of coronary artery stenosis by magnetic resonance imaging. Heart 1996; 75: 127–33.

[193] Wang Y, Rossman PJ, Grimm RC, Riederer SJ, Ehman RL. Navigator-echo-based real-time respiratory gating and triggering for reduction of respiration effects in three-dimensional coronary MR angiography. Radiology 1996; 198: 55–60.

[194] Hofman MB, Paschal CB, Li D, Haacke EM, van Rossum AC, Sprenger M. MRI of coronary arteries: 2D breath-hold vs 3D respiratory-gated acquisition. J Comput Assist Tomogr 1995; 19: 56–62.

[195] Post JC, van Rossum AC, Hofman MBM, Valk J, Visser CA. Three-dimensional respiratory-gated MR angiography of coronary arteries: Comparison with conventional coronary angiography. Am J Roentgenol 1996; 166: 1399–404.

[196] Gomes AS, Lois JF, Drinkwater DC, Corday SR. Coronary artery bypass grafts: visualization with MR imaging. Radiology 1987; 162: 175–9.

[197] Jenkins JPR, Love HG, Foster CJ, Isherwood I, Rowlands DJ. Detection of coronary bypass graft patency as assessed by magnetic resonance imaging. Br J Radiol 1988; 61: 2–4.

[198] White RD, Caputo GR, Mark AS, Modin GW, Higgins CB. Coronary artery bypass graft-patency: noninvasive evaluation with MR imaging. Radiology 1987; 164: 681–6.

[199] Aurigemma GP, Reichek N, Axel L, Schiebler M, Harris C, Kressel HY. Noninvasive determination of coronary artery bypass graft patency by cine magnetic resonance imaging. Circulation 1989; 80; 1595–602.
[200] van Rossum AC, Galjee MA, Doesburg T, Hofman M, Valk J. The role of magnetic resonance in the evaluation of function results after CABG/PTCA Int J Card Imaging 1993; 1; 59–69.
[201] Hoogendoorn LI, Pattynama PM, Buis B, van der Geest RJ van der Wall EE, de Roos A. Noninvasive evaluation of aortocoronary bypass grafts with magnetic resonance imaging flow mapping. Am J Cardiol 1995; 75: 845–8.
[202] Galjee MA, van Rossum AC, Doesburg T, van Eenige MJ, Visser CA. Value of magnetic resonance imaging in assessing patency and function of coronary artery bypass grafts: An angiographically controlled study. Circulation 1996; 93: 660–6.
[203] Edelman RR, Manning WJ, Gervino E, Li W. Flow velocity quantification in human coronary arteries with fast, breath-hold MR angiography. J Magn Reson Imaging 1993; 3: 699–703.
[204] Keegan J, Firmin D, Gatehouse P, Longmore D. The application of breath hold phase velocity mapping techniques to the measurement of coronary artery blood flow velocity: phantom data and initial in vivo results. Magn Reson Med 1994; 31: 526–36.
[205] Hofman MB, Visser FC, van Rossum AC, Vink QM, Sprenger M, Westerhof N. In vivo validation of magnetic resonance blood volume flow measurements with limited spatial resolution in small vessels. Magn Reson Med 1995; 33: 778–84.
[206] Poncelet B, Weisskof RM, Wedeen VJ, Brady TJ, Kantor H. Time of flight quantification of coronary flow with echo-planar MRI Magn Reson Med 1993; 30: 447–57.
[207] Clarke GD, Eckels R, Chaney C *et al*. Measurement of absolute epicardial coronary artery flow and flow reserve with breath-hold cine phase-contrast magnetic resonance imaging. Circulation 1995; 91: 2627–34.
[208] Hundley WG, Lange RA, Clarke GD *et al*. Assessment of coronary arterial flow and flow reserve in humans with magnetic resonance imaging. Circulation 1996; 93: 1502–8.
[209] Pflugfelder PW, Wisenberg G, Prato FS, Carroll SE. Serial imaging of canine myocardial infarction by in vivo nuclear magnetic resonance. J Am Coll Cardiol 1986; 7; 843–9.
[210] Johnston DL, Thompson RC, Liu P *et al*. Magnetic resonance imaging during acute myocardial infarction. Am J Cardiol 1986; 57; 1059–65.
[211] Fisher MR, McNamara MT, Higgins CB. Acute myocardial infarction: MR evaluation in 29 patients. Am J Roentgenol 1987; 148: 247–51.
[212] Ahmad M, Johnson RJ, Fawcett HD, Schreiber MH. Magnetic resonance imaging in patients with unstable angina: comparisons with acute myocardial infarction and normals. Magn Reson Imaging 1988; 6: 527–34.
[213] Filipchuk NG, Peshock RM, Malloy CR *et al*. Detection and localization of recent myocardial infarction by magnetic resonance imaging. Am J Cardiol 1986; 58: 214–9.
[214] White RD, Holt WW, Cheitlin MD *et al*. Estimation of the functional and anatomic extent of myocardial infarction using magnetic resonance imaging. Am Heart J 1988; 115: 740–8.
[215] White RD, Cassidy MM, Cheitlin MD *et al*. Segmental evaluation of left ventricular wall motion after myocardial infarction: magnetic resonance imaging versus echocardiography. Am Heart J 1988; 115: 166–75.
[216] van Rossum AC, Visser FC, van Eenige MJ *et al*. Value of gadolinium-diethylene-triamine pentaacetic acid dynamics in magnetic resonance imaging of acute myocardial infarction with occluded and reperfused coronary arteries after thrombolysis. Am J Cardiol 1990; 65: 845–51.
[217] Holman ER, van Jonbergen HP, van Dijkman PR, van der Laarse A, de Roos A, van der Wall EE. Comparison of magnetic resonance imaging studies with enzymatic indexes of myocardial necrosis for quantification of myocardial infarct size. Am J Cardiol 1993; 71: 1036–40.
[218] de Roos A, Matheijssen NA, Doornbos J, van Dijkman PR, van Voorthuisen AE, van der Wall EE. Myocardial infarct size after reperfusion therapy: assessment with Gd-DTPA-enhanced MR imaging. Radiology 1990; 176: 517–21.
[219] Dubnow MH, Burchell HB, Titus JL. Postinfarction left ventricular aneurysm. A clinicomorphologic and electrocardiographic study of 80 cases. Am Heart J 1965; 70; 753–60.
[220] Perrone-Filardi P, Bacharach SL, Dilsizian V, Maurea S, Frank JA, Bonow RO. Regional left ventricular wall thickening. Relation to regional uptake of 18fluorodeoxyglucose and 201Tl in patients with chronic coronary artery disease and left ventricular dysfunction. Circulation 1992; 86; 1125–37.
[221] Baer FM, Schneider CA, Theissen P, Schicha H, Sechtem U. Comparison of low-dose dobutamine-gradient-echo magnetic resonance imaging and positron emission tomography with [18F]fluorodeoxyglucose in patients with chronic coronary artery disease. A functional and morphological approach to the detection of residual myocardial viability. Circulation 1995; 91: 1006–15.
[222] Sechtem U, Theissen P, Heindel W *et al*. Comparison of magnetic resonance imaging, computed tomography, echocardiography, and angiography in the diagnosis of left ventricular thrombi. Am J Cardiol 1989; 64: 1195–9.
[223] Jungehuelsing M, Sechtem U, Theissen P, Hilger HH, Schica H. Left ventricular thrombi: evaluation with spin-echo and gradient-echo MR imaging. Radiology 1992; 182: 225–9.
[224] Harrity P, Patel A, Bianco J, Subramanian R. Improved diagnosis and characterization of postinfarction left ventricular pseudoaneurysm by cardiac magnetic resonance imaging. Clin Cardiol 1991; 14: 603–6.
[225] Hetherington HP, Luney DJE, Vaughan JT *et al*. 3D 31P spectroscopic imaging of the human heart at 4.1 T. Magn Reson Med 1995; 33: 427–31
[226] Bottomley PA. MR spectroscopy of the human heart: the status and the challenges. Radiology 1994; 191: 593–612.
[227] Neubauer S, Krahe T, Schindler R *et al*. 31P magnetic resonance spectroscopy in dilated cardiomyopathy and coronary artery disease. Altered cardiac high-energy phosphate metabolism in heart failure. Circulation 1992; 86: 1810–8.
[228] Weiss RG, Bottomley PA, Hardy CJ, Gerstenblith G. Regional myocardial metabolism of high-energy phosphates during isometric exercise in patients with coronary artery disease. N Engl J Med 1990; 323: 1593–600.
[229] Yabe T, Mitsunami K, Okada M, Morikawa S, Inubushi T, Kinoshita M. Detection of myocardial ischemia by 31P magnetic resonance spectroscopy during handgrip exercise. Circulation 1994; 89: 1709–16.
[230] Yabe T, Mitsunami K, Inubushi T, Kinoshita M. Quantitative measurements of cardiac phosphorus metabolites in coronary artery disease by 31P magnetic resonance spectroscopy. Circulation 1995; 92; 15–23.
[231] Hardy CJ, Weiss RG, Bottomley PA, Gerstenblith G. Altered myocardial high-energy phosphate metabolism in patients with dilated cardiomyopathy. Am Heart J 1991; 122; 795–801.
[232] de Roos A, Doornbos J, Luyten PR, Oosterwaal LJ, van der Wall E, den Hollander J. Cardiac metabolism in patients with dilated and hypertrophic cardiomyopathy: assessment with proton-decoupled P-31 MR spectroscopy. J Magn Reson Imaging 1992; 2: 711–9.
[233] Neubauer S, Horn M, Goedde M *et al*. In patients with dilated cardiomyopathy the myocardial phosphocreatine-ATP ratio predicts mortality better than ejection fraction or NYHA class (abstr.). Circulation 1996; 94: I–30.
[234] Conway MA, Allis J, Ouwerkerk R, Niioka T, Rajagopalan B, Radda GK. Detection of low phosphocreatine to ATP ratio in failing hypertrophied human myocardium by 31P magnetic resonance spectroscopy. Lancet 1991; 338: 973–6.

Appendix

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