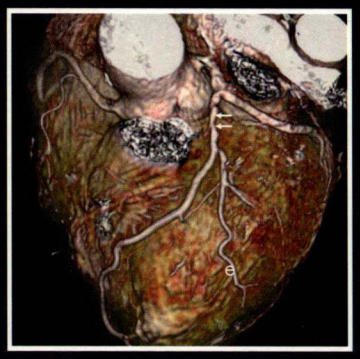
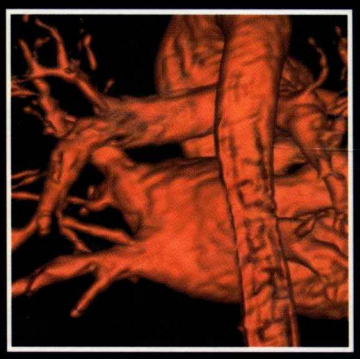
Christopher Kramer • W. Gregory Hundley


Atlas of Cardiovascular Magnetic Resonance *Imaging*

An Imaging Companion to Braunwald's Heart Disease

Series Editor: Robert O. Bonow

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ATLAS OF CARDIOVASCULAR MAGNETIC RESONANCE IMAGING AN IMAGING COMPANION TO BRAUNWALD'S HEART DISEASE

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Notice

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Foreword

The rapid advances in cardiology during the first half of the twentieth century may be fairly ascribed to the introduction of new techniques.

Paul Wood, 1951
Diseases of the Heart and Circulation

These prophetic words of Dr. Paul Wood, the preeminent London cardiologist of the 1950s, clearly have even a more meaningful relevance as we near the end of the first decade of the twenty-first century. Dr. Wood died prematurely from coronary artery disease at the age of 55, 11 years after publishing his textbook *Diseases of the Heart and Circulation*, and thus was not witness to the explosive growth of cardiovascular technology over the second half of the last century. In that same period of time, coronary heart disease deaths were cut in half.

It is unclear what role imaging has played in these improved outcomes. But it is clear that diagnostic imaging has increased more rapidly than any other component of medical care.

Cardiovascular magnetic resonance (CMR) is among the most exciting and most advanced of the imaging modalities, and its ability to visualize cardiac structures dynamically represents a true breakthrough in imaging technology. CMR has important real and potential applications for assessing vascular anatomy, the structure and function of the cardiac chambers (including ventricular mass and volume), myocardial perfusion, and myocardial scar.

Many of the applications of CMR are those that can be performed using more readily available and less expensive technology. For example, evaluation of left ventricular mass, shape, volume, and both region and global systolic function are obtained with echocardiography on a routine basis, and radionuclide imaging is in a strong leadership position for myocardial perfusion imaging. CMR is useful for these purposes when either echocardiography or nuclear perfusion imaging is unable to obtain adequate image quality or provides equivocal findings. However, CMR is not relegated to this second-tier position for many other indications in which CMR has been established as the gold standard. Most notably, the ability to visualize myocardial necrosis and fibrosis using late gadolinium hyperenhancement is an attribute that is unique to CMR. Contrast-enhanced CMR accurately identifies the location, transmural and circumferential extent, and mass of infarcted myocardium, in both acute and chronic settings. The detection of even small infarct zones detects previous myocardial infarction in patients in whom this diagnosis cannot be made by other methods. Infarct mass measured shortly after treatment for myocardial infarction predicts the degree of subsequent left ventricular remodeling and thus has important prognostic implications. As a marker of non-viable myocardium, contrast-enhanced CMR is an excellent method for determining the presence or absence of viable myocardium, which predicts the likelihood of reversal of regional and global dysfunction after revascularization.

Another means to assess myocardial viability is imaging regional left ventricular function during low-dose dobutamine administration to demonstrate contractile reserve, and studies

have demonstrated that the combination of low-dose dobutamine CMR and contrast-enhanced CMR provides diagnostic accuracy in identifying viable myocardium that is greater than either method alone.

Contrast hyperenhancement has also been observed in a number of other conditions beyond coronary artery disease, including myocarditits, dilated cardiomyopathy, hypertrophic cardiomyopathy, and infiltrative conditions such as amyloidosis and sarcoidosis, which reflect pathophysiologic processes affecting the myocardial extracellular space. In some disorders, detection of these processes has prognostic as well as diagnostic value.

Unlike perfusion imaging with single photon or positron emitting radionuclides, which has limited spatial resolution, CMR perfusion imaging with pharmacologic stress provides information regarding the transmural extent of myocardial ischemia. CMR is thus able to visualize small areas of ischemia (usually present in the subendocardial zone) and also detects subendocardial ischemia in patients with multivessel coronary artery disease who might be misdiagnosed as normal by nuclear imaging because of a uniform, balanced reduction in flow. Similar methods have detected diffuse subendocardial hypoperfusion during vasodilator stress in patients with microvascular abnormalities such as those with syndrome X. New methods have evolved for quantification of regional myocardial blood flow distribution from endocardium to epicardium. Such quantitative methods will be valuable for assessing therapies, such as those stimulating angiogenesis, that result in small increases in endocardial perfusion within the ischemic zones.

CMR has become established as the most accurate noninvasive method for measuring left ventricular mass and volume, and thus ejection fraction measurements also have a high degree of accuracy and reproducibility. Strain imaging using tagging techniques offer exciting possibilities to further the understanding of regional systolic and diastolic function in a variety of cardiac diseases.

Coronary magnetic resonance angiography (MRA) remains an elusive target as a procedure that can yield images of diagnostic quality on a uniform, reproducible basis. The small caliber and tortuosity of the vessels, combined with cardiac and respiratory motion, have presented hurdles that are yet to be surmounted. Nonetheless, progress is being made. In contrast, MRA of the larger and relatively stationary non-coronary vessels is now commonplace in clinical practice, providing excellent visualization of the vessel wall and lumen, with and without the use of contrast media. Arterial remodeling is readily apparent in atherosclerotic vessels with large plaque volumes before there is significant encroachment of the vascular lumen, and important progress has been made in tissue characterization of the atherosclerotic plaques. There is promise that, with further technical advances, similar inroads will be made in coronary MRA and coronary plaque characterization.

One of the major advantages of CMR is the ability to obtain images of such excellent spatial resolution without ionizing radiation. Thus, when future research ultimately achieves the goal of routine, high quality coronary artery imaging, coronary MRA will undoubtedly compete very favorably with coronary CT angiography as the preferred tool for noninvasive assessment of coronary atherosclerotic burden and severity of coronary stenosis.

Other unresolved issues still linger: Who should be studied? Who should interpret the study? Who will pay for the study? Who will train whom? How will guidelines be affected? How will quality be determined and maintained? Hopefully, these are not unresolvable, and the cardiovascular societies are collectively addressing these complex and inter-related questions. Measuring performance in cardiac imaging is inherently difficult as it is not possible to connect the results of an imaging test to health-related outcomes. Patient selection is a key variable as it impacts importantly on downstream management decisions including further testing, interventions and costs.

On the other hand, cardiovascular imaging has transformed, and will continue to transform, cardiovascular care. CMR in particular represents a revolutionary imaging modality that creates a unique opportunity to improve diagnosis and streamline clinical management strategies but also creates challenges in patient selection, clinical training, resource utilization and cost effectiveness. That will be our challenge going forward.

The editorial team of *Braunwald's Heart Disease* is delighted to launch a series of four imaging companions, each dedicated to one of the key cardiac imaging modalities. This companion on cardiovascular magnetic resonance, expertly edited by Drs. Kramer and

Hundley, covers all of the important technical and clinical aspects of this exciting field and provides a unique case-based perspective into the tremendous potential for magnetic resonance imaging to enhance patient diagnosis and management. We believe that this companion will be a highly valuable resource for clinicians, imaging subspecialists and cardiovascular trainees and that it will contribute in a significant manner to the care of the patients they serve.

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Abbreviations

A = atrium
AA = aortic arch
ACC = American College of Cardiology
ACE = angiotension converting enzyme
AHA = American Heart Association
AL = anterolateral
AO = aorta
Ao S = aortic sinus
ARVC = arrhythmogenic right ventricular cardiomyopathy
ARVC/D = arrhythmogenic right ventricular cardiomyopathy/dysplasia
Asc Ao = ascending aorta
ASD = atrial septal defect
AV = atrioventricular valves
AVS = antrioventricular septum
Az V = azygous vein
BMI = body mass index
BP = blood pressure
BSA = body surface area
CA = conus arteriosus
CABG = coronary artery bypass graft
CAD = coronary artery disease
Circ = Circumflex
CMR = Cardiovascular magnetic resonance
COPD = Chronic Obstructive Pulmonary Disease
CRT = cardiac resynchronization therapy
CS = coronary sinus
CT = computed tomography
DE = delayed enhancement
DE-CMR = delayed enhancement cardiovascular magnetic resonance
Desc Ao = descending aorta
DSCMR = dobutamine stress cardiovascular magnetic resonance
ECG = electrocardiogram
EDV = end-diastolic volume
EEST = electrocardiogram exercise stress testing
Eso = esophagus
ESRD = end-stage renal disease
ESV = end-systolic volume
F = Fontan conduit
FDG – PET = fludeoxyglucose positron emission tomography
FO = foramen ovale
FOV = field of view
Gd = gadolinium
Gd-DTPA = gadolinium diethyltriaminepentaacetic acid
HASTE = half-Fourier acquisition single-shot turbo spin echo
HCM = hypertrophic cardiomyopathy
Hep V = hepatic vein
HLA = horizontal long axis
HR = heart rate
i = index
IB = inferior baffle
IDCM = idiopathic dilated cardiomyopathy
IF = inflow tract
ILB = inferior limbic band
Inf = infundibulum
innom = innominate / brachiocephalic artery
IVC = inferior vena cava
LA = left atrium
LAA = left atrial appendage
LAD = left anterior descending
LCC = left common carotid artery
LCX = left coronary artery
LGE = late gadolinium enhancement
LLPV = left lower pulmonary vein
LM = left main coronary artery
LMB = left main bronchi
LPA = left pulmonary artery
LPV = left pulmonary vein
LSA = left subclavian artery
LUPV = left upper pulmonary
LV = left ventricular
LVA = left ventricular inferior wall aneurysm

LVEF = left ventricular ejection fraction
LVIDD = left ventricular internal diameter in diastole
LVM = left ventricular mass
LVOT = left ventricular outflow tract
LVV = left ventricular volume
MACE = major adverse cardiac events
MET = metabolic equivalent
MI = myocardial infarction
MIP = maximum intensity projection
MPA = main pulmonary artery
MPHRR = maximum predicted heart rate response
MPR = multi-planar reformatted
MSCT = multislice spical computed tomography
NYHA = New York Heart Association
OM = obtuse marginal
PA = pulmonary artery
PAPVC = partially anomalous pulmonary venous connection
PCI = percutaneous intervention
PDA = patent ductus arterious
PDA = posterior descending artery
PeriC = pericardium
PET = positron emission tomography
PFO = patent foramen ovale
PM = posteromedial
PV = pulmonary valve
PVA = pulmonary venous atrium
Qp = pulmonary blood flow
Qs = systemic blood flow
RA = right atrium
RAO = right anterior oblique
RCA = right coronary artery
Res = respiration
RF = radio frequency
RLPV = right lower pulmonary vein
RMB = right main bronchi
RPA = right pulmonary artery
RPV = right pulmonary vein
RUPV = right upper pulmonary vein
RV = right ventricular
RVOT = right ventricular outflow
SA = short axis
SA = sinoatrial
SB = superior baffle
SCD = sudden cardiac death
SE = spin echo
SLB = superior limbic band
SP = saturation pulse
SNR = signal-to-noise ratio
SPECT = single photon emission computed tomography
SSFP = steady-state free procession
ST = systolic wall thickening
STIR = short tau inversion recovery
SV = stroke volume
SVA = systemic venous atrium
SVC = superior vena cava
SVD = sinus venous defect
TD = trigger delay
TE = echo time
TGA = transposition of great arteries
TGrE = turbo-gradient echo imaging
TI = inversion time
TR = repetition time
Tr = trachea
TSE = turbo spin echo
TTC = triphenyl tetrazolium chloride
TV = tricuspid valve
VEC-CMR = velocity encoded CMR
VLA = vertical long axis
VSD = ventricular septal defect
VT = ventricular tachycardia
WMSI = wall motion score index

Contents

Chapter 1

Normal Cardiac Anatomy

Saul G. Myerson and Stefan Neubauer

KEY POINTS

- Knowledge of the anatomy of the heart and in particular the three-dimensional (3D) relationships of the various normal structures is essential.
- The heart lies at an oblique and variable angle within the chest, and standard cardiovascular magnetic resonance (CMR) imaging planes are relative to the long axis of the heart rather than the body. Standard image planes relative to body position (e.g., coronal, transaxial) can provide useful anatomic information, but it should be made clear how the image plane was positioned to avoid confusion.
- Three-dimensional spatial awareness is important in appreciating normal cardiac anatomy. Because of the oblique nature of many cardiac structures and the two-dimensional plane of a single CMR image slice, it is possible to "slice" through a structure at an oblique angle, which may appear abnormal. Further imaging in different planes (often perpendicular to the one with the apparent abnormality) is recommended to fully appreciate the nature of the anatomy and determine whether it is normal or abnormal.
- Modification of the image position may be required if the initial image is not ideal. Do not be afraid of repeating the sequence, having moved the image plane slightly or obtained other image slices to better position the image slice.
- Optimization of the sequence to each patient is important for obtaining the highest-quality images (e.g., the trade-off between spatial and temporal resolution may have to be adjusted individually). If the initial image is of poor quality, repeat with better parameters as necessary.
- For many images, cine imaging is recommended because of the continuously moving heart, because this provides a better appreciation of the anatomy in motion.
- Spin-echo images provide good contrast between tissues containing adipose tissue (e.g., pericardial fat) and tissues with high water content (e.g., myocardium) or fibrous tissue (e.g., pericardium).
- Beware of partial volume effects. The relatively thick slice thickness of CMR images (5 to 8 mm) can include parts of two structures combined in one image plane.

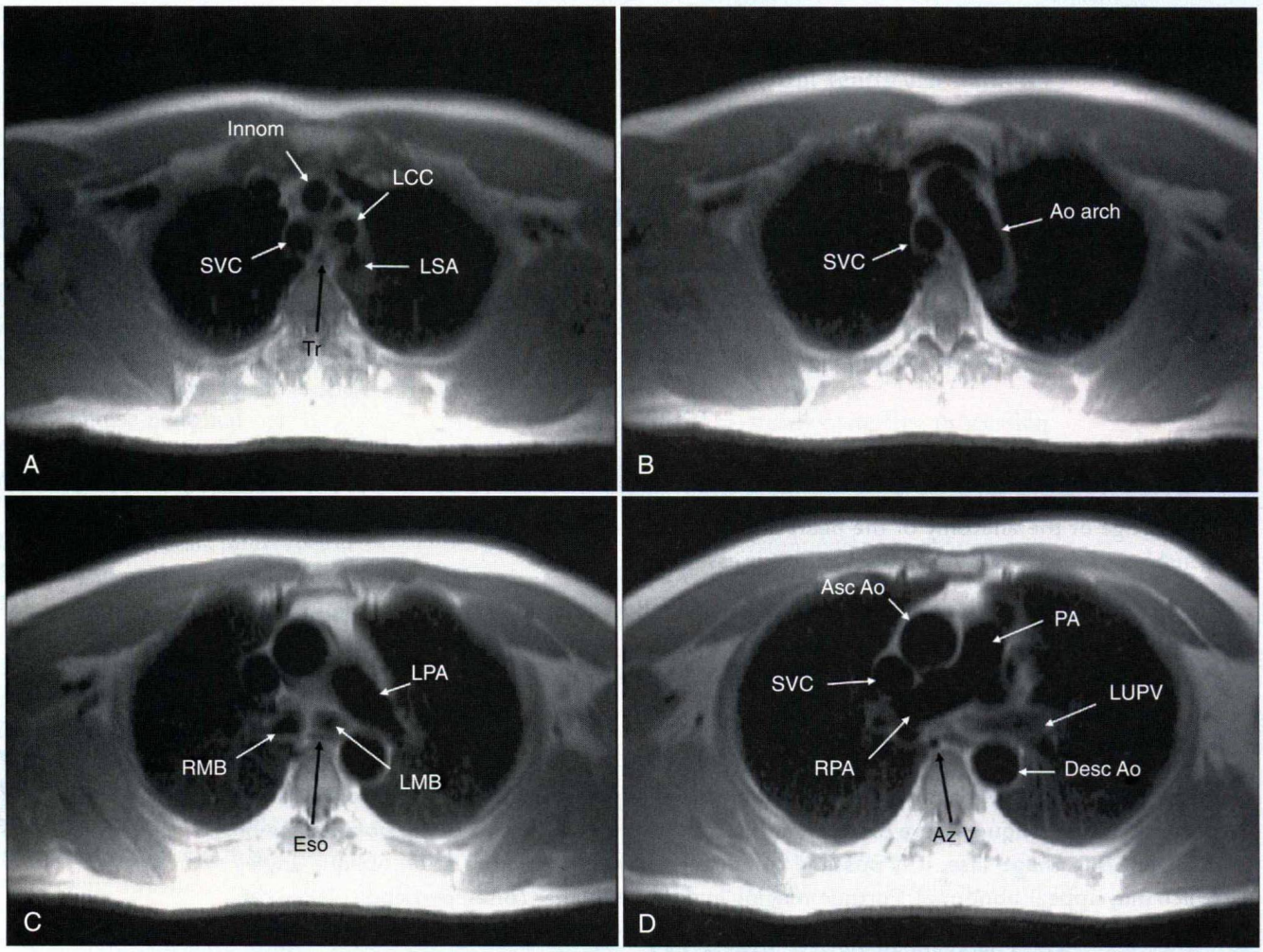

Figure 1-1 Transverse views from HASTE sequence in upper thorax, from superior (**A**) to inferior (**D**). Black-blood sequence, with adipose tissue appearing bright (high signal), air and flowing blood appearing dark (low signal), and most other tissues of mid-gray intensity (intermediate signal); slice thickness = 7 mm. In (**A**), the great vessel origins can be seen—innominate/brachiocephalic artery (innom), left common carotid artery (LCC), left subclavian artery (LSA), and superior vena cava (SVC), in addition to the trachea (Tr). The esophagus (Eso) is located posterior to the Tr, but is normally compressed when lying flat, and is difficult to visualize; it can be seen lower down in (**C**). The aortic arch (Ao arch) and SVC appear in (**B**). Just below the aortic arch in (**C**), the left pulmonary artery (LPA) can be seen along with the right (RMB) and left (LMB) main bronchi, highlighted against the mediastinal fat. Lower still in (**D**), the main pulmonary artery/trunk (PA) and right pulmonary artery (RPA) and left upper pulmonary vein (LUPV) are visible between the ascending and descending (Desc Ao) limbs of the thoracic aorta. The pulmonary veins are often better visualized on coronal imaging because of their thin wall and angulated course but ideally imaged with magnetic resonance (MR) contrast angiography. The azygous vein (Az V) can also be seen just anterior to the spine on the right side.

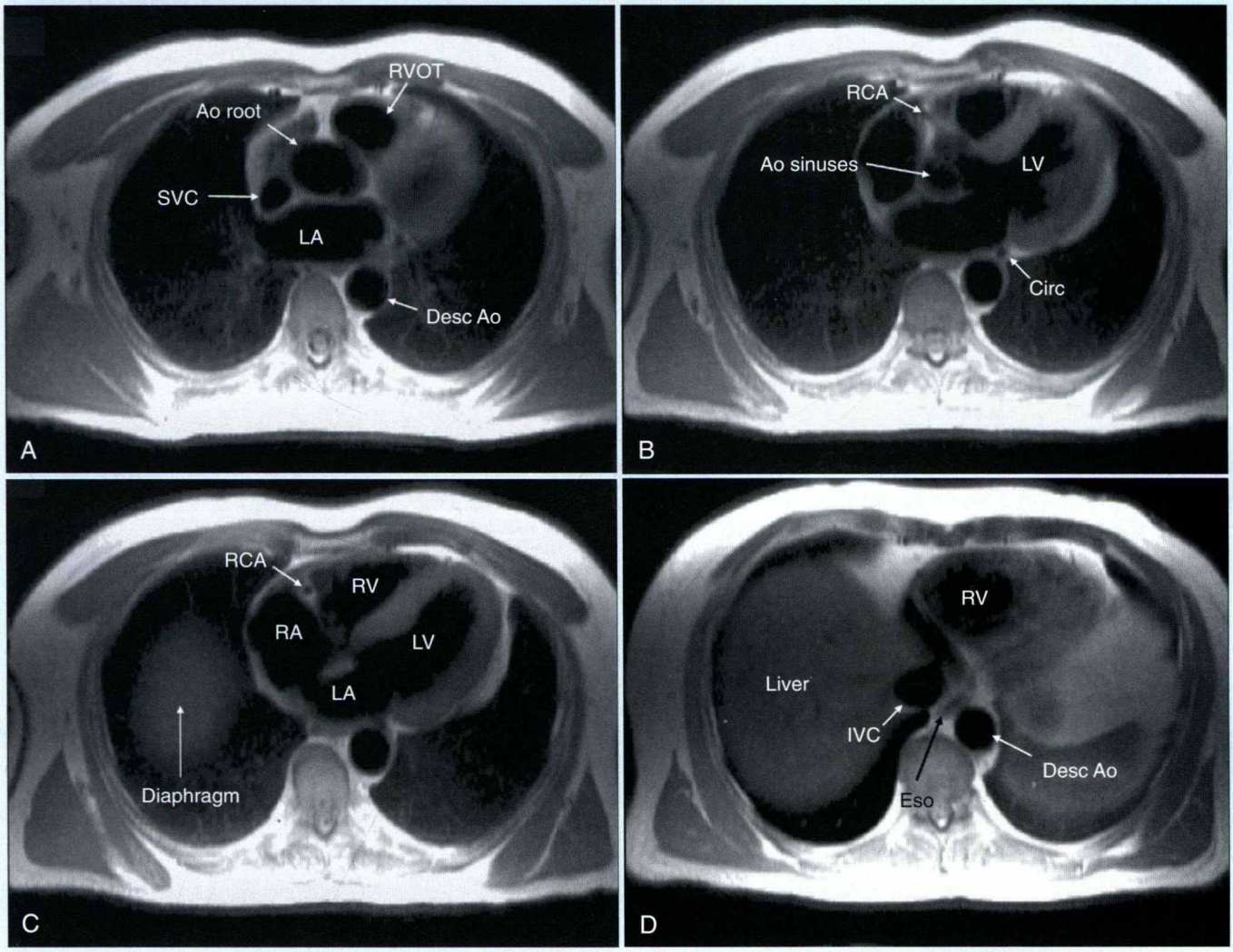

Figure 1-2 Transverse views from HASTE sequence in lower thorax, from superior (**A**) to inferior (**D**). The sequence characteristics are as for Figure 1-1. **A**, The superior aspects of the heart are now in plane, aortic root (Ao root), right ventricular outflow tract (RVOT) and left atrium (LA) visible, along with the SVC. The very top of the left ventricle (LV) can also be seen adjacent to the RVOT, although is better appreciated in the slightly lower slices. **B**, The aortic sinuses (Ao sinuses) and LV are visible, and the circumflex artery (Circ) is highlighted as a small circular black void within the fat in the left atrioventricular groove. The origin of the right coronary artery (RCA) can be seen arising from the right coronary cusp. **C**, The RCA is further seen, highlighted in a similar fashion to the circumflex, within the right atrioventricular groove. The main cardiac chambers are also seen—LA, LV, right atrium (RA), right ventricle (RV), and the dome of the diaphragm. At the lowest thoracic level (**D**), the liver can be seen because of the more superior position of the right diaphragm, along with the inferior vena cava (IVC). The Eso can be seen again adjacent to the descending aorta (Desc Ao), because both penetrate the diaphragm on entering the abdomen.