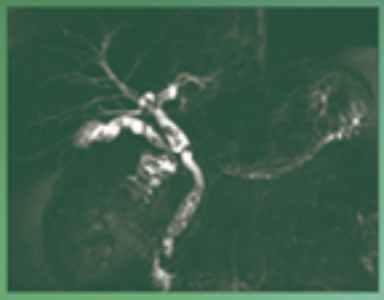
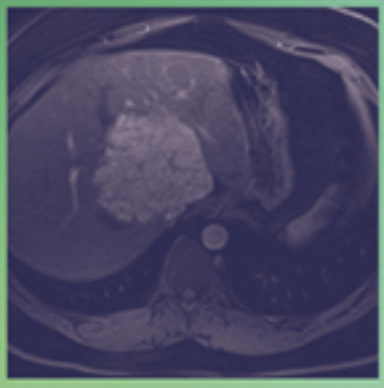
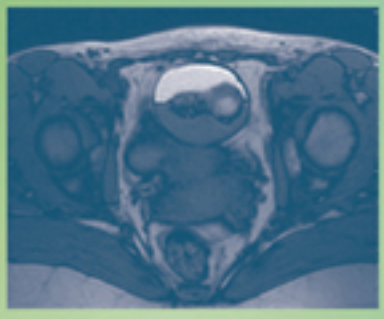
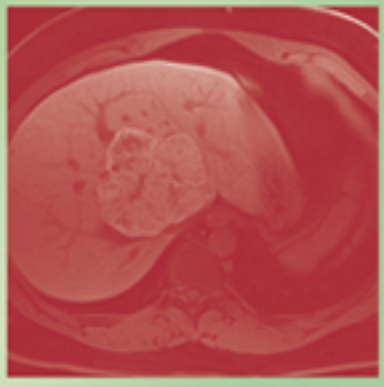
Practical Body MRI

Protocols, Applications and Image Interpretation









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To Charlie and Zoe: May you never need an MRI.
David J. Grand, MD

To my husband, Roland, and my parents, Karen and Richard, thank you for all your love and support.

To my children, Sophia and Liam, the joys of my life. You were both by my side while I worked on this book.
Courtney A. Woodfield, MD

To Margaret and Bill, who cultivated intellectual curiosity and prepared me for the world.

To dear Leslie who has supported me throughout.

And to James, Andrew, and Chris, who keep it all in perspective.
William W. Mayo-Smith, MD

Contents

Preface

Body Imaging Divisions in the United States tend to be biased to either CT *or* MRI. This was the case at Brown/Rhode Island Hospital where CT dominated. Our Chairman, John Cronan, sought to remedy this imbalance by hiring recently trained experts in body and pelvic MRI, Drs. Grand and Woodfield.

My job as division director was to encourage the development of imaging algorithms and MRI protocols that optimized patient care. Like many academic institutions, we had had no hesitation about adding the latest sequence, but seldom took any away. Thus our protocols were lengthy and often it was not clear exactly what each sequence was used for. We needed to pare down extra sequences, minimizing imaging time without sacrificing quality or patient care. If we couldn't explain why we were running a sequence or if it was redundant, it was eliminated. This is a subjective matter and, like making sausages, not always a pretty scene.

What started as a handout for residents on their first tour of duty in body MRI became this book. It is not intended to be a reference and cover all aspects of every disease, but rather to quickly get a neophyte from "zero to fifty." David and Courtney have done a great job of writing a short, practical book that teaches residents, fellows (and attendings) with minimal exposure to body MRI the most common MRI findings in the abdomen and pelvis. It is punctuated with David's subtle sense of humor. We hope you enjoy it!

William W. Mayo-Smith, MD FACR

To the reader

Reading abdominal and pelvic MRI isn't that hard.

Yet I still have colleagues who let body MRIs languish on the worklist. And, too often, normally brash, fearless residents (and fellows) arrive at the MRI service looking as though their mother is about to leave them on their first day of kindergarten.

I was one of them.

MRI has all the ingredients to instill fear into the bravest radiology warrior. The underlying physics is complicated and, to make matters worse, it is often taught by people to whom it comes naturally.

I wish I were one of them.

Toss in a spattering of seemingly incomprehensible abbreviations, lengthy lists of protocols each with more series than the next, and more adjustable parameters than a modern office chair, and it's no wonder that the uninitiated can be intimidated.

Sadly, when we allow ourselves to be intimidated, we miss out on the wonder of MRI and its evolution into a powerful problem-solving tool.

This book is for the inexperienced. It is an accessible introduction to abdominal and pelvic MRI. It is short, but it is not particularly abbreviated. It is simply written, but it is not simplified.

It is simply what you need to know to interpret abdominal and pelvic MRI.

In it you will find all the background, protocols, and imaging features that you need to approach MRI with confidence.

When you're finished, we think you'll agree: it just isn't that hard.

Each chapter of this book follows the same format:

(1) Name of the imaging protocol.
(2) Pulse sequences in each protocol and a brief description of why each is done.
(3) Approach to image interpretation and specific pathologic entities.

David J Grand, MD

Acknowledgments

No book is written in a vacuum and, while there are three authors listed, many people contributed to the text. We thank our families for their support and our Chairman, John Cronan, who has been a constant champion of academics at Brown. We also thank all our radiologic colleagues, particularly John Pezzullo, who granted us academic time to complete this task. And finally, we thank the medical students, residents, and fellows who keep asking the questions that make academics so stimulating.

WWM-S

Glossary of terms and abbreviations used in Body MRI

2D FL	Two-dimensional FLASH (fast low-angle shot)
3D	Three-dimensional
ACR	American College of Radiology
ADC	Apparent diffusion coefficient
AML	Angiomyolipoma
AVM	Arterio-venous malformation
B	B value
BH	Breath-hold
DWI	Diffusion-weighted imaging
ERCP	Endoscopic Retrograde Cholnagiopancreatography
FISP	Fast imaging with steady precession
FNA	Fine needle aspiration
FNH	Focal nodular hyperplasia
FS	Fat saturation
FSE	Fast-spin echo
GAD(O)	Gadolinium
HASTE	Half-Fourier acquisition single-shot turbo spin echo
HCC	Hepatocellular carcinoma
IMA	Inferior mesenteric artery
IP	In phase
IPMN	Intraductal papillary mucinous neoplasm
IV	Intravenous
IVC	Inferior vena cava
L	Liter
MIP	Maximum intensity projection
MRCP	Magnetic Resonance Cholangiopancreatography
MRA	Magnetic resonance angiography
MRV	Magnetic resonance venography
MS	milliseconds
NSF	Nephrogenic systemic fibrosis
OOP	Out of phase
PCKD	Polycystic kidney disease
PD	Proton density
POST	Post administration of intravenous contrast
PRE	Before administration of intravenous contrast
PSC	Primary sclerosing cholangitis
RCC	Renal cell carcinoma
SE	Spin echo
SMA	Superior mesenteric artery
SPEN	Solid and papillary epithelial neoplasm
SPGR	Spoiled gradient echo
STIR	Short-tau inversion recovery
SUB	Subtracted sequence (subtract pre-contrast image from post-contrast image)
T	Tesla
T1	Time required for tissue to recover longitudinal magnetization
T1W	T1 weighted: Sequence in which TE and TR are set to accentuate T1 characteristics of tissue
T2	Time in which tissues lose phase coherence
T2*	T2-star weighted sequence, highly dependent on magnetic field inhomogeneity and the decay of transverse magnetization
T2W	T2 weighted: Sequence in which TE and TR are set to accentuate T2 characteristics of tissue
TE	Echo time
TNF	Tumor necrosis factor
TOF	Time of flight
TR	Repetition time
True FISP	True fast imaging with steady-state precession
TSE	Turbo spin echo
UAE	Uterine artery embolization
VIBE	Volumetric interpolated breath-hold examination

Chapter 1

Introduction

Introduction

Body MRI is a dynamic, exciting modality. If read carefully, you will find in this small book the essentials to protocol, understand, and interpret abdominal/pelvic MRI. This is not a tome. It is light on physics. This sentence contains the only mention of *k*-space.

The utility of MRI for evaluation of the chest, abdomen, and pelvis has improved dramatically in the past decade due to more powerful scanners, better pulse sequences, and improved coils. It is the test of choice for evaluation of focal and geographic liver disease and the biliary tree. It is also commonly used to evaluate lesions of the kidneys, adrenal glands, and, more recently, the small bowel. With unparalleled soft tissue resolution, MRI has become the gold-standard imaging exam for evaluation of the female pelvis and staging of pelvic malignancies in either gender.

Fundamentally, MRI remains a problem-solving modality. Exams are/should be targeted to a specific diagnostic problem. The goal of MRI interpretation is to put a diagnostic issue to rest with one final test. It is critical that all previous imaging studies, as well as laboratory data and patient history, be thoroughly reviewed before protocoling and interpreting MRI studies. If the purpose of an exam cannot be determined from all available data, the referring clinician should be contacted.

When we attempt to interpret studies in a vacuum, everyone loses. Many clinicians hold fast to the belief that an ultrasound is better than a plain film, a CT is better than an ultrasound, and an MRI is the best there is. Therefore MRI is typically the last imaging stop, the final frontier – nowhere to go from here except biopsy. We need to be as definitive as possible.

Remember, the geniuses are the ones who thought it might be possible to create an image of the inside of the body using a really big magnet – and then made it happen. Imagine if they were asked to write a guide to creating and building an MRI scanner. It would be a heck of a lot more complicated than this book.

The interpretation is the easy part.

How MRI works in a few paragraphs (and T1 and T2 in a few more)

The MRI scanner works by placing the patient in a strong magnetic field, sending multiple radiofrequency (RF) signals into the patient, and then waiting, measuring, and localizing what comes out. The RF signal entering the patient comes from RF coils built into the magnet. The antennae or receiver coil can be the same "body coil" which is also built into the magnet, but we try whenever possible to use a "surface coil" – a coil placed on the surface (usually the belly) of the patient. (We call them coils, really they look more like foam pillows.)

Now let's tackle the basic MRI terms.

TE stands for echo time. It is the time we wait after sending the RF signal into the patient before we "listen" to the signal coming out. It is measured in milliseconds.

TR stands for repetition time. It is the time between sending the first RF signal into the patient and the next one. It is also measured in milliseconds.

That's it! *TE and TR are the two basic parameters that we change at the scanner console to determine what the image will look like.*

What about T1 and T2? These are *not* parameters we vary on the magnet, but *are innate physical properties of matter* that describe how tissues behave in a magnetic field. T1 is the time for tissues to recover longitudinal magnetization in a magnetic field and T2 is the time to lose phase coherence. Please now feel free to forget the last sentence (if you haven't already).

But, *do not forget that T1 and T2 are innate physical properties of matter*. We are born with them. MRI generates valuable images because there is tremendous

natural variation in the T1 and T2 of different tissues; the result is magnificent soft-tissue contrast.

There is much more variation in the T1 and T2 times of various tissues than there is in density, which is what we measure and "see" on CT. That's why a CT of the knee shows the bone as bright, the fat as black, and all the muscles and tendons as a gray mush, whereas MRI can separate each soft tissue from its neighbor.

By placing the patient in the magnet and varying the TR and TE to specific parameters (figured out by MRI physicists, not you and me) we can generate an image that emphasizes the T1 properties of the tissue. This is called a T1-"weighted" image. By changing the TR and TE, we can instead emphasize the T2 characteristics of the tissue. This image is then called T2-"weighted". By learning what different tissues and pathologies look like on T1-weighted and T2-weighted images, we can not only detect abnormalities, but also characterize the lesions that we detect.

It is because MRI has more variables than CT that we can be more specific. (CT has essentially only two variables – attenuation and enhancement.)

On T1-weighted images, fluid is dark. On T2-weighted images, fluid is bright. Thus, fluid in the bladder or CSF space is generally dark on T1-weighted images and bright on T2-weighted images. If you are looking at a set of images and don't know if they are T1- or T2-weighted, first find a fluid containing structure in the image (bladder or CSF space.) If it is dark, you are looking at a T1-weighted image, if it is bright, you are looking at a T2-weighted image (Figure 1.1).

Of course, "weighting" is not all or nothing. Most modern pulse sequences have a combination of T1- and T2-weighting. The beginning reader should think of T2-weighted sequences as fluid-sensitive – that is, *if fluid is bright, consider it a T2-weighted image*. Don't lose the forest for the trees.

So. Fluid is bright on T2-weighted images. What is bright on T1-weighted images?

The list of things that are bright on T1-weighted images is one of the great lists in radiology – great, because it is short. This is not the differential diagnosis of white-matter disease in the brain or, my favorite, a lecture I (twice) attended entitled "The twenty-one mimickers of pneumonia on chest x-ray." There are only four things:

(1) *Blood* Blood is very important. Don't miss blood.
One nice thing about body imaging is that the signal of blood products within the abdomen/pelvis does not necessarily follow the complex, mnemonic-ridden sequence it does in the brain. Don't try to "age" blood products in the abdomen/pelvis. Just be happy you detected them. Note also that flowing blood within vessels may be variably T1-bright as well.
(2) *Fat* Fat is bright on T1-weighted images. That is, as long as there is no fat saturation.
(3) *Protein* Various proteins are bright on T1. This includes melanin. Metastatic melanoma can be bright on T1.
(4) Enhancement with gadolinium-based contrast agents.

Fat is inherently bright on T1-weighted images and may be variably bright on T2-weighted images. Many sequences may be performed with or without fat saturation. Fat saturation is a parameter modified on the scanner to make fat lose its bright signal. Chemically selective fat saturation causes *macroscopic* fat to lose signal and become black. This is considered proof beyond any reasonable doubt that the tissue contains fat.

One final topic of unnecessary confusion. Pulse sequences are proprietary, they are developed and owned by the vendors, and therefore the vendor gets to name them. Commercially available scanners, from whichever vendor, all have essentially the same basic array of sequences for clinical use, but will name them differently. (This is analogous to generic and brand-name drugs. The generic name is most important, it describes what the pulse sequence actually is. The brand name is designed to sound good.)

For example, all vendors have a T1-weighted fat-saturated sequence used before and after gadolinium administration. Siemens calls it VIBE, General Electric liked the sound of LAVA, Philips named theirs THRIVE. In this section, we will list the sequences by "generic" names with "brand" names in parentheses.

Each sequence in each exam should be performed for a specific reason. *When looking at a series of images, ask yourself specifically what that sequence adds to the overall exam*. If you don't know why a sequence is performed or what it adds to the exam – ask someone else. *If no one knows, it should be eliminated*.

So, without further ado, commonly used sequences. . .

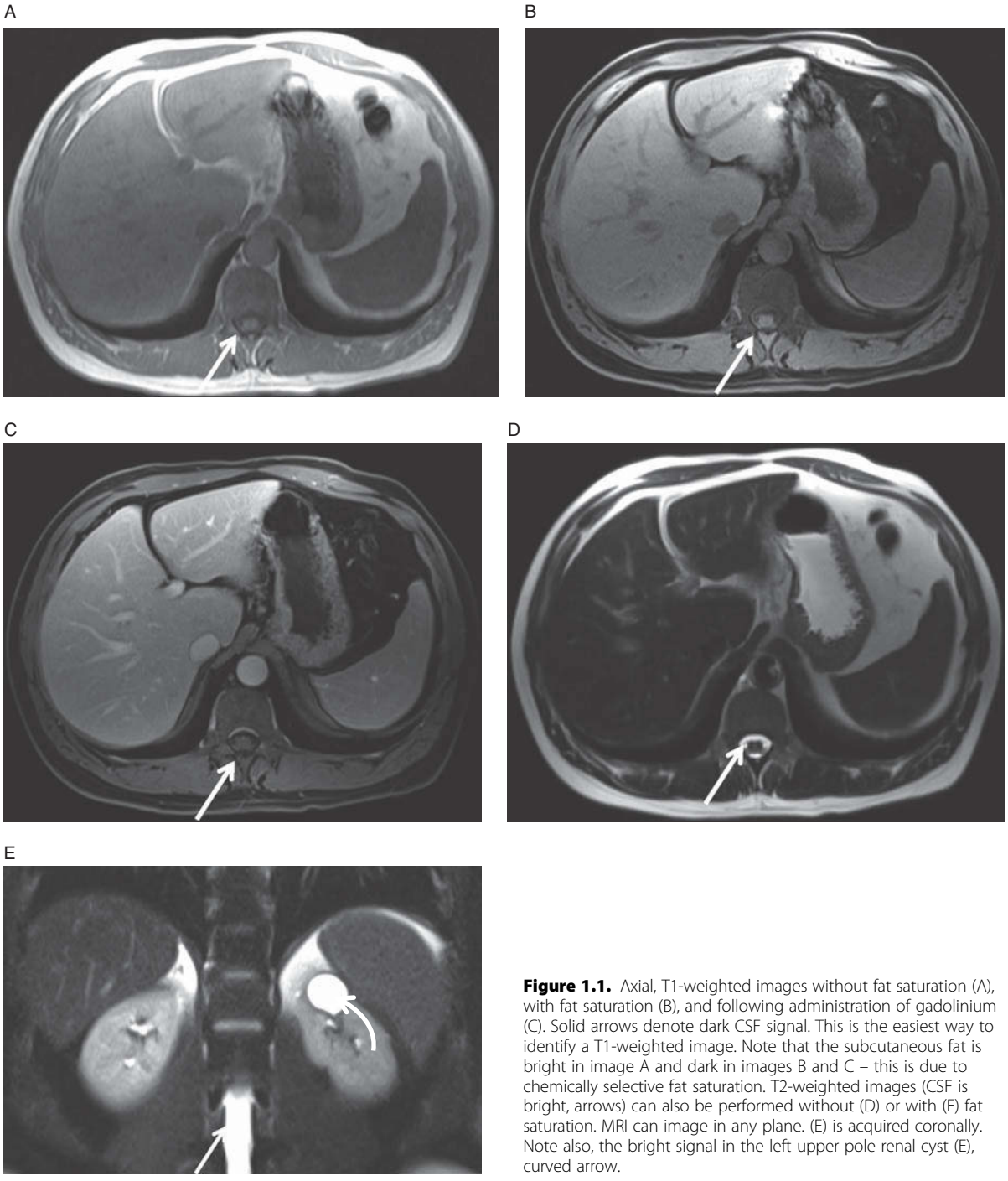

Figure 1.1. Axial, T1-weighted images without fat saturation (A), with fat saturation (B), and following administration of gadolinium (C). Solid arrows denote dark CSF signal. This is the easiest way to identify a T1-weighted image. Note that the subcutaneous fat is bright in image A and dark in images B and C – this is due to chemically selective fat saturation. T2-weighted images (CSF is bright, arrows) can also be performed without (D) or with (E) fat saturation. MRI can image in any plane. (E) is acquired coronally. Note also, the bright signal in the left upper pole renal cyst (E), curved arrow.

Commonly used pulse sequences

Many are intimidated by the seemingly never-ending and often-changing array of pulse sequences. Do not fear. In body imaging there is really only T1, T2, and diffusion. That's it.

There is no T3.

Below are the pulse sequences we commonly use as well as a brief description of what they are used for. Since there's only T1, T2, and diffusion, first we will discuss T1-weighted sequences, then T2-weighted sequences and then diffusion. Then we'll stop.

Nearly all the sequences used in body MRI are performed while the patient holds their breath. (There are rare exceptions which will be described later.) Good breath-holding is absolutely critical to generating quality images, particularly post-contrast images which are often the most time-consuming and therefore motion sensitive. It is critical that the MRI technologist coach and coax the patient, offer encouragement (and breaks when necessary) in order to achieve the best possible breath-holds. When the images are clear, the interpretation is easy.

T1-weighted sequences

The original T1-weighted images were spin echo sequences. This is the classic sequence described in radiology physics classes with the 90° pulses, etc. . . . Forget it. Takes too long. Not relevant to body imaging (unless the patient can hold their breath for 12 minutes).

The T1-weighted images we perform now are gradient echo. The physics of this is not particularly important for our needs. But, you should know that they are gradient echoes because you'll look stupid if you ever tell someone they are spin echoes. One of the reasons that gradient echo images are so fast is that we can use very small flip angles – less than 90°. This is the last time we will mention flip angles. In clinical practice, you will never change the flip angle.

We don't just get a T1-weighted gradient echo image. What we actually get is called a dual-echo or chemical-shift image.

In- and out-of-phase/chemical-shift images (General Electric = GRE, Philips = FFE, Siemens T1-weighted images, no fat saturation)

Chemical-shift imaging is also known is in/out-of phase imaging (IP/OOP). These are T1-weighted images without fat saturation acquired with two different TEs. You get both sets of images during the same acquisition. The difference is the TE, i.e. when we "listen" for the echo. Both are T1-weighted, but one set is in phase and the other set is out of phase.

Remember high-school physics? Did you ever make waves with jump ropes in a hallway or in little pools of water?

When two waves collide and their amplitudes are in the same direction (up or down) the amplitudes are additive. If the waves collide when one is up and one is down, they cancel.

The signals coming back to the scanner from the patient are also a wave. The signal is generated by protons, but the frequency of that signal changes based upon the microenvironment of each proton.

A proton bound to water emits a signal with a different frequency than a proton bound to fat. People smarter than you, me, or anyone either of us know figured out the precise frequencies of these signals. Of course, they vary with magnetic field strength, but at 1.5 T waves from protons bound to fat and protons bound to water will be out of phase at a TE of approximately 2.2 ms and in phase at 4.4 ms.

It is important to remember that we are talking about microscopic fat and the signal cancellation is on a per voxel basis.[1] That is, when protons bound to water and protons bound to fat are in the same voxel, their signals will be out of phase if imaged at a TE of 2.2 ms and the scanner will actually receive little signal. This voxel will be dark on the out-of-phase images and bright on the in-phase images (waves additive).

Our solid organs contain a great deal of water and are surrounded by fat. Thus, at the borders of solid organs there are protons bound to water (within the organ) and protons bound to fat (outside the organ). On an out-of-phase image, these "border" voxels have both protons bound to water and protons bound to fat and will therefore lose signal (turn black). This creates "etching" or "India-ink" artifact surrounding the solid organs.

The presence of microscopic fat is extremely useful for characterization of both focal and diffuse disease. If an adrenal mass has microscopic fat, if its signal drops significantly on the out-of-phase images compared to the in-phase images, it is an adenoma (Figure 1.2). If geographic areas of the liver lose signal on out-of-phase images this is diagnostic of hepatic steatosis.

[1] A voxel is a three-dimensional pixel.

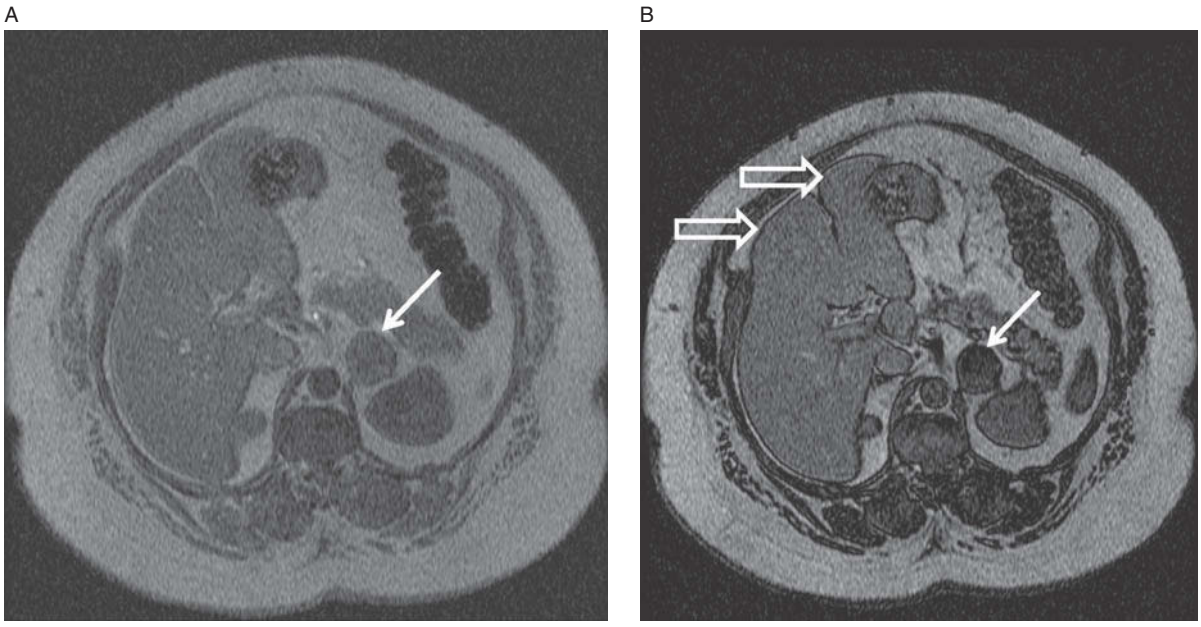

Figure 1.2. Adrenal adenoma. Solid arrows demonstrate marked signal loss in the left adrenal mass on the out-of-phase image (B) compared to the in-phase image (A). This proves that it contains microscopic fat, diagnostic of an adrenal adenoma. Open arrows demonstrate the appearance of "etching" artifact at the interface of the liver (which contains lots of water) and the peritoneal fat on the out-of-phase image.

Now, for the advanced student . . . those striving for mediocrity can proceed to the next section (you know who you are). Tissues that contain microscopic fat lose signal on out-of-phase images. What might lose signal on in-phase images?

Remember that the TE of the in-phase image (the time we wait before we listen for the signal back from the patient) is twice as long as that of the out-of-phase image. Iron deposition causes inhomogeneity in the magnetic field and inhomogeneity causes signal loss. The longer you wait to image, the more signal loss. Therefore, in patients with iron overload, the sites of iron deposition will lose signal as the TE lengthens (Figure 1.3). The degree of signal loss is directly related to the amount of iron in mg/dl.

Remember, in-/out-of-phase images assess microscopic fat. Signal loss occurs on the out-of-phase image when water and fat share the same voxel. To evaluate macroscopic fat, look for a sequence which utilizes a chemically selective fat-saturation pulse (typically, the pre-contrast T1-weighted images – see below). Signal drop on a chemically selective fat-saturated image is considered histologic proof of macroscopic fat, such as in an ovarian dermoid.

Volume-interpolated gradient echo (General Electric = LAVA, Philips = THRIVE, Siemens = VIBE) T1-weighted, fat-saturated images

These are our pre- and post-contrast images.

In the United States, all currently approved MRI contrast agents are chelates of gadolinium. These agents function by increasing the T1-relaxivity (shortening the T1 time) of blood and tissues.

Therefore, contrast-enhanced images are always T1-weighted.

Gadolinium is bright on T1-weighted images.

You can run a T2-weighted sequence post-contrast – it will look remarkably similar to a T2-weighted sequence run pre-contrast. The gadolinium (with few exceptions) has no noticeable effect on a T2-weighted image.

As we've discussed, fat is inherently bright on T1-weighted images. Pre- and post-contrast images are performed with chemically selective fat saturation so that the enhancement stands out prominently against a dark background. On modern scanners, this is accomplished by pressing a button on the scanner which squashes the spectral peak of fat so that we are essentially imaging only water. Macroscopic fat, which is bright on the in-/out-of-phase images, becomes black.

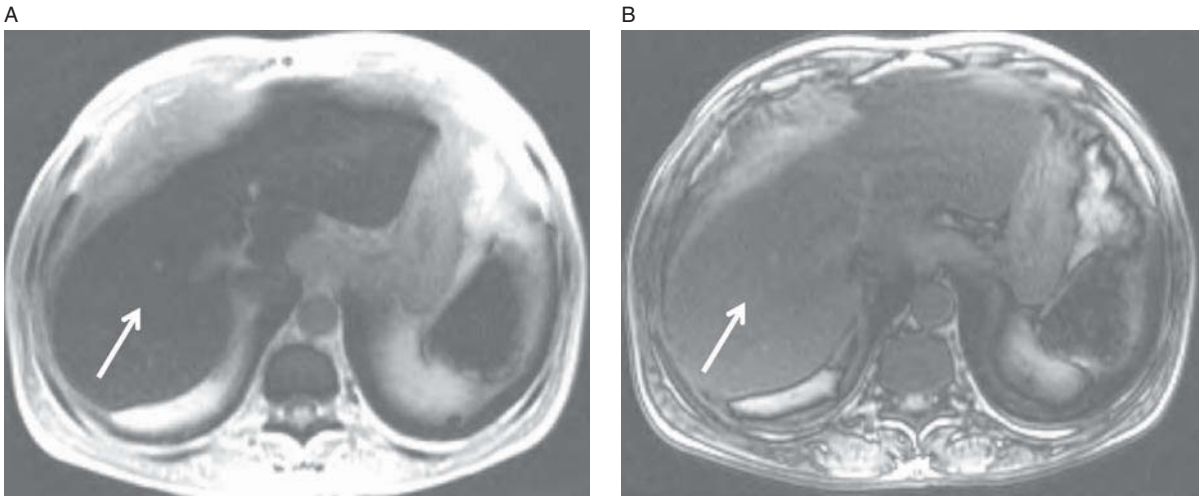

Figure 1.3. Iron overload, chemical shift imaging. Arrows demonstrate dark signal in the liver, spleen, and bone marrow on the axial out-of-phase image (B) which drops significantly on the in-phase image (A). This is due to the longer echo time of the in-phase image. The longer you wait to listen, the more signal you lose due to the inhomogeneity introduced by the iron in the magnetic field.

Whenever giving contrast, pre-contrast images should *always* be obtained. Imaging parameters must remain identical for pre- and post-contrast images so that subtraction images can be generated. Pre-contrast fat saturated images are performed to:

(1) Assure adequate coverage of intended imaging area.
(2) Assess (and correct) artifacts.
(3) Identify lesions that are T1-bright before contrast so that we *don't mistake them for enhancing lesions!* (This is a significant advantage of MRI over CT. You wouldn't routinely obtain pre-contrast imaging with CT because of the additional radiation dose. With only one phase, it can be difficult to tell enhancement from calcification, etc.)
(4) Identify macroscopic fat by comparing to in-phase images which are also T1-weighted but are performed without chemically selective fat saturation.

Subtraction images

Based on all the variables we have discussed, the scanner assigns a numeric value to each voxel in an image which is then displayed as a dot of variable brightness.[2] Subtraction images are generated by subtracting the value of each voxel of the pre-contrast images from the corresponding voxel on the post-contrast images and then displaying this "new" value.

For example, let's suppose a renal lesion is bright on T1-weighted images before contrast with a signal intensity of 200. It will also be bright on post-contrast images, but did it enhance? The subtraction image takes the post-contrast signal intensity and subtracts the initial value of 200. So, if the signal after contrast is 200, the subtracted pixel value will be 200 (post) – 200 (pre) = 0. The lesion will appear black. If, post-contrast, the signal intensity is greater than 200, it will appear bright.

Subtractions allow confident assessment of the presence or absence of true enhancement of lesions which are T1-bright before contrast administration

[2] This is a critical distinction between the way CT and MRI work. Hounsfield units are a measure of density. They are a physical property of matter based on the ability of a substance to attenuate x-rays. The density of water is defined as zero Hounsfield units. We can then "window" the image, alter the parameters by which the image is displayed, to accentuate voxels of different values. For example, lung windows accentuate voxels of very low number. However, changing the windows does not change the voxel value in Hounsfield units. Signal intensity values on MRI are not uniform, standard, or necessarily consistent. Fat will always be bright on a T1-weighted image, but its numeric value can change. This is because the scanner, in an effort to do us a favor, automatically scales each image for us before displaying it to create the "optimal" image. The scale typically changes only when a new pulse sequence is run but it can also change suddenly, without warning or explanation. Better not to draw ROI's unless you are prepared to also draw them on an internal standard and perform a more complicated calculation.

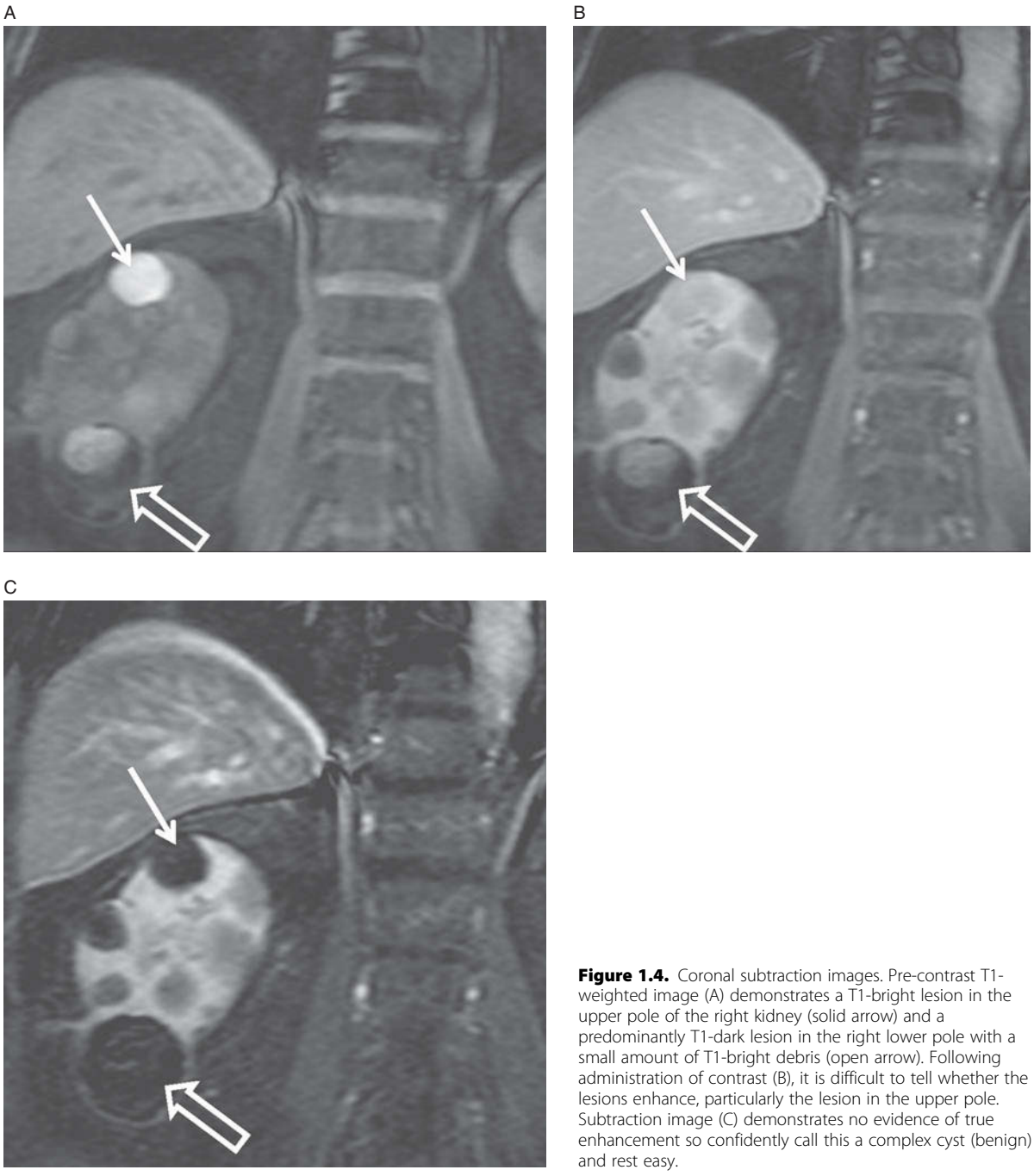

Figure 1.4. Coronal subtraction images. Pre-contrast T1-weighted image (A) demonstrates a T1-bright lesion in the upper pole of the right kidney (solid arrow) and a predominantly T1-dark lesion in the right lower pole with a small amount of T1-bright debris (open arrow). Following administration of contrast (B), it is difficult to tell whether the lesions enhance, particularly the lesion in the upper pole. Subtraction image (C) demonstrates no evidence of true enhancement so confidently call this a complex cyst (benign) and rest easy.

(Figure 1.4). They may also accentuate subtle hypervascularity which can be difficult to appreciate on conventional contrast-enhanced images. Subtraction images can only be created if all imaging parameters are *identical* on the pre- and post-contrast sequences.

T2-weighted sequences

Just as with T1-weighted images, the original T2-weighted images were spin echo. Forget about them. They take too long. We don't use them in body imaging.

Fast-spin echo (General Electric = FSE, Philips = TSE, Siemens = TSE) T2-weighted images; can be performed with or without fat saturation

This was the first, practical T2-weighted sequence for body imaging. It has the potential to generate excellent resolution with unparalleled T2-weighting. At many institutions, including possibly your own, they are a part of every body MRI exam. Unfortunately, for my taste, they still take too long, which makes them prone to motion artifacts when the patient breathes. We typically only use them in the pelvis, which, at least while in the MRI scanner, is relatively motionless.

Single-shot fast-spin echo (General Electric = SSFSE, Philips = SSTSE, Siemens = HASTE) T2-weighted images; can be performed with or without fat saturation

Now we're talking fast.

SSFSE is fast because only 1 RF pulse is used. We then "listen" for numerous echoes to create the image.

SSFSE images are very fast, T2-weighted images which may be obtained with or without fat saturation. Because the acquisition time is short, they are almost always free of motion. Initially they were (and are) the backbone of MRI cholangiopancreatography (MRCP) but they have now replaced TSE/FSE at many institutions (including our own). There's no free lunch here.

The pro: they are fast and therefore motion free.

The con: they are not as heavily T2-weighted as TSE/FSE. (In later chapters we will discuss why we are willing to sacrifice the T2-weighting for the sake of speed.)

As discussed above, T2-weighted images are "fluid-sensitive" – i.e., fluid is bright. But, beware, not all that is bright represents fluid. Classic mistakes include mucinous and neuroendocrine tumors which can be bright on T2. Close inspection will show that these lesions are not quite *as* bright as fluid – correlation with other sequences is critical.

Diffusion-weighted images

Diffusion-weighted images and ADC (General Electric = to be announced, Philips = DWIBS, Siemens = Reveal)

We like diffusion-weighted images (DWI), but you should know that this sentiment is not shared by everyone. They are not strictly necessary for good body MRI and therefore not everyone uses them. I think they make our job easier and I like things that make our job easier.

DWI is an exciting, relatively new area of body MRI which attempts to bridge anatomic and functional imaging. DWI measures and displays the ability of water molecules to diffuse through tissue. The ADC (actual diffusion coefficient) map is a plot of the actual diffusion coefficient.

Normal tissues consist predominantly of interstitial or extracellular space through which water can easily diffuse. Tumors are hypercellular when compared with normal tissues and this increased number and concentration of cells decreases the amount of free interstitial space for water to diffuse through easily, replacing it with intracellular space which creates boundaries and obstacles to diffusion. The result is restricted diffusion which is displayed as bright on DWI and dark on the ADC map (Figure 1.5).

Fundamentally, conventionally acquired DWI are T2-weighted – it is therefore critical to correlate DWI findings with the ADC map. Lesions that are bright on DWI but also bright on ADC do *not* represent true restricted diffusion, but "T2 shine-through."

Currently, the best use for DWI is lesion detection. DWI is now widely considered to be the most sensitive sequence for the detection of liver lesions (this may simply be due to their extreme T2-weighting and one reason we are willing to sacrifice the heavily T2-weighted FSE in favor of the single-shot).

It is tempting to use DWI to characterize masses; however, do so with caution. Despite the early excitement of many investigators, benign and malignant lesions can demonstrate significant overlap in ADC. DWI should therefore be viewed as one tool in our armamentarium – excellent for lesion detection, and one of many tools which should be evaluated together for lesion characterization.

The sensitivity of DWI to water motion can be varied depending on the gradient amplitude, duration of applied gradient, and time interval between paired gradients. The "b-value" can be changed on modern MRI scanners to increase sensitivity to diffusion, which is proportional to the above three factors, with emphasis on the gradient amplitude.

Technically speaking, the ADC is a curve, and the more b-values one uses, the more accurately that curve will reflect the true ADC. Some institutions will therefore use ever-increasing numbers of b-values to more accurately define the true ADC. The drawback to this is that more b-values typically mean increased acquisition time.

But, the actual ADC value doesn't much matter. As we've discussed, you can't use it as a strict predictor of histology. So if the specific number doesn't exactly matter, why measure it exactly?

We currently use b-values of 50 s/mm^2 and 500 s/mm^2. Obviously, with only two points we get

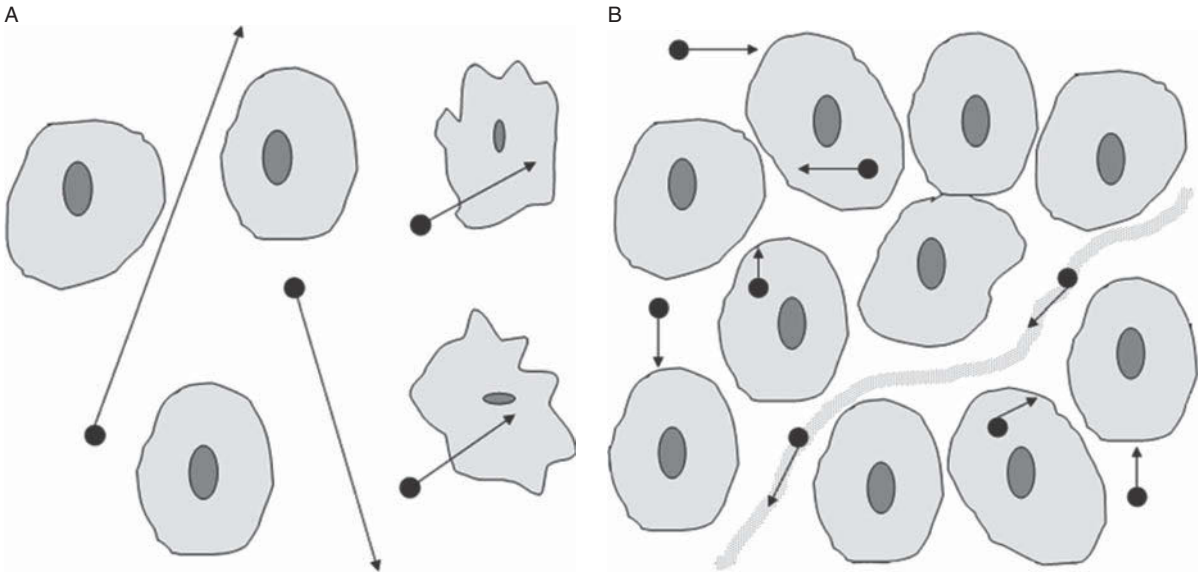

Figure 1.5. Diffusion-weighted imaging. Diagram of normal cells (A) demonstrating extensive extracellular space through which water can easily diffuse. Diagram of abnormal tissue (B) showing decreased extracellular space due to cellular crowding. Water cannot easily diffuse in the space around these cells. This is restricted diffusion. (Reprinted with permission from *AJR*, 6/2007 **188**: 1622–1635.)

a line, not a true curve. But, what's bright is bright and since all we really care about is detection (not characterization) it is accurate enough.

Water molecules with a large degree of motion or great diffusion distance will be hyperintense at low b-values. Alternatively, larger b-values (e.g. 500 s/mm^2), are required to perceive slow-moving water molecules or small diffusion distances.

Steady-State Free-Precession (General Electric = FIESTA, Philips = Balanced-FFE, Siemens = TruFISP)

Consider this a T2 weighted-sequence (fluid is bright).

This sequence features a very short acquisition time and can be used to generate cine images for cardiac or bowel imaging. It also depicts flowing blood within vessels as high signal intensity ("bright-blood" MRI). In the current era of nephrogenic systemic fibrosis (NSF) hysteria, there is renewed interest in these sequences as non-contrast MRAs.

And . . . well, that's it. Don't be intimidated by MRI pulse sequences and protocols. Fundamentally, there just isn't that much to it. You've got T1, T2, DWI, and +/− fat saturation. Each of these provides one more little piece of information which will lead us to our diagnosis.

RF coils used in body MRI

The coil is the antenna used to receive MRI signals from inside the body. The MRI signal which we are trying to capture is quite weak so the closer we can place our antenna to the body part we wish to image, the more signal we obtain (i.e., the better our signal-to-noise ratio).

Body coil

The MRI scanner arrives equipped with a send/receive coil within it – the "body" coil. While always used to send RF pulses into the body, it can also be used as an antenna to receive signal back. Obviously, this coil is as far from the body part we wish to image as a coil can be (while still being in the scanner) and thus should only be used when a surface coil will not fit between the patient and the bore of the magnet (i.e. obese patients).

Phased-array coils

Whenever possible, a phased-array surface coil is used. The term "phased-array" means that the surface coil is actually made of multiple (currently up to 32) individual coils which can each receive signal independently (these are receive-only coils). Having lots of individual coils within the larger coil allows us to perform parallel imaging. The physics of this is beyond the scope of this book, but parallel imaging decreases the acquisition time of pulse sequences by factors of 2 and beyond.

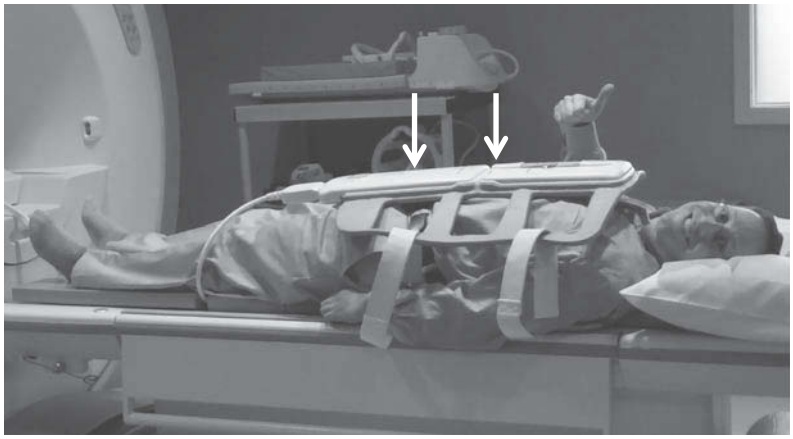

Figure 1.6. Surface coil placement. Arrows indicate the phased-array surface coil on the ventral surface of the patient (author). Depending on the manufacturer, either the patient lies directly on the posterior surface coil, or it is embedded in the table.

The surface coil is placed on the ventral surface of the patient. We call them "coils" but they look like foam pads. The coils are hidden on the inside (Figure 1.6). Modern scanners combine this information with signal information from the phased-array spine coil which either the patient lies directly on or is embedded within the table. Careful observation of images acquired particularly from larger patients will show brighter signal at the ventral and dorsal surfaces of the patient with a decrease in signal toward the center of the patient as the coils each become more distant.

Endocavitary coils

Everyone's favorite topic. Knowledge of these coils is more useful to disgust your friends than for the actual practice of radiology. But they do currently have a few clinical indications.

Remember that the signal we are listening for is very weak and diminishes rapidly with distance. The closer we can place our coil to the area of interest, the better the image. Hence, endocavitary coils placed within the body.

There are a variety of these coils, but the most commonly used is the endorectal coil which is used to image the prostate. Suffice it so, you can't get much closer to the prostate than this, but it often doesn't get there without a fight. Endocervical and even endourethral coils are available but are not widely used outside of large academic centers.

Contrast agents used in body MRI

Intravenous contrast agents

All intravenous MRI contrast agents available in the United States are chelates of gadolinium. They function by increasing the T1 relaxivity of blood and tissue, rendering them bright on T1-weighted images. They are typically power-injected and imaging may be performed dynamically, i.e. in multiple phases. Practically, there are two categories of gadolinium agents.

Extracellular or non-specific

These are the most commonly used agents. They are used for all types of MRI. Prior to the advent of Nephrogenic Systemic Fibrosis (NSF), they were essentially interchangeable.

Gadolinium-based contrast agents are extremely safe because the gadolinium ion is bound to something else. Free gadolinium is bad. The strength with which the gadolinium moiety is bound to its chelate varies between contrast agents, and the working theory of NSF is that it occurs when the gadolinium ion breaks free from its chelate and wreaks havoc. Theoretically, the tighter the binding, the safer the agent.

Gadopentetate dimeglumine (Magnevist)
Gadoteridol (ProHance)
Gadoversetamide (OptiMARK)

Liver specific

There are currently two available agents in the United States that are considered liver specific.

Gd-BOPTA (MultiHance) and gadoxetate disodium (Eovist) are taken up by functioning hepatocytes in the liver and have variable biliary excretion, as opposed to the renal only excretion of the non-specific agents.

Perhaps the main value of these agents is in distinguishing focal nodular hyperplasia (FNH) from hepatic adenoma. The imaging features of these two lesions overlap when using non-specific gadolinium

agents. Both are hypervascular on arterial-phase imaging and are essentially isointense to liver parenchyma on portal-venous and delayed-phase imaging. Despite all the textbook chatter regarding central scars in FNH and heterogeneity due to internal blood and fat within adenomas, these features may only be found in 50% of lesions (as is typical of appearances described as "classic").

On delayed images, FNH will retain these liver-specific agents and be isointense or hyperintense compared to liver whereas adenomas will be hypointense.

Like most explanations, the following is postulated rather than known, but it makes us feel better and gives us something official sounding to tell the referring clinicians. Both FNH and adenoma have functioning hepatocytes; however, whereas FNH has abnormal, blind-ending bile ducts, adenomas have no ducts at all. FNH will therefore take up the contrast hoping to excrete it, but because its ducts are abnormal the contrast has nowhere to go and gets trapped in the lesion.

Since adenomas have no ducts at all, biliary metabolism is blocked entirely and its hepatocytes will neither take up nor excrete the contrast. Hence, FNH will be isointense to liver or bright on delayed images (gadolinium is trapped in it), and adenoma will be hypointense (gadolinium is not taken up by it.)

What will other focal liver lesions look like? Any lesion without normal hepatocytes will be black on delayed images with these agents. That includes: cysts, hemangiomas, and metastases. There is some evidence to suggest that delayed imaging with gadoxetate disodium may increase sensitivity for detection of metastatic disease.

Because they are partially excreted through the biliary tree (in addition to renal excretion) it is possible to use these agents to make very pretty T1-weighted MRCPs. While interesting in principle, this technique has not proven particularly useful . . . yet.

Intravenous contrast dosing

The dosing of intravenous (IV) contrast agents should be determined by weight. A standard or "single" dose of a conventional, non-specific gadolinium agent is 0.1 mmol/kg.[3] A "double" dose is 0.2 mmol/kg. Traditionally, MRAs and cardiac exams were performed with a double dose, although this is changing with better scanners, better contrast agents, and fear of NSF.

The two available hepatocyte-specific agents have a higher T1 relaxivity than the other gadolinium agents. That is, they give you more enhancement per mmol.

At the time of writing, the optimal dose of gadoxetate disodium is controversial. We use 10 cc regardless of weight because it comes in a 10-cc vial.

Intravenous contrast agents are typically power-injected at 2 cc/s followed by a 20-cc saline "chaser" to flush the contrast from the tubing into the vasculature.

Oral contrast agents

Oral contrast is currently used at our institution for two indications. First, we routinely use ferumoxsil (GastroMARK) for MRCPs. Ferumoxsil is a suspension of iron oxide which is dark on both T1- and T2-weighted images. This helps to eliminate fluid signal within the duodenum which could otherwise obscure the ductal anatomy on MRCP images (which are heavily T2-weighted).

Secondly, we use a barium sulfate suspension (0.1% weight/volume, VoLumen) which is a low-concentration barium and sorbitol solution to distend the small bowel for MRI enterography exams. This agent is not absorbed, and yields (variable) distension of the entire small bowel. Because it is not absorbed, what goes in also comes out. It's nice to warn your patients beforehand.

MRI contrast use (gadolinium) in patients with renal dysfunction

During my MRI training, elevated creatinine (poor renal function) was probably the single most common indication for performing MRI. Times have changed.

Gadolinium contrast agents had been thought to be innocuous, despite the fact that the gadolinium ion itself was known to be quite toxic. It is rendered inert by binding to another molecule.

Remember that all or nearly all of the excretion of the most commonly used agents is renal. It is postulated that patients with poor renal function may not be able to excrete the contrast before the gadolinium ion manages to dissociate itself from its larger molecule. It is then free to wreak havoc.[4]

[3] The manufacturers have actually done us a favor here. The concentrations of all listed IV contrast agents except gadoxetate disodium (Eovist) are such that the standard dose of 0.1 mmol/kg is roughly equal in cc to the patient's weight in pounds divided by ten. Hence, a single dose of gadolinium for a 150-lb person is 15 cc. A double dose would be 30 cc.

[4] This is also why we *never, never, never* give gadolinium to a pregnant patient. The contrast agent can diffuse into the amniotic fluid and stay there – with plenty of time for the gadolinium ions to disassociate and cause trouble.

Recent studies have linked a serious (potentially fatal) group of diseases known as nephrogenic systemic fibrosis/nephrogenic fibrosing dermopathy (NSF/NFD) to the administration of gadolinium-based contrast agents in dialysis-dependent patients and those with severe renal failure, and an estimated glomerular filtration rate (eGFR) less than 30 ml/min. Use of gadolinium in patients with moderately reduced renal function, eGFR of 30–60 ml/min, has also been cautioned by the FDA.

Our current institutional guidelines are not listed because they are too darn complicated.

Here is how I approach gadolinium administration in the NSF era:

(1) If the patient is an otherwise healthy outpatient, without known or suspected kidney disease, give gadolinium and sleep easy at night. Use any gadolinium agent.
(2) If the patient is an inpatient or has known or suspected renal dysfunction, calculate an eGFR:
    (a) If eGFR >60 ml/min, use any gadolinium agent and sleep peacefully
    (b) If eGFR <60 ml/min but >30 ml/min try to use an agent with the tightest binding that your institution stocks
    (c) If eGFR <30 ml/min don't give gadolinium unless absolutely necessary. If absolutely necessary use ½ dose of an agent with a high T1 relaxivity.
(3) If patient is on dialysis: don't give gadolinium.

To my knowledge, no one with an eGFR ≥40 has ever gotten NSF.

It is important to think beyond the hysteria and remember that everything we do in medicine involves some calculation of risk vs. benefit. If you look at the number of cases of NSF compared to the number of MRIs performed in the world, statistically it essentially doesn't exist. Additionally, nearly all of the reported cases of NSF attributed to gadolinium are thought to be due to one contrast agent, gadodiamide (Omniscan), which is no longer in widespread use.

The point is this: unless a patient is on dialysis, their actual risk of contracting NSF from gadolinium is extremely low.

We tend to lose sight of this risk-vs.-benefit calculation in radiology because our tests and procedures are typically quite safe. However, there is risk to the patient in everything we do, and our willingness to perform a test/procedure implies that after careful consideration we believe the benefit of the test/procedure to outweigh the risk. It is important to remember that to date not a single person with normal renal function has acquired NSF. NSF has certainly increased the risk in the risk/benefit equation; however, we should use our judgment in addition to institutional guidelines to calculate the risk/benefit ratio for each patient. With improved protocols in place, NSF has essentially been eradicated.

Individual body protocols

There is no single right way to perform an MRI exam of the abdomen or pelvis. There is no magic protocol. The protocols listed here are examples. They work for us. We get the information we need in a reasonable amount of scanner time. All exams have both T1- and T2-weighted images. Pre- and post-contrast images should always be performed with fat saturation. We like DWI and use it routinely.

The exact protocol is not important. What is critical is to understand the purpose of each sequence, what diagnostic information it adds.

A note on field strength

1.5 T (tesla) is currently the standard field strength used for body imaging. 3 T scanners are becoming increasingly commonplace and with increased field strength is the potential for improved signal-to-noise ratios and resolution. Like everything else, though, there's no free lunch here. With increased field strength come a new slew of potential artifacts. Don't worry, let the physicists figure it out. The basics of performing and interpreting exams is the same.

Pearls and pitfalls

(1) TE and TR are parameters we vary on the scanner to create T1- and T2-weighted images.
(2) The T1 and T2 times of tissue are innate and don't change (except following contrast).
(3) Always use a surface coil.
(4) If you can't figure out what new, specific piece of information a given sequence adds to the exam, eliminate it.
(5) Always obtain pre-contrast images and don't vary any parameters when you do the post-contrast images.
(6) Don't give gadolinium to patients with eGFR <30 unless absolutely necessary.
(7) Don't give gadolinium to patients on dialysis.
(8) Don't give gadolinium to pregnant patients.

Chapter 2

Liver

Routine liver protocol

Indications

This protocol is used to evaluate the liver parenchyma for suspected mass, including screening for hepatocellular carcinoma, evaluation for metastatic disease, and characterization of lesions detected on other modalities (CT or ultrasound). Other common indications include elevated liver function tests and evaluation of tumor extent (i.e. multifocality).

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: none
- 2 L nasal oxygen
- At least 24-gauge IV; connect to power injector
- Cover from dome thru entire liver

Exam sequences and what we are looking for

(1) Diffusion-weighted imaging b50, 500/ADC – Very sensitive sequence for lesion detection.
(2) Coronal T2 single-shot fast-spin echo FS BH – Evaluate biliary tree. Detect T2-bright lesions.
(3) Axial T2 SSFSE BH – Identify T2-bright lesions.
(4–5) Axial T1 in- and out-of-phase (IP/OOP) – Identify geographic and microscopic focal fat.
(6) Axial T1-weighted volume-interpolated gradient echo BH pre – Identify anything that is T1-bright before contrast administration, so we don't mistake it for enhancement.
(7) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 20 s – Evaluate for hypervascular lesions.
(8) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 1 minute.
(9) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 2 minutes.
(10) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 3 minutes.

Gadoxetate disodium: liver protocol

Indications

This protocol is used for characterization of known liver lesion(s), typically to differentiate focal nodular hyperplasia (FNH) from hepatic adenoma, or to evaluate for metastatic disease. Gadoxetate disodium is a hepatocyte-specific contrast agent. It is taken up and excreted (partially) by hepatocytes.

Preparation

- **IV contrast agent**: 10 cc gadoxetate disodium at 2 cc/s
- **Oral contrast agent**: none
- 2 L nasal oxygen
- At least 24-gauge IV; connect to power injector
- Cover from dome thru entire liver.

Exam sequences

(1) Axial T1 in- and out-of-phase (IP/OOP) – Identify geographic and microscopic focal fat.
(2) Axial T1-weighted volume-interpolated gradient echo BH pre– Identify anything that is T1-bright before contrast administration, so we don't mistake it for enhancement.
(3) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 20 seconds– Evaluate for hypervascular lesions.
(4) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 1 minute.
(5) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 2 minutes.
(6) Coronal T2 single-shot fast-spin echo FS BH – Evaluate biliary tree.

(7) Axial T2 single-shot fast-spin echo BH – Identify T2-bright lesions.
(8) Diffusion-weighted imaging b50, 500/ADC – Very sensitive sequence for lesion detection.
(9) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 20 minutes – Look for dark lesions within liver. Dark signal on delayed images demonstrate lack of normal hepatocytes.
(10) Coronal T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 20 minutes.

Liver and MRCP protocol

Indications

This protocol is used to evaluate the liver and biliary tree. The difference between this protocol and the MRCP/pancreas protocol is that the post-gadolinium images cover the entire liver rather than thin slice coverage of the pancreas.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: Mixture 3 oz. ferumoxsil and 3 oz. barium sulfate 30 minutes prior to study. If 30 minutes is not available, have patient lie on right side for 5 minutes prior to study
- 2 L nasal oxygen
- At least 24-gauge IV; connect to power injector
- Cover from liver dome thru entire pancreas
- Find the best visualization of the ducts and repeat coronal haste slab 30 mm thick 15 times to differentiate between peristalsis and stricture.

Exam sequences

(1) Diffusion-weighted imaging b50, 500/ADC – Very sensitive sequence for lesion detection.
(2) Axial thin slice T2 single-shot fast-spin echo FS BH (coverage = biliary tree) – Evaluate for choledocholithiasis.
(3) Coronal thin slice T2 single-shot fast-spin echo FS BH (coverage = biliary tree) – Evaluate ductal anatomy and confirm stones.
(4) Coronal MRCP = (T2 single-shot fast-spin echo Slab 30 mm thick – repeat 15×) – Assess distal common bile duct (ampulla), nice overall images of biliary tree.
(5) Axial T1 in- and out-of-phase (IP/OOP) (coverage = liver and pancreas) – Identify focal and geographic microscopic fat.
(6) Axial T1-weighted volume-interpolated gradient echo BH pre (coverage = liver and pancreas).
(7) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 20 seconds.
(8) Axial T1-weighted volume-interpolated gradient echo post IV administration of contrast at 1 minute.
(9) Axial T1-weighted volume-interpolated gradient echo post IV administration of contrast at 2 minutes.
(10) Axial T1-weighted volume-interpolated gradient echo post IV administration of contrast at 3 minutes.

Hemochromatosis: liver protocol

Indications

This protocol is used to quantify iron deposition within the liver. Iron causes local fixed inhomogeneities which result in signal loss on both T1- and T2-weighted images (the liver is dark). By using the specialized sequences below, the amount of iron can be quantified.

Preparation

- IV contrast agent: none
- Give patient 2 L nasal oxygen
- No need to cover entire liver: nine slices placed through thickest part of the liver
- Scanned using *body coil*.

Exam sequences

(1) Diffusion-weighted imaging b50, 500/ADC – Very sensitive sequence for lesion detection.
(2) T1 axial 2D spoiled gradient echo (FLASH) TR 120 ms – TE 4 ms – 90° flip angle.
(3) Proton density axial TR 120 ms – TE 4 ms 20° flip angle.
(4) T2 axial TR 120 ms – TE 9 ms 20° flip angle.
(5) T2* axial TR 120 ms – TE 14 ms 20° flip angle.
(6) T2* axial TR 120 ms – TE 21 ms 20° flip angle.

Post processing

- For calculation of iron content we use the website listed below. Enter the magnet strength and respective values:

  www.radio.univ-rennes1.fr/Sources/EN/HemoCalc15.html

Approach to liver MRI exam interpretation

Liver MRI is the most commonly performed body MRI exam. It is the most sensitive and specific non-invasive radiologic examination for the detection and characterization of liver lesions. As in all body MRI exams, each sequence is designed and performed to add a specific piece of information.

Do not be overwhelmed. Do not fear. All we need to know is: what are we looking for on each sequence?

Coronal and axial single-shot fast-spin echo T2-weighted images

Coronal performed with fat saturation, axial without. This decision is arbitrary. But it's nice to have the axial without fat saturation for motion-free depiction of abdominal anatomy.

T2-weighted images are fluid-sensitive – that is, fluid is bright. The advantage of using SSFSE instead of fast-spin echo (FSE) is time. SSFSE takes far less time than FSE which enables imaging of the abdomen in a single breath-hold. Also, because it is faster, it is less prone to motion artifact. Many institutions still use FSE. We quit.

Like all things in MRI, there's no free lunch here. The images are significantly less T2-weighted than FSE. They have been described as a "tumor-hiding" sequence for the liver. So why use them?

We are not using them to detect tumors. We use the gadolinium-enhanced images and the DWI to detect tumors. We use the SSFSEs to help characterize lesions and hopefully help prove that they are benign. For this purpose they are good enough . . . and much quicker.

Cysts are the most commonly encountered liver lesions. They are simply sacs of fluid and so should be light-bulb bright on T2-weighted images. So are benign biliary hamartomas (also known as von Meyenburg complexes) and most hemangiomas. *Tumors, whether primary liver tumors or metastases, will not be as bright*. They may be bright, but not that bright. Remember, no single sequence characterizes any lesion on MRI.

Cysts will be as bright as CSF on T2-weighted images and will not enhance. Biliary hamartomas will be as bright as cysts and also do not enhance, but are typically small and scattered throughout the liver. So how do we definitively differentiate numerous cysts from biliary hamartomas? We don't. They're both benign so who cares?

If there are a few lesions of differentiated sizes, I call them cysts. If there are innumerable lesions of approximately the same size, I call them biliary hamartomas. (Also, cysts are *extremely* common and biliary hamartomas are not.)

Biliary hamartomas are often reported to have subtle, peripheral, continuous enhancement which is thought to be due to compression of the adjacent liver parenchyma, not true enhancement of the hamartoma itself. The key is that small lesions that are exquisitely T2-bright, and non-enhancing, are always benign (Figures 2.1–2.2).

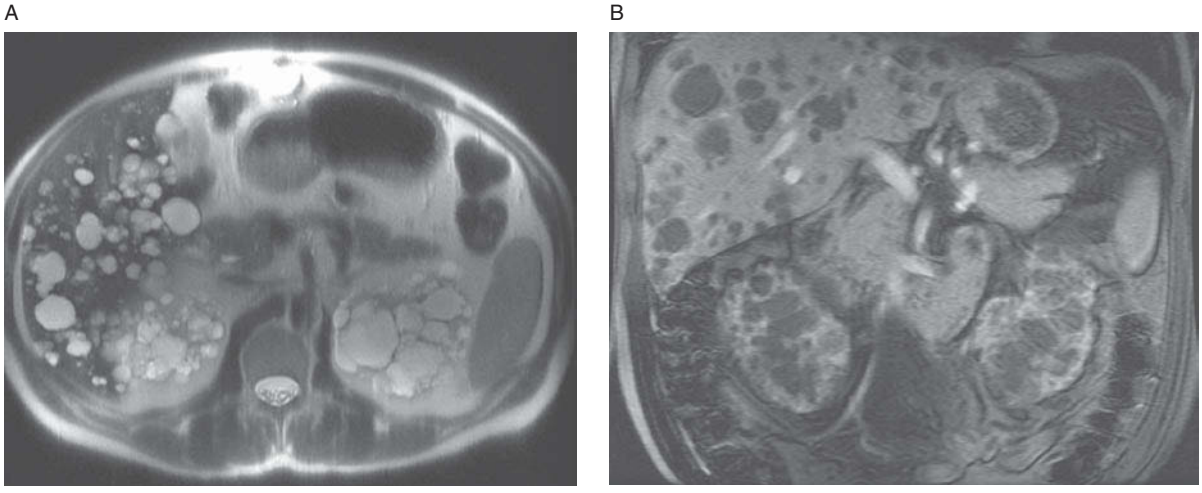

Figure 2.1. Liver cysts. Axial T2-weighted image (A) demonstrates numerous T2-bright lesions of various sizes within the liver and kidneys. Coronal, T1-weighted, fat-saturated contrast-enhanced image (B) demonstrates no evidence of enhancement. This patient has polycystic disease involving the liver and kidneys.

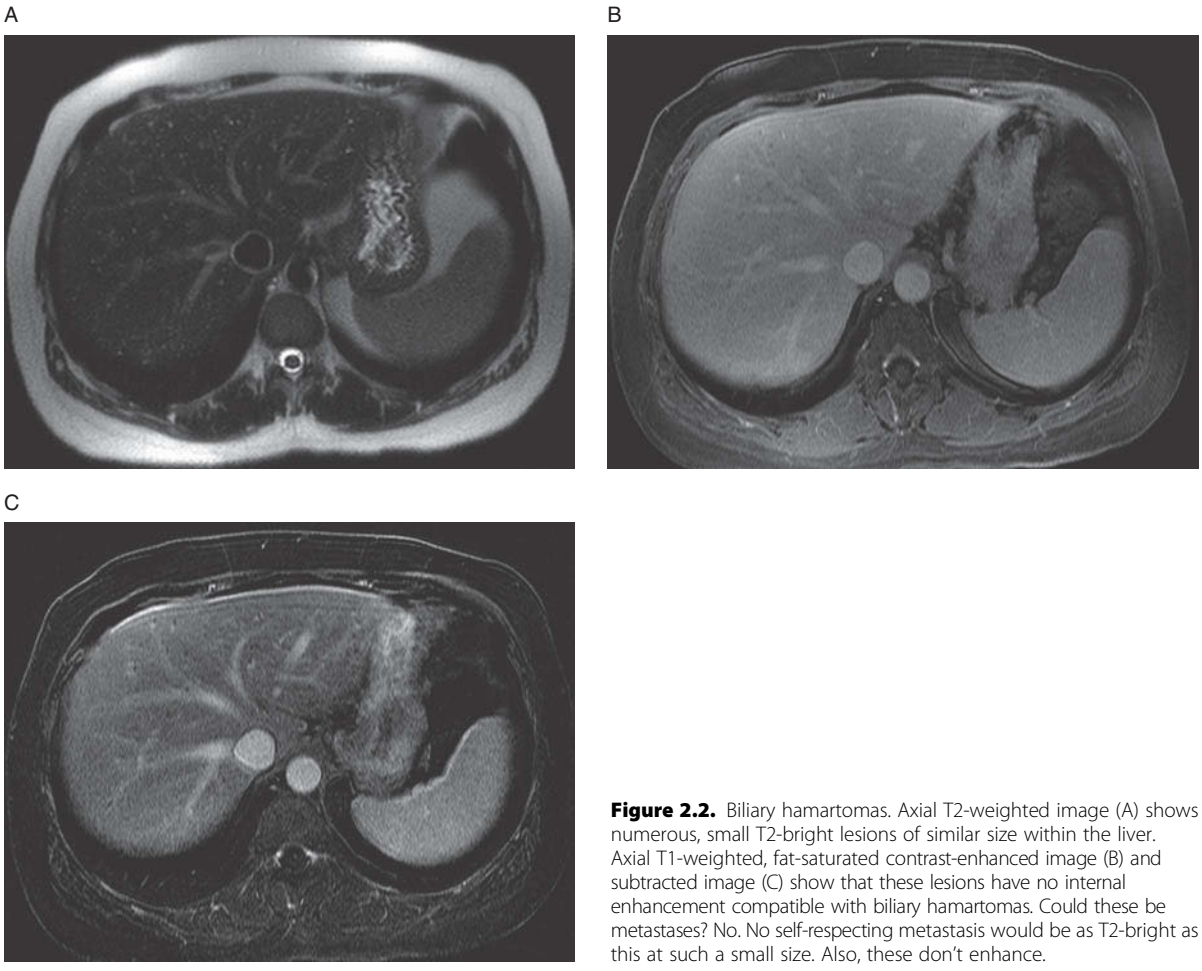

Figure 2.2. Biliary hamartomas. Axial T2-weighted image (A) shows numerous, small T2-bright lesions of similar size within the liver. Axial T1-weighted, fat-saturated contrast-enhanced image (B) and subtracted image (C) show that these lesions have no internal enhancement compatible with biliary hamartomas. Could these be metastases? No. No self-respecting metastasis would be as T2-bright as this at such a small size. Also, these don't enhance.

Hemangiomas are almost always very T2-bright as well, nearly as bright as CSF. The key to making the definitive diagnosis of a hemangioma is its enhancement pattern. Classic or typical hemangiomas demonstrate *peripheral, nodular, discontinuous enhancement* (Figure 2.3). They will (usually) progressively fill in with contrast over time, however *don't hang your hat on this*. Fill-in can be quite variable and some tumors can appear to fill in as well. Hang your hat on the peripheral, nodular, discontinuous enhancement.

For practical purposes, there are two types of atypical hemangiomas. The more common type are small lesions, typically <1 cm, which are nearly as bright as CSF on T2 but homogeneously fill with contrast on arterial-phase imaging. These are referred to as "flash-filling" hemangiomas. To be certain of the diagnosis, check the portal-venous and delayed phases. The enhancement of these lesions should follow blood pool on all phases, not wash out (Figure 2.4). Hemangiomas *do not* wash out. Washout is bad.

The second and less common type are thrombosed/sclerosed hemangiomas. *Do not make a definitive diagnosis of a thrombosed hemangioma on MRI*. If a lesion does not meet the criteria above for definitive diagnosis of a hemangioma (either typical or flash-filling) it should be followed or biopsied depending on the clinical history.

Thus, cysts and biliary hamartomas are very bright on T2-weighted images and do not enhance. Hemangiomas greater than 1 cm are T2-bright and demonstrate peripheral, nodular, discontinuous enhancement. If it does not, *do not call it a hemangioma*. This is one situation where you must follow the rules or suffer the consequences (Figure 2.5). Atypical

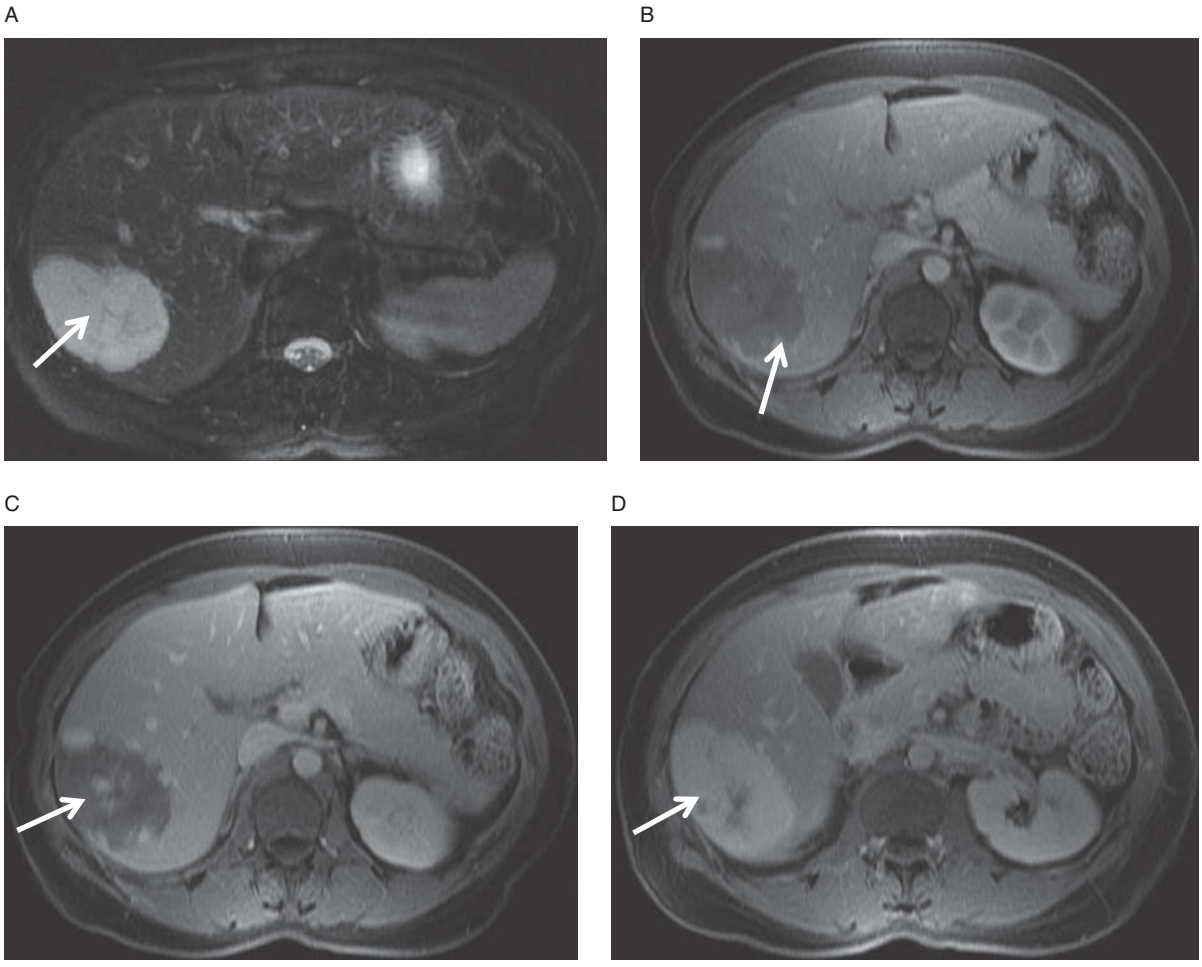

Figure 2.3. Classic hemangioma. Axial, fat-suppressed T2-weighted image (A) shows markedly bright, lobulated liver lesion. Axial, T1-weighted fat-saturated post-contrast enhanced images obtained in the arterial phase (B), portal venous phase (C), and delayed phase (D) demonstrate peripheral, nodular, discontinuous enhancement with progressive fill-in of contrast over time. (See arrows).

hemangiomas are less than 1 cm, flash-fill with contrast, and follow blood pool on all subsequent phases.

In-/out-of-phase (IP/OOP) chemical-shift images; T1-weighted images, no fat saturation

As discussed in greater detail in the introduction, if a voxel contains both protons bound to water and protons bound to fat, it will become dark on out-of-phase images compared to its signal on in-phase images. That is, *microscopic fat loses signal on out-of-phase images*. Out-of-phase images can be recognized by the "etching" or "India-ink" artifact surrounding the solid organs, where the voxels contain both protons bound to water (the solid organ) and protons bound to fat (the surrounding, peritoneal fat). (If there's any confusion, simply check the TE. An out-of-phase image will have a TE of approximately 2.2 and an in-phase-image will have a TE of approximately 4.4 at 1.5 Tesla.)

Chemical-shift images are most commonly used to make the diagnosis of hepatic steatosis and to differentiate focal fatty infiltration or focal fatty sparing from worrisome lesions. In patients with hepatic steatosis, the liver will be brighter than the spleen on the in-phase image and lose signal compared to the spleen on the out-of-phase images. Classically, focal fatty sparing and focal fatty infiltration are geographic and occur in the liver adjacent to the falciform ligament and the gall-bladder fossa (Figure 2.6). However, in reality, they can occur anywhere and can even be somewhat nodular (Figure 2.7).

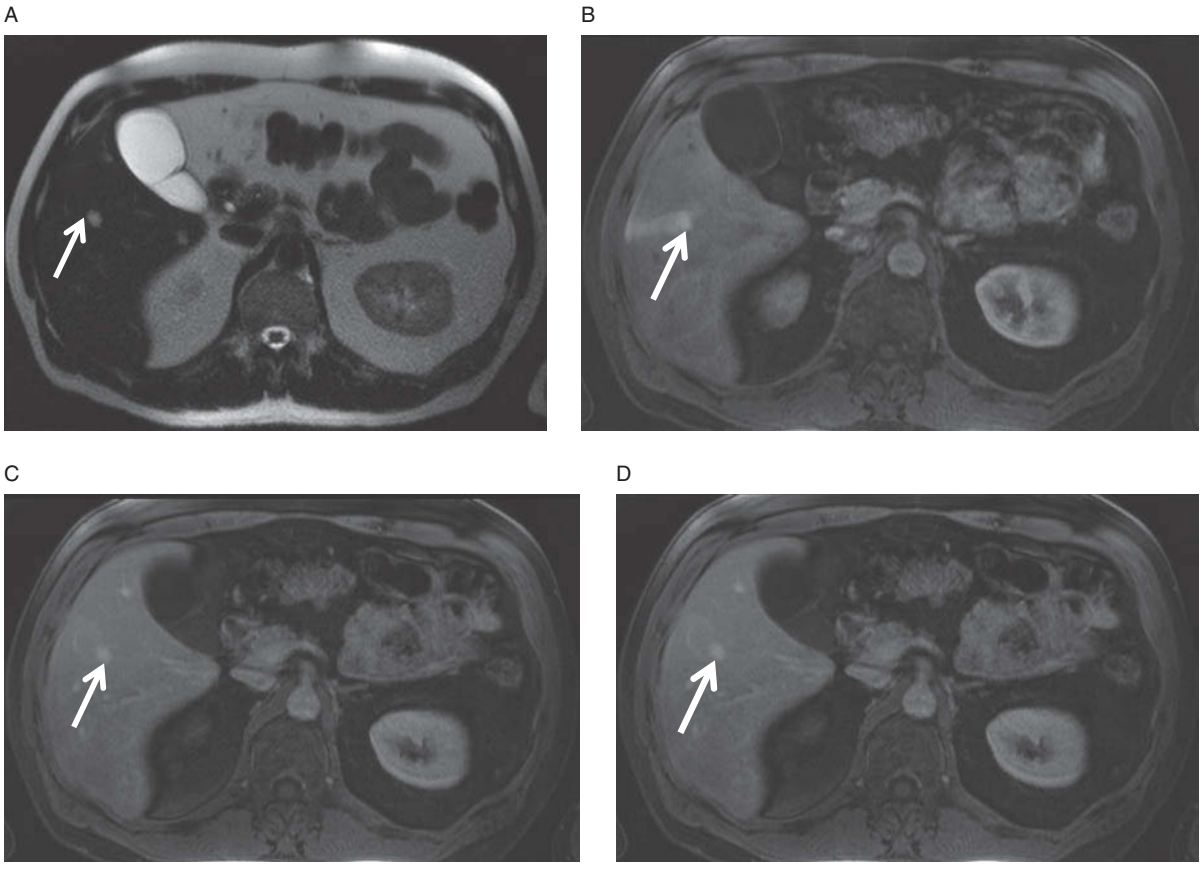

Figure 2.4. Atypical, flash-filling hemangioma. Axial T2-weighted image (A) demonstrates a very bright lesion within the liver measuring less than 1 cm. Axial, T1-weighted, fat-saturated arterial-phase image (B) demonstrates immediate, homogeneous enhancement of the lesion which follows the enhancement of the blood pool and portal venous (C) and delayed images (D). The vascular changes at the periphery of the lesion (seen best on image B) are common in atypical hemangiomas. Most importantly, the lesion follows blood pool on successive phases.

If a focal lesion loses signal on out-of-phase images, we know that it histologically contains microscopic fat. Most liver lesions that contain fat are benign. Not all (rare hepatocellular carcinomas can contain fat), that would be too easy, but most. Classically, hepatic adenomas contain some fat (Figure 2.8).

The presence of **macroscopic** fat can be assessed by comparing the in-phase images (T1-weighted without fat saturation) to the pre-contrast images (T1-weighted images with fat saturation). The astute observer will notice that livers which are chock-full of microscopic fat, and are very dark on the out-of-phase images, will sometimes contain enough microscopic fat to actually lose signal on the fat-saturated images. This is probably due both to the fat-saturation pulse and to the fact that these images are usually performed with an out-of-phase TE (to save time).

As we discussed in the introduction, just as intravoxel fat will cause signal loss on out-of-phase images, iron deposition will cause signal loss on in-phase images. Iron deposition acts like having small magnets within the overall magnetic field of the MRI scanner and causes inhomogeneity. The longer we wait after the TR to "listen" for our TE, the greater the effect of this inhomogeneity. Essentially, the longer we wait, the more signal we lose. No signal = black voxel.

We can detect iron deposition by comparing routine out-of-phase and in-phase images. But, if we want to actually quantify it, we can use our hemochromatosis protocol which consists of sequences with varying TEs. We measure the signal loss by drawing multiple regions of interest. Entering these values into a formula (easily found on the web) we can then calculate liver iron content (we do this *very* rarely).

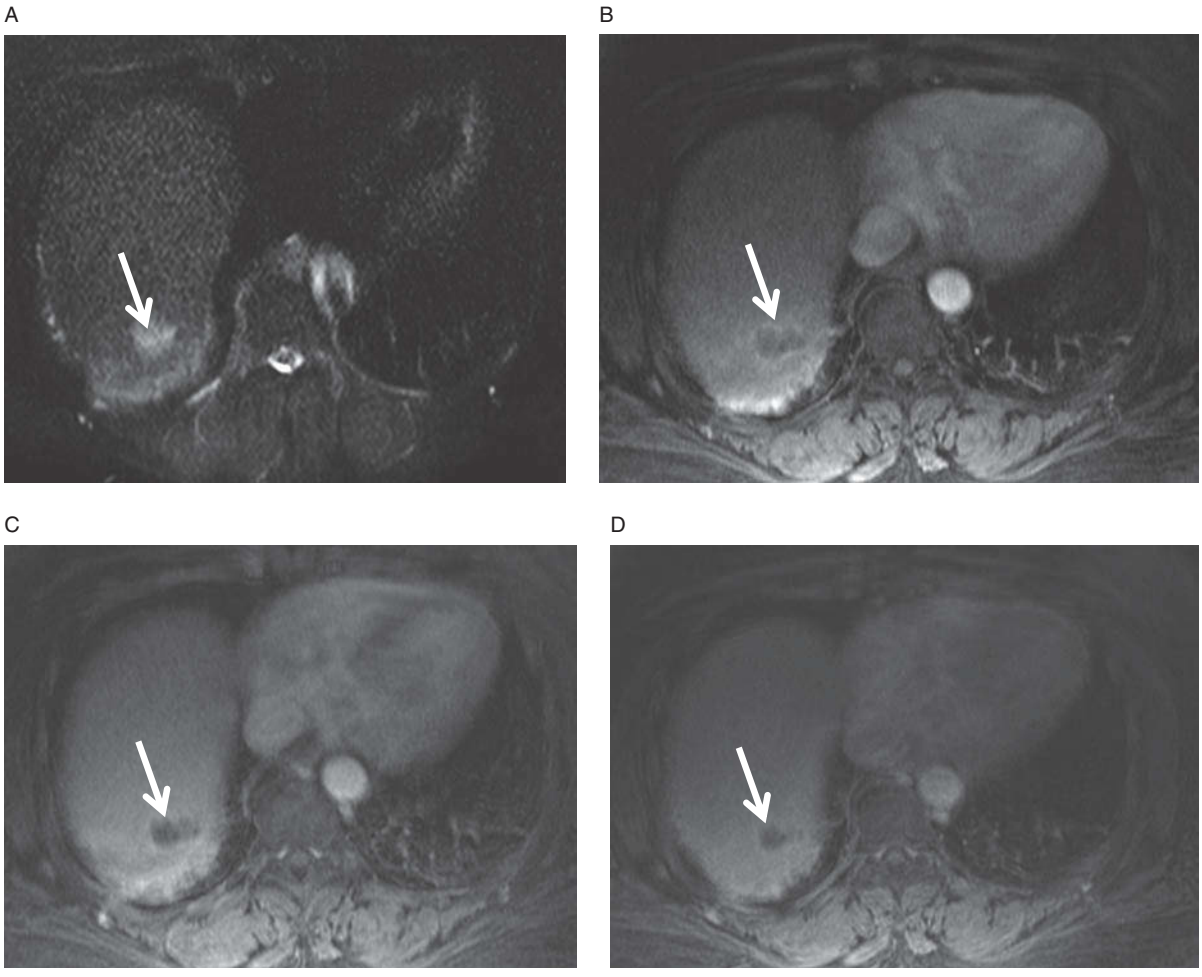

Figure 2.5. Is this a hemangioma? Axial fat-suppressed, T2-weighted images demonstrate a bright liver lesion (A). Axial, T1-weighted, fat-saturated arterial-phase image (B) demonstrates peripheral enhancement which appears to progressively fill in on delayed images (C, D). This is *not* a hemangioma. The arterial phase enhancement is not nodular or discontinuous, it is a complete ring. This is a biopsy-proven hepatocellular carcinoma (HCC). (See arrows).

Diffusion-weighted imaging and apparent diffusion coefficient

Diffusion-weighted imaging (DWI) is an exciting, relatively new area of body MRI which attempts to bridge anatomic and functional imaging. DWI measures and displays the ability of water molecules to diffuse through tissue. The apparent diffusion coefficient (ADC) map is a plot of the apparent diffusion coefficient. Normal tissues consist predominantly of interstitial or extracellular space through which water can easily diffuse. Tumors are hypercellular when compared with normal tissues and this increased number and concentration of cells decreases the amount of free interstitial space for water to diffuse through easily, replacing it with intracellular space which creates boundaries and obstacles to diffusion. This results in restricted diffusion which is displayed as bright on DWI and dark on the ADC map. (For more detail, but not too much more detail, see Introduction.)

Fundamentally, conventionally acquired DWI are T2-weighted – it is therefore critical to correlate DWI findings with the ADC map. Lesions that are bright on DWI but also bright on ADC do *not* represent true restricted diffusion, but "T2 shine-through."

Currently, the best use for DWI is lesion detection. DWI is now considered by many to be the most sensitive sequence for the detection of liver lesions (which may simply be due to their extreme T2-weighting) (Figure 2.13). It is very tempting to use DWI to characterize liver

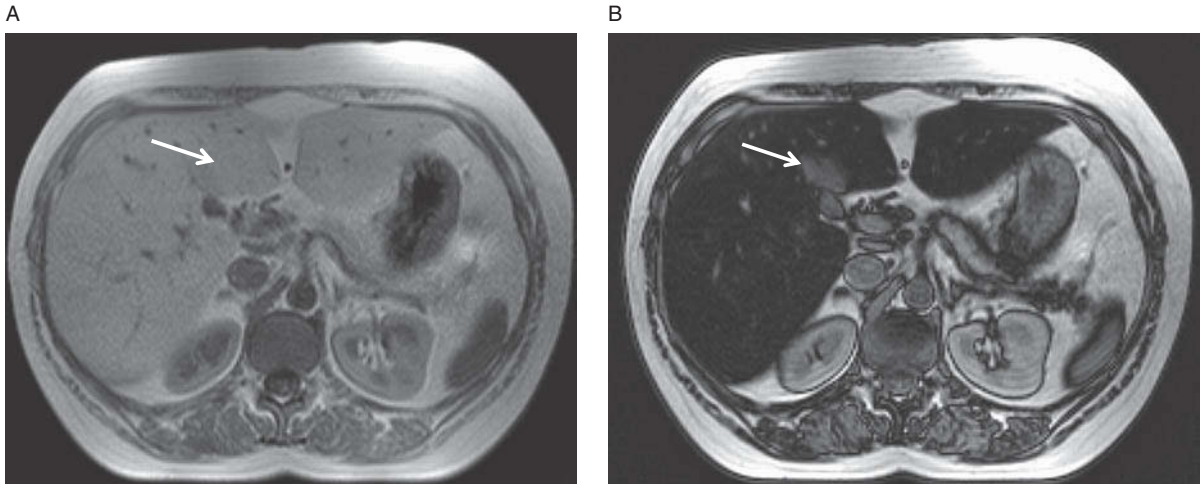

Figure 2.6. Focal fatty sparing. Axial, T1-weighted, fat-saturated in-phase (A) and out-of-phase (B) images demonstrate marked loss in hepatic signal compared to the spleen compatible with hepatic steatosis. Arrows indicate geographic area of bright signal (B) compatible with focal fatty sparing.

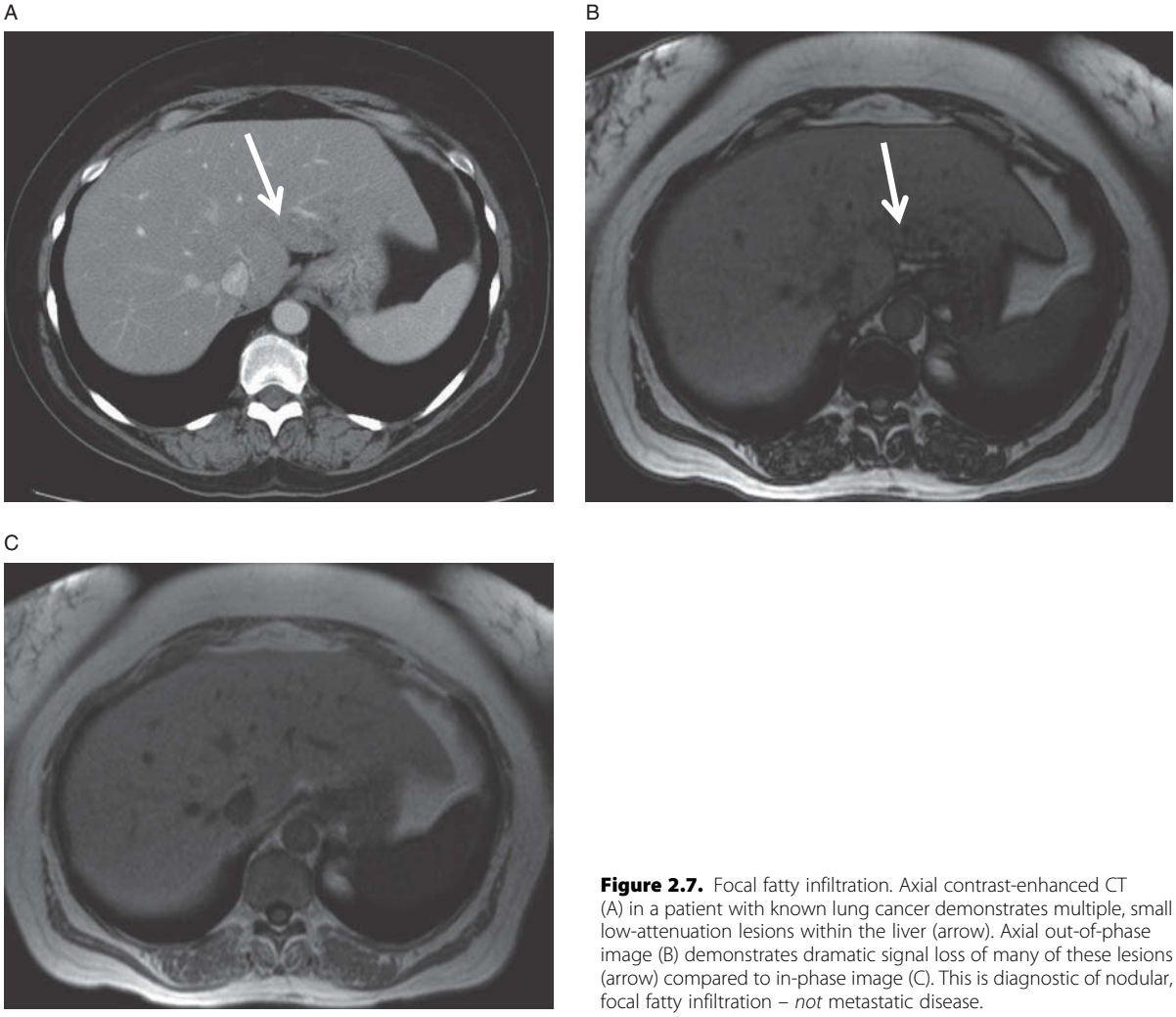

Figure 2.7. Focal fatty infiltration. Axial contrast-enhanced CT (A) in a patient with known lung cancer demonstrates multiple, small low-attenuation lesions within the liver (arrow). Axial out-of-phase image (B) demonstrates dramatic signal loss of many of these lesions (arrow) compared to in-phase image (C). This is diagnostic of nodular, focal fatty infiltration – *not* metastatic disease.

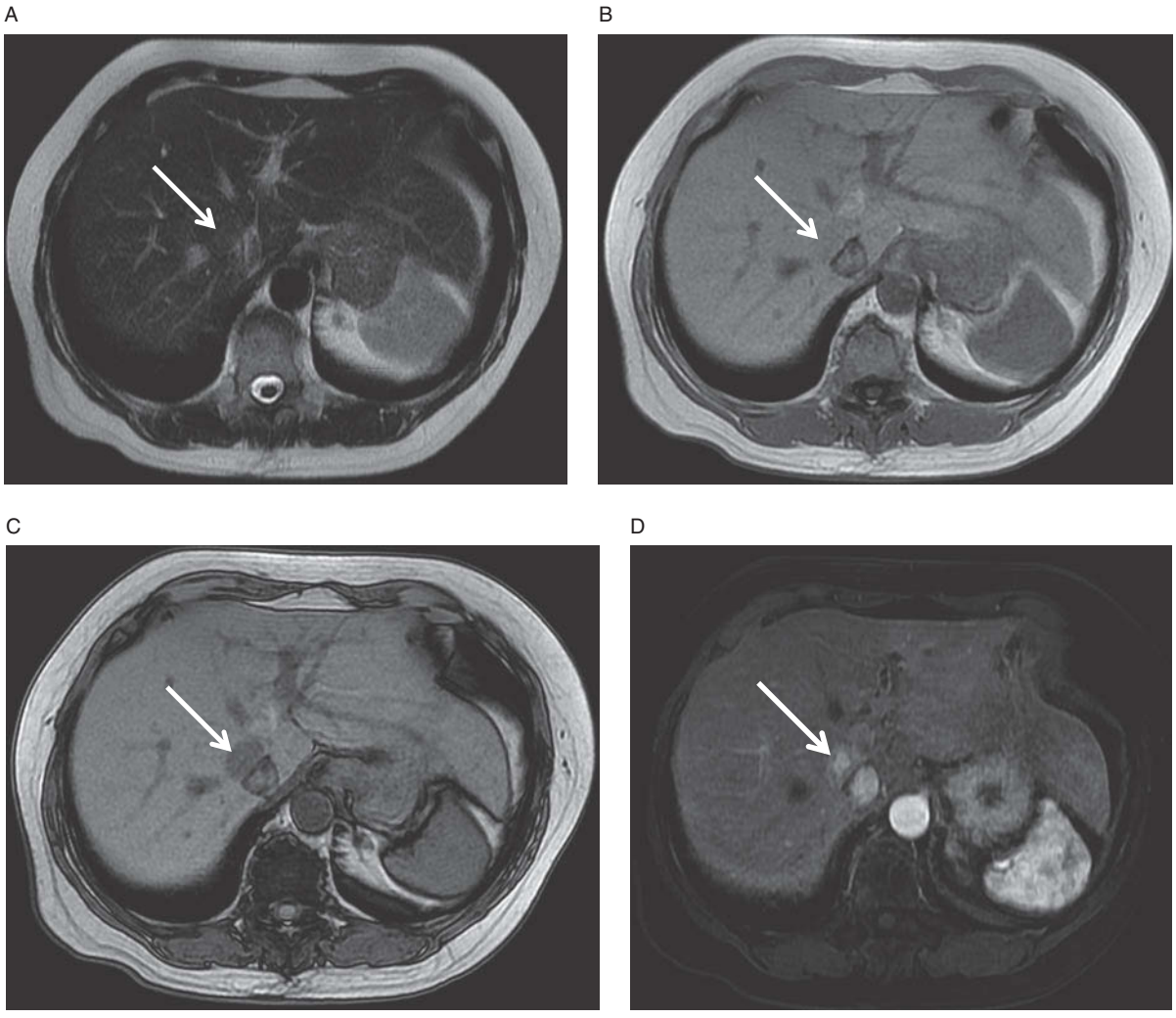

Figure 2.8. Hepatic adenoma. Lesion is not prospectively discernible on axial T2-weighted image (A). Most importantly, the lesion demonstrates marked signal loss on out-of-phase image (C) compared to in-phase image (B). Given that it is also hypervascular on arterial-phase image (D), it is most likely an adenoma. (See arrows).

lesions; however, this should be approached with caution. Unfortunately, benign and malignant lesions can demonstrate significant overlap in ADC. For example, focal nodular hyperplasia (FNH), a benign lesion, can demonstrate restricted diffusion. DWI should therefore be viewed as one tool in our armamentarium – excellent for lesion detection, and one of many tools which should be evaluated together for lesion characterization.

Contrast-enhanced images: T1-weighted volume-interpolated gradient echo

Remember that gadolinium agents shorten the T1 time of tissues, which makes them brighter on T1-weighted images. To accentuate this change in signal we always use fat saturation on pre- and post-contrast images (if the fat is dark, the bright, enhancing things stand out). Volume interpolated gradient echo sequences apply chemically selective fat saturation.

And what do gadolinium agents do to T2-weighted images? Nothing. Nada. Bobkus. They work by changing T1.

The ability to routinely acquire pre-contrast (and dynamic post-contrast) imaging is one of the advantages of MRI compared to CT. Obviously, we can obtain multiple phases in all patients on MRI because of the lack of ionizing radiation. But what do we look for on the pre-

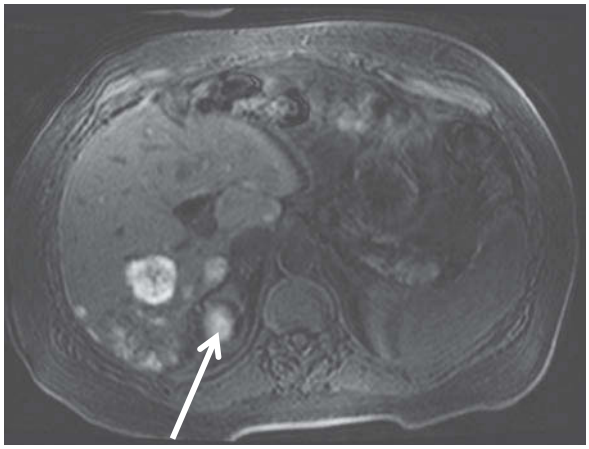

Figure 2.9. Melanoma metastases. Axial T1-weighted fat-saturated image obtained before administration of contrast demonstrate multiple T1-bright liver lesions compatible with metastatic melanoma. Arrow denotes additional metastatic lesion in right adrenal gland.

contrast images? Essentially, we use the pre-contrast images to make sure that lesions which are bright on the post-contrast images are truly enhancing and not just T1-bright.

As we discussed in the Introduction, the list of things which are T1-bright is delightfully short and when we're talking about liver lesions we're really only thinking of two of them:

(1) *Melanoma* Melanin is bright on T1 so melanoma metastases can be too (Figure 2.9).
(2) *Blood* There are essentially two liver lesions that bleed: hepatocellular carcinoma (HCC) and adenoma. More on this later. (Flowing blood can also be variably T1-bright.)

There are essentially two types of contrast agents currently available in the US for liver MRI. Both are chelates of gadolinium. They can broadly be considered:

(1) *Extracellular non-specific* Most commonly used. They wash in, they wash out, tissues enhance in much the same fashion as they do on CT. They are excreted by the kidneys. (This is why we no longer give them to people with renal failure.)
(2) *Hepatocyte-specific* Newer agents. Technically, both Gd-BOPTA (MultiHance) and gadoxetate disodium (Eovist) are hepatocyte-specific. They are both taken up, held onto, and excreted by normal hepatocytes. However, the degree of hepatocyte uptake (and excretion) is much higher with gadoxetate disodium, thus this agent is preferred for hepatocyte-specific imaging purposes.

IV contrast is a critical portion of liver MRI and should always be administered unless there is an eGFR issue or history of anaphylaxis. True anaphylaxis to gadolinium is very, very rare. Our current policy is that if the patient has a history of less severe allergy to a gadolinium agent, such as hives, we will simply administer a different gadolinium agent.

Post-contrast imaging is the gold standard for lesion detection. (DWI may be as sensitive, but this remains controversial.) It is also critical for characterizing lesions. For simplicity's sake, we can consider contrast enhancement in broad categories:

(1) *No enhancement* Liver cysts, biliary hamartomas = benign lesions.
(2) *Peripheral, nodular discontinuous enhancement* = Hemangioma.
(3) *Hypervascular lesions* These are defined as enhancing brighter than the liver parenchyma on the arterial phase. They include:
    (a) Atypical hemangiomas
    (b) Focal nodular hyperplasia
    (c) Adenoma
    (d) Hepatocellular carcinoma
    (e) Hypervascular metastases such as neuroendocrine tumors.
(4) *Hypovascular lesions* These lesions enhance, but less than the liver parenchyma and are typically best seen on the portal-venous phase. They include:
    (a) Hypovascular metastases
    (b) Cholangiocarcinoma
    (c) Liver abscess.

We've already discussed non-enhancing lesions and hemangiomas. So let's jump into hypervascular masses. A well-timed arterial phase is critical for detection of these tumors. How do we know if an arterial phase is technically adequate (i.e., well timed)? *On a perfect arterial phase image, the portal vein may enhance a bit but the hepatic veins do not*. (Figure 2.10).

Let's further divide hypervascular liver lesions into two categories:

(1) *Benign*: Hemangioma, focal nodular hyperplasia, adenoma
(2) *Malignant*: Hepatocellular carcinoma, hypervascular metastases.

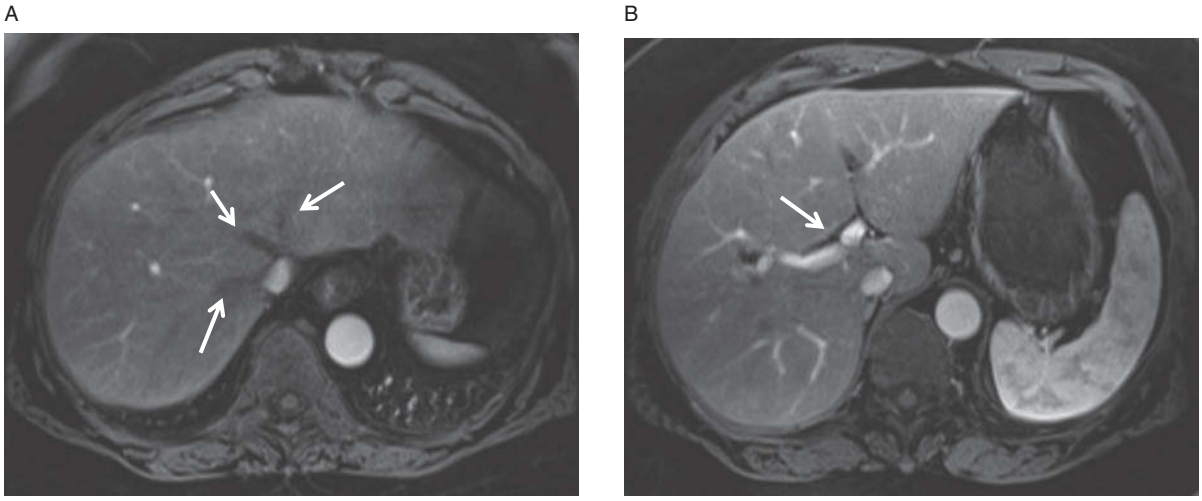

Figure 2.10. Axial T1-weighted post-contrast arterial phase images demonstrate lack of contrast enhancement of the hepatic veins (arrows A) with some enhancement of the portal vein (arrow B). Note also the heterogeneous enhancement of the spleen which is typical of an arterial phase image.

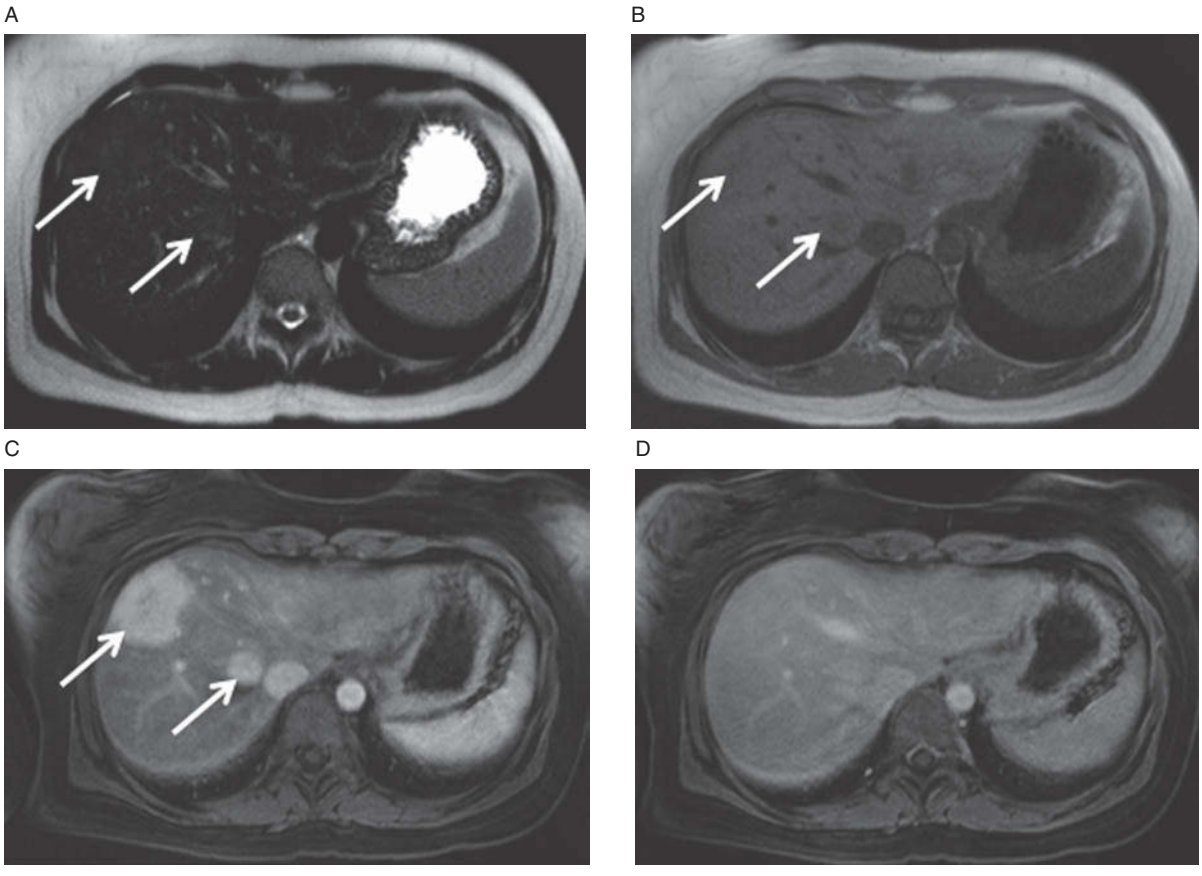

Figure 2.11. Multiple focal nodular hyperplasias (FNHs). T2-weighted image (A) and T1-weighted image (B) demonstrate two, barely perceptible lesions within the liver (arrows). These lesions enhance avidly on arterial-phase T1-weighted, fat-saturated image (arrows, C) and are imperceptible on delayed image (D).

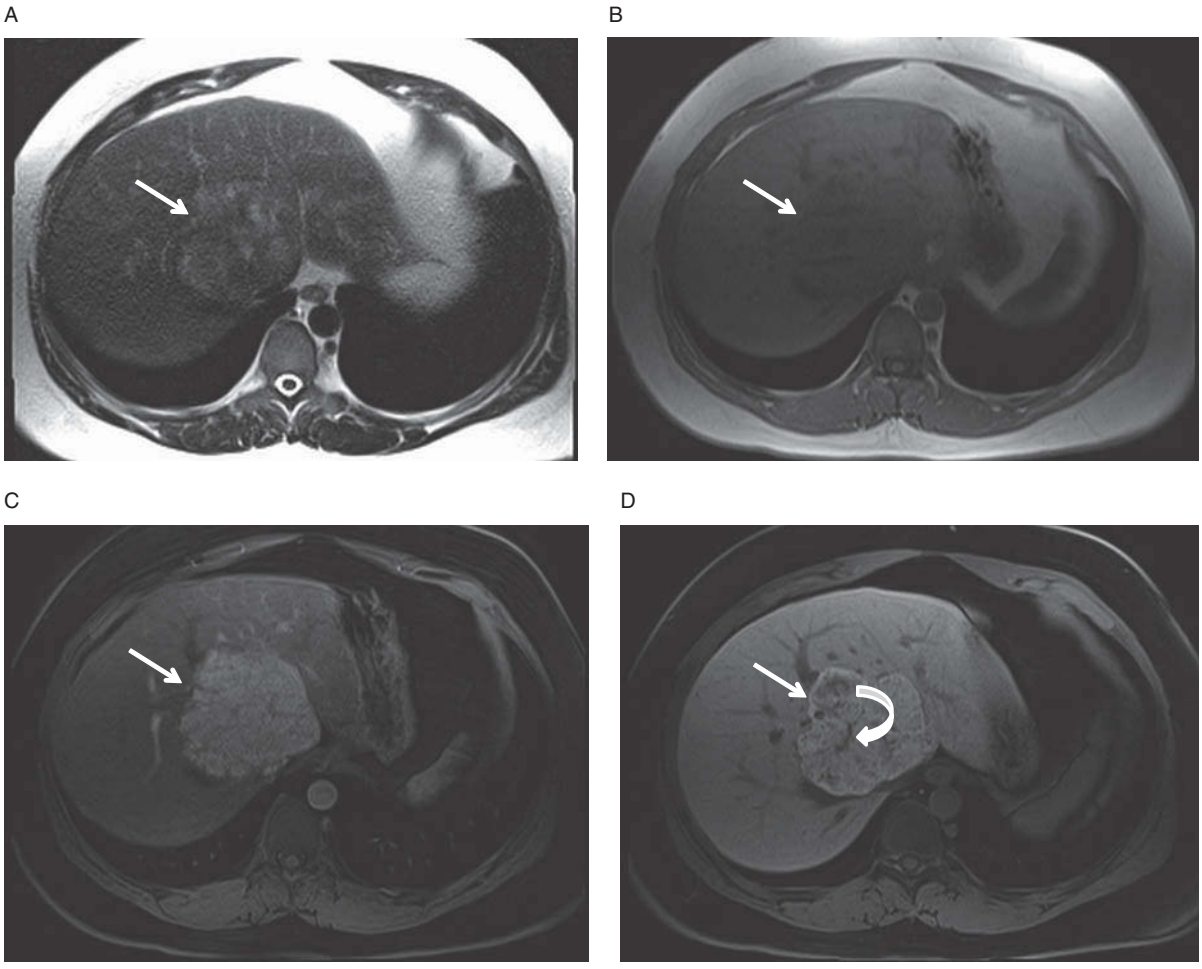

Figure 2.12. Focal nodular hyperplasia (FNH), hepatocyte-specific contrast agent gadoxetate disodium. Lesion slightly hyperintense on T2-weighted image (A), and is slightly hypointense to liver on T1-weighted image (B, arrow). The lesion enhances briskly on arterial-phase T1-weighted, fat-saturated image (C) and retains contrast material on 20-minute delayed image (D). Notice the often-discussed but rarely seen central scar (curved arrow.)

Benign hypervascular lesions

First, the benign lesions. It's always gratifying when we can prove a lesion is benign, sparing the patient a liver biopsy and diagnosis of a life-threatening disease. We've already discussed hemangiomas. Bright on T2, peripheral, nodular, discontinuous enhancement.

Focal nodular hyperplasia (FNH)

We alluded to FNH in the introductory section on hepatocyte-specific contrast agents. FNH is extremely common, often multiple, and always benign. We see more and more of them every year because we are imaging more people, performing more multi-phasic exams, and, because modern CT scanners are so fast, we often image in a "more" arterial phase than we used to.

Focal nodular hyperplasia pathologically is a disorganized jumble of normal hepatic tissue. This is why they look almost exactly like normal liver on most pulse sequences. It does not explain why they are hypervascular. They are . . . I don't know why.

A classic, well-behaved FNH is *slightly* darker than the liver parenchyma on T1, *slightly* brighter than the liver on T2, enhances avidly on the arterial phase, and blends in with the liver on the portal-venous and delayed phases. If a lesion has these features, I call it an FNH (Figure 2.11).

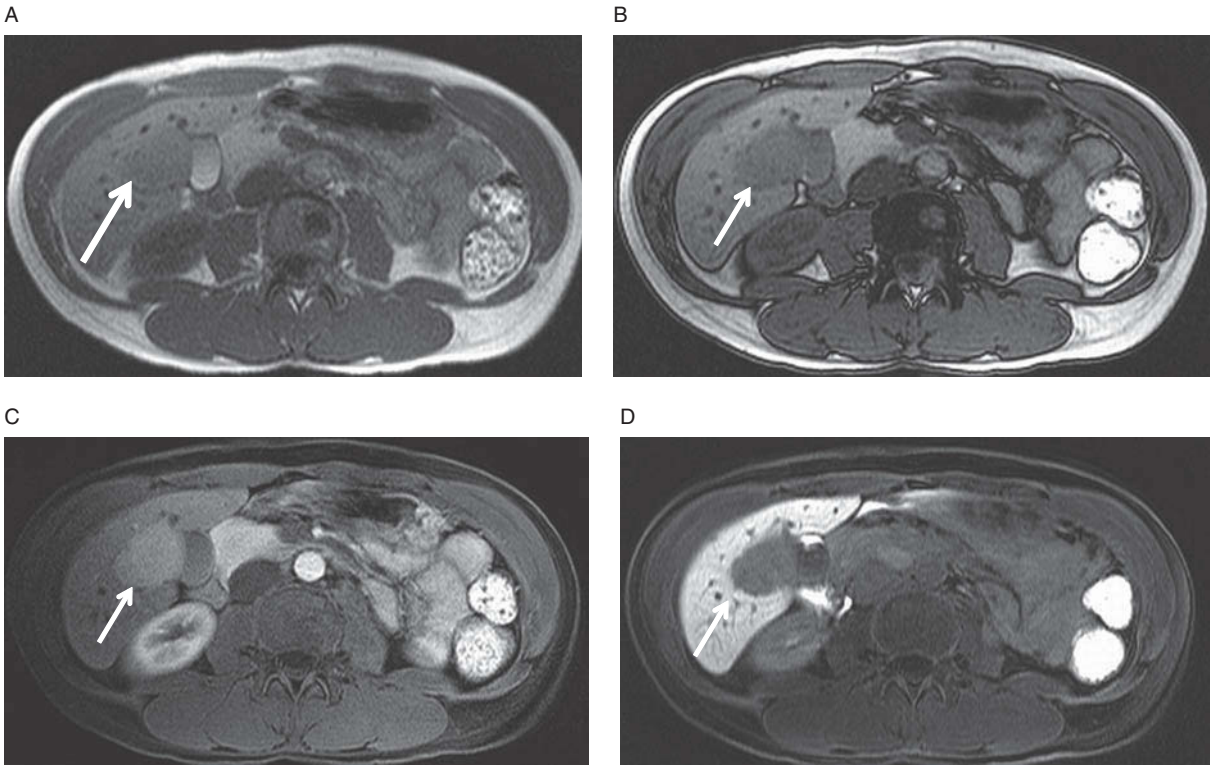

Figure 2.13. Hepatic adenoma. Hypointense liver lesion (arrows) does not significantly change in signal from in-phase image (A) to out-of-phase image (B). Gadoxetate disodium was administered IV. The lesion avidly enhances on arterial phase T1-weighted, fat-saturated post-contrast image (arrow, C), but completely washes-out on 20 minute delayed image (D). FNH would retain the contrast.

(In the course of daily work, finding FNHs typically goes as follows: I read the T2s and don't see anything. I look at the in- and out-of-phase images and don't see anything. Pre-contrast . . . you guessed it . . . nothing. Then comes the arterial phase and there is a lesion as bright as the sun which blends in on the portal-venous and delayed phases. I only see the "subtle" T1 and T2 changes when I go back.)

If a lesion fits the description above, it is an FNH. End of story. But, if for any reason the appearance is atypical or the patient is at high risk for developing a hypervascular malignancy or an adenoma, a hepatocyte-specific contrast agent (gadoxetate disodium) may be administered. With this agent, the arterial phase should still demonstrate brisk enhancement and a 20-minute delayed image should show the lesion to be isointense or hyperintense to the liver parenchyma (Figure 2.12). It is postulated that the lesion holds onto contrast because its normal hepatocytes take the agent up, but its blind-ending ducts cannot excrete it.

Hepatic adenoma

Hepatic adenomas are considered benign, epithelioid lesions of the liver. They occur in women on oral contraceptives, people on steroids and patients with glycogen-storage diseases. They may be multiple.

Classically, they are heterogeneous, and may contain fat (Figure 2.13) and even blood. Unfortunately, not all hepatic adenomas contain fat or blood and when they don't they can be difficult to distinguish from FNH. Here are a few things to keep in mind:

(1) FNH is very common. Hepatic adenoma is pretty rare. If you call all questionable lesions FNH, you will be right far more often than not.
(2) Hepatic adenomas typically occur in young women on oral contraceptives. Adenoma is pretty rare. If the patient isn't female and on oral contraceptives, it's even rarer.
(3) If you're still not sure, and a definitive diagnosis is necessary, use gadoxetate disodium. On the 20-minute delayed image, an adenoma should wash out.

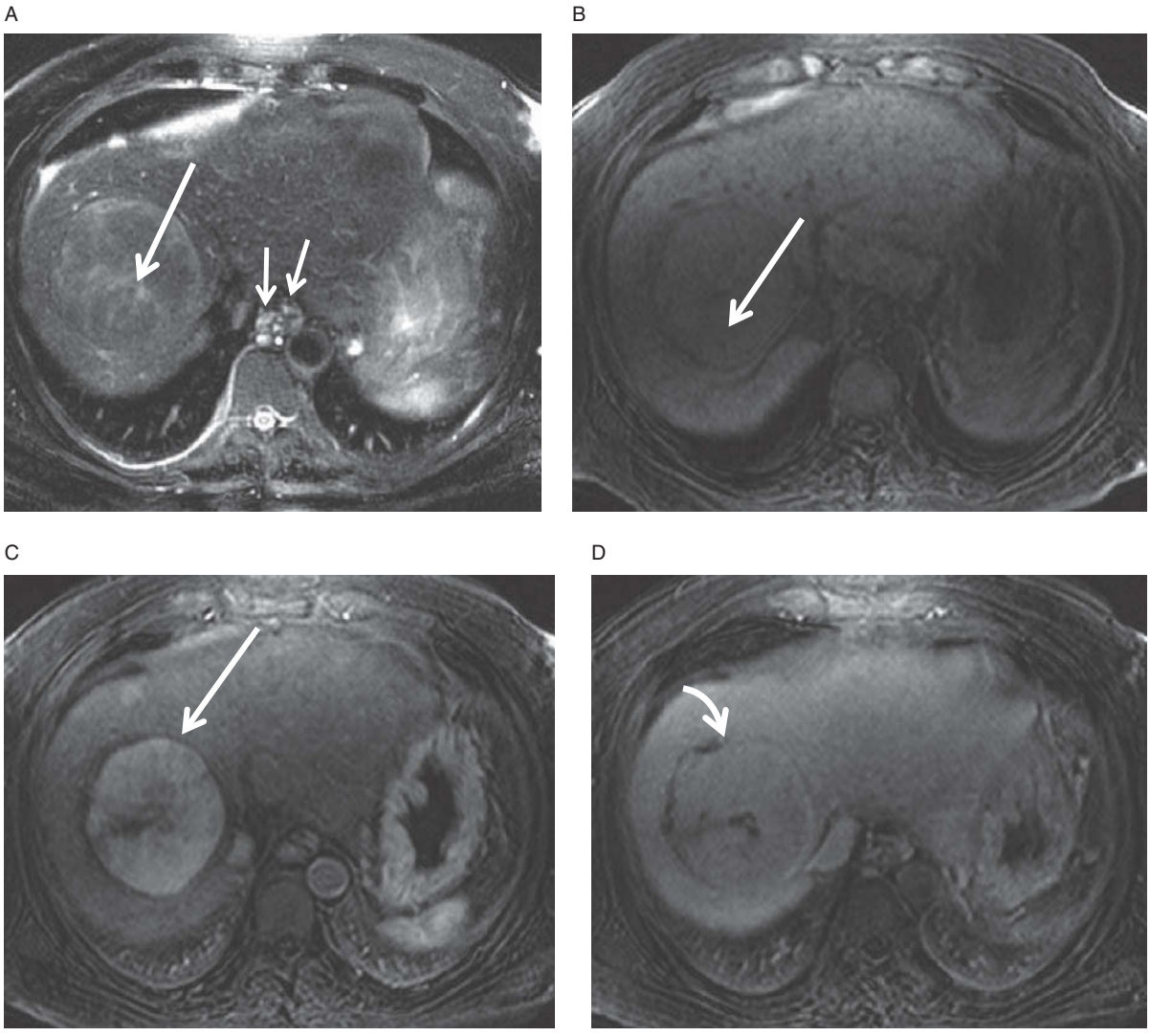

Figure 2.14. Well-encapsulated HCC. Axial T2-weighted image (A) shows mildly increased signal in a large hepatic mass (arrow). Axial, T1-weighted, fat-saturated pre-contrast image (B) shows nearly hypointense mass which demonstrates marked arterial-phase enhancement (C) and slight washout on delayed image (D). If you are thinking, wait a minute, couldn't this be an FNH with a central scar . . . look again. It's too heterogenous on T2. Also, notice the washout of the capsule on the delayed image (D) (curved arrow). Also, see T2-bright esophageal varices on image (double arrow) A. All of these features make this an HCC.

Malignant hypervascular lesions

Liver MRI is the most sensitive and specific examination for detection of hepatocellular carcinoma (HCC). HCC most commonly occurs in patients with cirrhosis which is, unfortunately, very common. The two most common causes are alcoholic cirrhosis and cirrhosis secondary to hepatitis B or C infection.

Cirrhosis is progressive fibrosis of the liver. As the normal liver undergoes fibrosis, the body attempts to make new, functioning liver, forming regenerative nodules. These regenerative nodules can become dysplastic nodules which are considered to be precursor lesions of HCC. (This is analogous to the progression of colon polyps.)

Textbooks and the literature spend a great deal of time discussing distinguishing between regenerative nodules, dysplastic nodules, and HCC. For our purposes, it boils down to this: we cannot always tell.

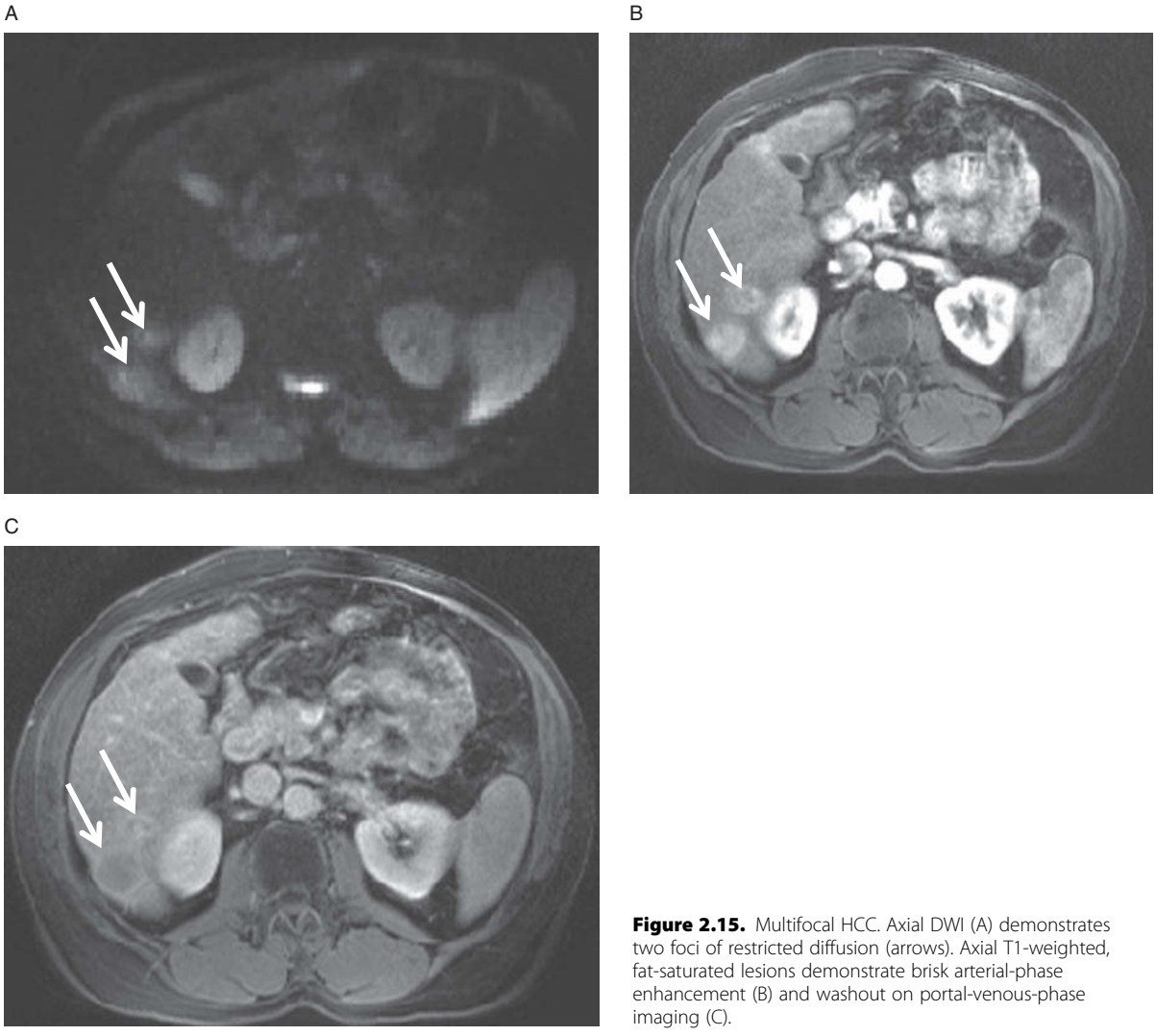

Figure 2.15. Multifocal HCC. Axial DWI (A) demonstrates two foci of restricted diffusion (arrows). Axial T1-weighted, fat-saturated lesions demonstrate brisk arterial-phase enhancement (B) and washout on portal-venous-phase imaging (C).

I don't ever call anything a regenerative nodule, because if it isn't cancer, who cares? There's a lot of overlap in the appearance of dysplastic nodules and HCC but essentially they're either about to be cancer or cancer, so who cares? They are treated the same and frankly, the pathologists often have a tough time telling the difference as well.

HCC is hypervascular on arterial phase and washes out on delayed phase imaging (Figures 2.14–2.15). Everyone talks about the arterial phase enhancement, but *washout* is just as bad as wash-in. Although they should be T2-bright, in practice their T2 signal is variable (partly, remember, because our T2s are a "tumor-hiding sequence" and partly because the T2 signal changes of small HCCs are not impressive).

If a patient has cirrhosis and ANY *hypervascular enhancing liver lesion* $\geq$*1 cm the first differential consideration should be HCC.* HCCs are very common and all other lesions are very rare in cirrhotic livers. *Never say a cirrhotic has an FNH.*

Not all nodules in a cirrhotic liver are HCC. Regenerative nodules, the body's response to cirrhosis, can also appear mass-like but are typically bright on T1 and demonstrate neither hyperenhancement nor washout.

There is one caveat, but, at least there's only one. Cirrhotic livers will commonly show tiny bright spots

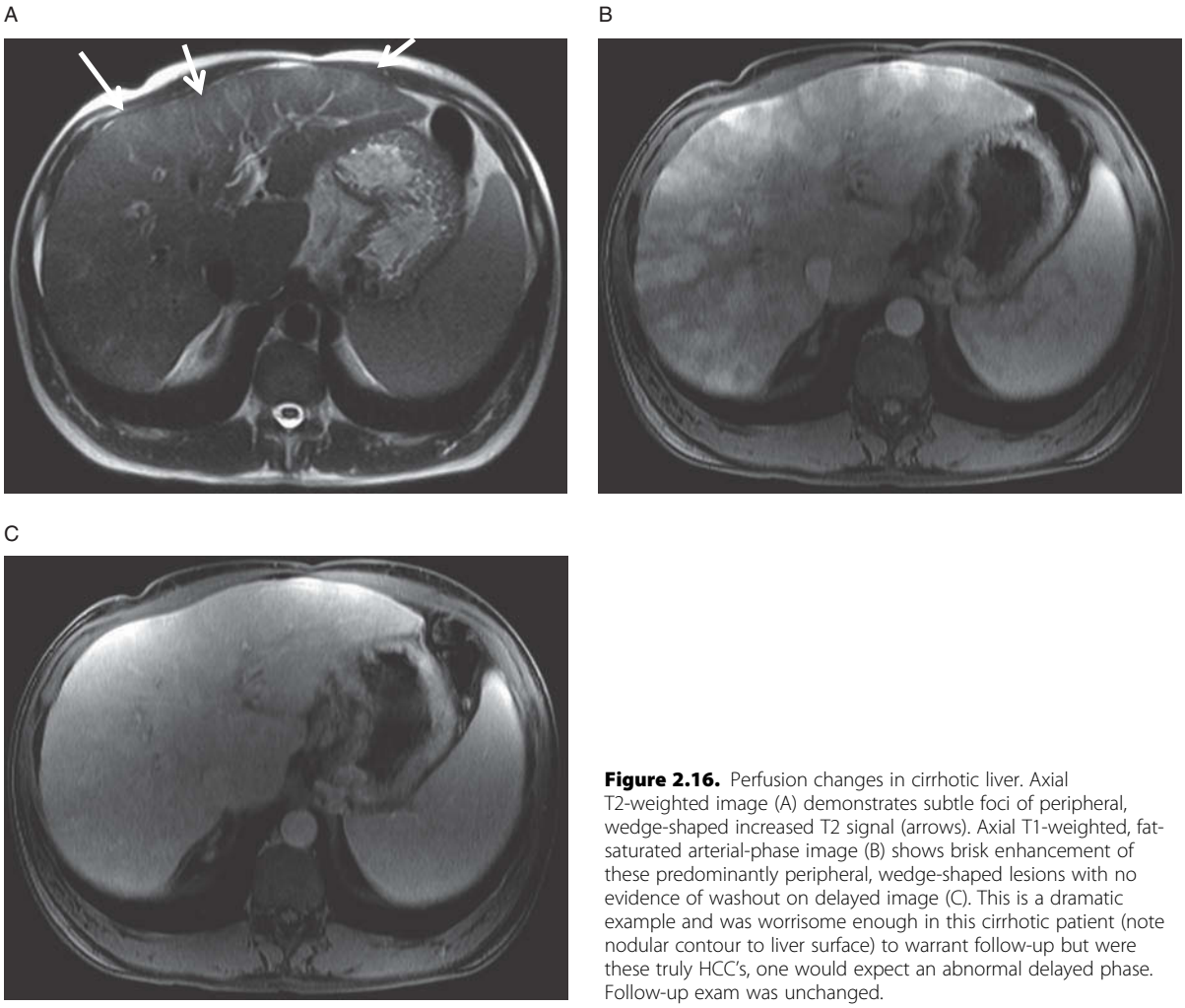

Figure 2.16. Perfusion changes in cirrhotic liver. Axial T2-weighted image (A) demonstrates subtle foci of peripheral, wedge-shaped increased T2 signal (arrows). Axial T1-weighted, fat-saturated arterial-phase image (B) shows brisk enhancement of these predominantly peripheral, wedge-shaped lesions with no evidence of washout on delayed image (C). This is a dramatic example and was worrisome enough in this cirrhotic patient (note nodular contour to liver surface) to warrant follow-up but were these truly HCC's, one would expect an abnormal delayed phase. Follow-up exam was unchanged.

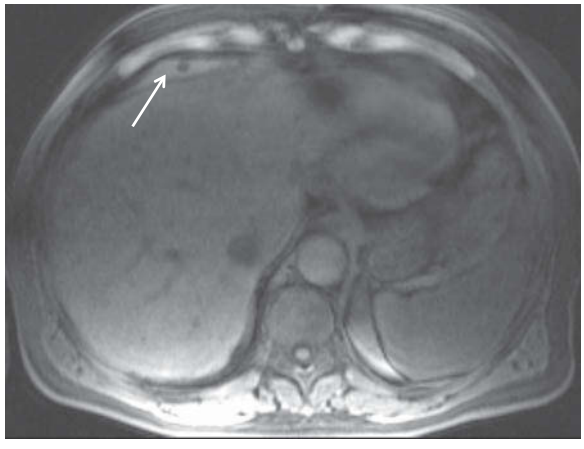

Figure 2.17. Cirrhotic liver with hemoperitoneum. Axial T1-weighted, fat-saturated pre-contrast image demonstrates a nodular cirrhotic liver with subcapsular, T1-bright material compatible with blood (arrow). Although a focal liver lesion was not definitively detected on post-contrast imaging, the radiologist should strongly suspect underlying HCC (which was later surgically confirmed).

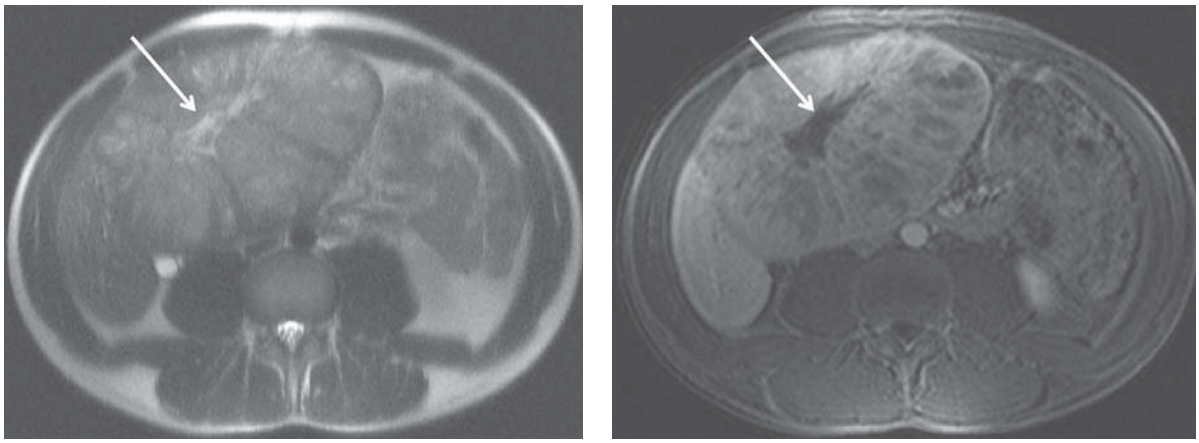

Figure 2.18. Fibrolamellar HCC. Axial T2-weighted image (A) demonstrates heterogeneous lesion within the liver. Arrow denotes T2-bright scar. Axial, T1-weighted, fat-saturated post-contrast image (B) demonstrate same heterogeneous lesion with areas of hypervascularity. Arrow again denotes central scar. Note lack of enhancement of central scar. The scar in FNH typically shows delayed enhancement.

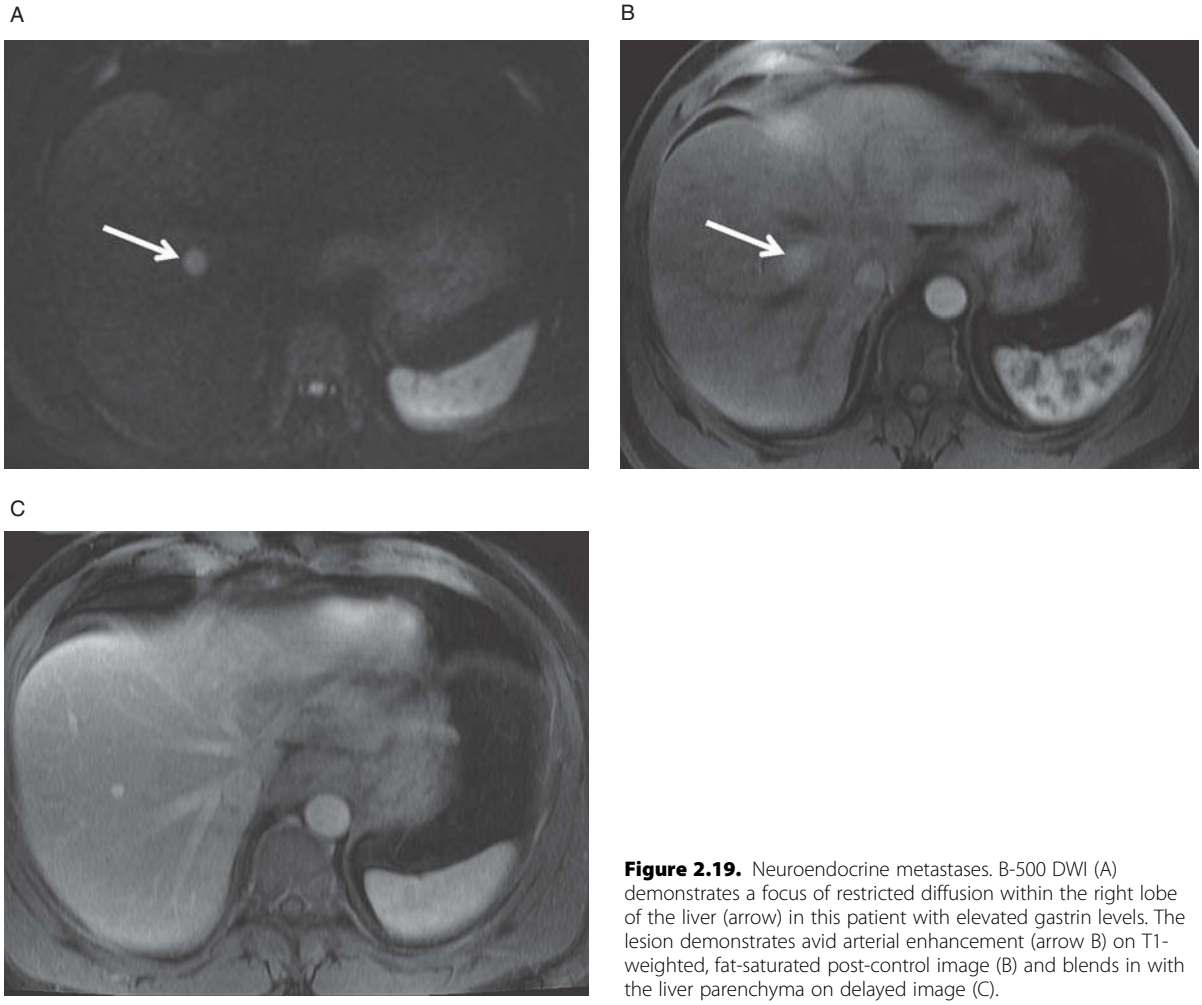

Figure 2.19. Neuroendocrine metastases. B-500 DWI (A) demonstrates a focus of restricted diffusion within the right lobe of the liver (arrow) in this patient with elevated gastrin levels. The lesion demonstrates avid arterial enhancement (arrow B) on T1-weighted, fat-saturated post-control image (B) and blends in with the liver parenchyma on delayed image (C).

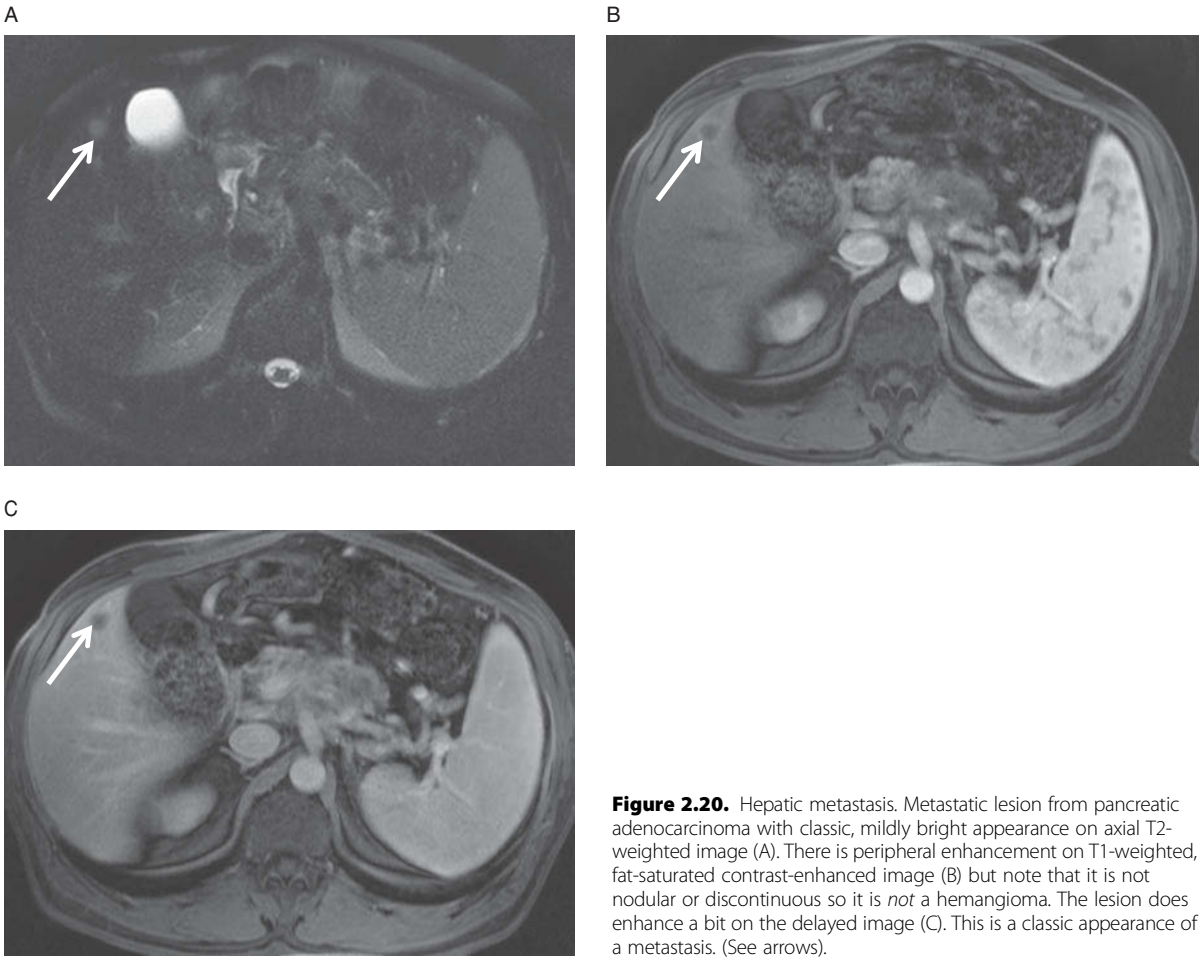

Figure 2.20. Hepatic metastasis. Metastatic lesion from pancreatic adenocarcinoma with classic, mildly bright appearance on axial T2-weighted image (A). There is peripheral enhancement on T1-weighted, fat-saturated contrast-enhanced image (B) but note that it is not nodular or discontinuous so it is *not* a hemangioma. The lesion does enhance a bit on the delayed image (C). This is a classic appearance of a metastasis. (See arrows).

on arterial-phase imaging which are referred to as "perfusion changes or THIDS (transient hepatic intensity differences)." These are not HCC. They are smaller than 1 cm and *do not wash out*. If you call these HCC you will be very sensitive, but not so specific. I hesitate to call anything under 1 cm HCC (unless I see washout in addition to wash-in) (Figure 2.16).

It is important to remember that HCC is one of the few liver tumors that commonly bleed (adenoma is the other). *If a cirrhotic patient presents with bleeding from the liver, you should suspect HCC whether or not you see a discrete mass* (Figure 2.17).

Previously, we mentioned that fat-containing liver lesions are typically benign. This is true. However, some HCCs can contain small amounts of fat. This does not complicate matters as much as it may sound. If the patient is a young woman on oral contraceptives and has a fat-containing liver lesion, it is an adenoma. If the patient has cirrhosis and has a fat-containing liver lesion, it is an HCC.

You may have heard of a variant of HCC called fibrolamellar HCC. This is a classic radiology lesion which receives much more attention in print, case conference, and on exams than it deserves; it is exceedingly rare. Your chances of being asked about it are far greater than actually seeing one (other than here, Figure 2.18). They typically occur in young women. The good news is that they have a much better prognosis than a typical HCC. If you see a lesion that looks like a well-encapsulated HCC with a central scar in a young person with *no history of hepatitis or cirrhosis*, you can suggest this diagnosis. Biopsy is required.

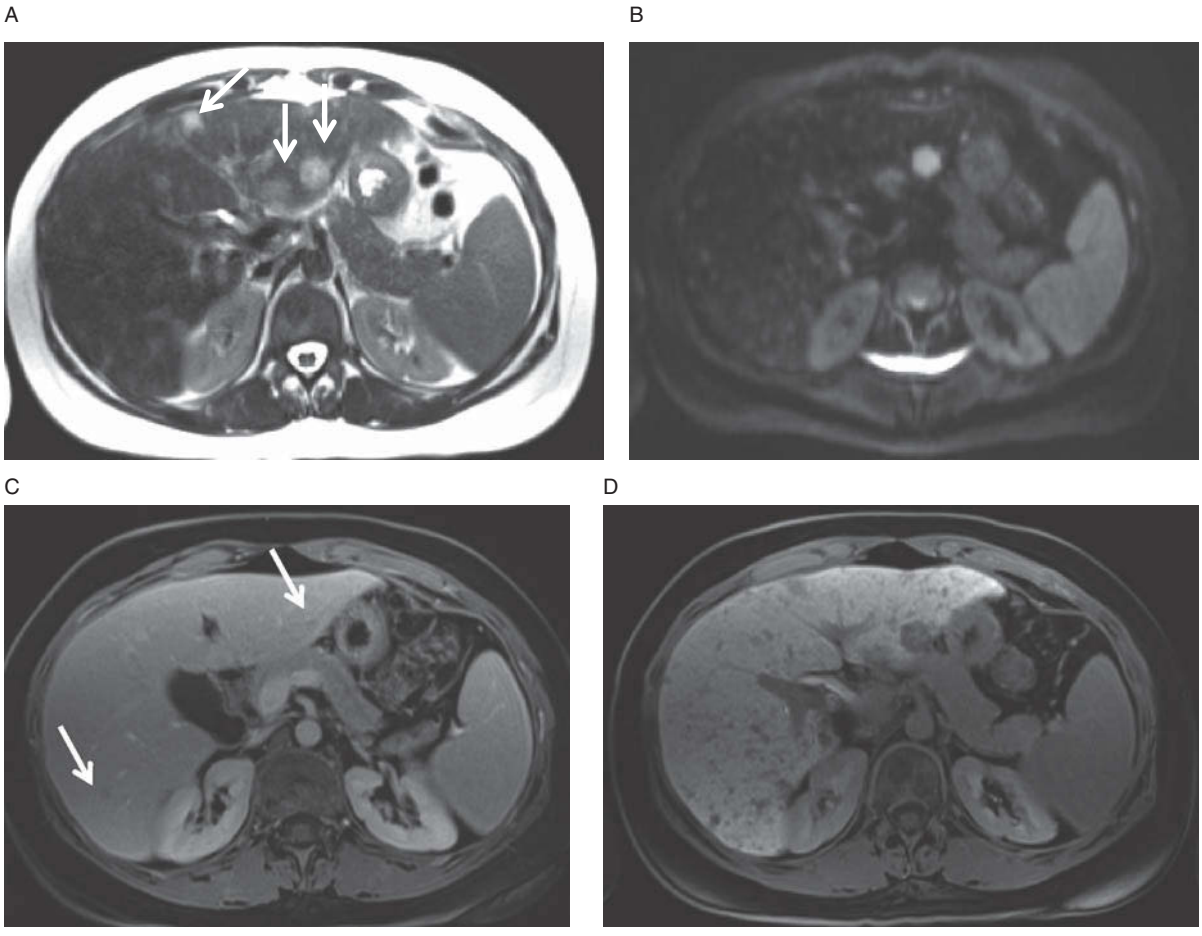

Figure 2.21. Hepatic metastases with gadoxetate disodium. Multiple, moderately T2-bright lesions are seen throughout the liver (arrows) (A) which demonstrate restricted diffusion (B). These lesions are also seen on portal-venous phase (C). Note however, that on the delayed image (D) we see numerous small, dark lesions with washout of contrast T1-weighted, fat-saturated representing metastatic disease, much more prominent than on the portal-venous phase.

Hypervascular metastases

Metastases from neuroendocrine tumors and (very rarely) renal cell carcinoma may be hypervascular.

They are classically hypervascular on arterial-phase imaging and may blend in with the hepatic parenchyma on delayed phases (Figure 2.19). Most commonly, patients with metastatic neuroendocrine tumors have numerous, small lesions in their livers, rather than a large mass as is typical in HCC.

Hypovascular (but enhancing) liver lesions

Lesions in this category do enhance, but they enhance less than the background liver parenchyma. They include: metastatic disease, liver abscesses, and cholangiocarcinoma.

Metastatic disease is certainly the most commonly encountered hypovascular lesion within the liver. They are variably T2-bright, but less so than cysts and hemangiomas. They are typically seen best on the portal-venous phase (and DWI). They are usually relatively small and multiple. (A quick pearl: For some reason they often follow the signal of the spleen.) They often demonstrate peripheral enhancement, but it will not be peripheral, nodular, and discontinuous (Figure 2.20).

There is some evidence that the hepatocyte-specific contrast agent gadoxetate disodium is more sensitive than traditional gadolinium agents for

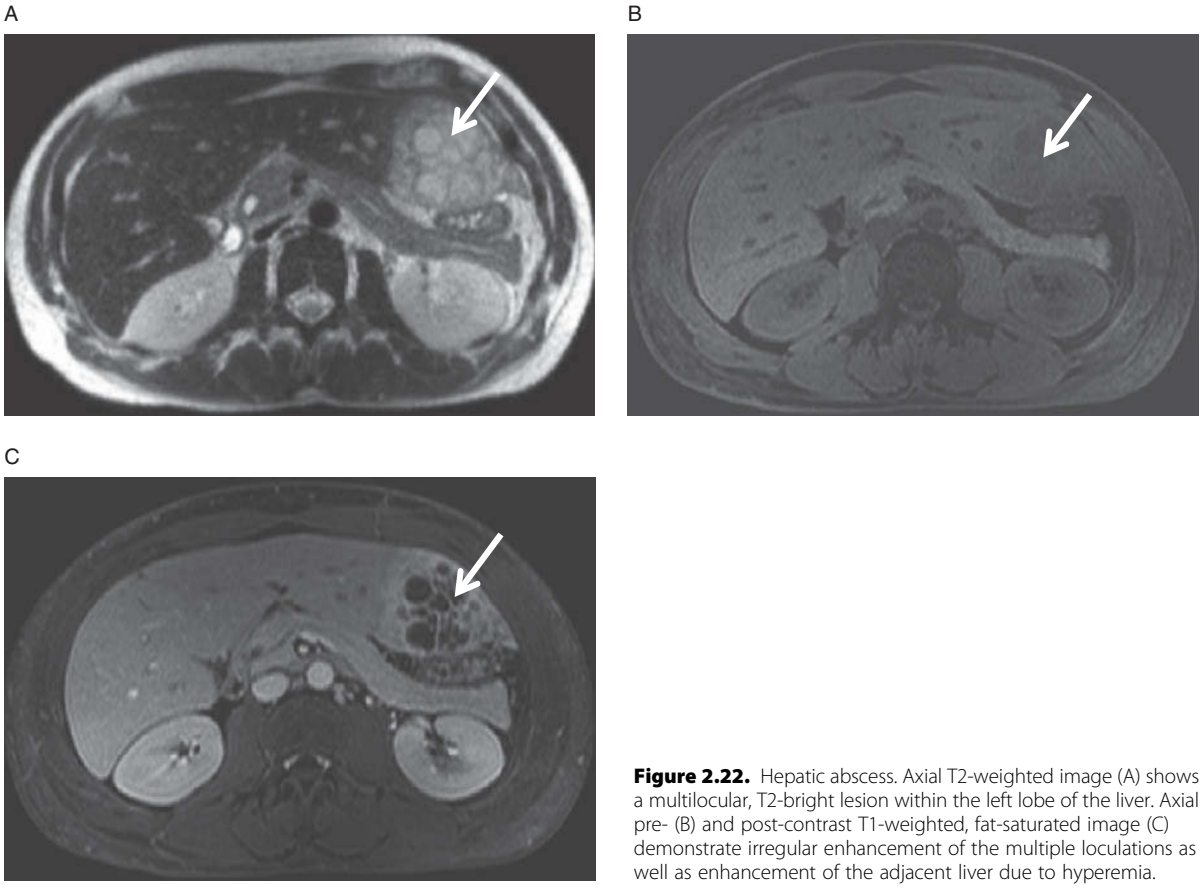

Figure 2.22. Hepatic abscess. Axial T2-weighted image (A) shows a multilocular, T2-bright lesion within the left lobe of the liver. Axial pre- (B) and post-contrast T1-weighted, fat-saturated image (C) demonstrate irregular enhancement of the multiple loculations as well as enhancement of the adjacent liver due to hyperemia.

detecting small metastases. And, dear reader, what will they look like? The dynamic phases will be identical. But, since they do not have functioning hepatocytes, they will be black on the delayed images (Figure 2.21).

Liver abscesses can be impossible to differentiate from liver metastases without a needle. They are also variably T2-bright and enhance peripherally (Figure 2.22). This appearance overlaps with that of a metastasis with central necrosis. Clinical history (if you're lucky enough to get some) can be helpful. Does the patient have cancer? Does the patient have a fever? Has the patient recently had a bout of diverticulitis? These are some questions worth asking the referring doctor. In the end, every year there are a few "metastases" we try to biopsy and instead get pus (in which case, I say to the patient, "great news, pus!"), and a few "abscesses" we attempt to aspirate but instead are forced to biopsy. It happens.

Our final hypovascular liver lesion worthy of discussion is cholangiocarcinoma, a primary malignancy of the biliary tree. Most commonly, cholangiocarcinoma is extrahepatic and presents with biliary ductal dilatation. The classic description of cholangiocarcinoma is that it is mildly T2-bright, T1-dark, and enhances prominently on very delayed images (some centers will perform a 10- or even 20-minute delayed post-contrast series when cholangiocarcinoma is suspected) (Figure 2.23). The delayed enhancement is thought to be due to the fibrous nature of cholangiocarcinoma, and fibrous tissue tends to hold on to gadolinium agents.

In reality, cholangiocarcinomas can be extremely difficult to see. Often, one cannot draw a circle around them like you can with a metastasis or even a HCC. This is due to the infiltrative nature of the tumor which grows along the bile ducts. *The most common imaging finding in cholangiocarcinoma is dilated ducts which abruptly taper/terminate*. The lesion is where the dilated ducts end.

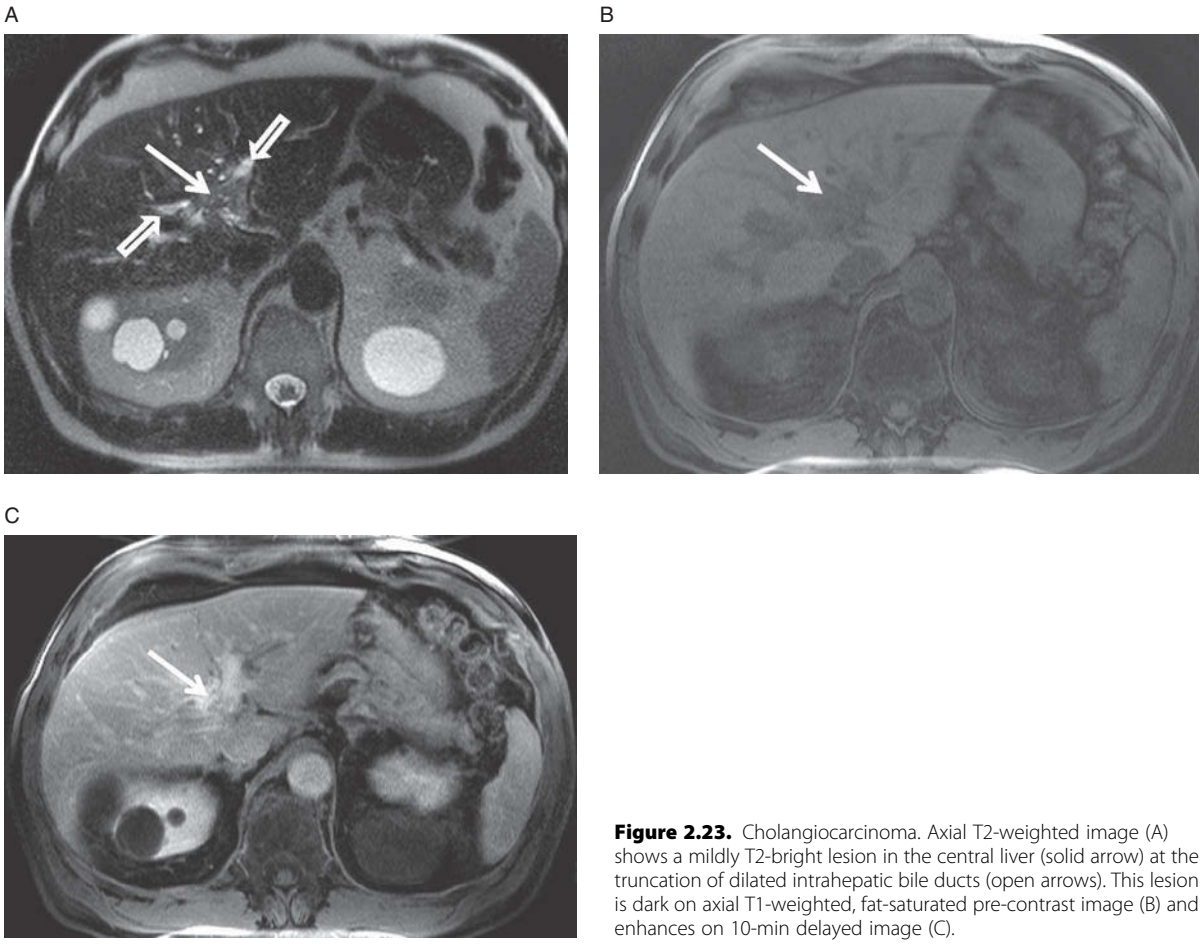

Figure 2.23. Cholangiocarcinoma. Axial T2-weighted image (A) shows a mildly T2-bright lesion in the central liver (solid arrow) at the truncation of dilated intrahepatic bile ducts (open arrows). This lesion is dark on axial T1-weighted, fat-saturated pre-contrast image (B) and enhances on 10-min delayed image (C).

One final word on cholangiocarcinoma: There are times when it can be difficult to distinguish an intrahepatic cholangiocarcinoma from an HCC. Remember:

(1) HCCs are hypervascular
(2) Cholangiocarcinoma tends to have more biliary ductal dilatation
(3) If the portal vein is invaded, it is HCC not cholangiocarcinoma.

The table below summarizes what we have learned about focal liver lesions. It is critical to know the common appearances of these lesions and, fundamentally, it isn't that hard.

But, after all we've been through, it's also useful to take a step back at this point and consider the following simple approach to liver lesions:

If a focal liver lesion does not meet criteria for a cyst, hemangioma, or FNH then it does not meet imaging criteria for a benign lesion and should be at least followed and usually biopsied. (Given that this sentence sums up the entire chapter, please read one more time before proceeding.)

Summary of liver lesions

Table 2.1

	T1	T2	Fat	DWI, ADC	Arterial	Venous	Delayed
Cyst	↓↓	↑↑	None	↑↑,↑↑	↓↓	↓↓	↓↓
Hemangioma	↓↓	↑↑	None	↑↑,↑↑	↑[a]	Iso or ↑	Iso or ↑
Metastasis (general)	↓	↑	None	↑,↓	↓[b]	↓	↓
Metastasis (hypervascular)[c]	↓	↑–↑↑	None	↑,↓	↑↑	Iso	Iso or↓
FNH	Iso to ↓	Iso to ↑	None	Variable	↑↑	Iso	Iso
Adenoma[d]	Iso to ↓	↑	Often	Variable	↑↑	Iso	Iso
HCC[e]	↓	↑	Occasional	↑,↓	↑↑	↓ to ↓↓	↓ to ↓↓

[a]Arterial phase enhancement must be peripheral, nodular, and *discontinuous*. Venous and delayed enhancement may be slightly brighter than the liver parenchyma but will typically follow the enhancement of the portal venous system.
[b]Lesions often have a *continuous* ring of peripheral enhancement. Please note that these lesions do enhance, but enhance less than the liver and are therefore hypointense.
[c]Hypervascular metastases include neuroendocrine tumors, RCC, melanoma, satellite lesions of HCC.
[d]The astute reader will notice the similarity between the signal characteristics of FNH and adenoma. Some things to keep in mind: FNH is extremely common, adenoma is not. Adenoma occurs in women on oral contraceptives. Adenoma is typically more heterogeneous than FNH and may contain blood products and fat. FNH classically has a T2-bright, hypoenhancing scar. Liver-specific imaging agents may be helpful to increase confidence.
[e]HCCs may not be bright on T2 until they are greater than 2 cm. Essentially, hypervascular lesions in a cirrhotic liver are HCC until proven otherwise. Below 1 cm the specificity of this "rule" significantly decreases and lesions should probably be followed rather than called HCC unless there are other suspicious features. These may be clinical features (elevated alpha-fetoprotein) or imaging features, including restricted diffusion and washout on delayed images. Remember: *Washout is just as important and just as bad as early wash-in* and should be considered indicative of malignancy.

Pearls and pitfalls

(1) T2-bright, non-enhancing lesions are benign.
(2) Hemangiomas >1 cm must demonstrate peripheral, nodular, discontinuous enhancement. *If not, DO NOT call it a hemangioma*. Follow the rules.
(3) FNH is very, very common. Adenoma is not.
    (a) FNH retains gadoxetate disodium at 20 minutes. Adenoma does not.
(4) For any enhancing lesion in a cirrhotic liver, first think HCC.
(5) Hemoperitoneum + cirrhosis = HCC whether or not you can see a mass.
(6) To differentiate cholangiocarcinoma from HCC:
    (a) HCC is hypervascular
    (b) Cholangiocarcinoma typically has markedly dilated ducts
    (c) Portal venous invasion = HCC.

Further reading

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Chapter 3

Pancreas and biliary tree

MRI cholangiopancreatography (MRCP)/pancreas protocol

Indications

This protocol is used for evaluation of the biliary tree and pancreas. Indications include biliary ductal dilatation, suspicion of choledocholithiasis, jaundice, pancreatitis, pancreatic mass.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: Mixture 3 oz. ferumoxsil and 3 oz. barium sulfate 30 minutes prior to study. If 30 minutes is not available, have patient lie on right side for 5 minutes prior to study
- 2 L nasal oxygen
- At least 24-gauge IV; connect to power injector
- Cover from dome thru entire liver
- Find the best visualization of the ducts and repeat coronal single-shot fast-spin echo slab 30 mm thick 10 times.

Exam sequences

(1) Diffusion-weighted imaging b50, 500/ADC – Very sensitive sequence for lesion detection.
(2) Axial thin slice single-shot fast-spin echo FS BH – Evaluate for choledocholithiasis.
(3) Coronal thin slice single-shot fast-spin echo FS BH – Confirm biliary duct stones, evaluate for ductal stricture.
(4) Coronal single-shot fast-spin echo thick slab (30 mm) – Repeat 10× – Assess distal CBD (ampulla), nice overall image of biliary tree. Repeated images to assure at least one with open sphincter of Oddi.
(5–6) Axial T1 in- and out-of-phase (IP/OOP) – Identify focal fat in the pancreas, don't mistake it for a mass.
(7) Axial T1-weighted volume-interpolated gradient echo pre-contrast BH – Identify masses/material which are T1-bright before contrast administration. The fat saturation of this sequence increases relative signal in the pancreas and is good for identifying more subtle pancreatic masses.
(8) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 20 seconds. Look for hypervascular lesions such as islet cell tumours.
(9) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 1 minute.
(10) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 2 minutes.
(11) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 3 minutes.

Approach to MRCP/MRI pancreas exam interpretation

MRCP/MRI of the pancreas is one of my favorite exams. If you've ever had the pleasure of seeing an endoscopic retrograde cholangiopancreatography (ERCP) performed, it's a tough procedure (for the patient and gastroenterologist). And while we can't remove a stone if we find one, keep two things in mind:

(1) It's rare for a patient to actually have what the ordering physician is looking for (particularly if ordered from the Emergency Room).
(2) No one ever got pancreatitis from an MRCP.

The incidence of pancreatitis from an ERCP is somewhere in the range of 5% – in the best hands. Pancreatitis is extremely painful and can result in prolonged hospitalization and even death. Thus the MRCP is the gastroenterologists' friend – not enemy. If a patient has known (or very high suspicion for)

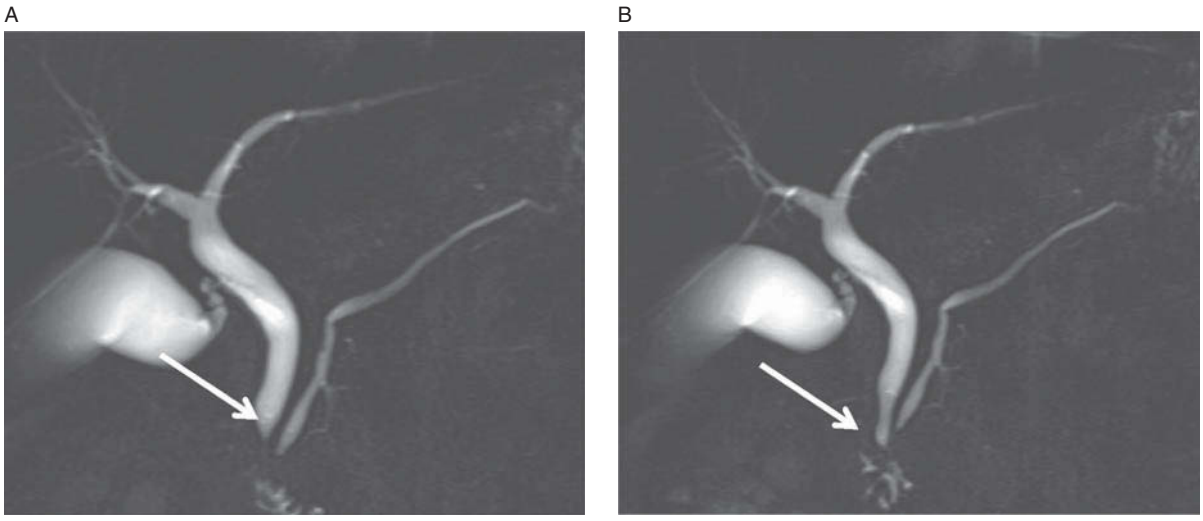

Figure 3.1. Sphincter of Oddi. Coronal thick-slab MRCP image (A) demonstrates what could be mistaken as a stricture of the distal common bile duct (arrow). Repeat image (B) acquired in same location 2 seconds later demonstrates the full length of a normal common bile duct. This is due to normal opening and closing of the sphincter of Oddi.

choledocholithiasis, the first line exam is an ERCP as the common duct stone can be removed at the time of the procedure. Otherwise, do no harm . . . do an MRCP.

We perform all of our MRCPs with IV contrast to provide a comprehensive evaluation of the pancreas and biliary tree. Most of our MRCP patients come to us with dilated ducts or elevated liver function tests. *While dilated ducts may be due to CBD stones, they can also be due to pancreatic cancer – which kills people*. If we do not want to miss pancreatic cancer, we need IV contrast.

We also give oral contrast to all patients who can tolerate it. It is helpful, but it is not helpful enough to justify making an already miserable patient even more miserable. The concoction we have settled on, a 6-oz. mixture of ferumoxsil and barium, renders the enteric contents dark on T2-weighted images, leaving the biliary tree the only bright structure. (Otherwise, fluid in the stomach/duodenum would be bright and could obscure the biliary tree.)

Sequences and approach

Multiplanar SSFSE: T2-weighted; currently obtained with and without fat saturation

Single-shot fast-spin echo (SSFSE) images are the backbone of the MRCP. Most of what we are looking for can be found or excluded here.

We currently perform thick slabs as well as thin slices. The thick slabs are obtained 10× at 1-second intervals. The sphincter of Oddi opens approximately once every 10 seconds, thus these repetitive thick slabs ensure demonstration of the entire duct on at least one image (Figure 3.1). They also provide a nice anatomic overview; we rely on the thin slabs for more precise anatomic localization.

A few words on 3D MRCP. Since all of you likely jump out of your chairs and run to the mailbox whenever the latest journal arrives, you are undoubtedly familiar with the spectacular images generated by 3D MRCP sequences. These are typically respiratory-navigated 3D SSFSE sequences acquired with extremely small, isotropic voxels, allowing multiplanar reconstruction.[1] Theoretically, one could perform a standard MRCP with only this one sequence.

To date, our experience with 3D MRCP has been disappointing. It is time-consuming (at least 5–6 minutes) because while the patient breathes freely, the scanner only obtains useful information when

[1] A note on respiratory navigation. The technologist obtains a scout image and places a cursor on the diaphragm. The scanner then continuously images the diaphragm waiting for end-expiration. At end-expiration it images. The goal is to eliminate respiratory artifacts. However, nothing comes for free. If you image only at end-expiration the sequence is inherently inefficient as it obtains no information during the remainder of the respiratory cycle. Also, while the vendors may deny it and call me nasty names, this sequence currently has a relatively high rate of failure in patients with irregular breathing.

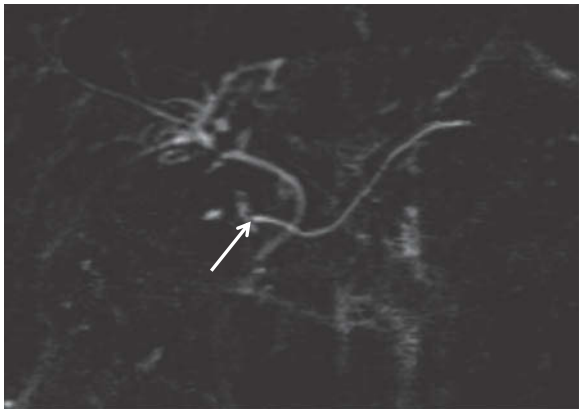

Figure 3.2. Pancreas divisum. Coronal T2-weighted image shows a non-dilated main pancreatic duct draining into the minor papilla (arrow). This is diagnostic of pancreas divisum.

the patient is in end-expiration. In patients whose breathing is irregular the sequence may take significantly longer and, even worse, may fail entirely – yielding no diagnostic information.

So, until the sequences are more robust and reliable, we are sticking with 2D. It is fast, it is diagnostic and it is often quite pretty. 3D is spectacular . . . when it works. My chairman, division head, and I don't have the patience for inconsistency.

Biliary and pancreatic ductal pathology

The first thing I do when I open an MRCP is to check for pancreas divisum. Pancreas divisum is an embryologic failure of fusion of the dorsal and ventral pancreatic buds. That's right, some of us are failures while still in the womb.

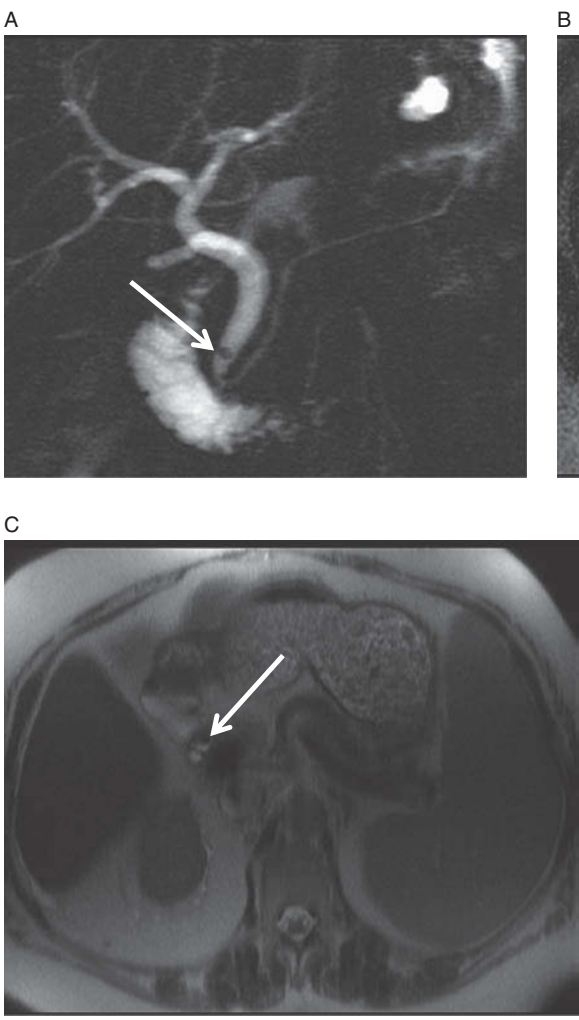

Figure 3.3. Filling defects, CBD. Coronal T2-weighted image (A) demonstrates a filling defect within the CBD (arrow) which sinks dependently (arrow) on axial T2-weighted image (B). Axial T2-weighted image without fat saturation (C) from a *different* patient shows a filling defect within the CBD which floats (arrow), diagnostic of an air bubble.

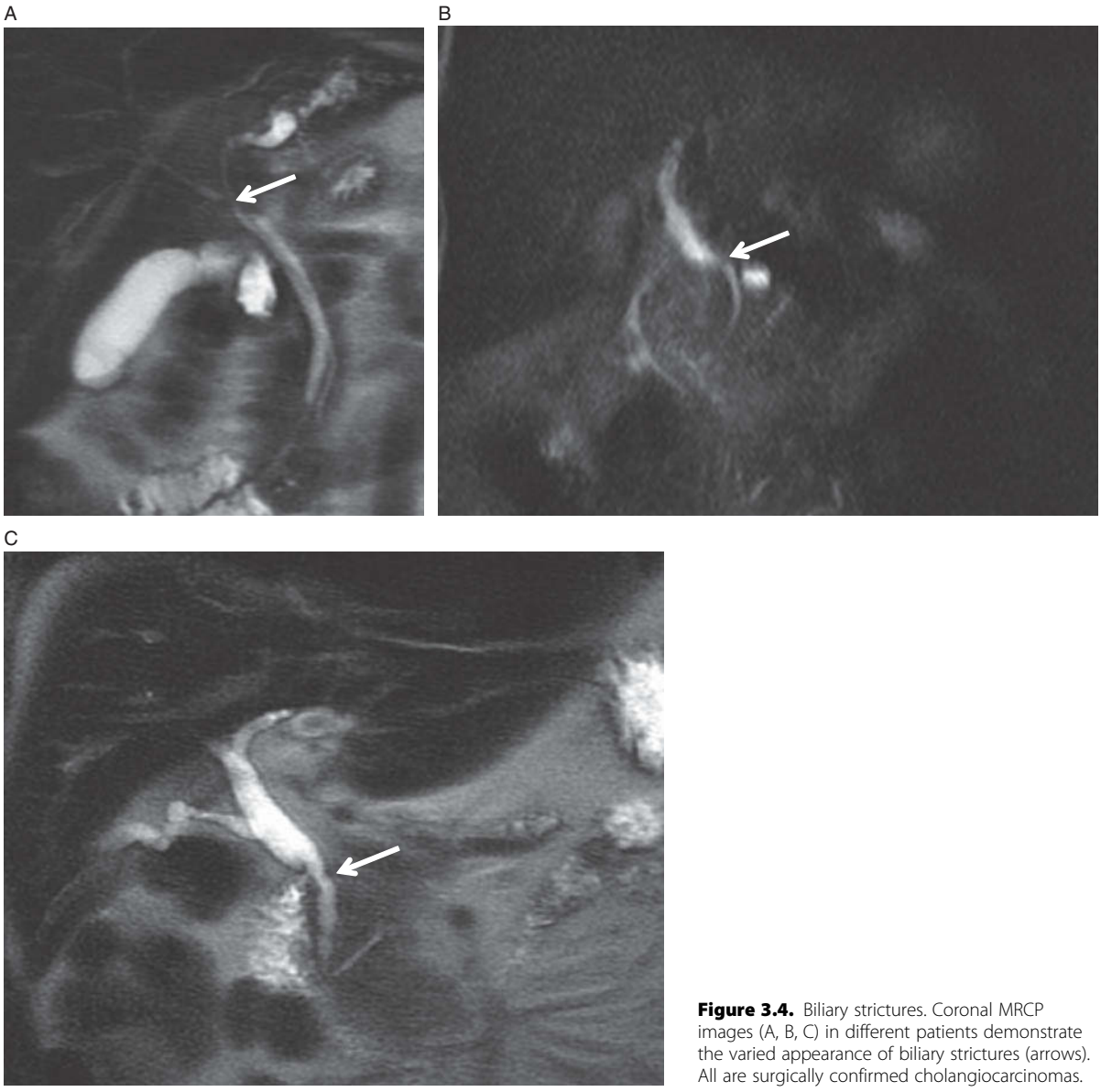

Figure 3.4. Biliary strictures. Coronal MRCP images (A, B, C) in different patients demonstrate the varied appearance of biliary strictures (arrows). All are surgically confirmed cholangiocarcinomas.

Failure to fuse leaves the main pancreatic duct draining the bulk of the pancreatic secretions into the minor papilla which, the theory goes, is not equipped to handle the volume coming its way and predisposes the patient to recurrent bouts of pancreatitis.

(By the way, don't worry about the eponyms associated with the pancreatic ducts. I don't and I seem to be getting along just fine. I call them the main duct and the ventral duct and no gastroenterologist or hepatobiliary surgeon has ever complained.)

If the main pancreatic duct drains into the minor papilla *and* there's no communication with the ventral duct, the patient has pancreas divisum (Figure 3.2). Just seeing a pancreatic duct cross the common bile duct (CBD) doesn't count because the ventral duct will always cross the CBD. *The key is that the main pancreatic duct drains in to the minor papilla and there's no communication with the ventral duct.*

Next, evaluate for stones. Remember that a filling defect on a coronal image is not necessarily indicative

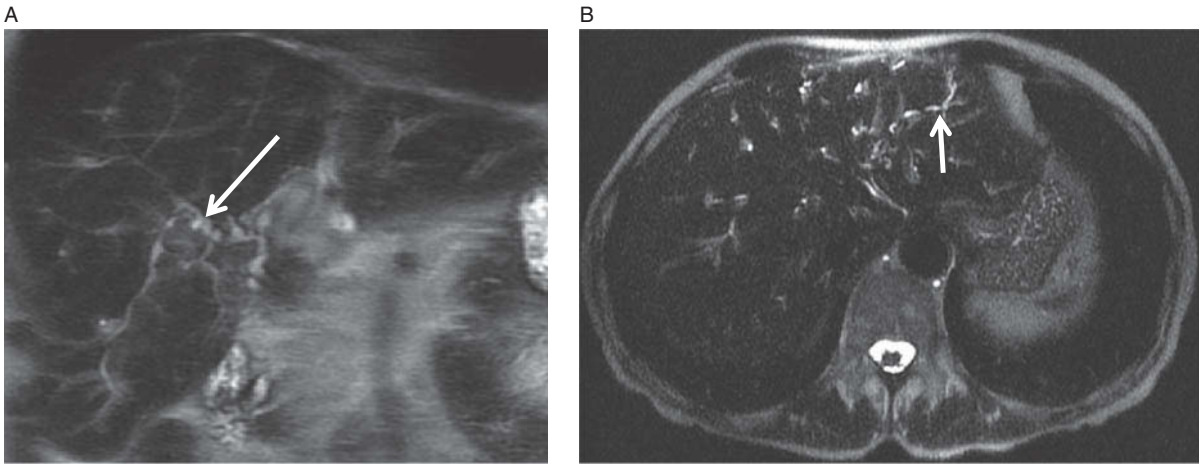

Figure 3.5. Primary sclerosing cholangitis (PSC). Coronal T2-weighted image (A) demonstrates marked irregularity and beading of the CBD (arrow). Axial T2-weighted image (B) shows irregular, dilated intrahepatic biliary ducts which extend to the periphery of the left lobe of the liver (arrow). For some reason, this appearance is PSC every time.

of a stone. Both stones and air bubbles are T2-dark. Filling defects must be confirmed on axial images – stones will sink dependently within the duct and air bubbles will float (Figure 3.3). Air bubbles are more common than one might think – many patients have had sphincterotomies. They may also occur in patients who have recently passed a stone. The distinction between a common duct stone and biliary air is critically important. The last thing these patients need after dutifully passing their stone is to undergo ERCP.

Strictures are also fairly common abnormalities and may occur in the biliary or pancreatic ducts. Our job is to identify them. They may be due to prior infection, inflammation, stone disease, or malignancy. We report whether they appear smooth or jagged/shouldered and, if you have too much time on your hands you can wax poetic about the likelihood of cancer, but *any patient with a biliary stricture should probably undergo ERCP with brushing to exclude malignancy* (Figure 3.3).

Cholangiocarcinoma, primary cancer of the biliary ducts, can affect any portion of the biliary tree. Whenever a dilated duct or dilated ducts abruptly terminate, without evidence of stone, cholangiocarcinoma must be suspected (Figure 3.4).

Beaded ducts, which look just like it sounds, or ducts with multiple, small, segmental strictures should raise concern for primary sclerosing cholangitis (PSC). This entity should also pop into your mind whenever you see peripherally dilated ducts, particularly (for some unknown reason) in the left lobe (Figure 3.5). Patients with PSC are at high risk for developing cholangiocarcinoma which is yet another reason to use intravenous contrast for all MRCP patients.

And now, to conclude our discussion of the biliary tree, a few words on a confusing topic: choledochal cysts. Choledochal cysts, like many commonly discussed topics in radiology, are extremely uncommon, but get lots of press in radiology conferences. If that is not bad enough, there are five different "types," all rarer than the next.

Choledochal cysts are congenital abnormalities of the biliary tree which are rare but greatly increase one's risk of cholangiocarcinoma. They are classified as follows:

Type I: Cystic dilatation of the common bile duct.
Type II: Diverticulum of the common bile duct.
Type III: Cystic dilatation of the intraduodenal portion of the common bile duct.
Type IV: Cystic dilatation of the intra- and extrahepatic biliary ducts.
Type V: Cystic dilatation of the intrahepatic biliary ducts only (Caroli's disease).

Type I is the most common type of choledochal cyst and is also the most challenging to diagnose. Quite frankly, this diagnosis still gives me a little chest pain. Whenever I'm at a radiology conference or have had the pleasure of a long conversation with a gastroenterologist, I ask them how they make this diagnosis. So, far ... no good answer. Stay tuned for our second edition.

I suggest choledochal cyst when MRCP shows an enlarged CBD (without a definite cause from a stone or stricture) and normal intrahepatic ducts (Figure 3.6). These patients may go to ERCP

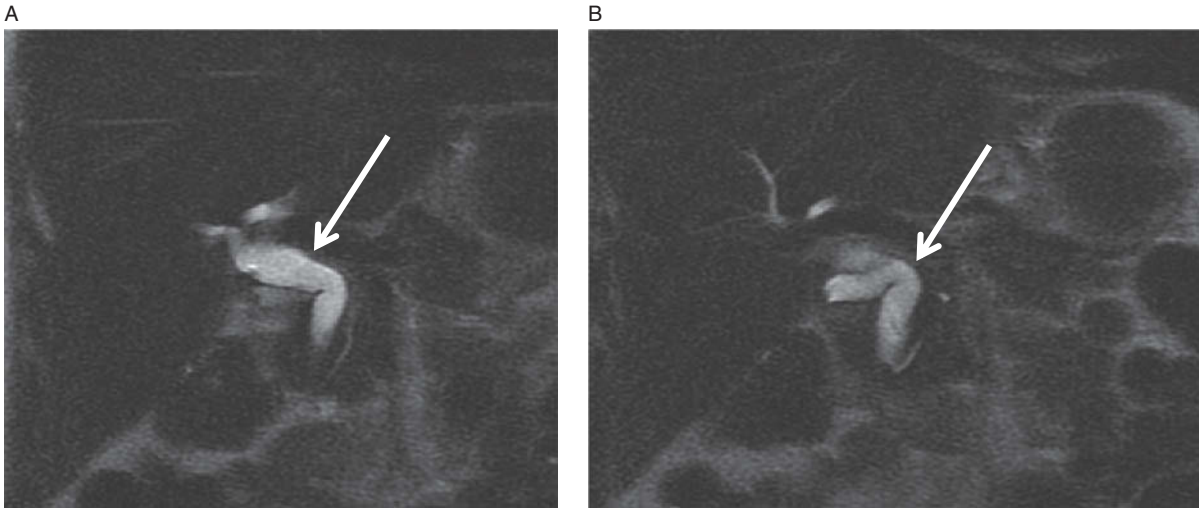

Figure 3.6. Type I choledochal cyst. Consecutive, coronal T2-weighted images (A, B) showing dilatation of the CBD without definitive etiology (such as stone or stricture). (See arrows). The intrahepatic ducts are normal.

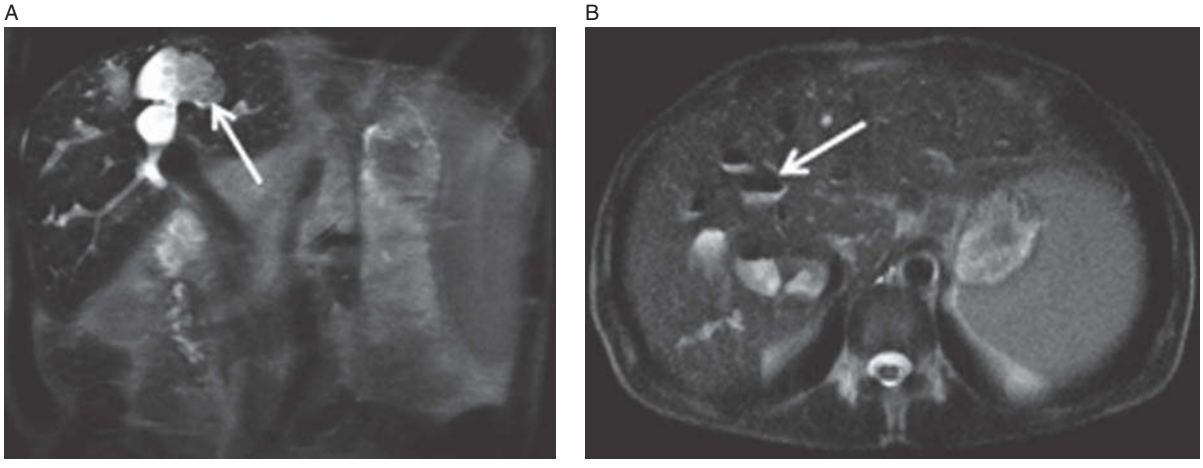

Figure 3.7. Caroli's disease. Coronal T2-weighted image (A) shows saccular, cystic dilatation of the intrahepatic biliary tree with multiple filling defects (arrow). Are these stones? Axial T2-weighted image (B) demonstrates that these filling defects float and therefore represent air (arrow). Patient has already undergone hepaticojejunostomy.

to confirm lack of underlying stricture and to obtain brushings, but essentially the lesion looks the same to the endoscopist as it does to us – a big CBD. Unfortunately, definitive treatment is a hepaticojejunostomy to reduce the risk of cholangiocarcinoma.

The only other form of choledochal cyst worth discussing is Caroli's disease which is cystic dilatation of the intrahepatic biliary tree. It is typically quite dramatic. As long as you've heard of the entity, you won't miss it when you see it. The profound stasis within the intrahepatic biliary tree often leads to stone formation (Figure 3.7).

Pancreatitis

It is worth taking a minute to familiarize oneself with the MRI appearance of acute pancreatitis. Acute pancreatitis is characterized by mild expansion of the pancreas with surrounding elevated T2 signal (Figure 3.8). Remember, calcification is often invisible on MRI. In acute pancreatis, the duct is typically normal in size or small due to edema. In chronic pancreatis we see a T1-dark, atrophic gland with dilated pancreatic ducts (Figure 3.9).

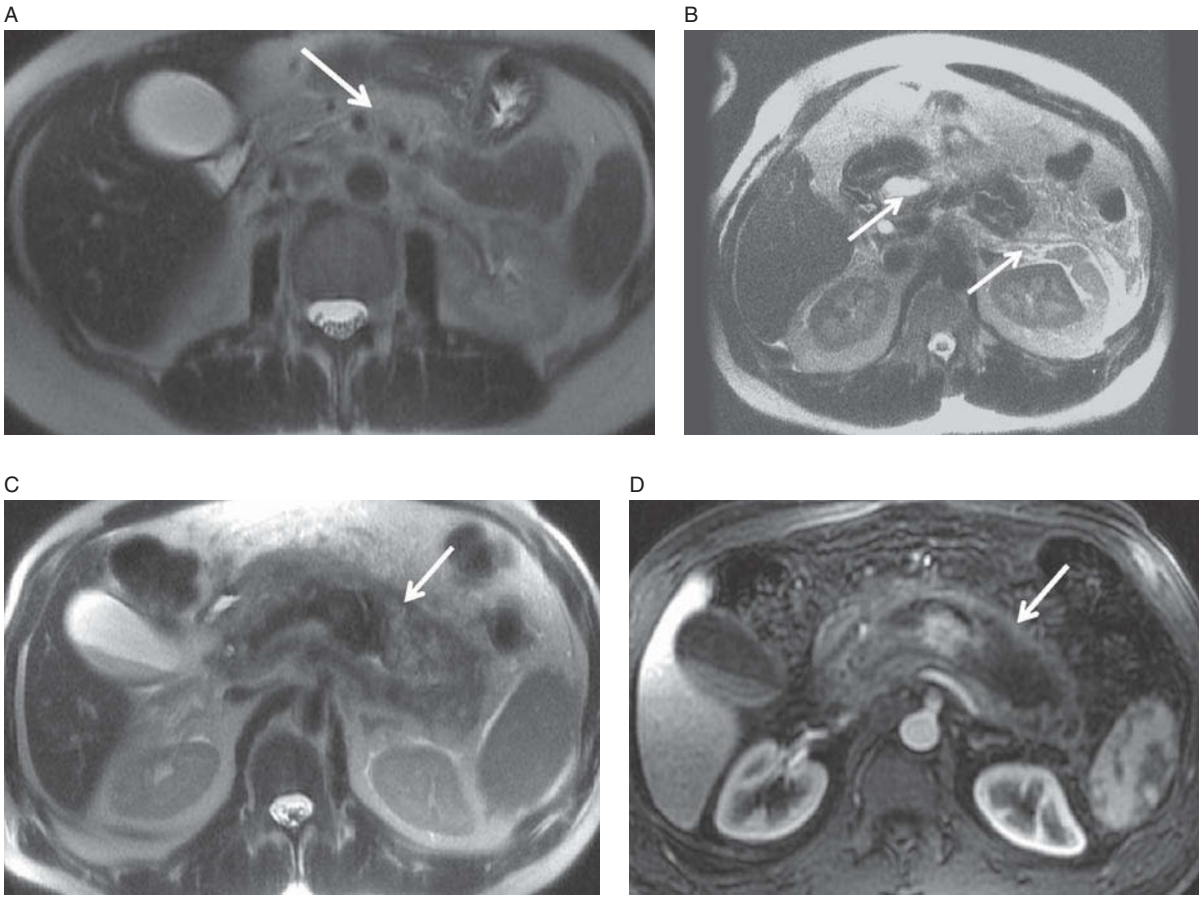

Figure 3.8. Spectrum of acute pancreatitis. Axial T2-weighted image (A) demonstrates mildly increased T2 signal within the pancreatic body (arrow). Axial T2-weighted image (B – different patient) demonstrates more severe pancreatitis with mild expansion of the pancreas and extensive surrounding fluid (arrows). Axial T2-weighted image (C – different patient) shows extensive replacement of the pancreatic body and tail with heterogeneous T2-bright signal. Axial post-gadolinium enhanced image (D) demonstrates a T1-dark heterogeneous fluid collection replacing large segments of the pancreatic body and tail compatible with pancreatic necrosis.

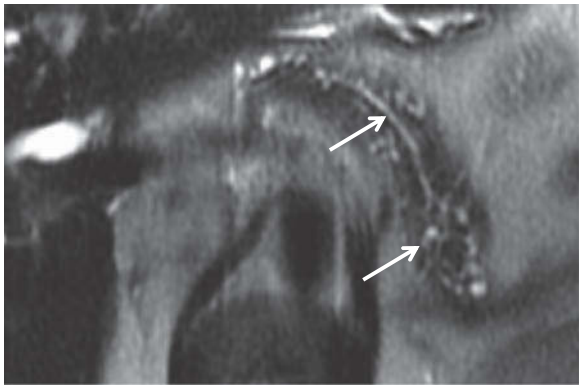

Figure 3.9. Chronic pancreatitis. Axial T2-weighted image demonstrates dilatation of the main pancreatic duct and side-branches (arrows).

Cystic lesions of the pancreas

Cystic lesions of the pancreas are common, confusing, frustrating, and poorly understood. As we image more and more people, we are finding more of these lesions. Because they are so common, and typically indolent, guidelines have been prepared to minimize unnecessary follow-up and intervention. These are summarized in Figure 3.10.

We said these lesions are confusing, but, really, they aren't. The differential diagnosis of a cystic lesion of the pancreas consists of pseudocysts and cystic neoplasms. That's it.

Until recently it was thought that pseudocysts were common and cystic neoplasms were rare. In one of those wonderfully dramatic flip-flops possible only in medicine, this thinking has now completely reversed and

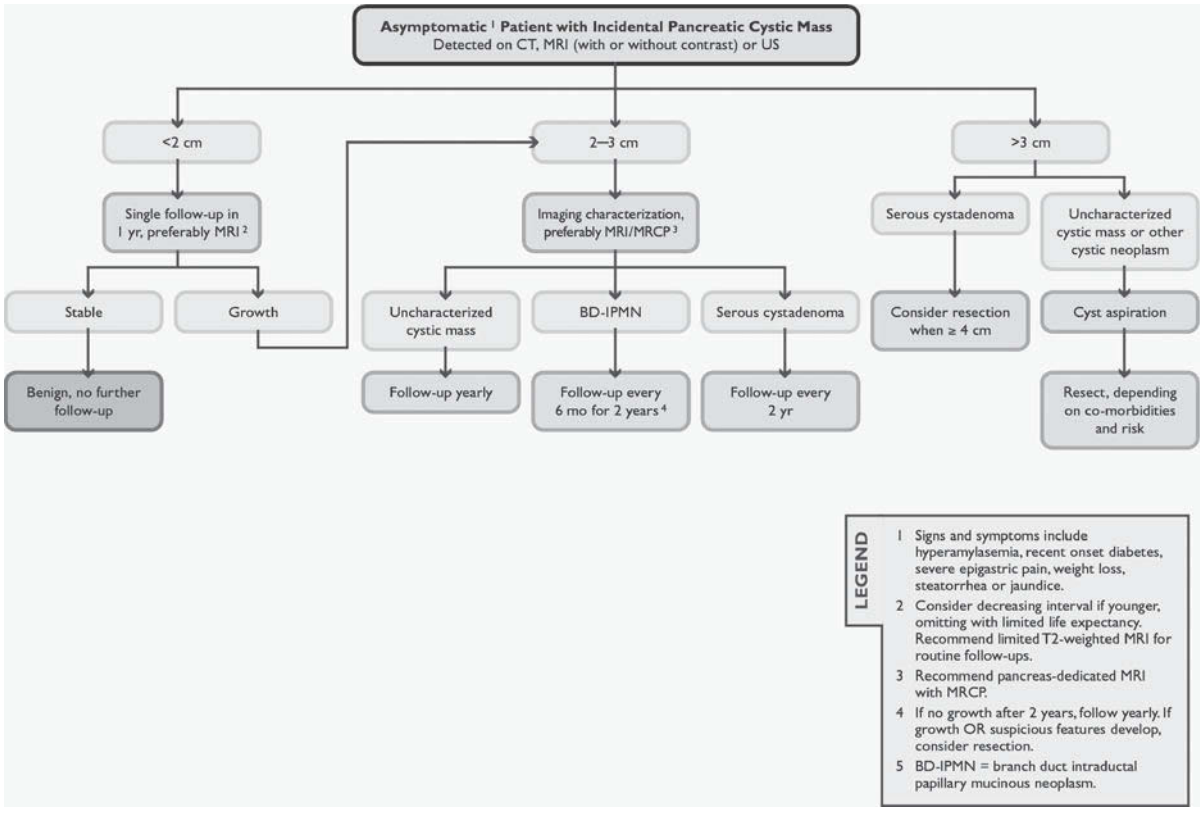

Figure 3.10. Flowchart depicting recommended guidelines for incidentally detected, asymptomatic cystic lesions of the pancreas. Source: *Journal of the American College of Radiology* (2010) 7:754–73 (doi:10.1016/j.jacr.2010.06.013), reprinted with permission.

cystic neoplasms are thought to be more common and pseudocysts uncommon (unless the patient has a convincing history of pancreatitis). High-resolution abdominal imaging has caused this change of thinking. We now routinely see things that were not known before.

Pseudocysts are the sequelae of a prior bout of pancreatitis and are benign. They are cysts without an epithelial lining. If the patient has a history of pancreatitis, they are very common and you do not need to mention cystic neoplasm. Most pseudocysts are T2-bright, as we would expect. However, they may have internal debris and even hemorrhage, resulting in T1-bright signal. The key is the absence of enhancement. (*Anytime anything is T1-bright, remember to look at subtractions to be sure there's no internal enhancement.*)

It is critical to look at all prior imaging exams when attempting to differentiate pseudocysts from cystic pancreatic neoplasms. A look back will frequently show a prior CT with acute pancreatitis or sequelae of chronic pancreatitis (an atrophic gland with dilated ducts and pancreatic calcifications). If you see acute or chronic pancreatitis on prior exams and you now have a pancreatic cyst, call it pseudocyst and sleep well.

Cystic neoplasms include serous cystadenoma (benign), mucinous cystadenoma/cystadenocarcinoma (potentially malignant/malignant), intraductal papillary mucinous neoplasm (IPMN, potentially malignant), and solid and papillary epithelial neoplasm (SPEN, low-grade malignant potential).

At first read, this may seem like a lot of lesions with a lot of confusing names, but radiologically they are fairly straightforward.

Serous cystadenomas are benign lesions which typically occur in middle-aged women. Classically they consist of innumerable small cysts often with central calcification (Figure 3.11). If asymptomatic, biopsy can prove that these lesions are benign (and are not mucinous cystadenocarcinomas) but, if large or if the patient has symptoms, many pancreatic surgeons will resect them.

Mucinous cystadenocarcinomas are classically macrocystic lesions with internal complexity including septations and nodular enhancement. They are

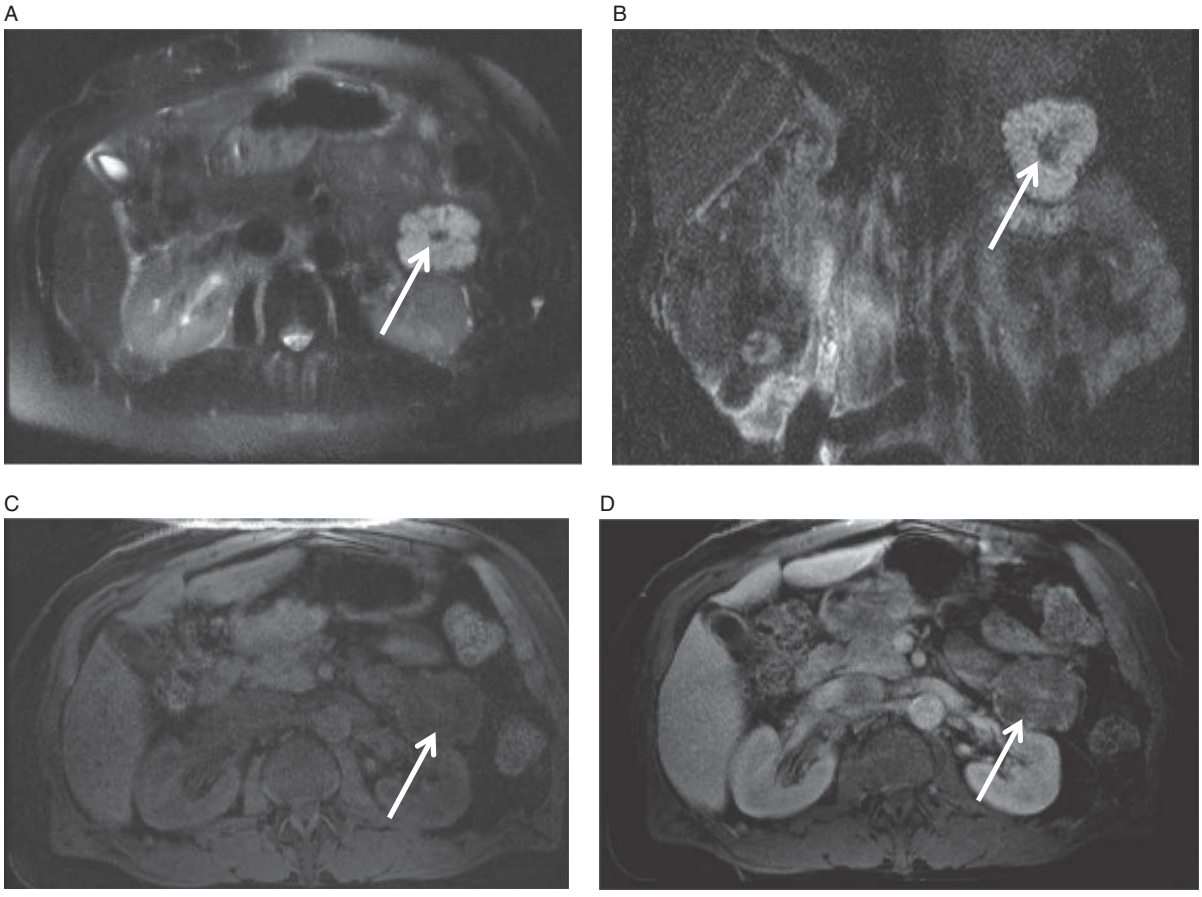

Figure 3.11. Serous cystadenoma. Axial and coronal T2-weighted images (A, B) demonstrate a multicystic lesion within the tail of the pancreas. Arrows indicate dark signal compatible with central calcification. Axial, T1-weighted, fat-saturated pre- and post-contrast images (C, D) demonstrate internal septal enhancement (arrows).

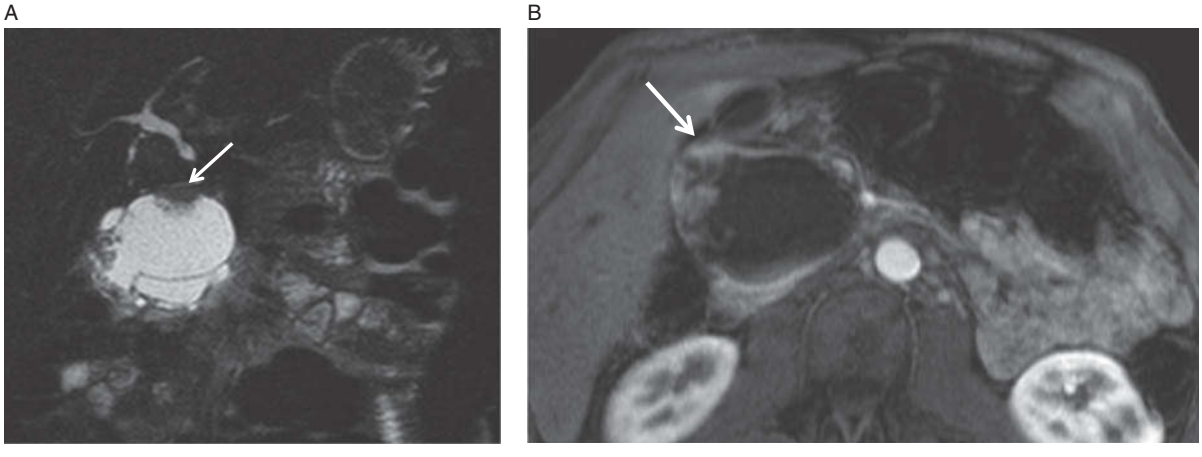

Figure 3.12. Mucinous cystadenocarcinoma. Coronal T2-weighted image (A) demonstrates a macrocystic lesion in the pancreas with internal septations and nodularity (arrow). Axial T1-weighted, fat-saturated post-contrast image (B) again demonstrates the cystic lesion with enhancing, peripheral nodularity (arrow).

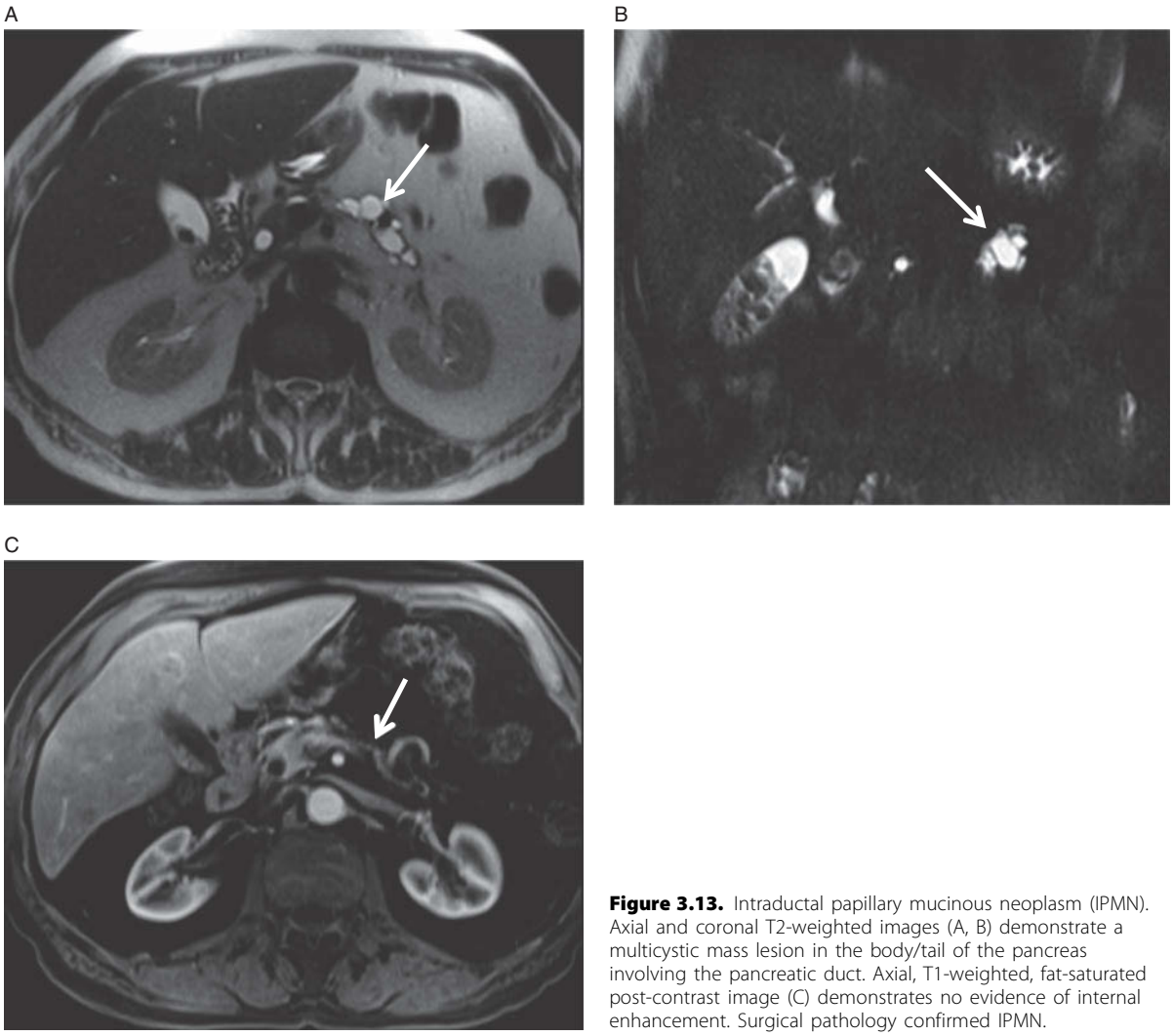

Figure 3.13. Intraductal papillary mucinous neoplasm (IPMN). Axial and coronal T2-weighted images (A, B) demonstrate a multicystic mass lesion in the body/tail of the pancreas involving the pancreatic duct. Axial, T1-weighted, fat-saturated post-contrast image (C) demonstrates no evidence of internal enhancement. Surgical pathology confirmed IPMN.

more common in women and should be resected due to malignant potential (Figure 3.12).

Small IPMNs are generally simple cystic lesions which often communicate with the main pancreatic duct and are radiologically indistinguishable from small pseudocysts. When you encounter them, and you will encounter them often, follow the previously described guidelines in Figure 3.10. When the cystic lesions begin to replace the pancreatic parenchyma and the pancreatic duct and its pancreatic ductal branches dilate, then I lean strongly on the diagnosis of IPMN and recommend surgical consultation (Figure 3.13). If the lesion communicates with the main pancreatic duct, it is far more likely to be malignant than if it communicates with a side branch.

Solid and papillary epithelial neoplasms (SPEN) are typically found incidentally in the tail of a young woman's pancreas. They, too, are cystic lesions with varying degrees of internal complexity (Figure 3.14). In terms of differential diagnosis, this diagnosis is favored simply based on young age. One interesting thing about SPENs is that they may be nearly entirely cystic.

So . . . Let's recap. Cystic lesions of the pancreas are extremely common. Most are simple, asymptomatic, incidentally discovered, and benign. Size matters – small cystic lesions are most frequently benign. The incidence of malignancy increases as they become complex internally. Follow the guidelines and you'll be fine.

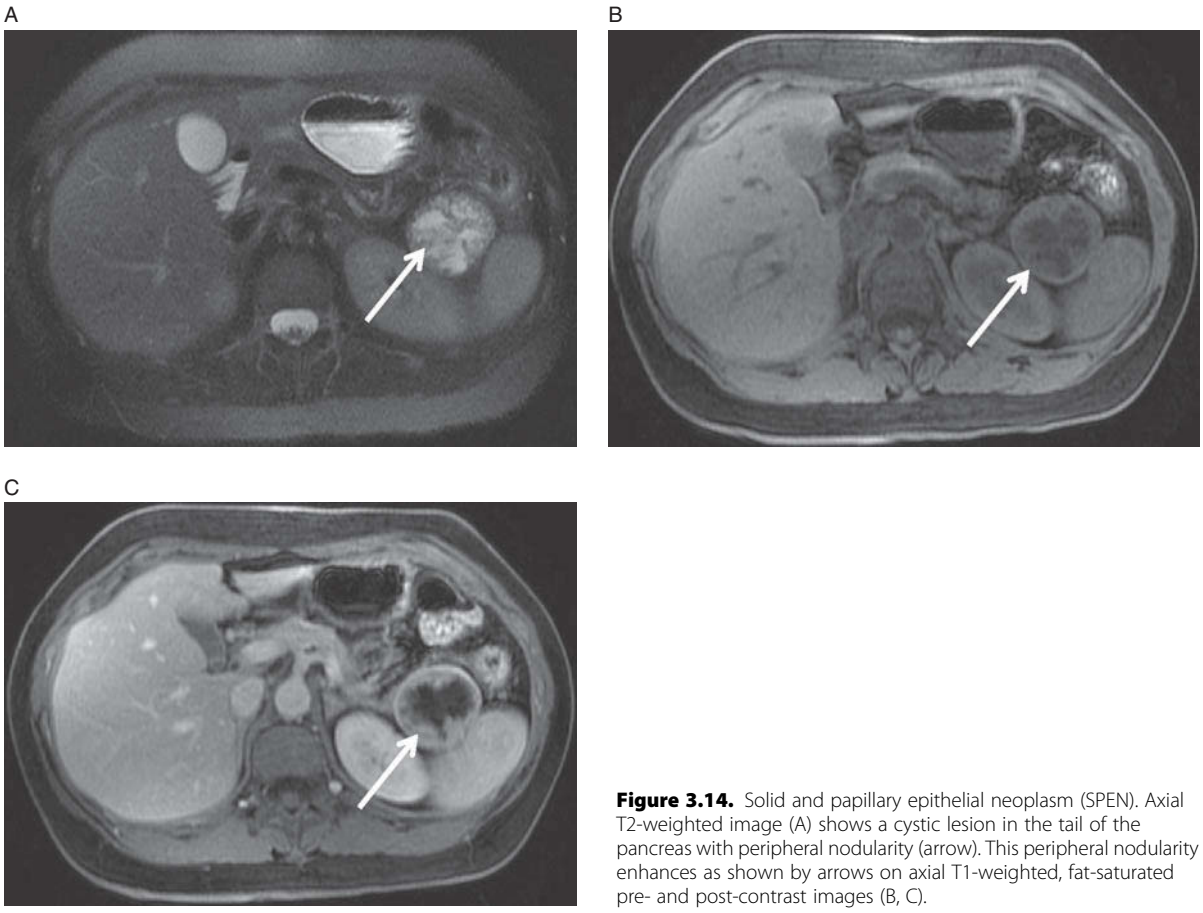

Figure 3.14. Solid and papillary epithelial neoplasm (SPEN). Axial T2-weighted image (A) shows a cystic lesion in the tail of the pancreas with peripheral nodularity (arrow). This peripheral nodularity enhances as shown by arrows on axial T1-weighted, fat-saturated pre- and post-contrast images (B, C).

In-/out-of-phase T1-weighted images, no fat saturation

True pancreatic lesions do not contain fat except for the occasional pancreatic lipoma which is composed of macroscopic fat and is not a diagnostic dilemma. However, it is not uncommon for a pancreatic lesion to be suspected on CT only to find that it loses signal on out-of-phase images and actually simply represented volume averaging with adjacent fat. I love these cases because we can give a definitive answer and be a hero. They are supremely satisfying. Imagine the difference between being told you had pancreatic adenocarcinoma and six months to live . . . or . . . well . . . just a little fat in the pancreatic head, nothing to worry about (Figure 3.15).

Remember, that, like iron in the liver, air too causes inhomogeneities in the magnetic field and will "bloom" (grow larger) on in-phase images due to their longer TE (approximately 4.4 ms vs. 2.2 ms). This can be useful in characterizing "masses" caused by duodenal diverticulae. Tumors don't contain air.

Contrast-enhanced images: Axial T1-weighted volume-interpolated gradient echo

Contrast-enhanced images are critical for evaluation of solid pancreatic masses. This is the big one. We don't want to miss it.

The normal pancreas is T1-bright. The precise reason is not known, but one theory is that it is due to the abundance of proteinaceous fluid in pancreatic enzymes. (My favorite theory is that it has to do with manganese. This may not be true, but who knows enough about manganese to dispute it?)

On pre-contrast, fat-saturated T1-weighted imaging, the normal pancreas is brighter than on in- and out-of-phase T1-weighted imaging. Remember, rather than using absolute values of density like CT Hounsfield units, the signal intensity in MRI is relative. When we use fat saturation to decrease the signal in retroperitoneal fat, we decrease the overall intensity of the image. The scanner will then automatically brighten the image,

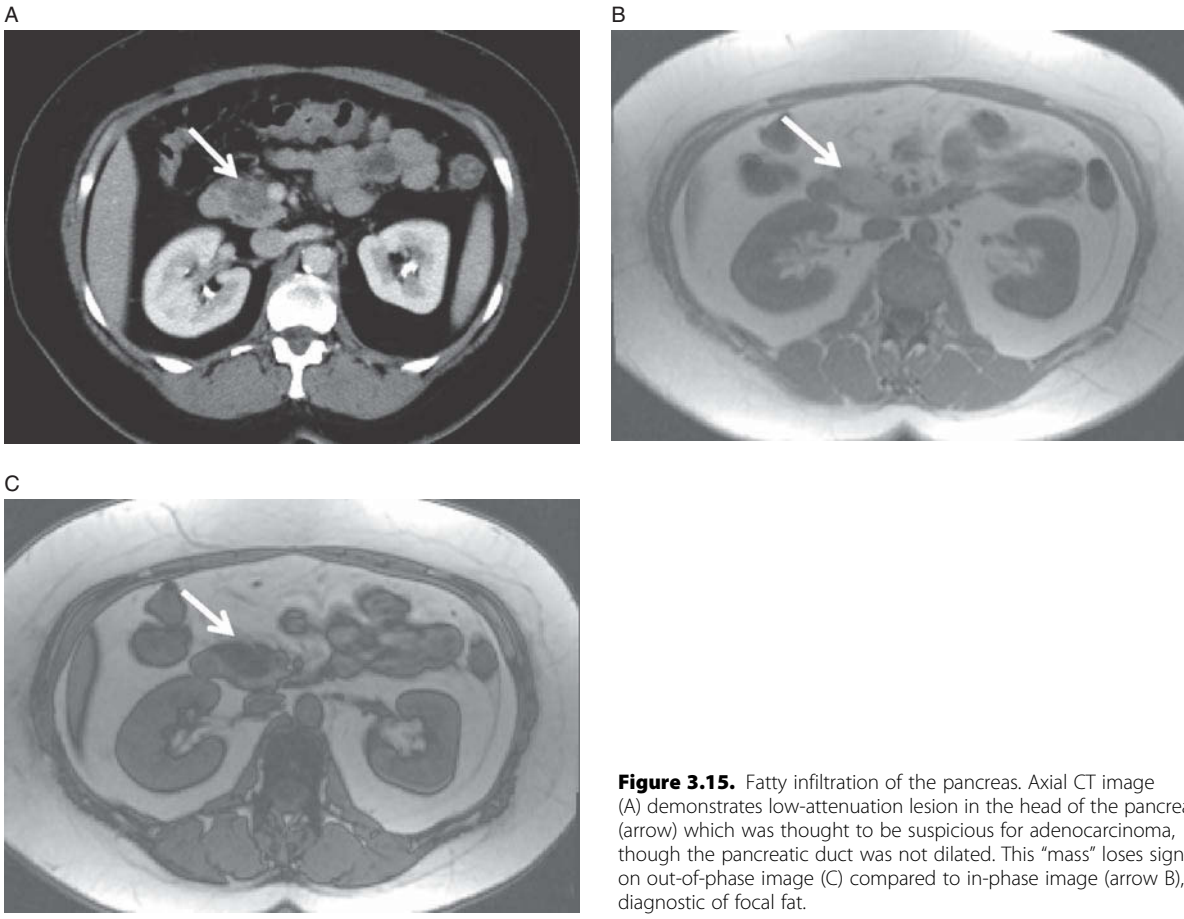

Figure 3.15. Fatty infiltration of the pancreas. Axial CT image (A) demonstrates low-attenuation lesion in the head of the pancreas (arrow) which was thought to be suspicious for adenocarcinoma, though the pancreatic duct was not dilated. This "mass" loses signal on out-of-phase image (C) compared to in-phase image (arrow B), diagnostic of focal fat.

making the pancreas look even brighter against the dark saturated background fat. This sequence is excellent to detect subtle dark lesions (such as adenocarcinoma) against the bright background normal pancreas.

Always review the in-/out-of-phase images and pre-contrast images to be sure that the signal intensity of the pancreas is consistently T1-bright – adenocarcinoma will be dark. It is also mildly bright on T2-weighted images although this is often very subtle. Adenocarcinoma does enhance on post-gadolinium enhanced images but less than normal adjacent pancreas (just like on CT) (Figure 3.16).

Neuroendocrine (islet cell) tumors of the pancreas are less common than adenocarcinoma or pancreatic cystic lesions. If they are large, they are likely "non-functioning" or they would have been discovered sooner. They are solid, hypervascular masses which light up brightly on arterial-phase images and are inconspicuous on delays. They may be heterogeneous on T2-weighted images if there is internal necrosis.

Functioning islet tumors are usually very small and are named for the hormone that they produce: insulinoma, glucagonoma and (my favorite) the VIPoma. Occasionally, patients will have elevated hormone levels and our job is to find the tumor. Look carefully for small enhancing masses on the arterial phase and diffusion-weighted images.

Summary of pancreatic lesions

Solid lesions:

(1) Pancreatic adenocarcinoma: T1 hyopintense, mildly T2-bright. Infiltrative. Look for dilatation of the pancreatic duct and atrophy of the pancreatic tail.
(2) Neuroendocrine (islet cell) tumors: T1 hyopintense, mildly T2-bright. Enhance avidly on arterial phase.

Cystic lesion:

(1) IPMN: T2-bright, T1-dark. Most common cystic lesion. Concerning features include large size and

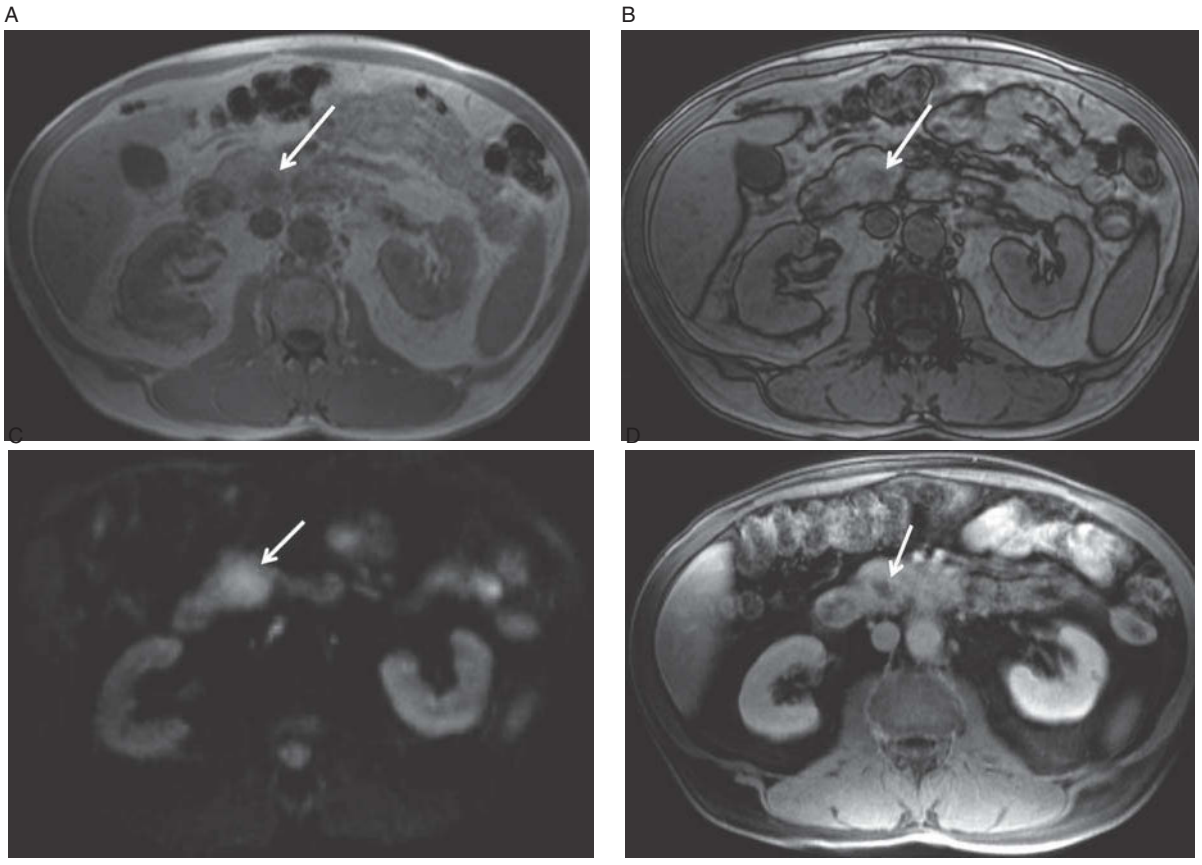

Figure 3.16. Pancreatic adenocarcinoma. Axial in-phase and out-of-phase images (A, B) demonstrate the normal bright T1 signal of the pancreas with a focal, rounded area of low signal (arrows). This should always raise your suspicion for pancreatic adenocarcinoma. Image (C) is a b-500 DWI demonstrating restricted diffusion of this mass. I include this image not because it is necessary or necessarily typical, but because it is gorgeous. Axial, T1-weighted, fat-saturated post-contrast image (D) shows the normal enhancing pancreatic parenchyma with the focal hypoenhancing mass (arrow).

enhancing mural nodules. Communicate with pancreatic duct.

(2) Pseudocyst: T2-bright, T1-dark. Usually simple. Patient should have history of pancreatitis.

(3) Serous cystadenomas: Classically microcystic and in the pancreatic head. These do not communicate with pancreatic duct.

(4) Mucinous cystadenoma/adenocarcinomas: Classically macrocystic with enhancing internal nodules and septations.

Pearls and pitfalls:

(1) Check every case for pancreas divisum.
(2) Stones sink, air floats.
(3) Don't get so lost looking for stones that you forget to look for strictures.
(4) Cystic neoplasms are *extremely* common and the vast majority are indolent.
   - The incidence of true malignancy increases with size and increasing complexity.
(5) The normal pancreas is T1-bright. Focal areas of T1-dark signal within the pancreas should put you on edge for adenocarcinoma.

Further Reading

Bilgin M, Shaikh F, and Semelka RC. Magnetic resonance imaging of gallbladder and biliary system. *Top Magn Reson Imaging* 2009 Feb; **20**(1): 31–42.

Ku YM, Shin SS, Lee CH, *et al.* Magnetic resonance imaging of cystic and endocrine pancreatic neoplasms. *Top Magn Reson Imaging* 2009 Feb; **20**(1): 11–18.

Vachiranubhap B, Kim YH, Balci NC, *et al.* Magnetic resonance imaging of adenocarcinoma of the pancreas. *Top Magn Reson Imaging* 2009 Feb; **20**(1): 3–9.

Chapter 4

Kidneys

Renal mass protocol

Indications

This protocol is used for evaluation of renal mass(es)/lesion(s), polycystic kidney disease, and hematuria.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- 2 L nasal oxygen
- At least 24-gauge IV; connect to power injector
- Subtract volume-interpolated gradient echo pre from each dynamic post volume-interpolated gradient echo post sequence.

Exam sequences

(1) Diffusion weighted imaging b50, 500/ADC – Very sensitive for lesion detection.
(2) Coronal T2 single-shot fast-spin echo FS BH – Identify T2-bright lesions and septations/nodularity within them.
(3) Axial single-shot fast-spin echo T2 non FS BH – Identify T2-bright lesions and septations/nodularity within them.
(4–5) Axial in- and out-of-phase BH (IP/OOP) – Evaluate masses for microscopic fat.
(6) Axial volume-interpolated gradient echo pre – Identify foci of T1-bright signal before contrast administration. Also assess for macroscopic fat via comparison to in-/out-of-phase images.
(7) Coronal volume-interpolated gradient echo pre – Identify foci of T1-bright signal before contrast administration.
(8) Coronal volume-interpolated gradient echo post at 30 seconds, 1 minute, and 2 minutes – Assess for enhancement.
(9) Axial volume-interpolated gradient echo at 3 minutes – Assess for enhancement.

Post processing

Subtractions corresponding to each phase of contrast enhancement above.

Approach to renal MRI interpretation

MRI is an excellent test for evaluation of renal lesions. Just like CT, the key to evaluating renal lesions is internal complexity and enhancement. MRI correctly upstages the Bosniak classification of cystic renal lesions by identifying subtle areas of enhancement. MRI also allows for more confident classification of benign lesions, typically hemorrhagic cysts. In large part, this is due to subtraction images. When masses are black on subtracted images, the lesion does not enhance, it is a benign cyst and we are happy.

Sequences

Multiplanar single-shot fast-spin echo: T2-weighted images

T2-weighted images are excellent for detecting internal complexity within cystic renal lesions as internal septations and nodularity will stand out (as dark) against the very bright cystic background. Hemorrhage within the renal lesions will be dark on T2-weighted images (and bright on T1-weighted images).

In/out-of-phase: T1-weighted images

Not all solid renal masses are malignant. Use these images to detect microscopic fat. Most renal masses which contain microscopic fat are angiomyolipomas (AML), but since clear cell renal carcinomas can also have microscopic fat, we biopsy them. In/out-of-phase images also serve as T1-weighted non-fat-saturated images which can be compared to the pre-contrast volume-interpolated gradient echo images to detect macroscopic fat (also indicative of AML) (Figures 4.1 and 4.2).

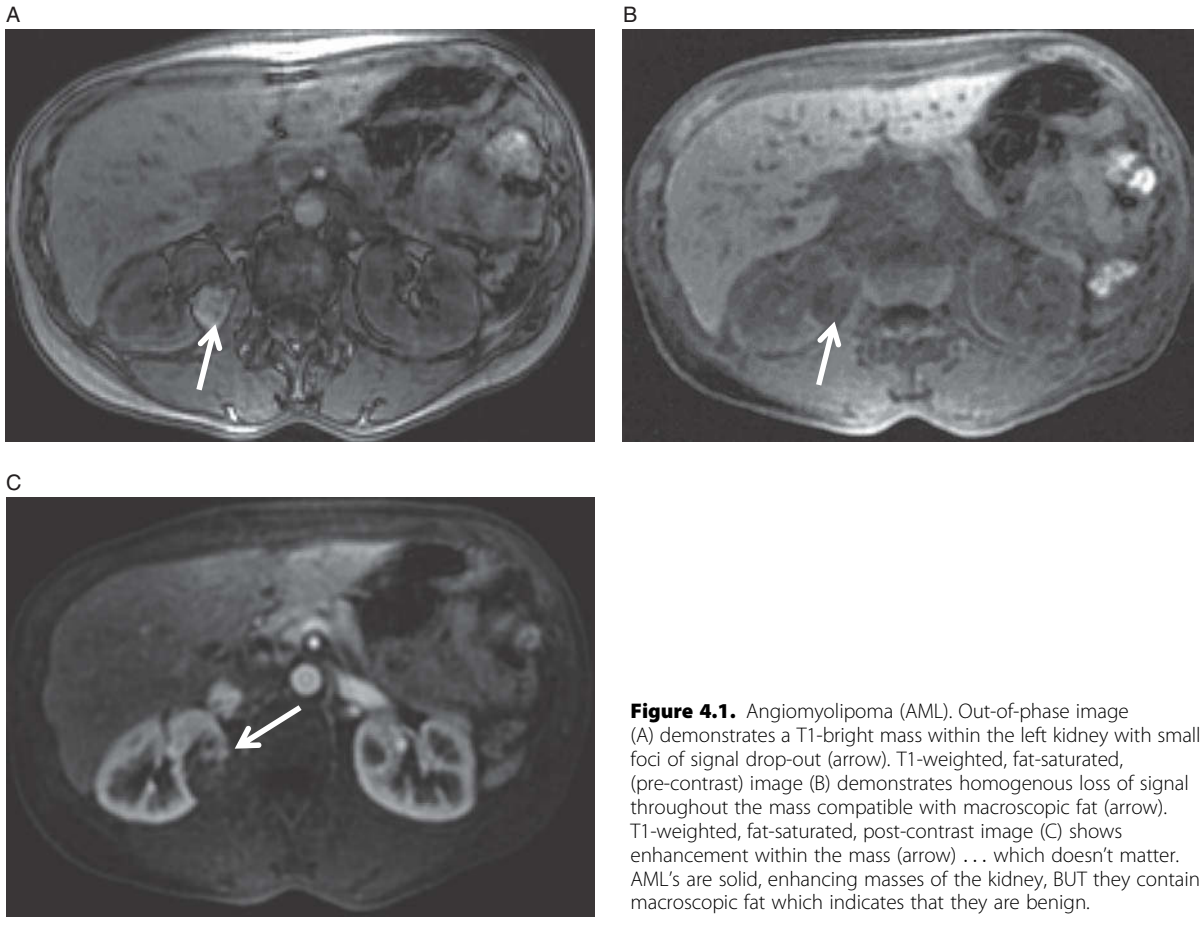

Figure 4.1. Angiomyolipoma (AML). Out-of-phase image (A) demonstrates a T1-bright mass within the left kidney with small foci of signal drop-out (arrow). T1-weighted, fat-saturated, (pre-contrast) image (B) demonstrates homogenous loss of signal throughout the mass compatible with macroscopic fat (arrow). T1-weighted, fat-saturated, post-contrast image (C) shows enhancement within the mass (arrow) . . . which doesn't matter. AML's are solid, enhancing masses of the kidney, BUT they contain macroscopic fat which indicates that they are benign.

Contrast-enhanced images: volume-interpolated gradient echo with fat saturation

Renal masses are best thought of in two broad categories: solid and cystic.

Solid renal masses

Differential diagnosis:

(1) Renal cell carcinoma (RCC)
(2) Oncocytoma
(3) Angiomyolipoma (AML)
(4) Lipid-poor AML

AMLs (#3) contain fat. The other solid renal masses are indistinguishable. Easy, right?

So, unless they contain fat, solid renal masses are considered malignant (there will, of course, be lipid-poor angiomyolipomas and oncocytomas that we will overcall). Every report of a solid renal mass should include mention of whether the tumor involves the renal veins or inferior vena cava (IVC) (Figures 4.3–4.5). (RCC is our third and final mass which can invade the IVC – the others are hepatocellular carcinoma and adrenocortical carcinoma.)

At times, it may be difficult to tell whether a tumor compresses or invades the IVC. In these cases, try real-time cine images in the sagittal plane. You'll see the blood flow through the IVC and can easily tell the difference between compression and invasion (Figure 4.6). This takes 5 minutes, but show the images in a genitourinary conference and you will have many happy (and repeat) referrers.

Patients with a known primary malignancy (other than RCC) present a bit more of a challenge. In these patients, we must also consider the possibility of

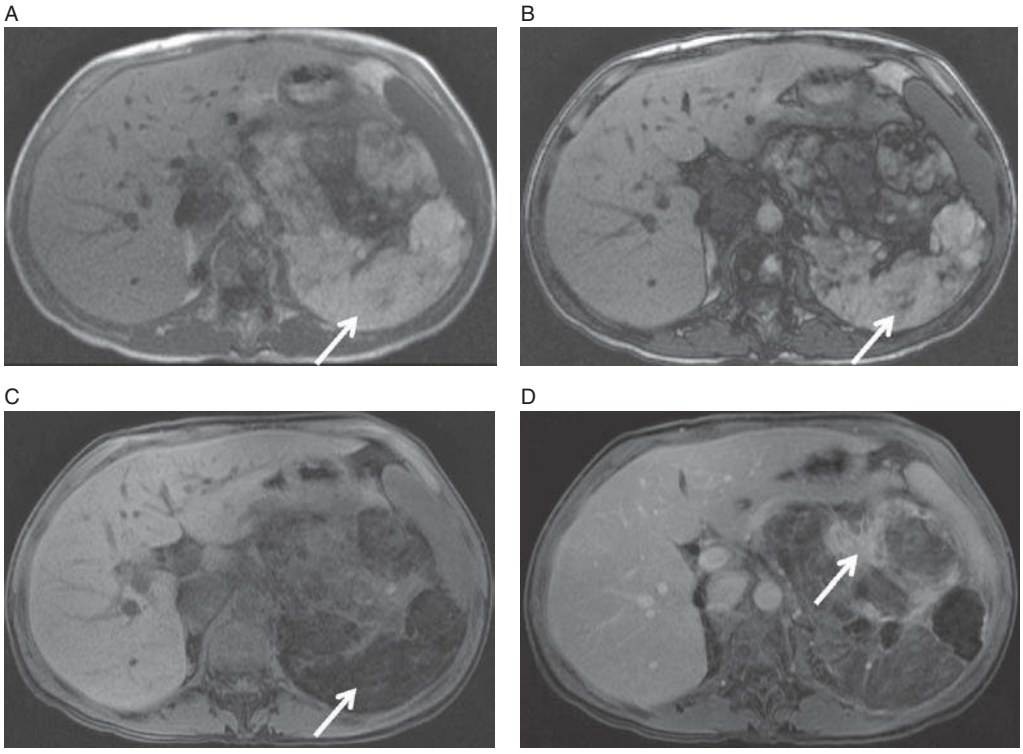

Figure 4.2. Tuberous sclerosis with multiple AMLs. In-phase image (A) demonstrates numerous fat-containing lesions within the left kidney which demonstrate signal loss on out-of-phase image (B). Pre-contrast axial T1-weighted, fat-saturated image (C) demonstrates near-complete loss of signal due to fat saturation. Post-contrast image (D) shows the residual, normally enhancing renal parenchyma (arrow).

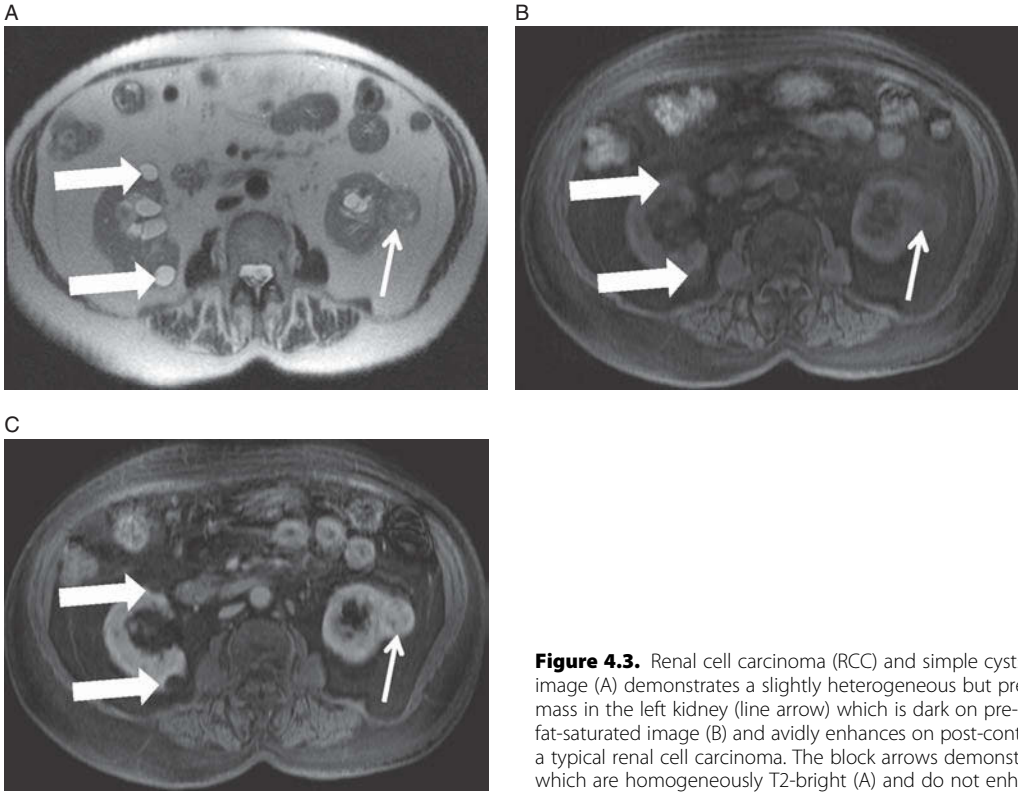

Figure 4.3. Renal cell carcinoma (RCC) and simple cysts. Axial T2-weighted image (A) demonstrates a slightly heterogeneous but predominantly T2-dark mass in the left kidney (line arrow) which is dark on pre-contrast T1-weighted fat-saturated image (B) and avidly enhances on post-contrast image (C). This is a typical renal cell carcinoma. The block arrows demonstrate simple cysts which are homogeneously T2-bright (A) and do not enhance (B, C).

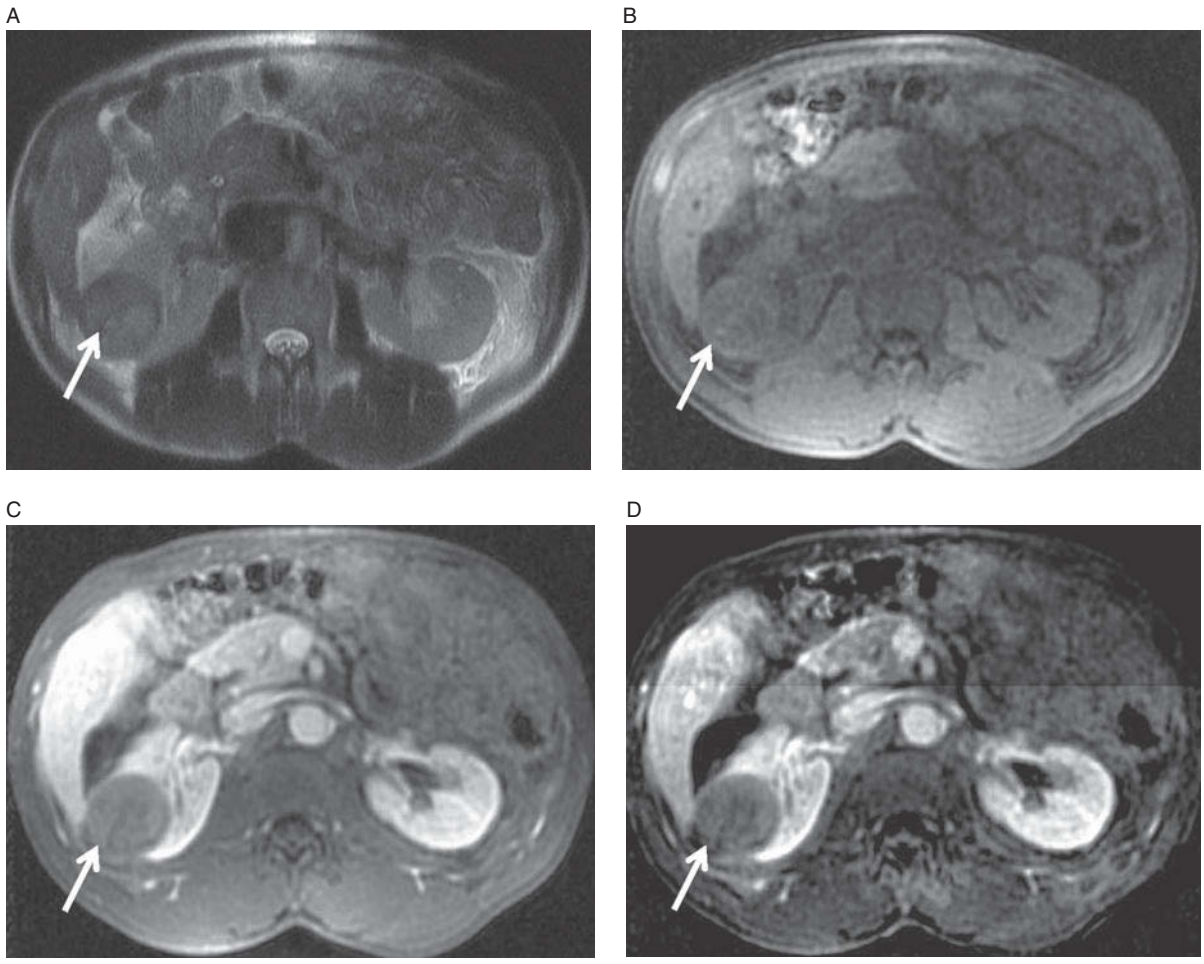

Figure 4.4. Papillary RCC (arrows). Axial T2-weighted image (A) shows a T2-dark mass in the right kidney which is dark on axial T1-weighted pre-contrast image (B) demonstrates solid, though not dramatic, enhancement on T1-weighted, fat-saturated post-contrast image (C). Subtracted image (D) shows solid, intralesional enhancement. Papillary renal cell carcinoma is frequently T2 dark.

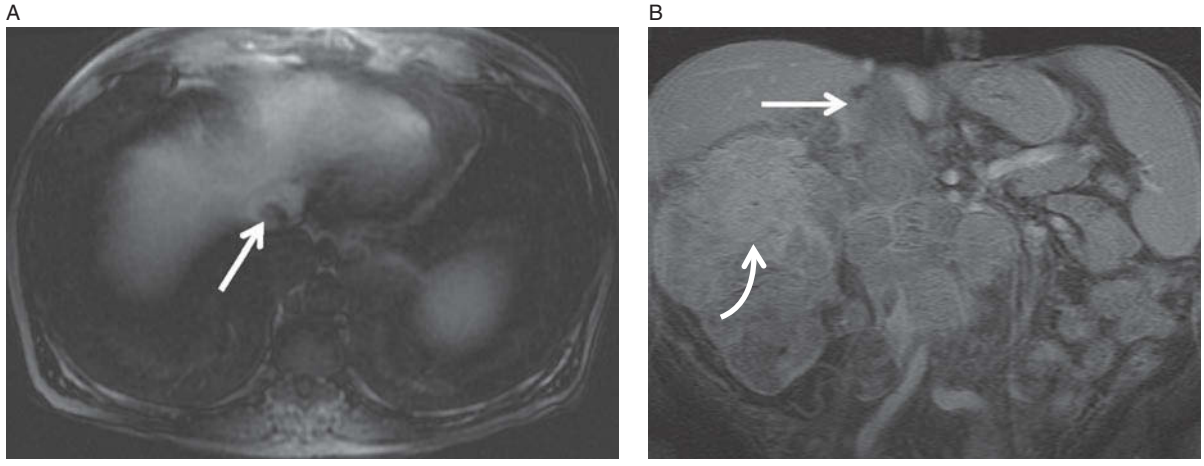

Figure 4.5. Renal cell carcinoma (RCC) invading the inferior vena cava (IVC). Axial post-contrast image (A) demonstrates filling defect within the IVC (arrow). Coronal post-contrast image (B) shows a large, heterogeneous mass replacing the right kidney (curved arrow) with tumor distending the IVC (arrow). Remember, there are three tumors that invade the IVC: hepatocellular carcinoma, renal cell carcinoma, and adrenocortical carcinoma.

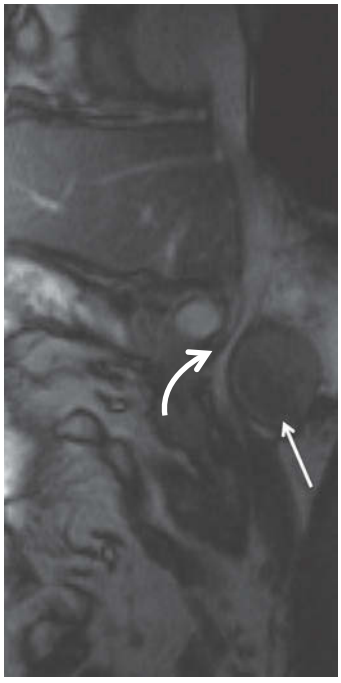

Figure 4.6. Renal cell carcinoma (arrow) compressing IVC (curved arrow). Sagittal SSFSE image taken from cine loop definitively shows compression but not invasion of the IVC.

metastatic disease to the kidney when we see an enhancing renal mass. A few words of advice:

(1) It is very rare for the kidney to be the only site of metastatic disease.
(2) Metastatic disease to the kidney enhances less than the typical RCC and is less likely to be round (Figure 4.7).
(3) If the histology of the renal lesion will change clinical management, biopsy it.

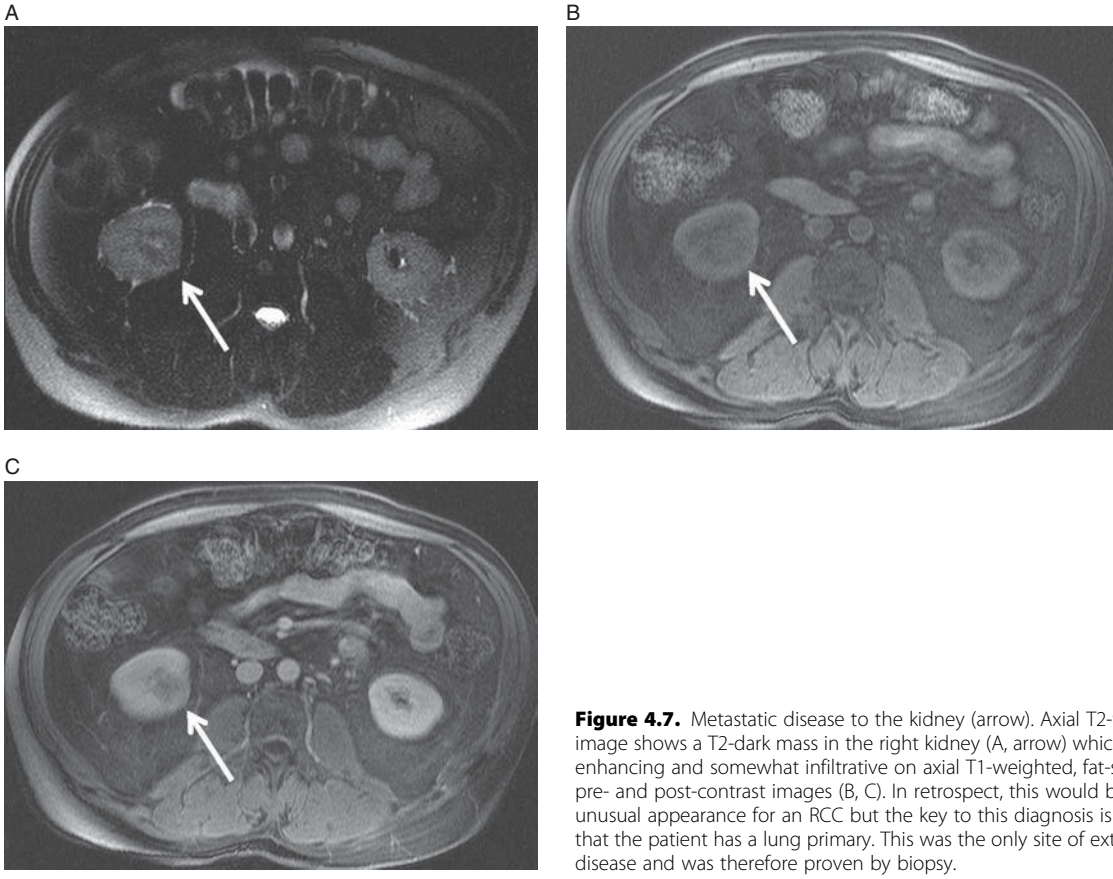

Figure 4.7. Metastatic disease to the kidney (arrow). Axial T2-weighted image shows a T2-dark mass in the right kidney (A, arrow) which is mildly enhancing and somewhat infiltrative on axial T1-weighted, fat-saturated pre- and post-contrast images (B, C). In retrospect, this would be an unusual appearance for an RCC but the key to this diagnosis is to know that the patient has a lung primary. This was the only site of extrathoracic disease and was therefore proven by biopsy.

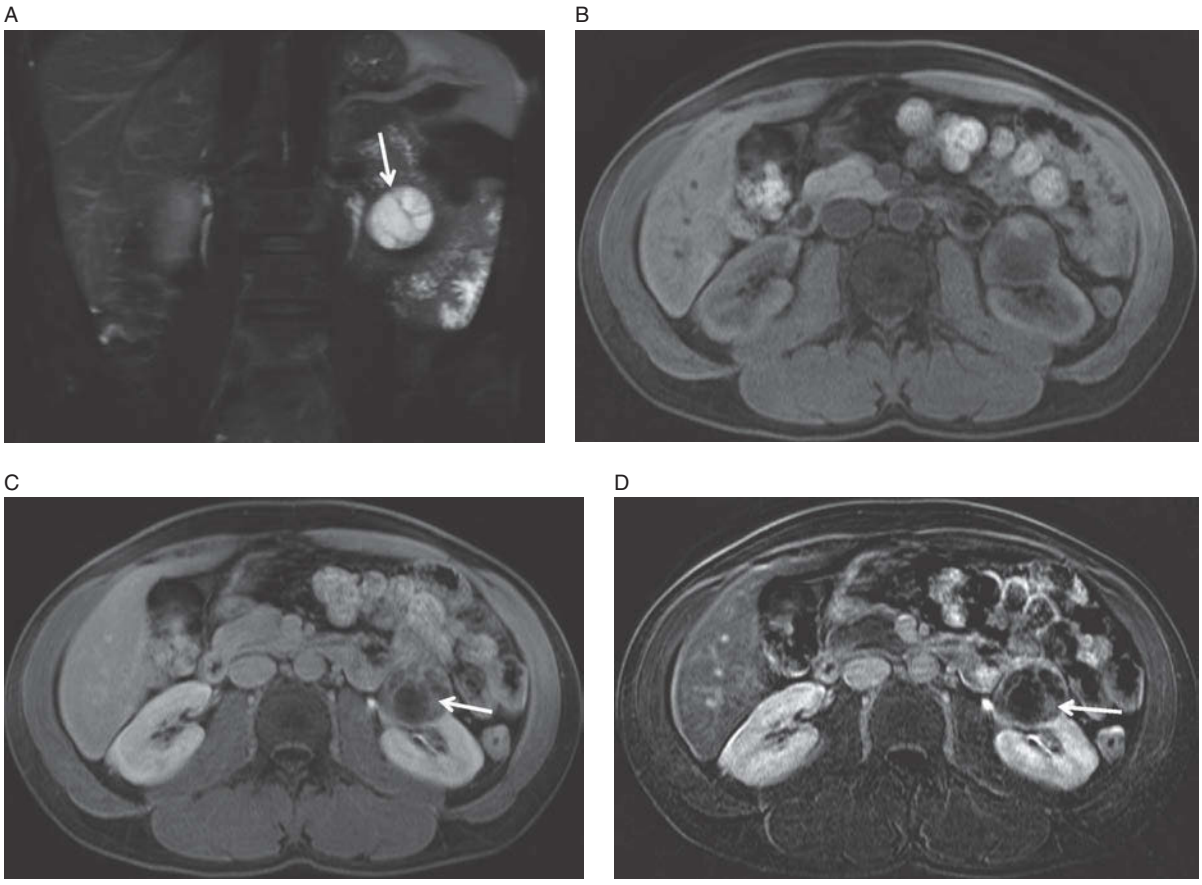

Figure 4.8. Bosniak III renal lesion (arrow). Coronal T2-weighted image (A) shows cystic lesion with multiple septations. These are too thick. Axial pre-contrast image (B) and post-contrast image (C) demonstrate enhancement of the septations with nodularity (arrow). Subtraction image (D) is provided in case there's any question of whether the enhancement is real.

Cystic masses

Cystic masses are a bit more challenging, but the Bosniak criteria apply as well to MRI as they do to CT. MRI is superior to CT at detecting subtle enhancement and nodularity which allow MRI to correctly upstage lesions compared to CT. Summarized (and blasphemed) Bosniak criteria:

Bosniak I: Simple cyst. Benign.

Bosniak II: Cyst with one or two thin septations.[1] Benign.

Bosniak IIF: A little more complex than Bosniak II, but not as complex as Bosniak III or IV. Follow in 6 months. The "F" of Bosniak IIF stands for follow.

Bosniak III and IV: Thick, enhancing septations, nodular or mass-like enhancement (Figure 4.8). Bosniak III lesions have an approximately 50% risk of malignancy. Bosniak IV lesions have a >50% chance of malignancy. Surgeons tend to treat them both as malignant.

Remember that the Bosniak criteria are for cystic lesions. Not solid masses.

(The careful reader will note that this blasphemed description of the Bosniak criteria does not even mention calcification. That's because calcification is

[1] A word on "thin." Thin means that you could draw it with a freshly sharpened #2 pencil. If it's thicker than this, it's too thick.

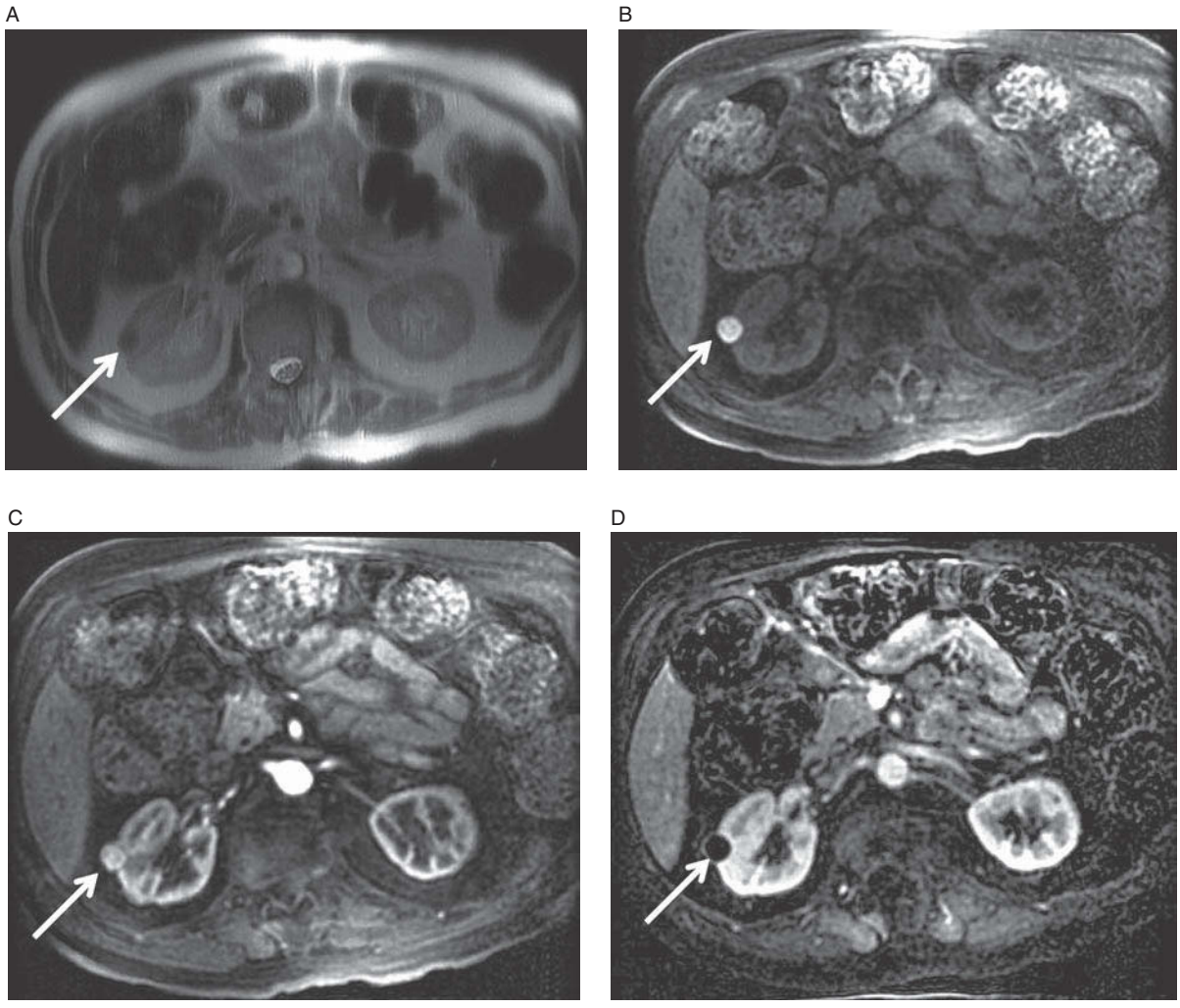

Figure 4.9. Complex renal cyst. Axial T2-weighted image (A) shows a small, T2-dark lesion within the upper pole of the right kidney. Axial T1-weighted, fat-saturated pre-contrast image (B) demonstates homogeneous T1-bright signal. If all you had was post-contrast image (C) you might think this was a solidly enhancing (and therefore malignant) mass. But, we know that the lesion was bright before contrast administration. So, we check our subtracted image (D) and see that the lesion is black – there is no true enhancement. This is a complex (proteinaceous or hemorrhagic) cyst.

often invisible on MRI. Don't worry. It turns out that calcification isn't particularly important in determining whether a lesion is benign or malignant. Enhancement is king.)

Subtraction images

Subtraction images are critical for evaluation of the kidneys. Currently, this is the only protocol in which we routinely make/use them. Complex (hemorrhagic or proteinaceous) cysts are common and can be difficult to categorize on CT. The debris within these lesions renders them bright on T1-weighted images yet our most important task is to judge whether or not they enhance. If they enhance they are malignant. If not, benign.

Obviously, this is a critical juncture so we need to be sure. The subtracted images allow confident detection of the presence or absence of true enhancement. The most common mistake that I see when interpreting subtracted images is a willingness

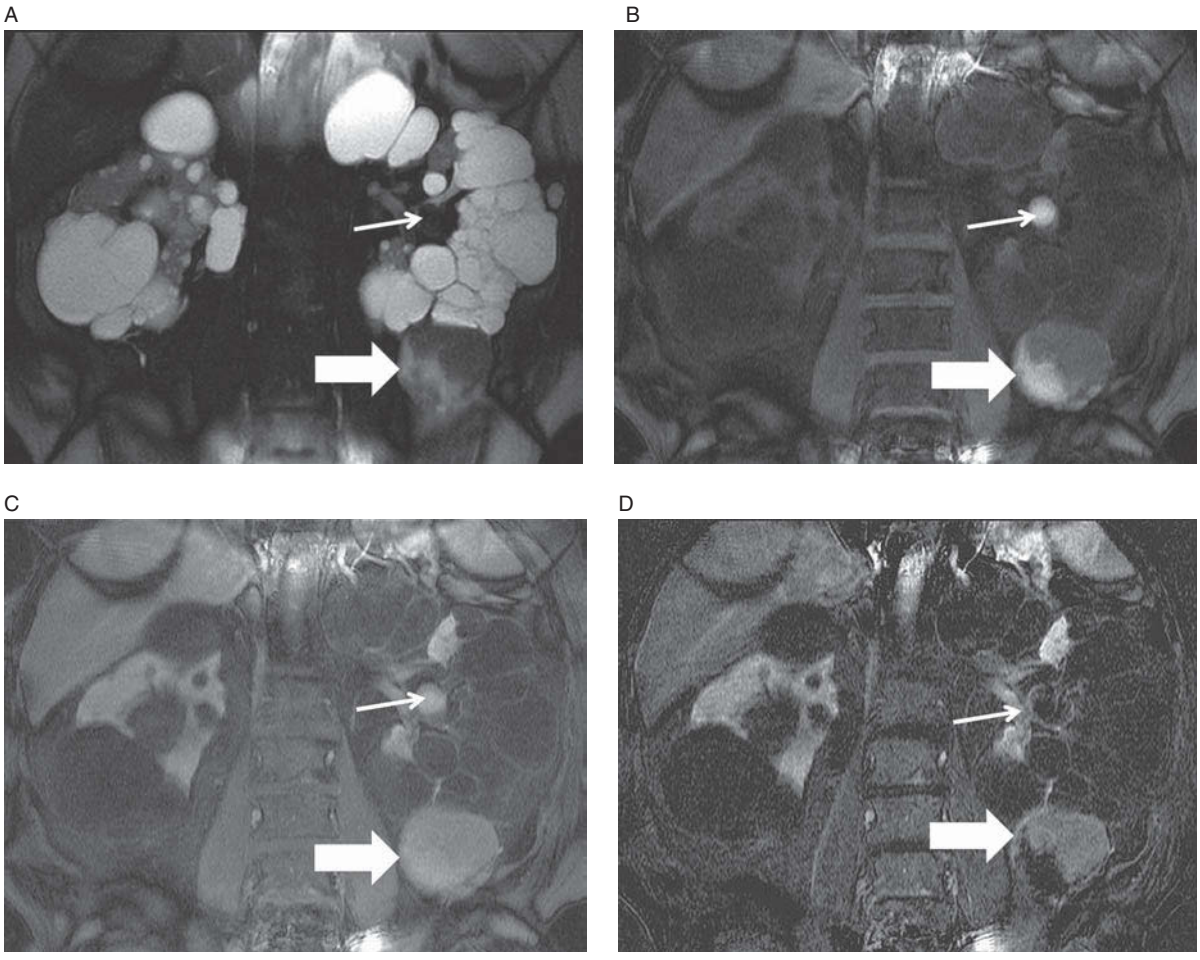

Figure 4.10. Polycystic kidney disease (PCKD) with complex cyst and renal cell carcinoma. This is quite simply the greatest example of renal subtraction imaging ever – take your time with it. Coronal T2-weighted image (A) demonstrates enlarged kidneys with innumerable renal cysts compatible with PCKD. There are innumerable simple renal cysts which are bright on T2-weighted image (A), and dark on T1-weighted, fat-saturated pre- and post-contrast images (B, C). Note the T2-dark lesion in the left interpolar region (line arrow) and the heterogeneous lesion in the left lower pole (block arrow). Axial T1-weighted, fat-saturated pre-contrast image (B) shows T1-bright signal within the interpolar lesion and heterogeneous signal within the lower pole lesion. Quite frankly, it's tough to figure out what's happening in post-contrast image (C). Is there any true enhancement . . . if so, where? Subtracted image (D) saves the day. The interpolar lesion loses all signal = no enhancement = benign, complex cyst. The lower pole lesion demonstrates mass-like enhancement in its superior portion = renal cell carcinoma.

to allow a bit of "grayness" pass for no enhancement. *Complex cysts should be black on subtracted images*. Otherwise, we must suspect that there is internal enhancement suspicious for malignancy (Figures 4.9 and 4.10).

For the sake of honesty, it is often not quite this easy because of misregistration artifacts which occur when the patient takes a slightly different breath before the pre- and post-contrast images. Although I don't often admit it, this is one of the reasons to do multiple phases after contrast administration. If you do three phases post-gadolinium, usually at least one will be relatively misregistration-free.

Pearls and pitfalls

(1) Solid, enhancing renal masses without fat (microscopic or macroscopic) suggest renal cell carcinomas.[2]
(2) Solid, enhancing masses macroscopic with fat are benign angiomyolipomas.
(3) Simple cysts or complex cysts which are black on subtracted images are benign.
(4) Cystic lesions with one or two pencil-thin enhancing septations are benign.
(5) Any complexity or nodularity beyond the above requires follow-up, biopsy, or resection.
(6) T1 bright, non-enhancing renal masses are benign hemorrhagic cysts.

[2] Some aren't. Some will be oncocytomas and some will be lipid-poor AMLs, but we call them all renal cell carcinomas.

Summary of lesions

(1) Solid masses:
  (a) No fat = renal cell carcinoma, oncocytoma, lipid-poor AML (can't tell between these three, >85% will be RCC)
  (b) Macroscopic fat = AML
(2) Cystic lesions:
  (a) Cysts +/− hemorrhage and proteinaceous debris = no enhancement = benign
  (b) Cystic lesions with enhancing internal complexity = apply Bosniak criteria

Further reading

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Chapter 5

Adrenal glands

Adrenal MRI protocol

Indications

This protocol is most frequently used to characterize incidentally detected adrenal masses (incidentalomas) found on other imaging modalities.

Preparation

- **IV contrast agent**: None used routinely. If radiologist chooses to administer contrast, use 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- At completion of exam, page radiologist to check if intravenous contrast is necessary
- Give patient 2 L nasal oxygen
- Continue axial single-shot fast-spin echo FS to level of bladder

Exam sequences

(1) Coronal T1 spoiled gradient echo BH – Anatomic overview. Assess for adrenal mass. Be certain that "adrenal" masses are really in the adrenal gland.

(2–3) Axial T1 in and out of phase (IP/OOP) – Assess for intracellular lipid (microscopic fat) within adrenal lesions. This is by far the most important sequence for the adrenal gland.

(4) Axial T2 single-shot fast-spin echo – Identify T2-bright lesions within the adrenal glands.

(5) Axial T2 single-shot fast-spin echo FS – Compare to prior sequence to identify *macro*scopic fat.

(6) Radiologist check for contrast. If contrast is to be used run:

(7) Volume-interpolated gradient echo BH pre.

(8) Volume-interpolated gradient echo BH post 20 seconds.

(9) Volume-interpolated gradient echo BH post 1 minute.

(10) Volume-interpolated gradient echo BH post 2 minutes.

Approach to adrenal MRI interpretation

Adrenal masses are extremely common, estimated to occur in up to 7% of the adult population. The overwhelming majority of adrenal masses are benign, non-functioning adenomas. Adrenal masses are most commonly detected on an imaging exam performed for other purposes, for example an abdominal CT performed for abdominal pain. They are the classic "incidentaloma."

I think of adrenal masses in two categories: patients without a history of malignancy and patients with a history of malignancy.

In patients without a known history of malignancy: a recent retrospective study of 1300 incidentally discovered adrenal masses found a zero incidence of malignancy, that's right – zero[1].

In patients with a *known* malignancy, incidentally detected adrenal masses are still almost always benign, and not a metastasis. In these patients, however, we must prove it.

Imaging characterization of an adrenal mass uses two separate physiologic properties to differentiate benign adrenal adenomas from more concerning lesions such as metastases or the very rare adrenocortical carcinoma.

The first is the presence of intracellular lipid (which is high in 80% of benign adrenal adenomas and is not present in metastases or adrenal carcinomas.) Both CT and MRI are excellent at this. The second property is adrenal perfusion, measured by assessing adrenal enhancement on dynamic imaging and washout on 15-minute delayed imaging. CT is excellent at this. While it is possible, we don't really do this with MRI.

Since both CT and MRI are useful at characterizing an incidentally discovered adrenal mass, how do you decide which one to use? At our institution, CT is the workhorse depending on patient age. (A non-contrast-enhanced exam is performed first

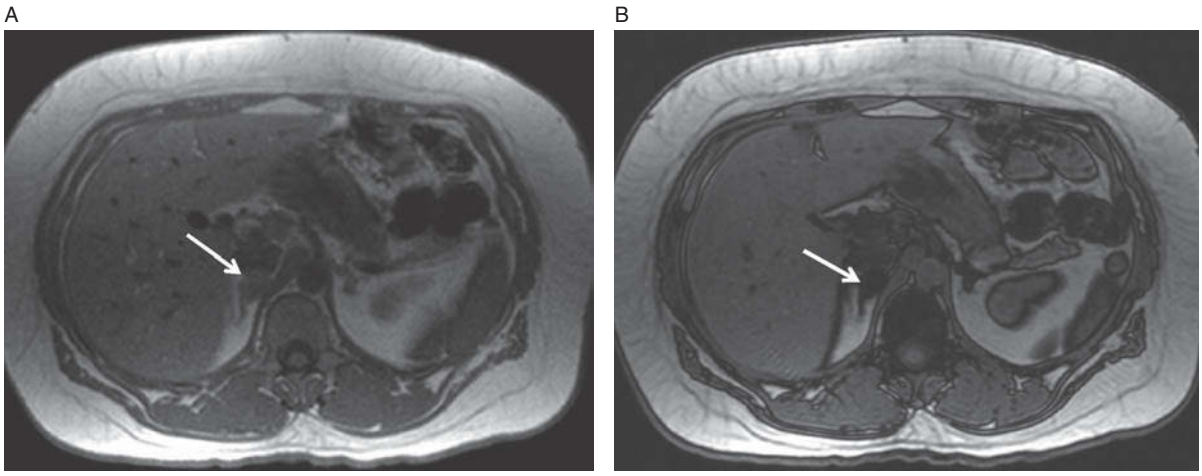


Figure 5.1. Adrenal adenoma. In-phase (A) and out-of-phase (B) images. The mass indicated by the arrows is brighter than the spleen in image A and darker than the spleen in image B. This is a classic adrenal adenoma. Based on imaging alone, we do not know whether this is functioning or non-functioning. That distinction requires biochemical analysis. Would this mass enhance if we gave contrast? Yes. Would seeing it enhance help us? No. So don't give contrast.

and if the HU $\leq$10, the lesion is an adenoma. If the HU is >10, then contrast is administered and washout is calculated.) We use MRI to characterize an incidentally discovered adrenal mass discovered in younger patients where radiation is a potential concern.

It is important to note that 80% of benign adenomas have increased intracellular lipid, which means 20% do not. Thus, the presence of intracellular lipid (low density on CT, signal drop-off on out-of-phase imaging) guarantees the lesion is an adenoma, but the absence of these findings does not mean the lesion is a metastasis or adrenocortical carcinoma. It could be either a lipid-poor adenoma (most likely) or a metastasis. In these cases, biopsy may be performed.

One final note. While imaging is very useful to differentiate adenomas from other adrenal tumors, it does *not* tell us whether the mass is functional (i.e. making hormones). Biochemical analysis is the only way to determine whether an adrenal mass is functional or not. Our job is to reassure the patient and the referring physician that the adrenal mass is benign.

Sequences

In/out of phase: T1-weighted, no fat-saturation

This is the key sequence for adrenal imaging. Although signal intensity calculations may be performed, it is easier and just as accurate to "eye" it. Use the spleen or skeletal muscle as an internal control. If a lesion is brighter than the spleen on in-phase imaging, and darker on out-of-phase imaging, it contains microscopic fat and is therefore an adenoma (Figure 5.1). As a general rule, if you are unsure whether the lesion dropped sufficiently in signal – it did not.

Beware of mistaking the etching artifact at the periphery of masses for signal drop. We are looking for loss of signal in the center of the mass. This can be tricky in small lesions. But, be reassured, in the absence of known cancer, they are almost all benign anyway.

Not all adenomas will demonstrate signal loss on in/out-of-phase imaging. Some adenomas simply do not have enough fat within them. These are referred to as lipid-poor adenomas. Some of these lesions may be characterized by "washout" CT as described above or simply followed.

Metastases are the most common malignant mass in the adrenal gland. I only suggest that an adrenal lesion may be a metastasis when there is *both*:

(1) An adrenal mass that does not contain fat (micro- or macroscopic)
(2) A history of malignancy.

Even if these criteria are met, it may not be a metastasis. It still may be a lipid-poor adenoma and biopsy is required (Figure 5.2).

Adrenocortical carcinoma is a primary malignancy of the adrenal gland. It is extremely rare. These masses are typically over 5 cm in size and quite ugly at the time of diagnosis (Figure 5.3). If you see a large,

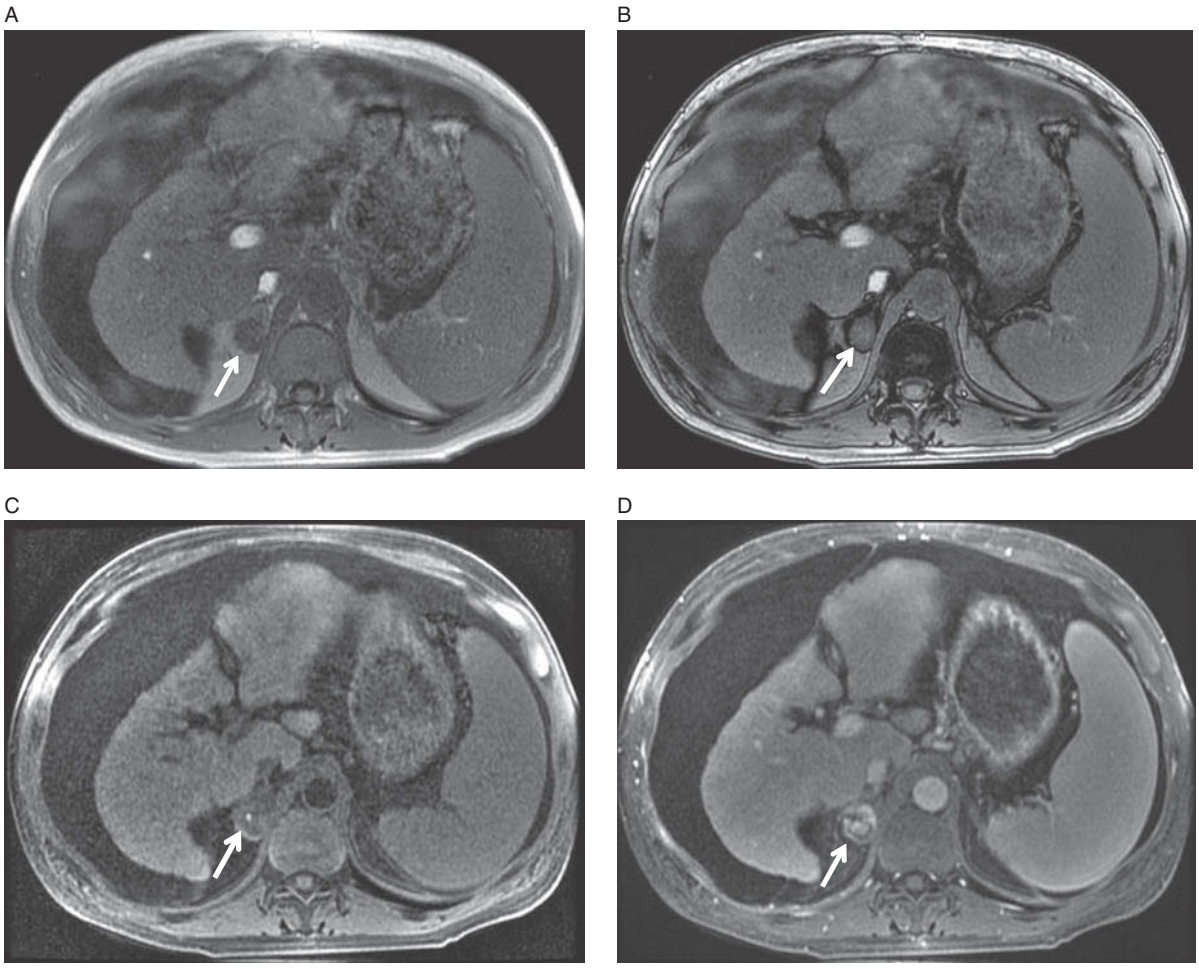


Figure 5.2. Adrenal metastatis. Arrows indicate right adrenal mass which does not change in signal between in-phase image (A) and out-of-phase image (B). Axial T1-weighted, fat-saturated pre- and post-contrast enhanced images (C, D) demonstrate significant enhancement. Differential diagnosis includes lipid-poor adenoma and metastatic disease. Biopsy revealed metastatic hepatocellular carcinoma; note cirrhosis and ascites.

heterogeneous mass with irregular margins in the adrenal gland in a patient with no history of cancer or other metastases, suggest this diagnosis.

(One other quick pearl: there are three tumors that classically invade the IVC. They are: hepatocellular carcinoma, renal cell carcinoma, and adrenocortical carcinoma.)

SSFSE: T2-weighted images

Pheochromocytomas are tumors of the adrenal medulla which may produce catecholamines. Most are benign and occur within the adrenal gland; however, they can occur at the organ of Zuckercandl at the level of the aortic bifurcation. This is why we always continue SSFSE imaging to the level of the bladder.

They are classically bright on T2-weighted images. Keep in mind, however, that they do not have to be "light-bulb" bright as is often described in textbooks. They may vary significantly, but most are at least slightly T2-bright (Figure 5.4). Like all other solid adrenal masses, they enhance if you give gadolinium. (There are cystic pheochromocytomas, but even these should have some solid, enhancing component.)

We currently perform SSFSE with and without fat saturation. It is important to compare these two sequences to detect macroscopic fat. If an adrenal mass contains macroscopic fat, it is a myelolipoma. Myelolipomas are nice because we can diagnose them definitively, and they have no malignant potential.

Remember adenomas contain *micro*scopic fat, myelolipomas contain *macro*scopic fat. Both are benign, but adenomas may make and secrete hormones.

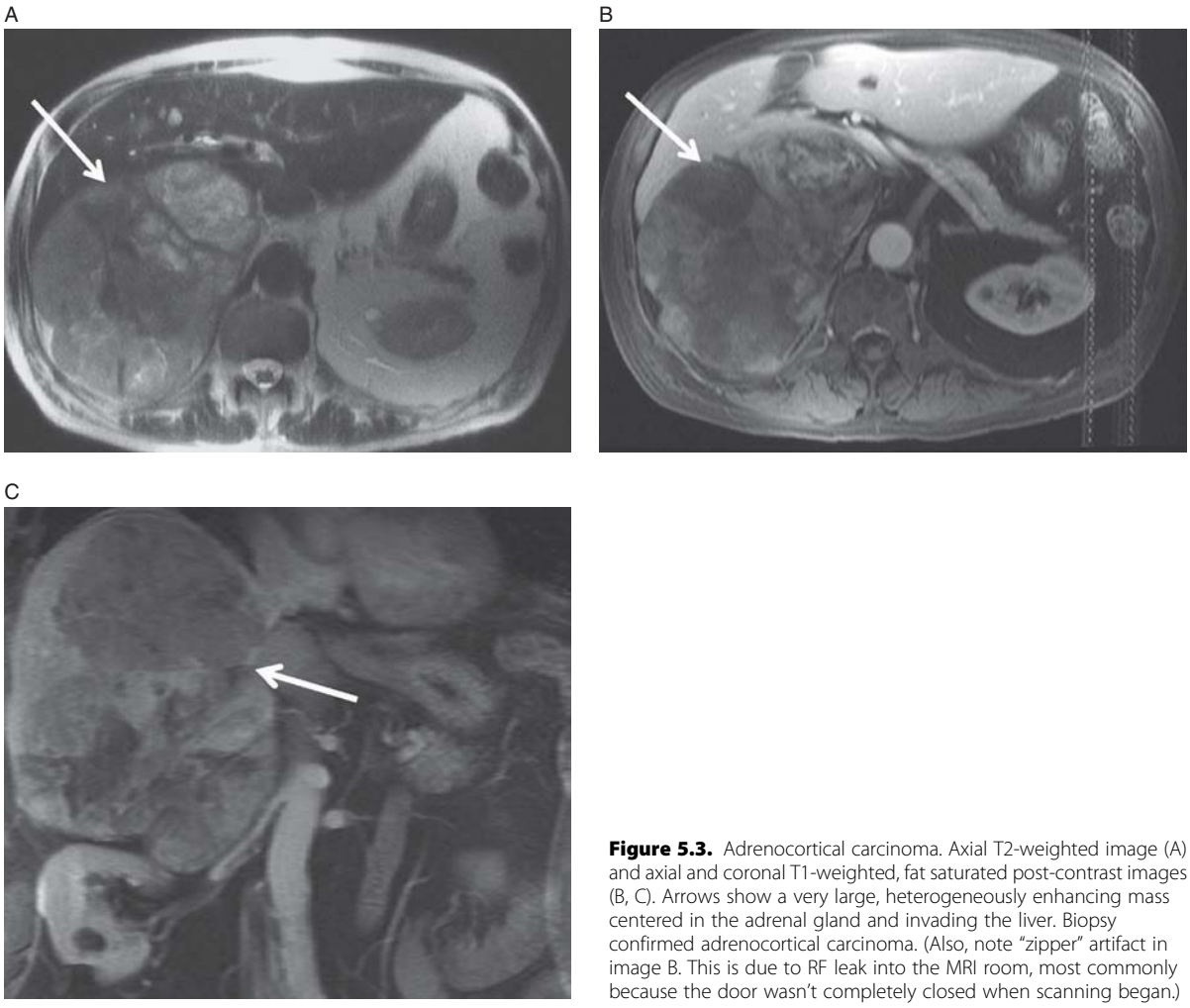


Figure 5.3. Adrenocortical carcinoma. Axial T2-weighted image (A) and axial and coronal T1-weighted, fat saturated post-contrast images (B, C). Arrows show a very large, heterogeneously enhancing mass centered in the adrenal gland and invading the liver. Biopsy confirmed adrenocortical carcinoma. (Also, note "zipper" artifact in image B. This is due to RF leak into the MRI room, most commonly because the door wasn't completely closed when scanning began.)

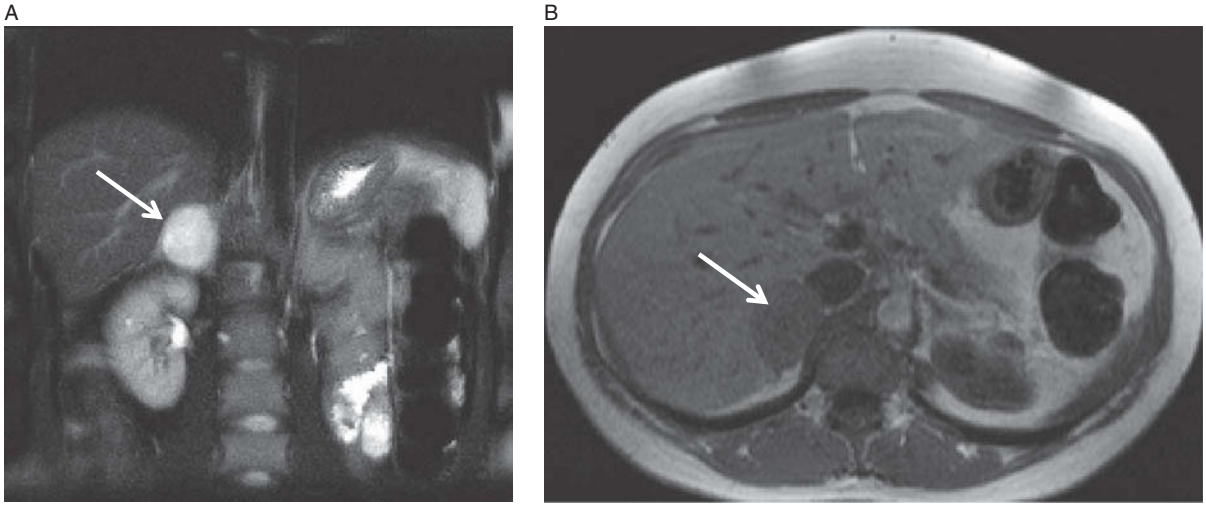


Figure 5.4. Pheochromocytoma (arrow). Coronal T2-weighted image (A) demonstrates a T2-bright mass within the right adrenal gland. Axial out-of-phase (C) when compared to IP imall (B) images demonstrate no significant signal loss to suggest microscopic fat. Would this mass enhance if we gave contrast? Yes. Would seeing it enhance help us? No. The appropriate next step is biochemical analysis *not* biopsy.

C

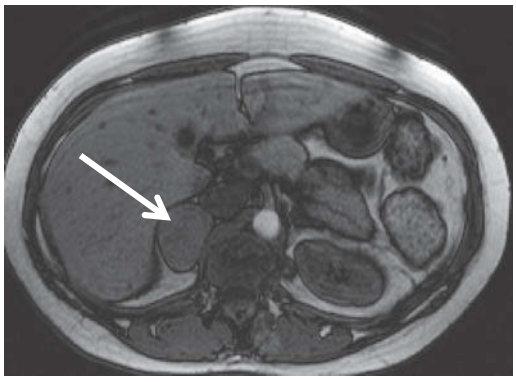


Figure 5.4. (cont.)

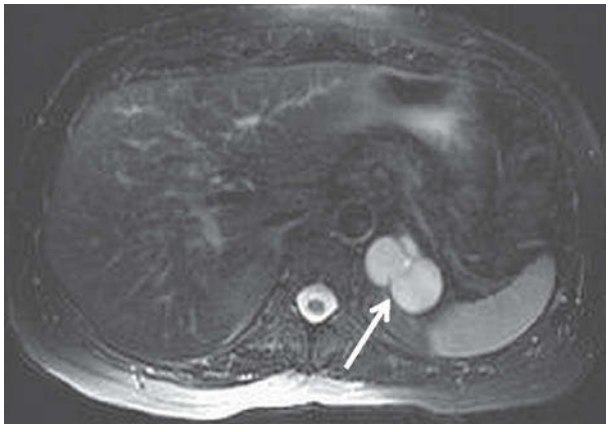
Volume-interpolated gradient echo

All solid adrenal masses enhance. Because they all enhance, giving gadolinium doesn't usually help us. We only give gadolinium in two scenarios:

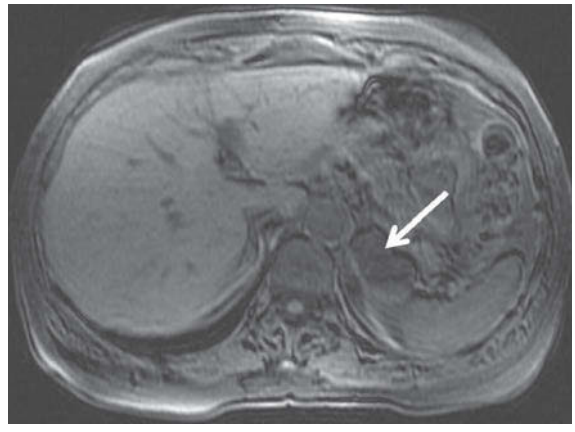
(1) The very, very T2-bright mass. In these cases, the differential diagnosis includes pheochromocytomas and the very rare adrenal cyst or pseudocyst. Pheochromocytomas will enhance and adrenal cysts will not (Figure 5.5).

(2) The T1-bright mass. A T1-bright mass within the adrenal gland is usually indicative of adrenal hemorrhage which is actually fairly common. Occasionally we will be asked if there is an underlying mass that bled or if the bleeding was "spontaneous." Contrast (and subtraction images) can help in these rare cases.

A



B



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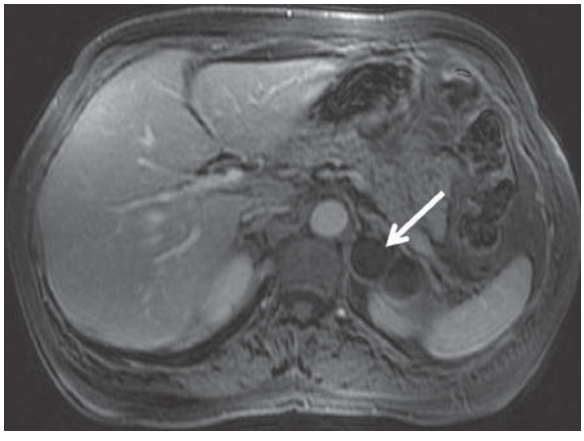

Figure 5.5. Adrenal cyst. This is really a great case. Axial T2-weighted image (A) shows a septated T2-bright mass in the left adrenal gland (arrow). This patient had been told that she had a pheochromocytoma and the mass had been followed for years. But, when a clever endocrinologist noticed that her biochemical profile was, and always had been, normal we decided to give gadolinium. Axial T1-weighted, fat saturated pre-contrast image (B) and post-contrast image (C) demonstrate no evidence of internal enhancement. Pheochromocytomas, despite being T2-bright, are solid masses and would therefore enhance.

Summary of lesions

(1) Fat-containing adrenal lesions = benign
   (a) Microscopic fat = adenoma, suggest biochemical analysis if patient has signs or symptoms
   (b) Macroscopic fat = myelolipoma, needs nothing

(2) No fat in lesion:
   (a) Lipid-poor adenoma = most common
   (b) Metastasis = should have known primary, biopsy may be necessary to confirm
   (c) Pheochromocytoma = T2-bright, abnormal metanephrines
   (d) Adrenal cortical carcinoma: large, very ugly, very rare.

Pearls and pitfalls

(1) Adrenal masses are almost always benign adenomas.
(2) Signal loss on out-of-phase images indicates intracellular lipid = adenoma.
(3) We cannot differentiate a functioning from a non-functioning adenoma. Biochemical testing is required.
(4) All solid adrenal masses enhance.
(5) T2-bright lesions may represent pheochromocytomas. If they are as bright as CSF, consider giving gadolinium to be certain it's a solid mass, then recommend biochemical testing.

Further reading

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Boland GWL, Blake MA, Hahn PF, and Mayo-Smith WW. Incidental adrenal lesions: principles, techniques, and algorithms for imaging characterization. *Radiology* 2008 Dec; **249**(3): 756–75.

Song JH, Chaudhry FS, and Mayo-Smith WW. The incidental adrenal mass on CT: prevalence of adrenal disease in 1,049 consecutive adrenal masses in patients with no known malignancy. *AJR* 2008 May; **190**(5): 1163–8.

Chapter 6

MRI enterography

MRI enterography protocol

Indications

This protocol is used to evaluate patients' with known or suspected Crohn's disease.

Preparation

- **IV contrast**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast**: Have patient begin drinking the first 450 cc barium sulfate 0.1% 30 minutes prior to exam time. Give patient second 450 cc barium sulfate 0.1% 15 minutes prior to exam time. At exam time, give 450 cc water
- 2 L nasal oxygen
- Start IV with at least 24-gauge needle; connect to power injector
- Use 2-phased array coils to cover entire abdomen and pelvis
- Glucagon IV 0.5 mg

Exam sequence

(1) Axial True FISP BH – Cover entire abdomen and pelvis. Anatomic overview. Identify suspicious bowel segments.
(2) Coronal True FISP BH – Cover entire abdomen and pelvis. Anatomic overview. Identify suspicious bowel segments.
(3) Axial T2 single-shot fast-spin echo FS BH – Cover entire abdomen and pelvis. Identify T2-bright signal within or adjacent to bowel wall. Evaluate for perianal disease.
(4) Coronal T2 single-shot fast-spin echo FS BH – Cover entire abdomen and pelvis. Identify T2-bright signal within or adjacent to bowel wall.
(5) Coronal volume-interpolated gradient echo BH pre-contrast – Identify anything T1-bright which could be mistaken later for enhancement.
(6) Coronal volume-interpolated gradient echo BH post-contrast 35 seconds – Identify abnormal mucosal hyperenhancement implying acute Crohn's disease.
(7) Axial volume-interpolated gradient echo BH post-contrast $\sim$ 2 minutes – Cover entire abdomen and pelvis.
(8) Coronal volume-interpolated gradient echo BH post 3 minutes – Identify delayed enhancement which, in the absence of early enhancement, indicates chronic, fibrotic disease.

Approach to interpretation of MRI enterography exams

They said it couldn't be done. Certainly, you would never use MRI to image the small bowel.

Times have changed. MRI enterography is a fairly new MRI application targeted at evaluation of known or suspected Crohn's disease (ulcerative colitis confines itself to the colon and is therefore well within the reach of the endoscope).

MRI enterography is an excellent test for two reasons. First, patients with Crohn's disease are often diagnosed at a young age and may require numerous examinations throughout their lifetime – MRI enterography minimizes their radiation exposure. Second, the holy grail of inflammatory bowel disease (IBD) imaging is to distinguish active inflammation from chronic fibrostenotic disease – this distinction is absolutely critical to patient management because the treatment is very different.

Patients with active inflammation are treated with steroids or "biologics" (powerful anti-inflammatory agents which to date are targeted against tumor necrosis factor. These drugs are highly effective for active inflammatory disease but useless for chronic, fibrostenotic disease. They are also expensive and potentially highly toxic (a small percentage of patients receiving these medications have been reported to develop

lymphoma). Patients with symptomatic, fibrostenotic disease may require surgical intervention to remove the stenotic segment. If we can prospectively predict who will respond to medical therapy and who will require surgery we can be heroes. Likely unknown heros, but heros none the less.

MRI is also the test of choice for detection of common complications of Crohn's disease such as abscess and fistula. These complications, particularly perianal fistulae, are well delineated with MRI and provide critical anatomic information for the surgeon (perianal fistulae are discussed in depth in the pelvic MRI section).

I love these studies, because I believe that we are doing good for patients. But they are not easy to read. There are a lot of images and the T2 hyperintensity in the bowel wall can be subtle and difficult to detect. My colleagues and the residents often groan when they arrive in the queue.

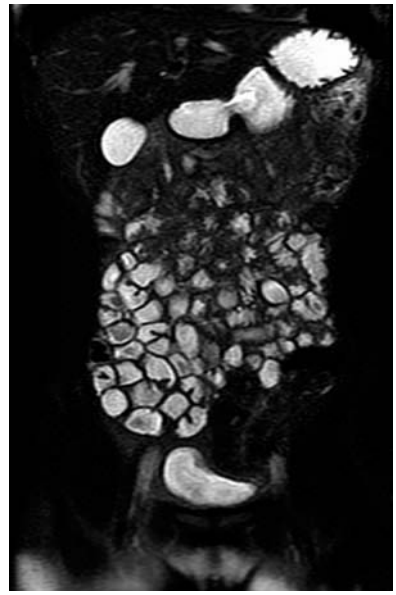

Figure 6.1. Coronal SSFSE with fat saturation demonstrates excellent small bowel distension. This is what we are hoping to achieve in all patients.

Sequences

Oral contrast

Barium sulfate 0.1% is currently administered as oral contrast to distend the small bowel. The contrast is dark on T1-weighted imaging and bright on T2-weighted imaging. Oral contrast agents vary across institutions, but all are hyperosmolar to distend the small bowel.

Don't tell the vendors, but the actual small bowel distension achieved is quite variable. However, adequate small bowel distension can be achieved in the majority of patients using the following protocol (Figure 6.1).

We ask patients to begin drinking 30 minutes before their exam begins. They drink the first bottle of 450 cc oral contrast over 15 minutes. They then drink a second bottle over the next 15 minutes followed by 450 cc water. The manufacturer recommends a third bottle of contrast rather than the water, but remember that the purpose of the contrast is to distend the distal small bowel without being resorbed. Water is just fine for distension of the upper tract, is cheaper, tastes better, and doesn't cause diarrhea.

It is worthwhile to inform your patients that the contrast is designed so that it will not be absorbed and is therefore diarrhea-genic. Most of these patients have diarrhea anyway and don't seem to mind but I think a warning is only fair, particularly when performing the exam in the outpatient setting.

Four final topics worthy of discussion:

(1) What do we do when patients will not or cannot drink the oral contrast? Do the study anyway. While not ideal, patients who cannot drink the oral contrast often have severe disease which you will see even without it.

(2) What about antimotility agents (glucagon)? We use them. We currently give 0.5 mg glucagon IV just before inserting contrast.

(3) What about pregnant patients? Do the study. Consent the patient about the potential, unknown risk of MRI in pregnancy (as per your institution's protocol), give the oral contrast. *Don't give gadolinium*. It can be challenging to differentiate the causes of abdominal pain during pregnancy. A flagrantly abnormal MRI enterography (on SSFP and SSFSE) exam can be very helpful.

(4) It is critical that the technologist continue to scan inferiorly all the way through the perineum. Perianal disease is extremely common and we do not want to miss it. In our practice, stopping too high is our most common technical error. I encourage the technologists to scan until air is seen between the thighs. Then, they know they are done.

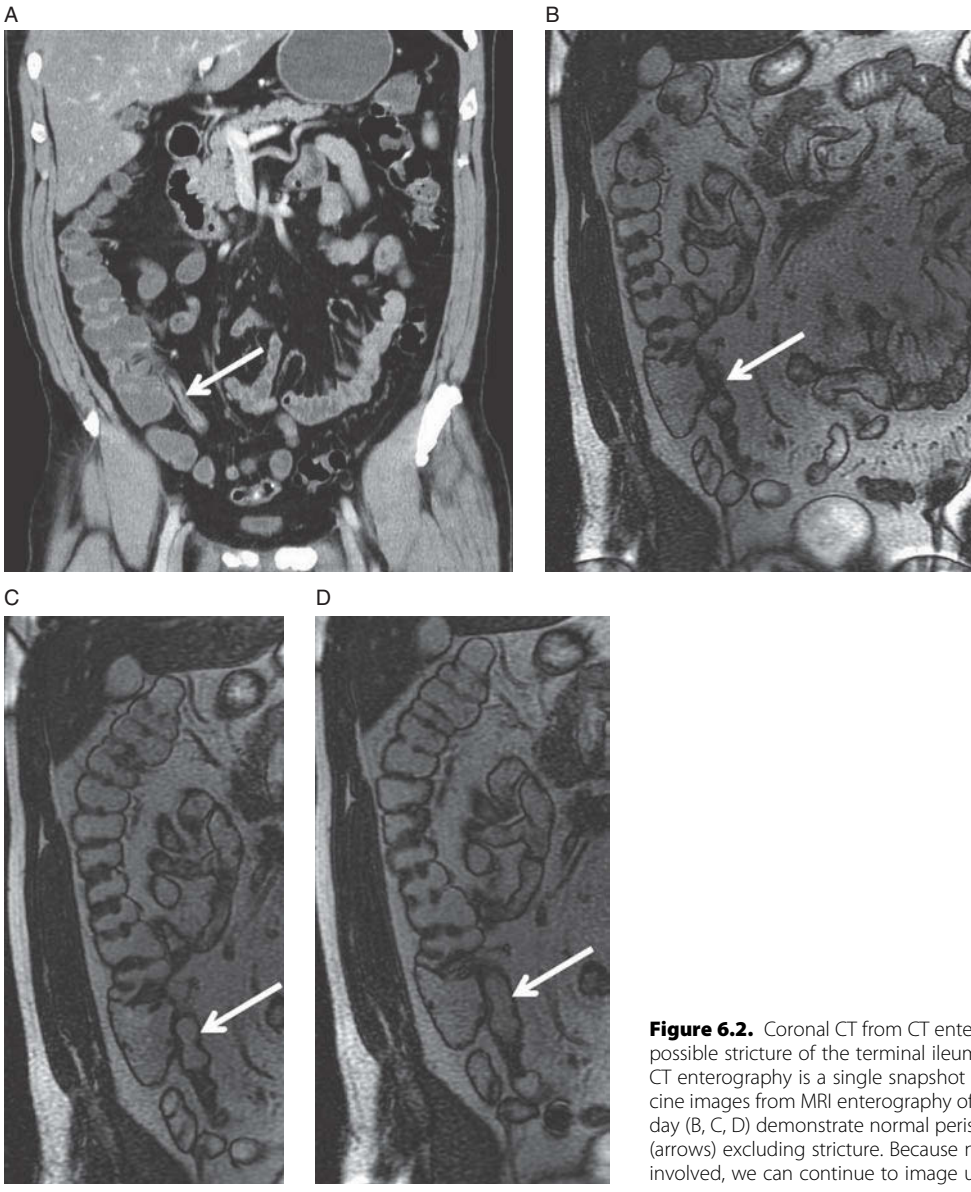

Figure 6.2. Coronal CT from CT enterography (A) demonstrates possible stricture of the terminal ileum (arrow). But, remember, CT enterography is a single snapshot in time. Coronal real-time cine images from MRI enterography of same patient on the same day (B, C, D) demonstrate normal peristalsis of the terminal ileum (arrows) excluding stricture. Because no ionizing radiation is involved, we can continue to image until we have our answer.

Multiplanar True FISP: steady-state free-precession images

This adapted cardiac sequence is extremely fast and provides a nice overview of intestinal anatomy as well as excellent depiction of bowel thickening. It is also used to create real-time cine images which nicely depict bowel peristalsis. Remember from your experience performing small bowel series that it can be impossible to distinguish a stricture from a collapsed loop of bowel without examining the loop in real-time to see if it demonstrates normal peristalsis. As MRI involves no ionizing radiation we can "watch" as long as we would like (or as long as the patient will tolerate) to see if a bowel loop "opens up" (Figure 6.2).

Multiplanar SSFSE: T2-weighted images with fat saturation

Performed with fat saturation these images are intended to demonstrate fluid within or adjacent to

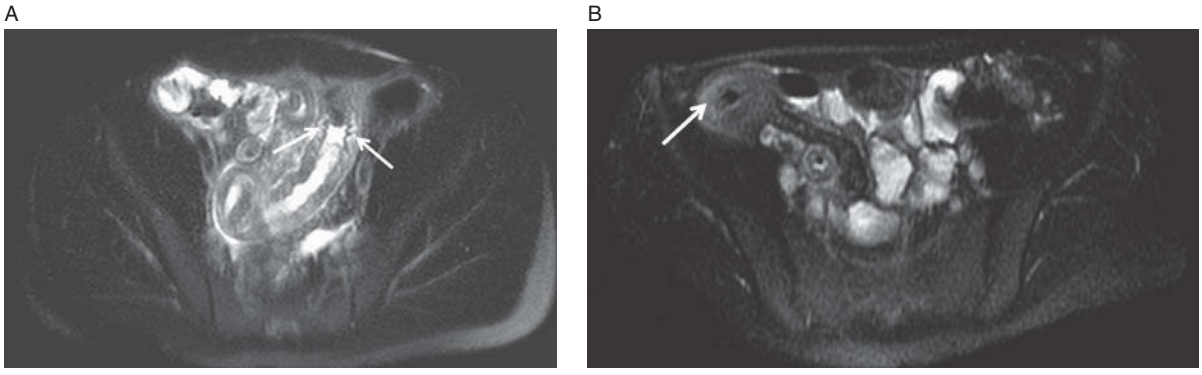

Figure 6.3. Axial SSFSE with fat saturation (A) demonstrates dramatic increased T2-signal within the bowel wall (arrows) as well as adjacent to the inflamed loop of ileum. Axial SSFSE (B – different patient) demonstrates more subtle increased T2-signal within the bowel wall. These are examples of active inflammatory Crohn's disease which warrants medical rather than surgical therapy. If only they were all this easy.

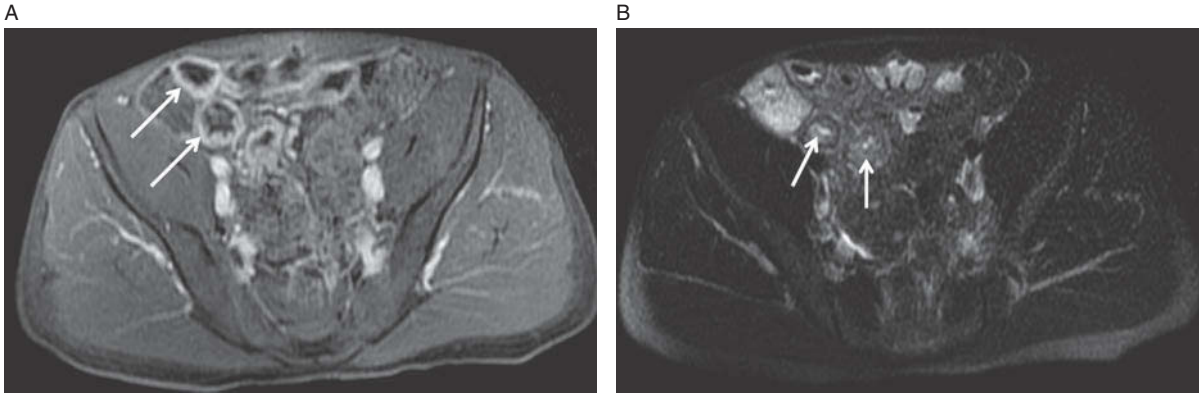

Figure 6.4. Axial T1-weighted, fat-saturated post-contrast image (A) shows multiple loops of abnormally enhancing distal ileum (arrows). The abnormalities are easiest to see on post-contrast images. Then, go back to the T2-weighted images to assess activity. In this patient, the T2-weighted image (B) demonstrates elevated T2-signal within the bowel wall indicating active inflammation.

the bowel wall. *Even subtle T2 changes within the wall or adjacent to the bowel are considered indicative of active inflammation*. The ability to distinguish between active and chronic disease, which will often demonstrate wall thickening without edema, is one of the critical advantages of MRI compared to CT (Figures 6.3–6.5).

Contrast-enhanced images: 3D SPGR, T1-weighted fat-saturated images (VIBE)

The enhancement of Crohn's disease is often dramatic; however, it is important to keep in mind that both active and chronic changes can enhance. Elevated T2 signal within a diseased segment of small bowel is the most specific indicator of active rather than chronic, fibrostenotic disease (Figure 6.6).

We perform our post-contrast images dynamically because investigators have shown that active disease will typically enhance early whereas both active and chronic disease will enhance on delayed images. Additionally, early enhancement of the mucosa out of proportion to the remainder of the bowel wall is considered indicative of active disease as is engorgement of the mesenteric vasculature (the comb sign) (Figure 6.7). Finally, contrast enhancement is critical for identification of abscesses and fistulae.

Homogenous, transmural enhancement in the absence of any T2 changes is thought to represent

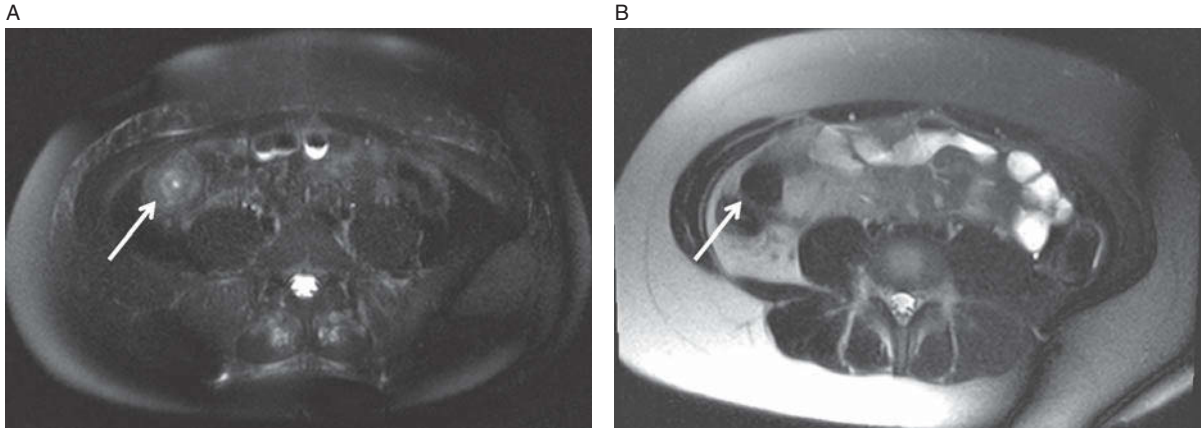

Figure 6.5. Active Crohn's disease. Axial T2-weighted image (A) shows a thickened terminal ileum with increased T2-signal within the wall (arrow) indicative of active disease. Axial T2-weighted image (B) from same patient 6 weeks following initiation of anti-TNF therapy. The terminal ileum remains thickened, but the active changes are gone. (Note also the technical difference between the two images. In image B the fat-saturation failed.)

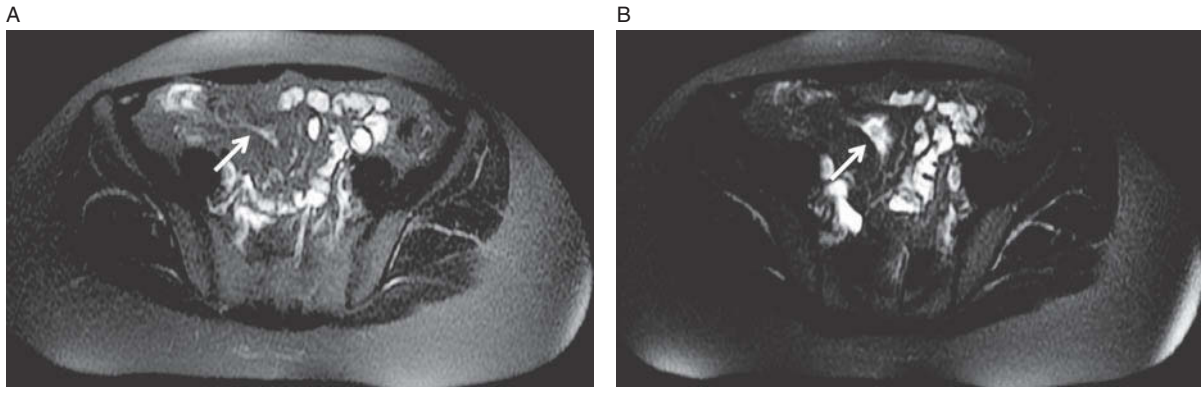

Figure 6.6. Chronic structure. Axial T2-weighted image (A) demonstrates marked narrowing of the terminal ileum which persisted on all sequences. Note the lack of elevated T2 signal. Axial T2-weighted image (B) shows prestenotic dilatation which confirms presence of stricture.

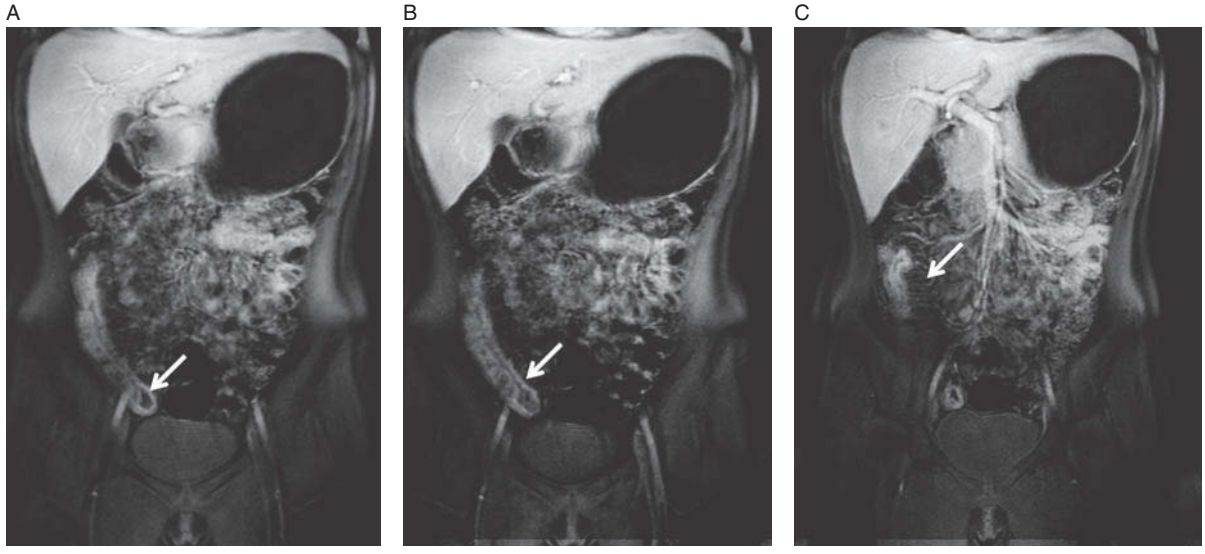

Figure 6.7. Active terminal ileitis. Contiguous coronal T1-weighted, fat-saturated images obtained 35 seconds following injection. Note the abnormal mucosal enhancement (A, B) as well as the engorgement of the mesenteric vasculature (the "comb" sign) (C).

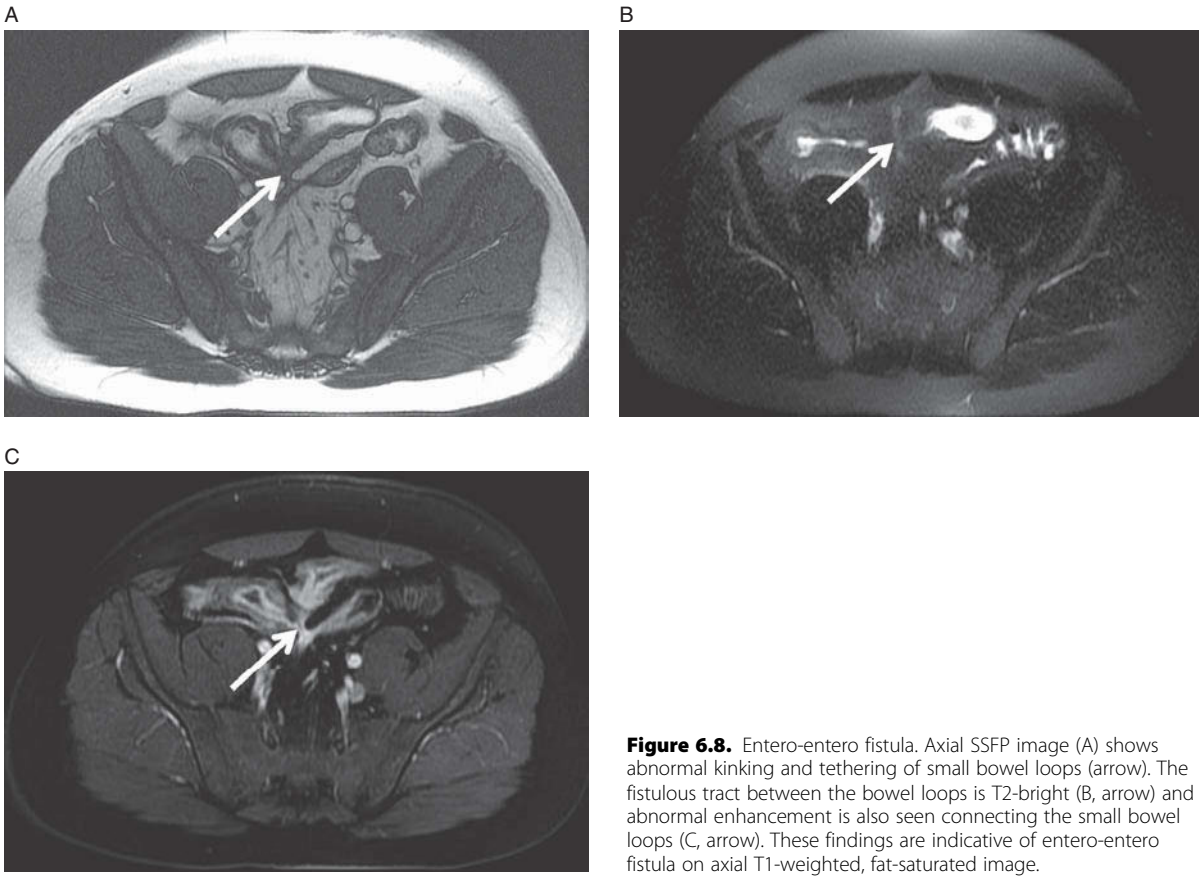

Figure 6.8. Entero-entero fistula. Axial SSFP image (A) shows abnormal kinking and tethering of small bowel loops (arrow). The fistulous tract between the bowel loops is T2-bright (B, arrow) and abnormal enhancement is also seen connecting the small bowel loops (C, arrow). These findings are indicative of entero-entero fistula on axial T1-weighted, fat-saturated image.

chronic changes, *though even very subtle T2 changes count as active disease*.

As discussed earlier, differentiation of active from chronic, fibrostenotic disease is the holy grail of IBD imaging and our best attempt is described above. Quite frankly, the distinction is often difficult with current imaging methods. If it makes you feel better, the pathologists can't really tell the difference either. When you read these cases, follow up their endoscopic biopsies: they are almost always read as "acute on chronic" disease. We must do better. This remains an area of active research.

Crohn's disease is a transmural inflammatory process which can lead to both fistula and abscess formation. Perianal fistulae are the most common and will be discussed in detail in the pelvic imaging section, but MRI enterography is a powerful tool for diagnosing entero-entero fistulae and abscesses. The key to making the diagnosis of entero-entero fistulae is tethering of bowel loops that often appear to kink at abnormal angles. The fistulous tracts between bowel loops will often be T2-bright and enhance (Figure 6.8).

Summary of lesions

There's only really one lesion here – Crohn's disease. Sites of Crohn's disease will demonstrate abnormal wall thickening and hyperenhancement. If the wall or adjacent tissue is T2-bright, call it active Crohn's. If the wall and adjacent tissue is T2-dark, consider chronic fibrostenotic disease.

Pearls and pitfalls

(1) Offer MRI enterography to your referring clinicians for young Crohn's patients. You will decrease their lifetime radiation dose and with experience will likely be able to distinguish active from chronic disease.
(2) Increased mural T2 signal is the most specific indicator of active inflammation, but is disappointingly insensitive (at least in my hands; some investigators seem to do better).
(3) Abnormal contrast enhancement is sensitive but is not specific for active disease unless it is distinctly early and mucosal.
(4) Tethering or bowel loops which form unusual angles should raise suspicion for entero-entero fistula.

Further reading

Lee SS, Kim AY, Yang SK, *et al*. Crohn disease of the small bowel: comparison of CT enterography, MRI enterography, and small-bowel-follow-through as diagnostic techniques. *Radiology* 2009 Jun; **251**(3): 751–61.

Siddiki HA, Fidler JL, Fletcher JG, *et al*. Prospective comparison of state-of-the-art MRI enterography and CT enterography in small-bowel Crohn's disease. *AJR* 2009 July; **193**(1): 113–21.

Tolan DJM, Greenhalgh R, Zealley IA, *et al*. MRI enterographic manifestations of small bowel Crohn disease. *Radio-graphics* 2010 Mar; **30** (2): 367–84.

Chapter 7

The female pelvis: uterus

1 Routine female pelvis protocol

Indications

The most commonly used protocol for MRI of the female pelvis. Indications include evaluation of adnexal masses, ovarian masses, fibroids, adenomyosis, endometriosis, and generalized or localized pelvic pain.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- Have the patient void prior to the start of the study.
- Start IV with at least 24-gauge needle; connect to power injector
- Subtract pre-contrast images from post-contrast images
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Diffusion-weighted imaging b50, 500/ADC – Excellent for lesion detection.
(2) Coronal T2 single-shot fast-spin echo BH (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis.
(3) Sagittal T2 fast-spin echo – Evaluate uterine anatomy. Identify T2-bright and T2-dark lesions.
(4) Axial T2 fast-spin echo – Identify T2-bright and T2-dark lesions.
(5) Coronal T2 fast-spin echo FS – Identify T2-bright and T2-dark lesions. Evaluate for pelvic fluid and T2-bright osseous lesions.
(6–7) Axial T1 in and out of phase (IP/OOP) – Identify T1-bright lesions and microscopic fat.
(8) Axial volume-interpolated gradient echo BH pre – Characterize T1-bright signal in lesions.
(9) Axial volume-interpolated gradient echo BH post 70 seconds. Determine enhancement.
(10) Sagittal volume-interpolated gradient echo BH post to follow.

Approach to female pelvis MRI exam interpretation: Uterus

Due to its superb soft-tissue resolution, MRI is the gold standard for localizing and characterizing female uterine pathology. Most commonly, the uterine female pelvis MRI protocols are performed for further evaluation of fibroids, adenomyosis, staging of endometrial and cervical carcinoma, and uterine anomalies. Remember that, just as in the abdomen, MRI predominantly remains a problem-solving modality and despite all the sequences and all the images, all we really have is T1- and T2-weighting performed in multiple planes.

Approach for the Uterus: Coronal single-shot fast spin echo: T2-weighted images

Every time you read a pelvic MRI, use this sequence to evaluate the presence and location of the kidneys. The genitourinary system shares a common embryologic origin, so anomalies of the uterus often involve the kidneys and vice versa. In addition, uterine pathology, especially fibroids, may result in compression of the distal ureters and hydronephrosis. This sequence also allows the technologist to assess correct coil position prior to proceeding. Most commonly, the coil needs to be moved superiorly to ensure complete coverage of a large fibroid uterus.

Sagittal, Axial, Coronal T2 fast-spin-echo: T2-weighted images

T2-weighted images best depict normal uterine anatomy (Figure 7.1) as well as uterine pathology. For example, T2-weighted images readily demonstrate the size, number and location of fibroids within the myometrium (submucosal, intramural, and subserosal). T2-weighted imaging is also useful to detect adenomyosis.

T2-weighted images are also the best sequences for staging endometrial and cervical carcinoma, and characterizing uterine anomalies with the use of oblique axial and coronal planes. Commonly, the orientation and origin of a uterine mass is only diagnostic in one plane, i.e. "neck" between an exophytic fibroid and the uterus. The addition of fat saturation in one plane (coronal) further highlights fluid and edema in the pelvis.

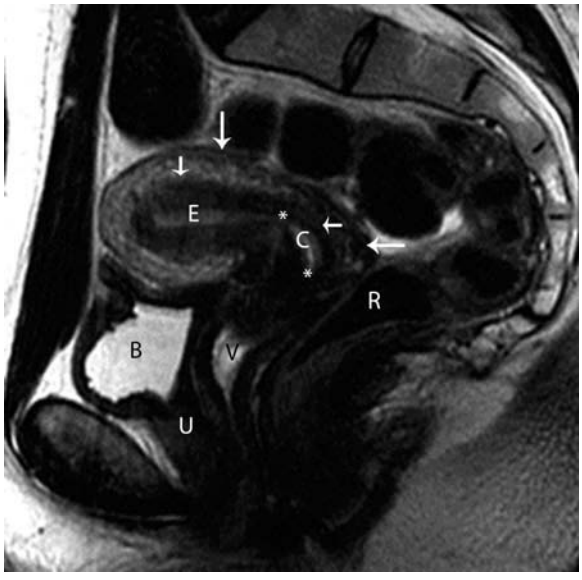

Figure 7.1. Normal uterus in a 32-year-old female. Sagittal T2-weighted image illustrates the normal zonal anatomy of the uterus and cervix. The length of the cervix is marked with asterisks (*). The endometrial canal of the uterus (E) is continuous with the endocervical canal (C). The inner myometrium or junctional zone of the uterus is continuous with the inner cervical stroma (short arrows). The outer myometrium of the uterus is continuous with the outer cervical stroma (long arrows). The vaginal canal (V) contains a small amount of high T2 signal intensity fluid. The bladder (B), urethra (U), and rectum (R) are also all well depicted in the sagittal plane.

In- and out-of-phase T1-weighted images: chemical-shift images; T1-weighted images, no fat saturation

This sequence is used for the detection of fat, blood and proteinaceous debris – fairly common findings in the female pelvis. Fibroids can undergo both hemorrhagic and fatty degeneration, and this sequence can help detect such degeneration. These images also beautifully depict pelvic anatomy, so be sure to search for adenopathy. The bone marrow should be evaluated as well – metastatic disease will be T1 dark.

Pre-Contrast volume-interpolated gradient echo BH: T1-weighted images with fat saturation

Because this sequence contains a fat-saturation pulse, compare it to the in-phase images: hemorrhage/protein is bright on the in-phase image and will remain bright on the volume-interpolated gradient echo.

Macroscopic fat is bright on the in-phase image but dark on the volume-interpolated gradient echo (due to fat saturation pulse).

This sequence also serves as a comparison for T1 fat-saturated images post-contrast.

Post-Contrast volume-interpolated gradient echo BH: T1-weighted images with fat saturation

This sequence is used to detect and characterize enhancement of uterine pathology. For the vast majority of applications, a single post-contrast phase is sufficient. It is typically performed at a standard 70-second delay. Post contrast evaluation of the uterus is helpful for determining the degree of enhancement/viability of uterine fibroids, and for staging endometrial and cervical carcinoma.

The only female pelvic protocols that require dynamic contrast injection and imaging are the endometrial cancer and MRI-MRA uterus protocols.

Endometrial cancer typically enhances less than the myometrium during the early arterial phase of enhancement, which helps detect the presence and depth of myometrial invasion. If evaluation of the uterine and ovarian arterial anatomy is requested (i.e. pre or post uterine artery embolization, post-partum bleeding to evaluate for an arteriovenous malformation (AVM) or

arteriovenous fistula (AVF)) 3D dynamic contrast enhanced imaging should also be performed.

Subtraction images: generated by subtracting the pre-contrast images from the post-contrast images

The presence of T1 bright pathology in the pelvis is a common finding. Subtraction images allow for evaluation of true enhancement in lesions that are inherently T1 bright, i.e. hemorrhagic fibroids. In order to create subtracted images, *all* scan parameters must remain identical between pre- and post-contrast imaging. (The most common mistake is that a technologist will "slightly" change the field of view after seeing the pre-contrast images. If the field of view needs to be changed, the pre-contrast images *must* be run again.)

DWI/ADC

This sequence is excellent for lesion detection, but not specific for characterization. In general, approach DWI of the uterus in the same manner in which you approach DWI of the abdomen.

(1) Identify bright lesions on b50. Lesions that remain bright on b500 are more likely to be malignant and lesions which drop in signal on b500 are more likely to be benign. Correlate with ADC.

   (a) DWI bright, ADC dark = restricted diffusion
   (b) DWI bright, ADC bright = T2 "shine through"

To date, a few small reports have described restricted diffusion in leiomyosarcomas, endometrial and cervical carcinomas. However, beware there is overlap in the DWI/ADC appearance of benign and malignant pelvic pathology.

ii Congenital abnormalities

2 Uterus anomaly protocol

Indications

This protocol is used for evaluation of known or suspected müllerian anomalies such as septate uterus, bicornuate uterus, and others.

Preparation

- **IV contrast agent**: None, unless protocoled otherwise by radiologist
- **Oral contrast agent**: None
- NPO for 4 hours prior to the exam
- Have the patient void prior to the start of the study
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Diffusion-weighted imaging b50, 500/ADC – Excellent for lesion detection.
(2) Coronal T2 single-shot fast-spin echo BH (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis.
(3) Sagittal T2 fast-spin echo – Evaluate uterine anatomy. Use to plan coronal and axial obliques.
(4) Coronal oblique T2 fast-spin echo: parallel to long axis of uterus – Evaluate outer uterine contour.
(5) Axial oblique T2 fast-spin echo: perpendicular to long axis of uterus – Evaluate for uterine septum.
(6–7) Axial T1 in and out of phase (IP/OOP) BH (true axial to pelvis) – Identify T1-bright lesions/material and microscopic fat.
(8) Axial volume-interpolated gradient echo BH (true axial to pelvis) – Evaluate for blood products.
(9) Optional post-contrast: axial volume-interpolated gradient echo BH post 70 seconds (true axial to pelvis).

NOTE

It is critical that the coronal and axial oblique sequences be run immediately after the sagittal T2 FSE as over time the bladder will fill and change the orientation of the uterus. If the examination has to be interrupted between the sagittal T2 and the start of the oblique T2 sequences, repeat the sagittal T2 to ensure accurate selection of the oblique planes prior to proceeding.

Uterine anomaly

Uterine anomalies are a common topic of confusion. They really aren't that confusing if we break them down by the number of endometrial cavities. I like to think of them this way:

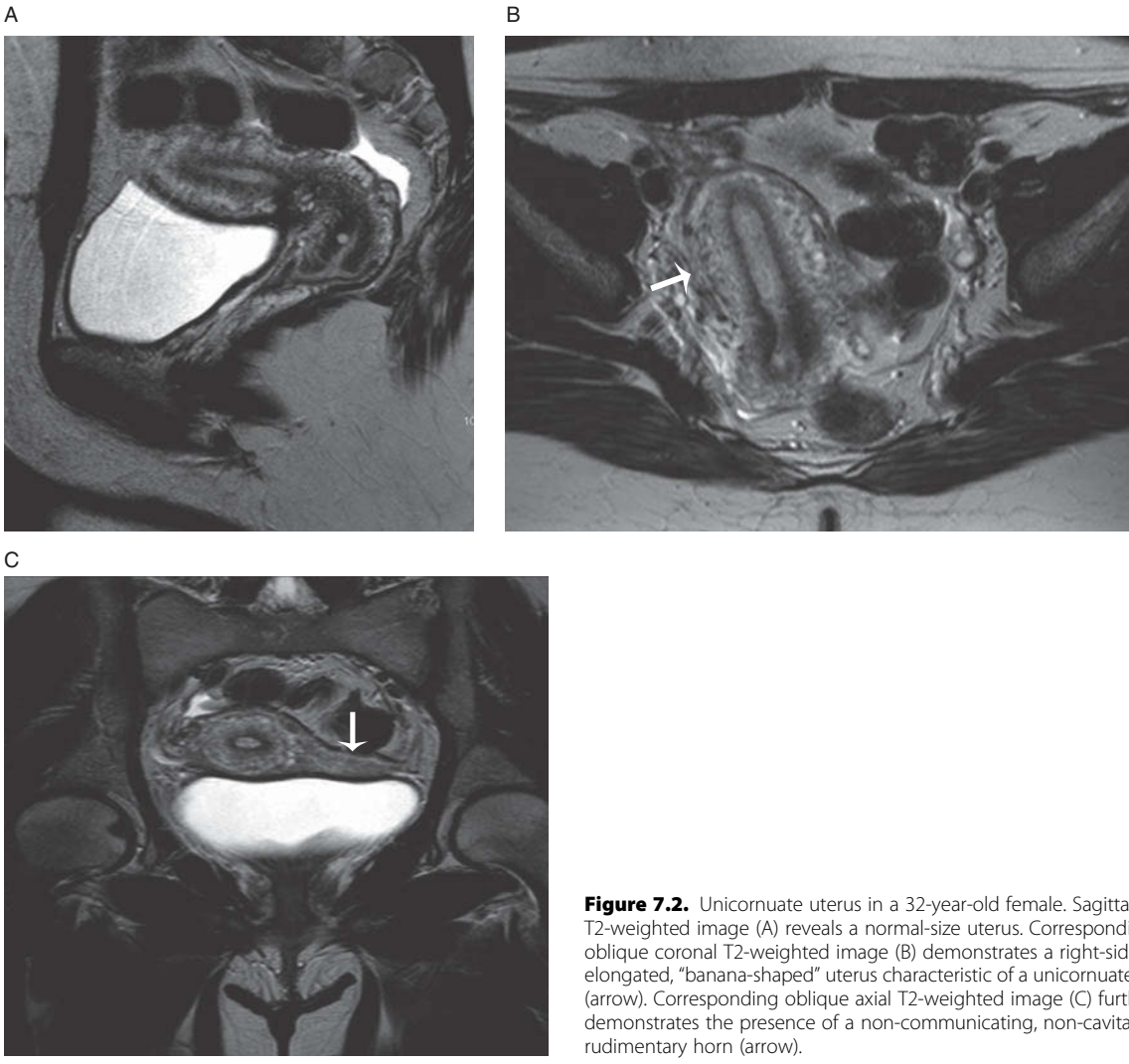

Figure 7.2. Unicornuate uterus in a 32-year-old female. Sagittal T2-weighted image (A) reveals a normal-size uterus. Corresponding oblique coronal T2-weighted image (B) demonstrates a right-sided, elongated, "banana-shaped" uterus characteristic of a unicornuate uterus (arrow). Corresponding oblique axial T2-weighted image (C) further demonstrates the presence of a non-communicating, non-cavitary left rudimentary horn (arrow).

If there is only one endometrial cavity:

(1) A unicornuate uterus has a single horn and is typically deviated off of midline. It looks like a banana. Contralateral rudimentary horns are common, so look very closely for them before dictating that there is no rudimentary horn (Figure 7.2).

If there is more than one endometrial cavity:

Evaluate the outer uterine fundal contour on the T2 coronal (long axis) oblique sequence. In the coronal plane, the normal outer uterine fundal contour is convex to minimally concave (depth of concavity measuring less than 10 mm).

(2) Two endometrial cavities with a normal outer uterine contour = septate uterus. The axial (short axis) oblique sequence can then be used to evaluate the composition and extent of a uterine septum (Figure 7.3).

(3) Two endometrial cavities with concavity of the outer uterine fundal contour >10 mm = bicornuate or didelphys uterus.

   (a) Bicornuate = always some myometrial bridging between the uterine horns even when it is bicornuate bicollis (two endocervical canals).

   (b) Didelphys = separate horns, no myometrial bridging, two separate endocervical canals.

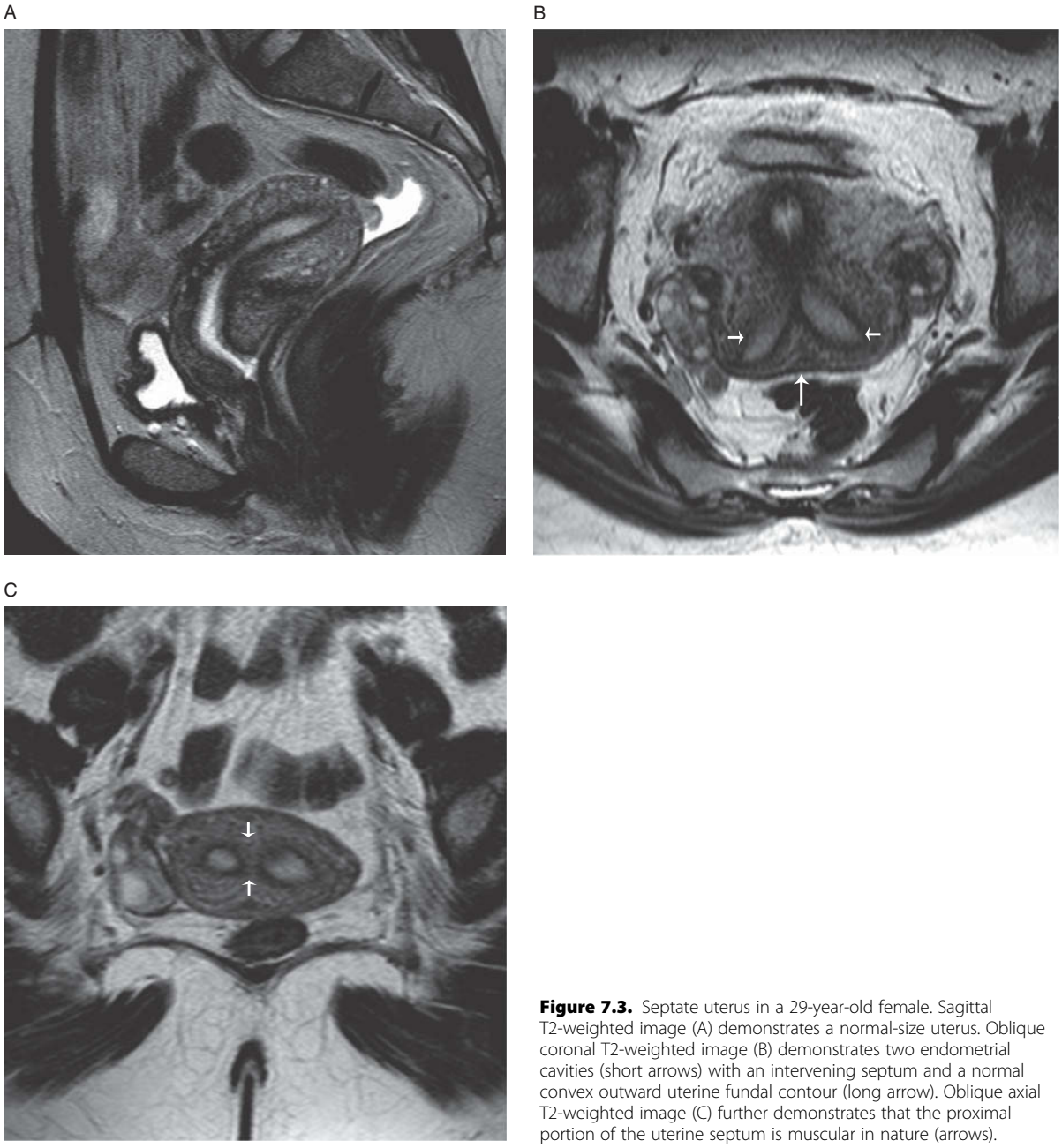

Figure 7.3. Septate uterus in a 29-year-old female. Sagittal T2-weighted image (A) demonstrates a normal-size uterus. Oblique coronal T2-weighted image (B) demonstrates two endometrial cavities (short arrows) with an intervening septum and a normal convex outward uterine fundal contour (long arrow). Oblique axial T2-weighted image (C) further demonstrates that the proximal portion of the uterine septum is muscular in nature (arrows).

Vertical vaginal septa are common (75% of didelphys uteri).

MRI can also readily depict uterine hypoplasia and agenesis. An arcuate uterus can be considered a normal variant, manifested by a normal outer uterine fundal contour and mild, smooth broad based impression on the fundal aspect of the endometrium, without two discrete separate endometrial cavities. Note that "T-shaped" DES drug related uterine anomalies are better depicted on hysterosalpingogram than MRI.

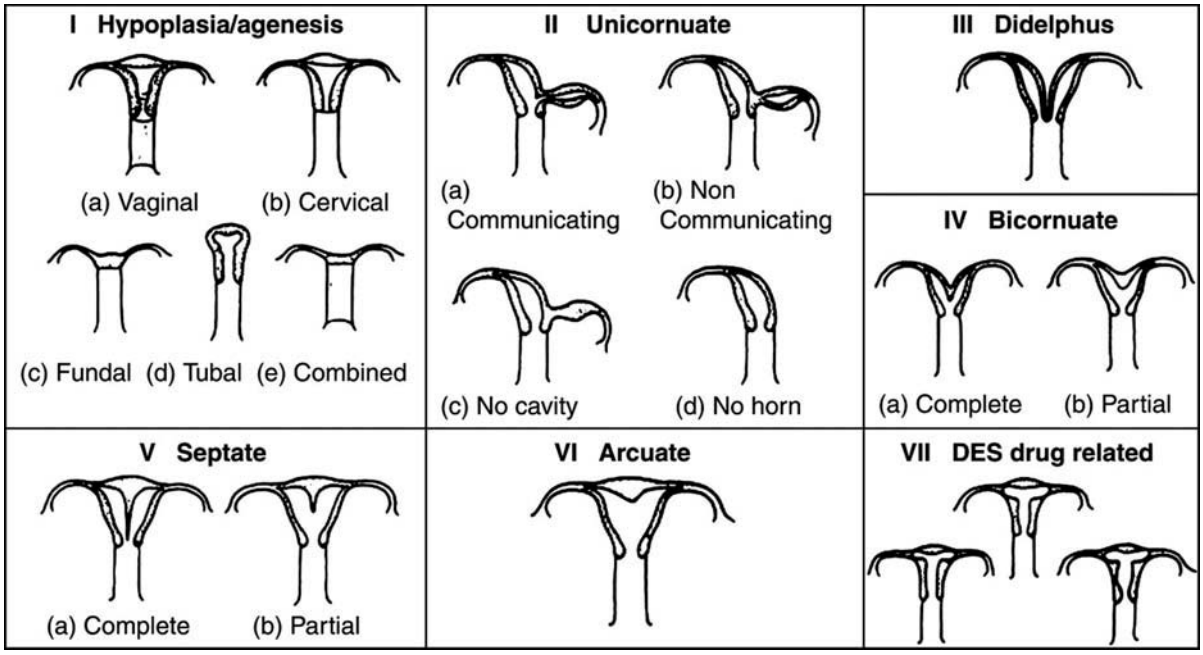

Figure 7.4. Classification of uterine anomalies. Reprinted with permission Fertil. Steril. 1988 June, **49**(6): 944–55.

Classification system of uterine (müllerian duct) anomalies (see Figure 7.4)

Class I: Hypoplasia/agenesis
Class II: Unicornuate
Class III: Didelphys
Class IV: Bicornuate
Class V: Septate
Class VI: Arcuate
Class VII: DES drug related

Developed by the American Fertility Society 1988

iii Fibroids

Fibroids are very common. They are the most common uterine neoplasm – we see them every day. In most cases, ultrasound is all the referring physician needs in order to determine how to manage a patient's fibroid(s). However, patients may proceed to MRI for further evaluation if the sonographic diagnosis is uncertain, or for treatment planning. MRI can exquisitely depict the exact number, size, and location of fibroids (Figures 7.5, 7.6, and 7.7). Often, there are multiple (sometimes too numerous to count) fibroids present. In order to avoid dictating a 10-page MRI report describing each individual fibroid, we have

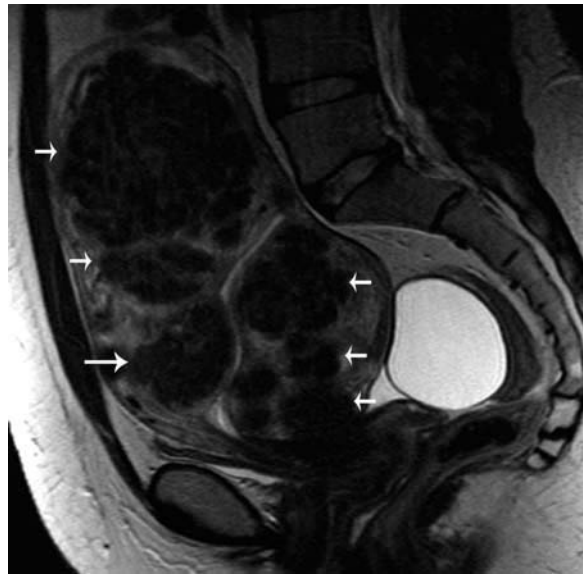

Figure 7.5. Myomatous uterus in a 35-year-old female. Sagittal T2-weighted image demonstrates multiple, intramural, low T2 signal intensity fibroids (short arrows). In addition, there is an intramural fibroid that is partially submucosal in location (long arrow).

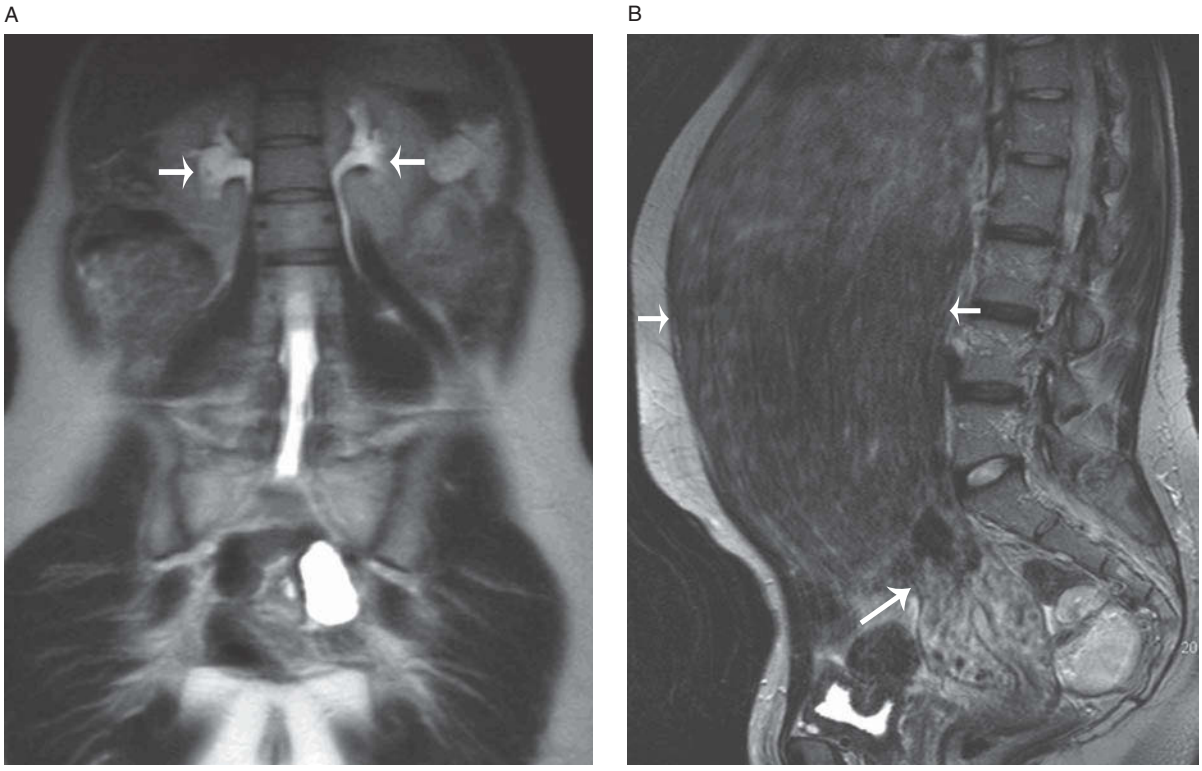

Figure 7.6. Mild bilateral hydronephrosis due to a large exophytic fundal fibroid in a 24-year-old female. Coronal T2-weighted image (A) demonstrates mild bilateral hydronephrosis (arrows). Sagittal T2-weighted image (B) further reveals a large exophytic fundal fibroid (short arrows) with a broad-based attachment to the uterus (long arrow) accounting for the hydronephrosis due to extrinsic ureteral compression.

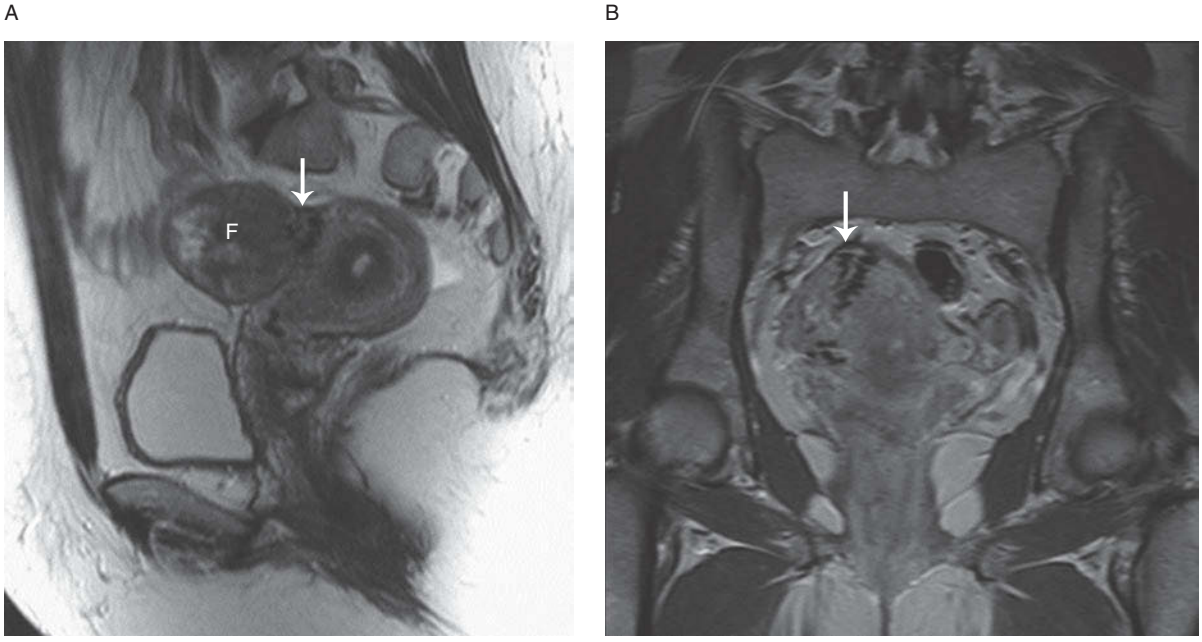

Figure 7.7. Exophytic fibroid in a 45-year-old female. Sagittal T2-weighted image (A) demonstrates the neck (arrow) between an exophytic fibroid (F) and uterus. Better demonstrated on a corresponding coronal T2-weighted image (B) are bridging low signal intensity vessels (arrow) between the uterus and fibroid.

to decide which fibroids are worth describing in detail.

Fibroids worth reporting include the largest fibroids (I limit the number of fibroid measurements in a report based on size alone to the three largest), submucosal (particularly those that are >50% submucosal or intracavitary), and exophytic fibroids (particularly those with narrow <50% of the fibroid diameter, uterine attachment). If surgery is planned, these >50% submucosal and intracavitary fibroids can be resected hysteroscopically. For a patient planning to undergo uterine artery embolization (UAE), predominantly submucosal and intracavitary fibroids are at risk of being "delivered" vaginally following UAE, something that we would like to be able to warn the patient about ahead of time. Exophytic fibroids with narrow attachments to the uterus are also at increased risk of uterine detachment following UAE. Other fibroid features that are important to recognize and describe include the presence of hemorrhage and the degree of enhancement. Fibroids with internal blood products and with minimal or no enhancement are less likely to have a favorable response to UAE compared to fibroids that are still viable, manifested by low T2 signal and homogeneous enhancement.

iv Adenomyosis

MRI is the gold standard for diagnosing adenomyosis and can be used to confirm the diagnosis when sonographic findings are indeterminate. The MRI criteria for adenomyosis are a junctional zone thickness ≥12 mm. High T2 signal intensity foci within the thickened junctional zone further supports the diagnosis (Figure 7.8). The high T2 foci represent ectopic endometrial glands and the low T2 junctional zone thickening represents secondary smooth muscle hyperplasia. Adenomyosis can be further described on MRI as focal or diffuse. Focal adenomyosis (or an adenomyoma) can be mistaken for a fibroid on ultrasound. However, there are clear MRI features that help distinguish between the two possibilities. On MRI, focal adenomyosis has more ill-defined borders and exerts minimal mass effect on the endometrium compared to a fibroid. Differentiation between adenomyosis and a fibroid is important in order to guide appropriate patient management.

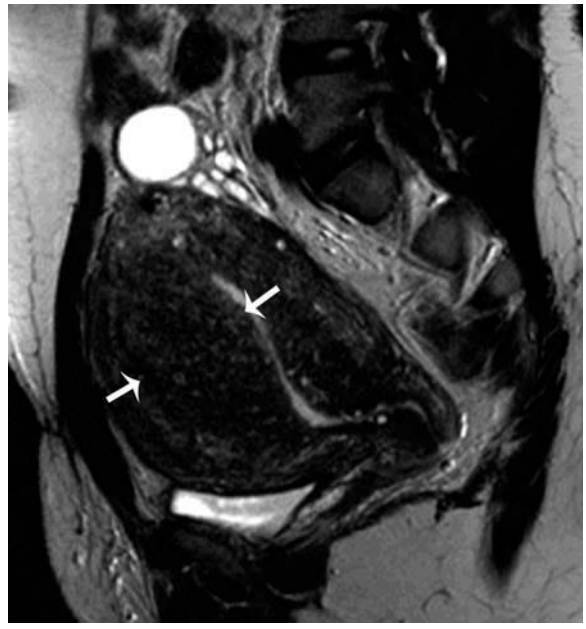

Figure 7.8. Adenomyosis in a 42-year-old female. Sagittal T2-weighted image illustrates marked thickening of the junctional zone, particularly along the anterior uterine body (arrows), and foci of T2 signal hyperintensity within the thickened junctional zone.

v Endometrial cancer

Endometrial cancer protocol

Indications

This protocol is used to stage known endometrial cancer, and to evaluate indeterminate endometrial masses and postmenopausal bleeding. (If MRI abdomen and pelvis is requested, to be combined with abdomen portion of abdomen and pelvis screening protocol [see below].)

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- NPO for 4 hours prior to the exam
- Have the patient void prior to the start of the study
- Give patient 2 L nasal oxygen
- Start IV with at least 24-gauge needle; connect to power injector
- Subtract pre-contrast images from post-contrast images
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Axial DWI b50, 500/ADC – Excellent for lesion detection.
(2) Coronal T2 single-short fast-spin echo BH (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis.
(3) Sagittal T2 fast-spin echo – Evaluate uterine anatomy. Use to plan axial and coronal obliques.
(4) Axial oblique T2 fast-spin echo: perpendicular to long axis of uterus – Evaluate location and extent of endometrial mass.
(5) Coronal oblique T2 fast-spin echo: parallel to long axis of uterus – Evaluate location and extent of endometrial mass invasion into the normal myometrium.
(6) Axial volume-interpolated gradient echo BH (true axial to pelvis) – Evaluate for pelvic lymphadenopathy.
(7) Sagittal volume-interpolated gradient echo BH pre.
(8) Sagittal dynamic volume-interpolated gradient echo BH post at 20 seconds, 1 minute, and 2 minutes after contrast administration.
(9) Coronal volume-interpolated gradient echo BH to follow.

If combined with screening abdomen, image in the following order:

(1) Screening abdomen up until contrast. Do not have to run separate abdominal coronal SSFSE.
(2) Endometrial cancer protocol including dynamic care bolus contrast injection in the pelvis.
(3) Delayed post-contrast axial volume-interpolated gradient echo BH through abdomen.

Endometrial cancer

Patients typically arrive for MRI with the diagnosis of endometrial carcinoma already established by endometrial biopsy. Finding the known endometrical carcinoma can be a challenge, so don't lose faith. On T2-weighted images, endometrial carcinoma is brighter than the myometrium, but darker than the normal endometrium. Endometrial carcinoma is typically hypovascular relative to the normal myometrium on dynamic post-contrast images (Figure 7.9, 7.10). Endometrial carcinoma may also demonstrate restricted diffusion (Figure 7.11). Once the tumor is located, it can be staged. The FIGO classification for endometrial carcinoma is as follows:

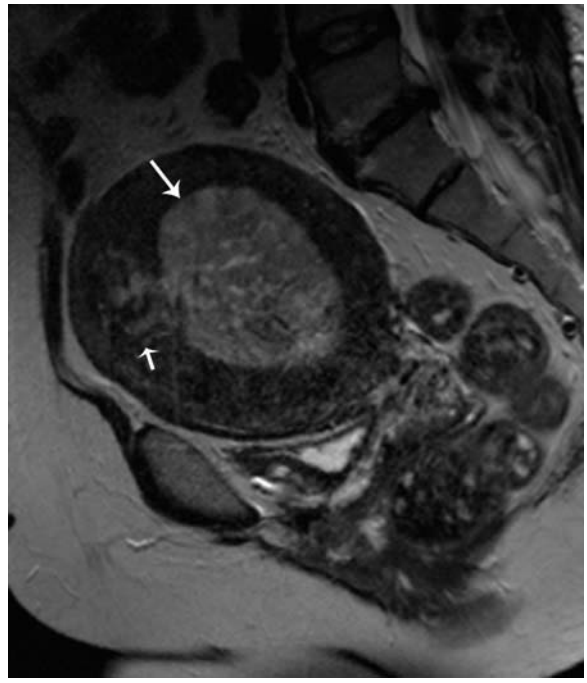

Figure 7.9. Stage 1C endometrial carcinoma in a 45-year-old female. Sagittal T2-weighted image illustrates intermediate T2 signal intensity material replacing and distending the endometrial cavity (long arrow). There is tumor extension into the anterior myometrium (short arrow) for a depth of at least 50% of the myometrium.

0: Carcinoma in situ
IA: No myometrial invasion*
IB: <50% myometrial invasion*
IC: >50% myometrial invasion*
IIA: Endocervical invasion
IIB: Cervical stroma invasion
IIIA: Invades serosa, adnexa, peritoneal seeding
IIIB: Vaginal invasion
IVA: Invasion of rectal mucosa or bladder
IVB: Distant metastasis

*The critical distinction to try to make on MRI is the depth of tumor invasion into the myometrium which may alter surgical planning and prognosis. The depth of myometrial invasion helps predict the risk of lymph node metastasis and may alter the extent of surgical lymph node resection.

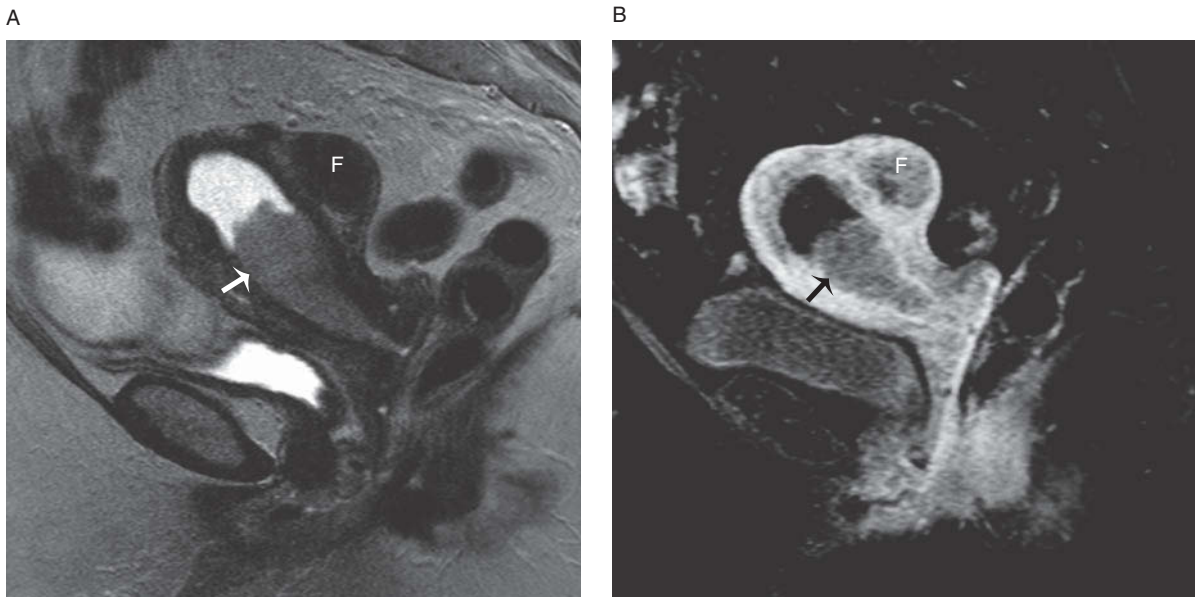

Figure 7.10. Stage 1A endometrial carcinoma in a 72-year-old female. Sagittal T2-weighted image (A) illustrates intermediate T2 signal intensity material (arrow) within the endometrial cavity which on the sagittal T1-weighted image with fat saturation (B), demonstrates a lower level of enhancement (arrow) than the adjacent myometrium. There is also a posterior body subserosal fibroid (F).

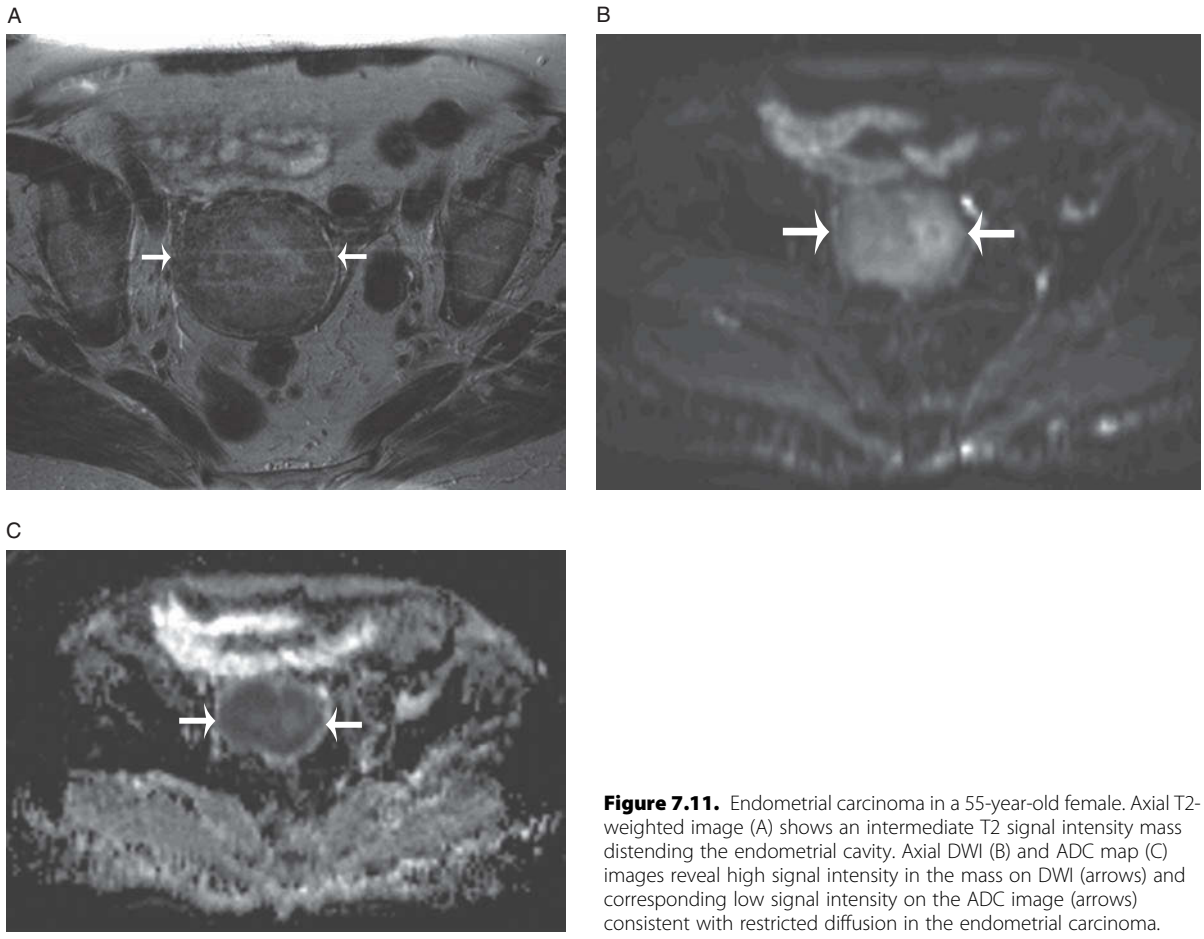

Figure 7.11. Endometrial carcinoma in a 55-year-old female. Axial T2-weighted image (A) shows an intermediate T2 signal intensity mass distending the endometrial cavity. Axial DWI (B) and ADC map (C) images reveal high signal intensity in the mass on DWI (arrows) and corresponding low signal intensity on the ADC image (arrows) consistent with restricted diffusion in the endometrial carcinoma.

vi Cervical cancer

Cervical cancer protocol

Indications

This protocol is used to stage known cervical cancer or to evaluate an indeterminate cervical mass. (If MRI abdomen and pelvis is requested, combine the below pelvic protocol with the abdomen portion of the abdomen and pelvis screening protocol [see below].)

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- NPO for 4 hours prior to the exam
- Have the patient void prior to the start of the study
- Give patient 2 L nasal oxygen
- Start IV with at least 24-gauge needle; connect to power injector
- Subtract pre-contrast images from post-contrast images
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Diffusion-weighted imaging b50, 500/ADC – Excellent for mass and lymph node detection.
(2) Coronal T2 single-shot fast-spin echo BH (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis.
(3) Sagittal T2 fast-spin echo – Evaluate uterine anatomy. Use to plan axial and coronal obliques.
(4) Axial oblique T2 fast-spin echo: perpendicular to cervix long axis – Evaluate location and extent of cervical mass.
(5) Coronal oblique T2 fast-spin echo: parallel to cervix long axis – Evaluate location and extent of cervical mass.
(6) Axial volume-interpolated gradient echo BH (to level of renal arteries) (true axial) – Evaluate for pelvic and retroperitoneal lymphadenopathy.
(7) Axial oblique volume-interpolated gradient echo BH pre: perpendicular to cervix long axis.
(8) Axial oblique volume-interpolated gradient echo BH post at 70 seconds: perpendicular to cervix long axis.
(9) Sagittal volume-interpolated gradient echo BH post to follow.

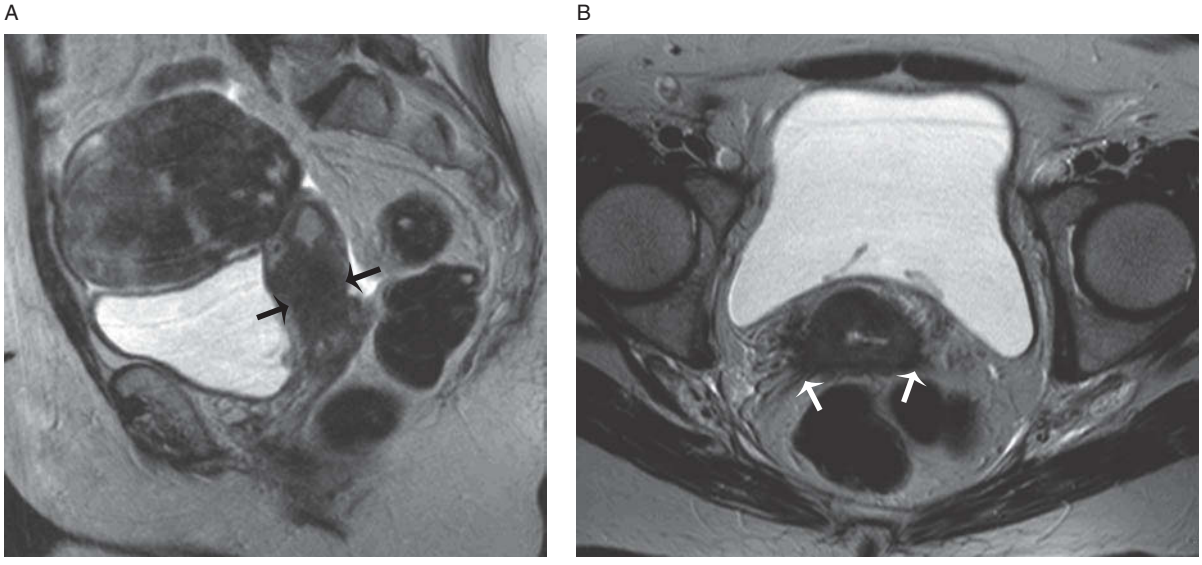

Figure 7.12. Stage IIB cervical carcinoma in a 72-year-old female. Sagittal T2-weighted image (A) demonstrates a low T2 signal intensity cervical mass with superior extension along the uterine body (arrows). Also incidentally seen superior to the uterus is a right ovarian fibroma. Corresponding oblique axial T2-weighted image (B) at the level of the cervix further demonstrates extension of the mass into the parametrium (arrows), right greater than left.

If combined with screening abdomen, image in the following order:

(1) Cervical cancer protocol up until contrast.
(2) Abdomen screening protocol with dynamic contrast injection in the abdomen. Do not have to run separate abdominal coronal T2 HASTE.
(3) Delayed post-contrast pelvic axial oblique and sagittal volume-interpolated gradient echo BH.

Cervical cancer

The diagnosis of cervical cancer is made by Pap smear and biopsy The patients that come to us already have a diagnosis, so our job is now tumor staging. Also, similar to endometrial carcinoma, simply finding the known cervical carcinoma can be difficult. Macroscopic carcinomas will be brighter than the adjacent normal cervical stroma, but darker than the endocervical canal glands on T2 weighted images. Cervical carcinoma also typically enhances less than the normal cervical stroma. Once the tumor is located, it can be staged. The FIGO classification for cervical carcinoma is as follows:

0: In situ
I: Confined to cervix
IIA: Into superior vagina
IIB: Parametrial invasion*
IIIA: Into lower vagina
IIIB: Invades pelvic wall and ureter (hydronephrosis)
IV: Invades adjacent organs: bladder/rectum
IVB: Distant metastasis

*The key here is to distinguish IIA from IIB, commonly remembered as "To be or not to be. . ." Parametrial extension triages patients to radiation rather than surgery. (Figure 7.12) Patients who have large tumors (>5 cm) may also undergo chemotherapy prior to tumor resection. It is also important to always remember to look for hydroureter and hydronephrosis, as hydroureteronephrosis is indicative of parametrial and pelvic sidewall extension even if such tumor extension outside the cervix is difficult to visualize.

Chapter 8

Adnexa

Routine female pelvis protocol

Indications

The most commonly used protocol for MRI of the female pelvis. Indications include evaluation of adnexal masses, ovarian masses, fibroids, adenomyosis, endometriosis, and generalized or localized pelvic pain.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- Have the patient void prior to the start of the study.
- Start IV with at least 24-gauge needle; connect to power injector
- Subtract pre-contrast images from post-contrast images
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Diffusion-weighted imaging b50, 500/ADC – Excellent for lesion detection.
(2) Coronal T2 single-shot fast-spin echo BH (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis.
(3) Sagittal T2 fast-spin echo – Evaluate uterine anatomy. Identify T2-bright and T2-dark lesions.
(4) Axial T2 fast-spin echo – Identify T2-bright and T2-dark lesions.
(5) Coronal T2 fast-spin echo FS – Identify T2-bright and T2-dark lesions. Evaluate for pelvic fluid and T2-bright osseous lesions.
(6–7) Axial T1 in and out of phase (IP/OOP) – Identify T1-bright lesions and microscopic fat.
(8) Axial volume-interpolated gradient echo BH pre – Characterize T1-bright signal in lesions.
(9) Axial volume-interpolated gradient echo BH post 70 seconds. Determine enhancement.
(10) Sagittal volume-interpolated gradient echo BH post to follow.

Approach to female pelvis MRI exam interpretation: Ovary and adnexa

Due to its superb soft-tissue resolution, MRI is also the gold standard for localizing and characterizing female ovarian and adnexal pathology. Most commonly, the routine female pelvis protocol is performed for evaluation of ovarian and adnexal masses and cysts when the initial sonographic examination is indeterminate. The approach to evaluating the ovaries and adnexa in the routine female pelvis protocol is similar to that previously detailed for uterine pathology with a few additional highlights as detailed below.

Coronal single-shot fast spin echo: T2-weighted images

Every time you read a pelvic MRI for ovarian or adnexal pathology, also use this sequence to evaluate the location and appearance of the kidneys. Large adnexal masses may result in compression of the distal ureters and hydronephrosis. This sequence also allows the technologist to assess correct coil position prior to proceeding. The coil may need to be moved superiorly to ensure complete coverage of a large ovarian or adnexal lesion.

Sagittal, Axial, Coronal T2 fast-spin-echo: T2-weighted images.

T2-weighted images best depict ovarian anatomy (Figure 8.1) as well as ovarian and adnexal pathology. For example, T2-weighted images highlight a variety

of T2-bright pathology in the adnexa such as ovarian and paraovarian cysts, and hydrosalpinx. The three orthogonal planes of T2 also aid in the localization and characterization of more complex pelvic pathology. Commonly, the orientation and origin of a pelvic structure is only diagnostic in one plane, i.e. the tubular configuration of hydrosalpinx (Figure 8.2). The addition of fat saturation in one plane (coronal) further highlights fluid and edema in the pelvis.

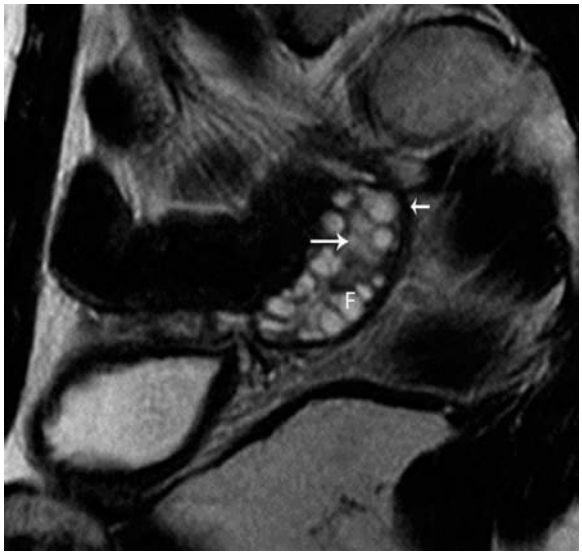

Figure 8.1. Normal ovary in a 27-year-old female. Sagittal T2-weighted image depicts the normal anatomy of the ovary with a low T2 signal intensity cortex (short arrow), higher T2 signal intensity medulla (long arrow), and multiple high T2 signal intensity and subcentimeter peripheral follicles (F).

In- and out-of-phase T1-weighted images: chemical-shift images; T1-weighted images, no fat saturation

This sequence is used for the detection of fat, blood and proteinaceous debris – findings that are even more common in the ovary than the uterus. The out-of-phase images allow for characterization of the rare pelvic mass containing microscopic fat (mature and immature ovarian teratoma) (Figure 8.3). Also, be sure to evaluate the pelvis for adenopathy and the bone marrow for metastatic disease (will be T1-dark) on this sequence.

Pre-Contrast volume-interpolated gradient echo BH: T1-weighted images with fat saturation

This sequence should again be compared to the non fat-saturated in-phase images as hemorrhage/protein

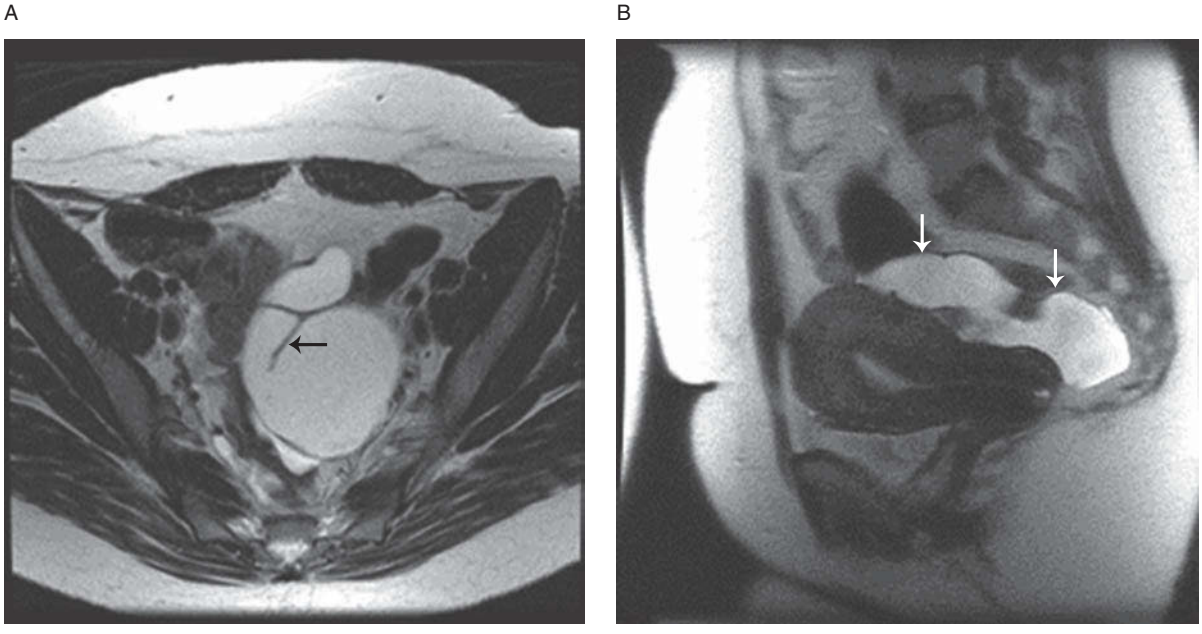

Figure 8.2. Simple hydrosalpinx in a 42-year-old female. Axial T2-weighted image (A) reveals an incomplete fold (arrow) characteristic of hydrosalpinx and the corresponding sagittal T2-weighted image (B) demonstrates the tubular configuration of hydrosalpinx (arrows).

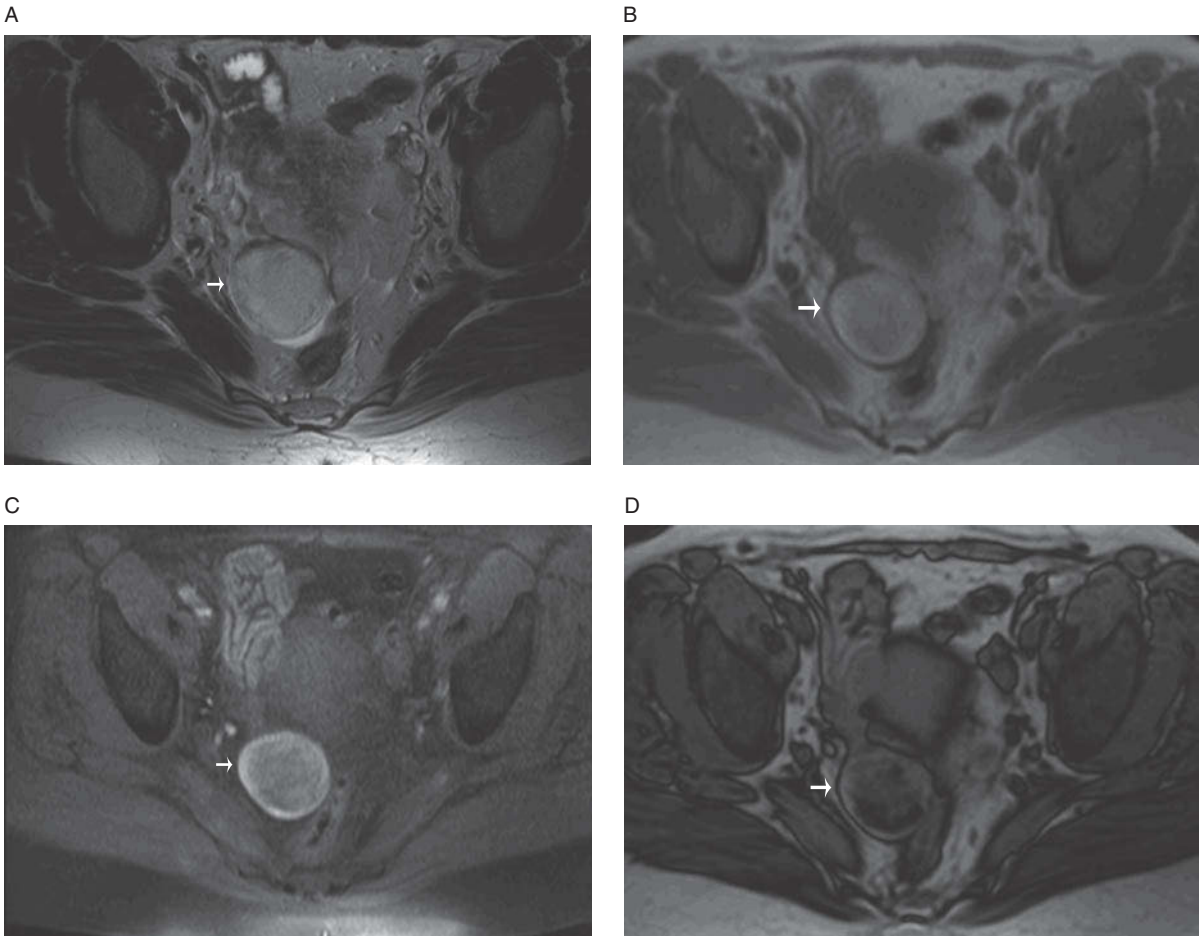

Figure 8.3. Mature teratoma containing microscopic fat in a 32-year-old female. Axial T2-weighted image (A) demonstrates a high T2 signal intensity, 3.5 cm, mass (arrow) in the right ovary with corresponding high T1 signal intensity (arrow) on the axial in phase T1-weighted image (B). There is no appreciable loss of T1 signal intensity within the mass (arrow) on the axial T1-weighted image with fat saturation (arrow, C). This suggests that the high T1 signal intensity in the mass is most likely due to blood or protein. However, the axial out-of-phase T1-weighted image (D) reveals signal loss within the mass (arrow) indicative of microscopic fat contents. Surgical pathology confirmed a benign mature teratoma containing microscopic fat.

is bright on the in-phase image and will remain bright on the volume-interpolated gradient echo.

- Macroscopic fat is bright on the in-phase image but dark on the volume-interpolated gradient echo (due to fat saturation pulse).
- This sequence also serves as a comparison for T1 fat-saturated images post-contrast.

Post-Contrast volume-interpolated gradient echo BH: T1-weighted images with fat saturation

This sequence is used to detect and characterize enhancement of ovarian and adnexal pathology. For ovarian and adnexal applications, a single post-contrast phase is sufficient. It is typically performed at a standard 70-second delay. Post contrast evaluation of the ovaries and adnexa is helpful for fully characterizing the nature and complexity of solid and cystic lesions.

Subtraction images: generated by subtracting the pre-contrast images from the post-contrast images

The presence of T1 bright pathology is even more common in the ovaries than the uterus. Subtraction images allow for evaluation of true enhancement in

lesions that are inherently T1 bright, i.e. hemorrhagic cysts. In order to create subtracted images, all scan parameters must remain identical between pre- and post-contrast imaging.

DWI/ADC

This sequence is excellent for lesion detection, but not specific for characterization. In general, approach DWI of the ovary in the same manner in which you approach DWI of the abdomen and uterus.

(1) Identify bright lesions on b50. Lesions that remain bright on b500 are more likely to be malignant and lesions which drop in signal on b500 are more likely to be benign. Correlate with ADC.

   (a) DWI bright, ADC dark = restricted diffusion

   (b) DWI bright, ADC bright = T2 "shine through"

To date, a few small reports have described restricted diffusion in ovarian carcinomas. However, beware; there is overlap in the DWI/ADC appearance of benign and malignant pelvic pathology, particularly for ovarian neoplasms.

Female pelvic pathology guidelines: Ovary

When the sonographic diagnosis of an ovarian lesion remains uncertain, MRI is the study of choice. Similar to MRI in the abdomen, we try to provide a definitive diagnosis, as the next step is typically surgery versus no surgery. Fortunately, MRI is capable of making several definitive benign diagnoses and helping guide surgical management. Even definitively benign ovarian lesions on MRI may be resected if they are large enough (i.e. >5 cm) to place the patient at risk for ovarian torsion.

i. Cystic ovarian lesions

There are a variety of cystic ovarian lesions that can be definitively characterized on MRI. In general, lesions that are uniformly cystic are benign. It is the mixed cystic and solid lesions that harbor the potential for malignancy.

An ovarian cyst that is uniformly low in signal on T1 and high in signal on T2, and lacks internal enhancement, can be diagnosed as a simple cyst. I call this simple high T2 signal intensity structure a follicle if it measures <2.5 cm and a cyst if it measures >2.5 cm (Figure 8.4).

A T1-bright, T2-bright lesion that does not enhance can be called a hemorrhagic cyst. If you see a T1-bright, T2-dark ovarian lesion that does not enhance, the differential diagnosis is a hemorrhagic cyst versus an endometrioma. A hemorrhagic cyst is a benign ovarian cyst that has blood in it. These usually resolve in 6 to 12 weeks.

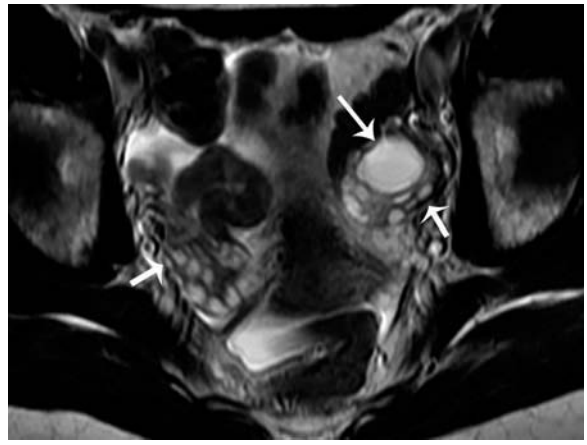

Figure 8.4. Ovarian follicle in a 37-year-old female. Axial T2-weighted image demonstrates normal bilateral ovaries (short arrows) with a high T2 signal intensity, 2.5 cm, dominant follicle in the left ovary (long arrow).

An endometrioma is a focus of endometrial tissue that has migrated from the endometrium into the pelvis. It also often has blood in it and remains present for longer than 6 to 12 weeks. Both may be painful. Differentiating between hemorrhagic cysts and endometriomas is a point of obsession for some radiologists, but even in the best of hands you just can't always tell them apart.

However, there are several features that can help lead us in the right direction. If you have serial imaging exams and the lesion is unchanged over several months, it is most likely an endometrioma. Low T2 signal intensity, "shading," is more characteristic of an endometrioma. Endometriomas are also more commonly multiple and bilateral (Figure 8.5). If you can find other features of endometriosis in the pelvis, such as hematosalpinx and endometrial implants, then you can be very confident in calling the lesion an endometrioma rather than a hemorrhagic cyst. In either case, both are benign, but an endometrioma may be resected if it is a source of chronic pain. In addition, endometriomas are associated with a small, but increased risk of clear cell and endometriod ovarian carcinoma (Figure 8.6).

A cystic ovarian lesion that loses its high T1 signal intensity with the application of fat saturation can be definitively characterized as a mature cystic teratoma (Figures 8.7). Despite being characterized as benign, a

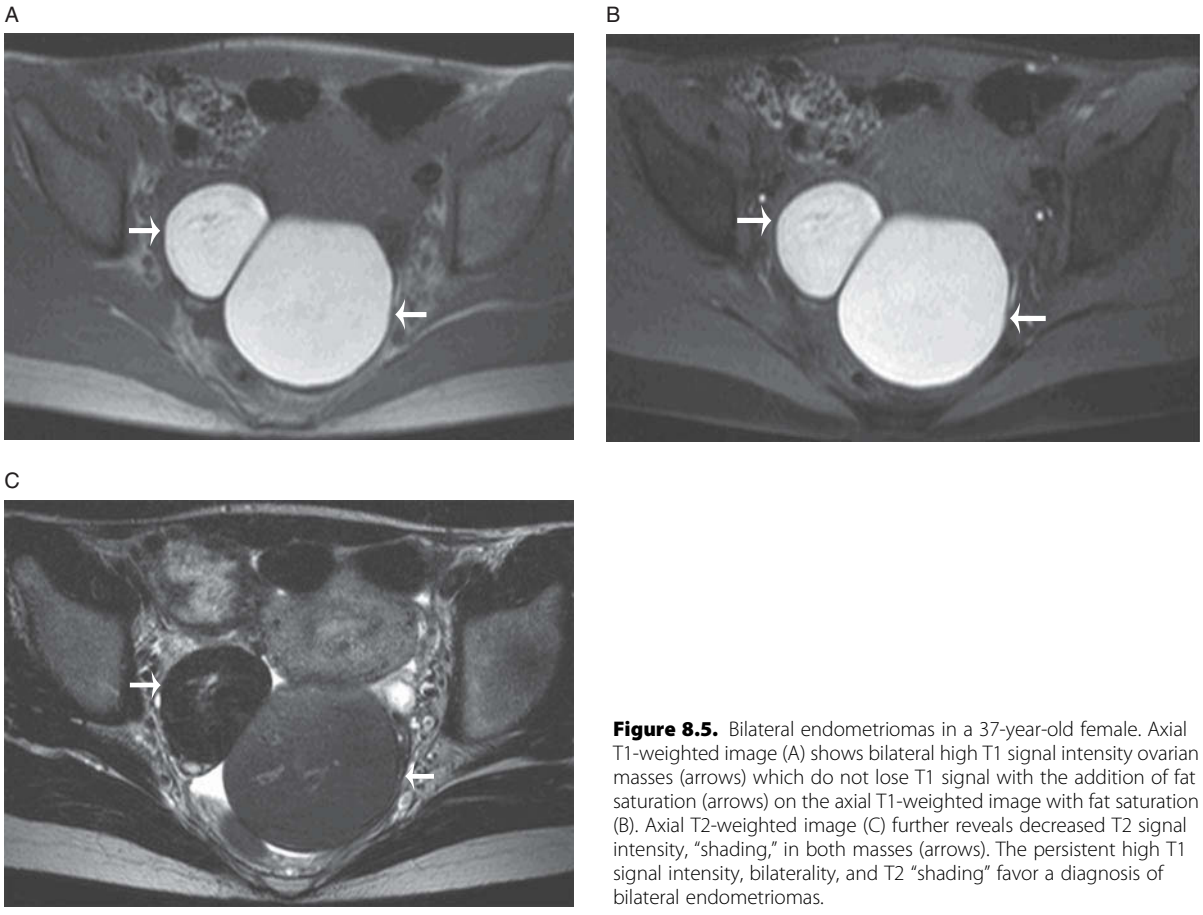

Figure 8.5. Bilateral endometriomas in a 37-year-old female. Axial T1-weighted image (A) shows bilateral high T1 signal intensity ovarian masses (arrows) which do not lose T1 signal with the addition of fat saturation (arrows) on the axial T1-weighted image with fat saturation (B). Axial T2-weighted image (C) further reveals decreased T2 signal intensity, "shading," in both masses (arrows). The persistent high T1 signal intensity, bilaterality, and T2 "shading" favor a diagnosis of bilateral endometriomas.

fat-containing mature teratoma may be resected based on size alone (>5 cm) due to an increased risk of ovarian torsion and rupture. There is a very rare (<2%) associated risk of developing malignancy within a mature teratoma. Immature teratomas are similarly a very rare malignant ovarian neoplasm which, compared to a benign mature teratoma, appear as a more aggressive solid lesion with only a small amount of internal fat.

Cystic ovarian lesions that cannot be characterized as simple or hemorrhagic cysts, endometriomas, or mature teratomas are indeterminate or potentially malignant cystic ovarian neoplasms. At this point, we need to look carefully for MRI features that would push us to say that an ovarian cyst may be malignant.

Well-described MRI features of a cystic ovarian malignancy (Table 8.1, Figure 8.8) include a mixed cystic and solid lesion, solid mural nodules, thick wall and/or septations (>3 mm), large size (>5 cm), and direct invasion of adjacent ovarian stroma. If a lesion has any of these features than gynecologic oncologic consultation and correlation with serum Ca-125 should be recommended. If a cystic lesion is more complicated than a simple cyst, but does not meet the above features of an ovarian malignancy, i.e. few thin internal septations, then close-interval follow-up imaging can be recommended. Often, this follow-up can be with ultrasound now that the lesion has been fully characterized with MRI. Whenever you are calling a cystic lesion a possible ovarian malignancy, take another look at the images so as not to overlook any associated ascites, implants, or adenopathy.

Don't worry about always providing a specific pathologic diagnosis for an ovarian malignancy. There is a lot of overlap in their MRI appearance. That being said, the two most common ovarian malignancies are serous and mucinous cystadenocarcinomas, both of epithelial origin. Compared to mucinous neoplasms, serous neoplasms are more commonly uniformly T1-dark and T2-bright, bilateral, smaller, and unilocular

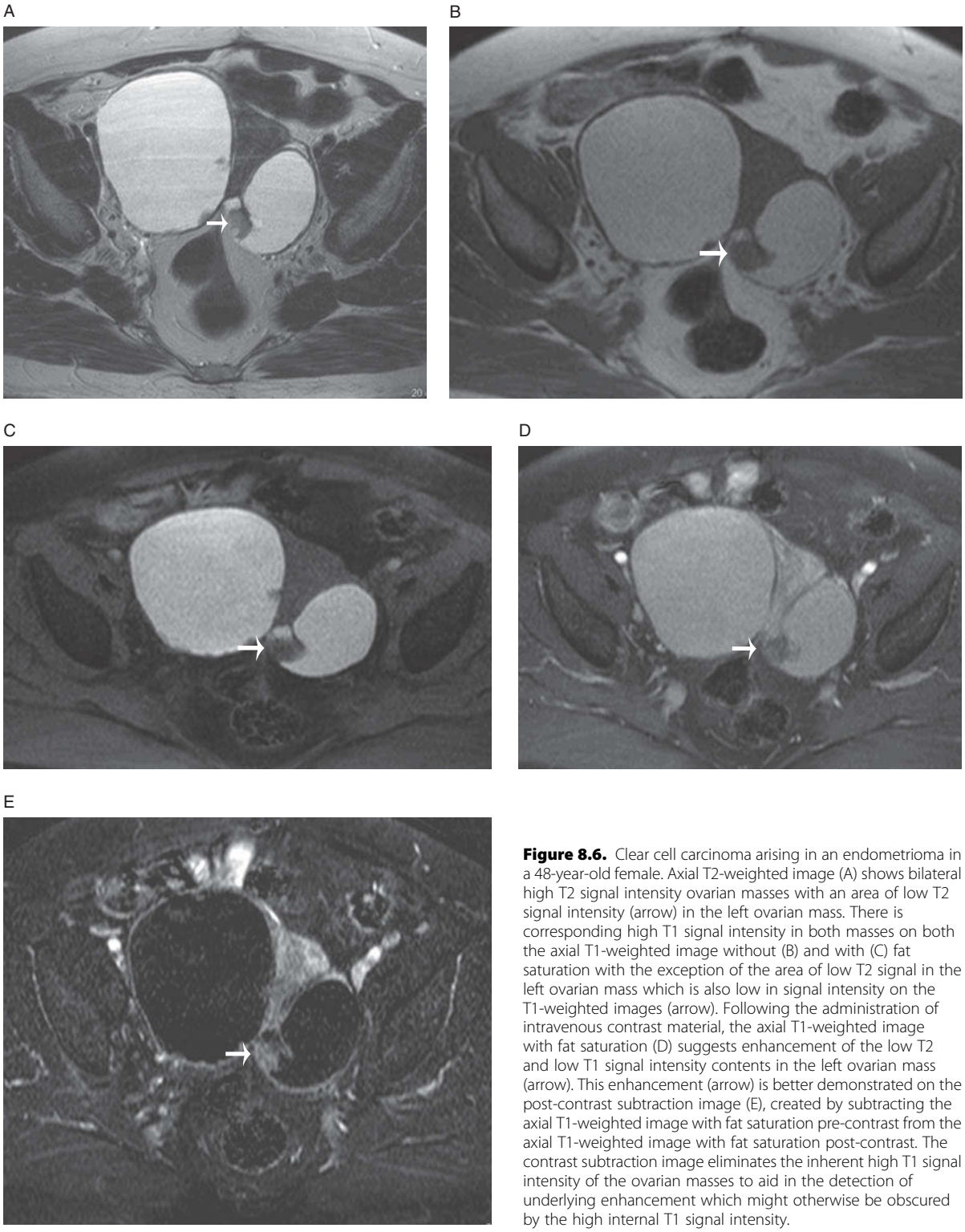

Figure 8.6. Clear cell carcinoma arising in an endometrioma in a 48-year-old female. Axial T2-weighted image (A) shows bilateral high T2 signal intensity ovarian masses with an area of low T2 signal intensity (arrow) in the left ovarian mass. There is corresponding high T1 signal intensity in both masses on both the axial T1-weighted image without (B) and with (C) fat saturation with the exception of the area of low T2 signal in the left ovarian mass which is also low in signal intensity on the T1-weighted images (arrow). Following the administration of intravenous contrast material, the axial T1-weighted image with fat saturation (D) suggests enhancement of the low T2 and low T1 signal intensity contents in the left ovarian mass (arrow). This enhancement (arrow) is better demonstrated on the post-contrast subtraction image (E), created by subtracting the axial T1-weighted image with fat saturation pre-contrast from the axial T1-weighted image with fat saturation post-contrast. The contrast subtraction image eliminates the inherent high T1 signal intensity of the ovarian masses to aid in the detection of underlying enhancement which might otherwise be obscured by the high internal T1 signal intensity.

Table 8.1 Ovarian lesions

	T1	T2	Fat	Enhancement
Simple cyst	Dark	↑↑	None	None or thin rim
Hemorrhagic cyst	↑	↑ to ↓	None	None or thin rim
Endometrioma	↑	↓	None	None or thin rim
Teratoma	↑	Variable	Characteristic	Variable
Malignancy[a]	Variable	Variable	None	Variable

[a] Adnexal masses: MRI features suggestive of malignancy:
(1) Mixed cystic and solid lesion, or solid mural nodules
(2) Thick wall and/or septations (>3 mm)
(3) Large size (>5 cm)
(4) Direct invasion of adjacent ovarian stroma
(5) *Papillary projections **are not** specific for malignancy*
(6) Ascites
(7) *Peritoneal implants **are** specific for malignancy.*

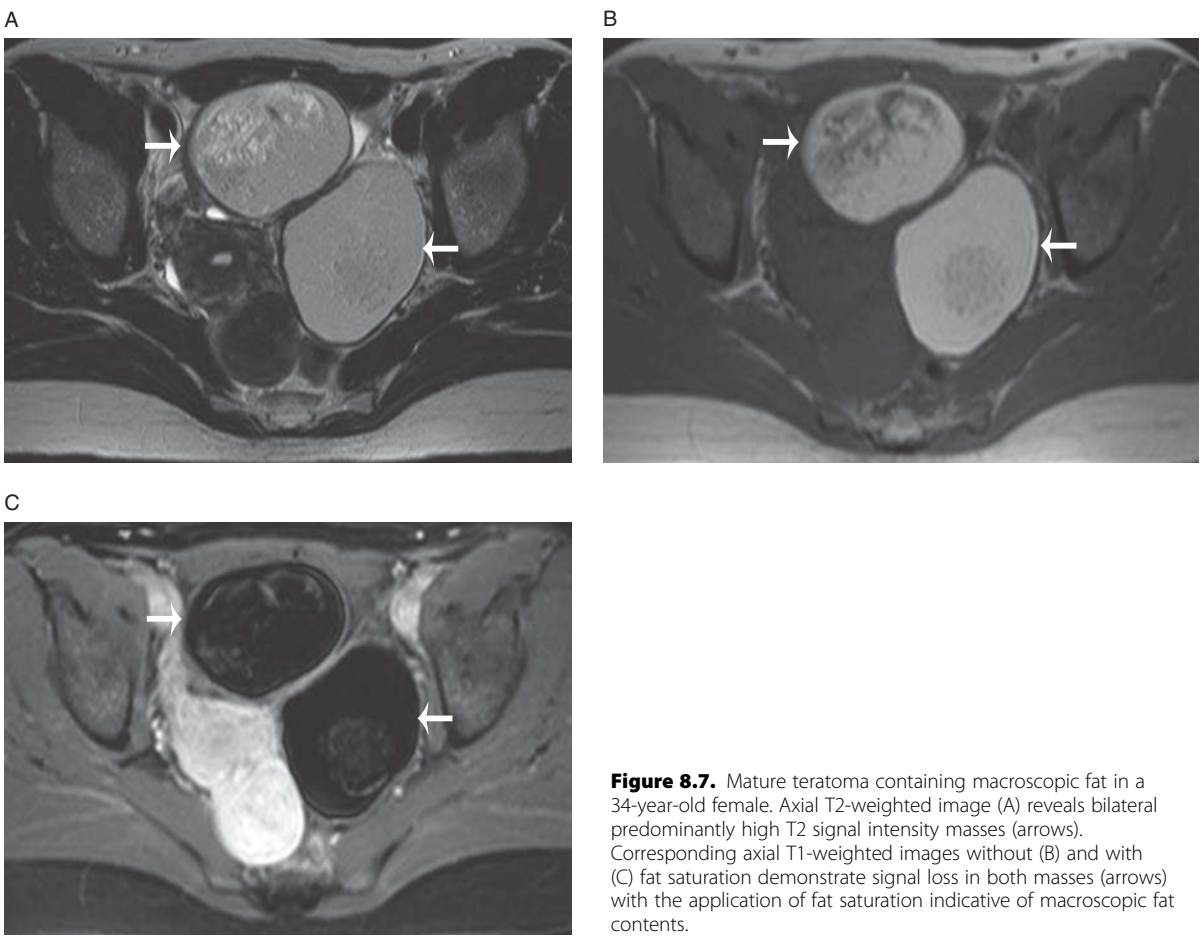

Figure 8.7. Mature teratoma containing macroscopic fat in a 34-year-old female. Axial T2-weighted image (A) reveals bilateral predominantly high T2 signal intensity masses (arrows). Corresponding axial T1-weighted images without (B) and with (C) fat saturation demonstrate signal loss in both masses (arrows) with the application of fat saturation indicative of macroscopic fat contents.

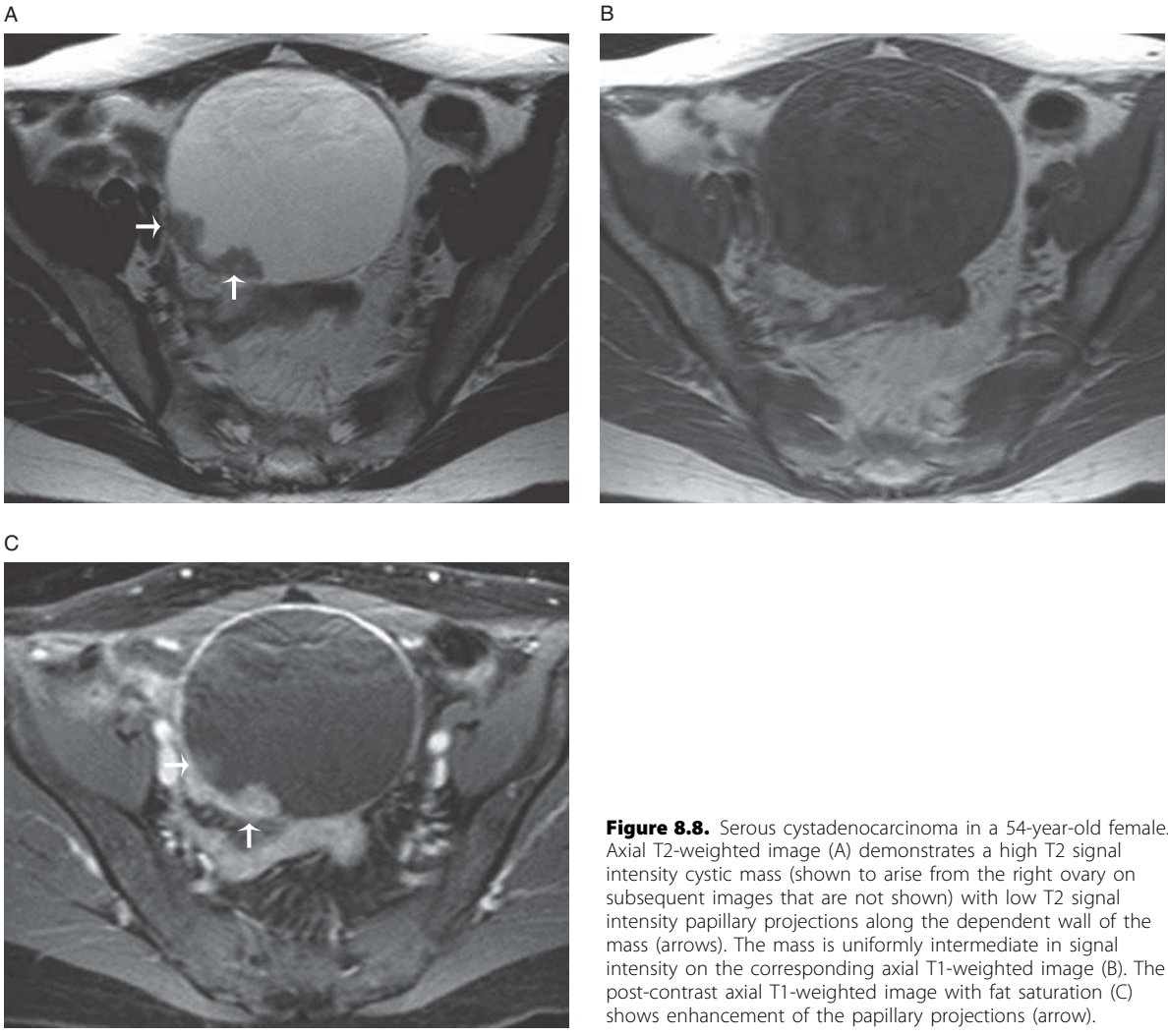

Figure 8.8. Serous cystadenocarcinoma in a 54-year-old female. Axial T2-weighted image (A) demonstrates a high T2 signal intensity cystic mass (shown to arise from the right ovary on subsequent images that are not shown) with low T2 signal intensity papillary projections along the dependent wall of the mass (arrows). The mass is uniformly intermediate in signal intensity on the corresponding axial T1-weighted image (B). The post-contrast axial T1-weighted image with fat saturation (C) shows enhancement of the papillary projections (arrow).

(Figure 8.8). Papillary projections are also more common in serous neoplasms, but do not necessarily qualify the serous lesion as a serous malignancy. Mucinous tumors are more commonly unilateral, larger, and multiloculated with variable T1 and T2 signal due to variable degrees of hydration of the mucinous contents (Figure 8.9). However, the most important thing to know and recognize are the general MRI features that make a cystic ovarian lesion suspicious enough for a malignancy to warrant surgical consultation.

ii. Solid ovarian lesions

In general, ovarian lesions that are uniformly solid on MRI are less ominous than those that are mixed cystic and solid. A solid, uniformly low T1 and low T2 signal intensity ovarian mass has a nice differential of an ovarian fibroma, fibrothecoma, or Brenner tumor. Evaluation of the endometrium can further help differentiate a fibroma from a fibrothecoma, as a thickened endometrium would be expected with an estrogen-producing fibrothecoma rather than the fibroma (Figure 8.10). All of these tumors are benign, but a fibrothecoma may be resected because of its estrogen production. In addition, Brenner tumors are often associated with another ovarian neoplasm, so the presence of an adjacent cystic ovarian neoplasm would favor the diagnosis of a Brenner tumor.

Another low T2 signal intensity solid ovarian mass to always consider in a patient with a history

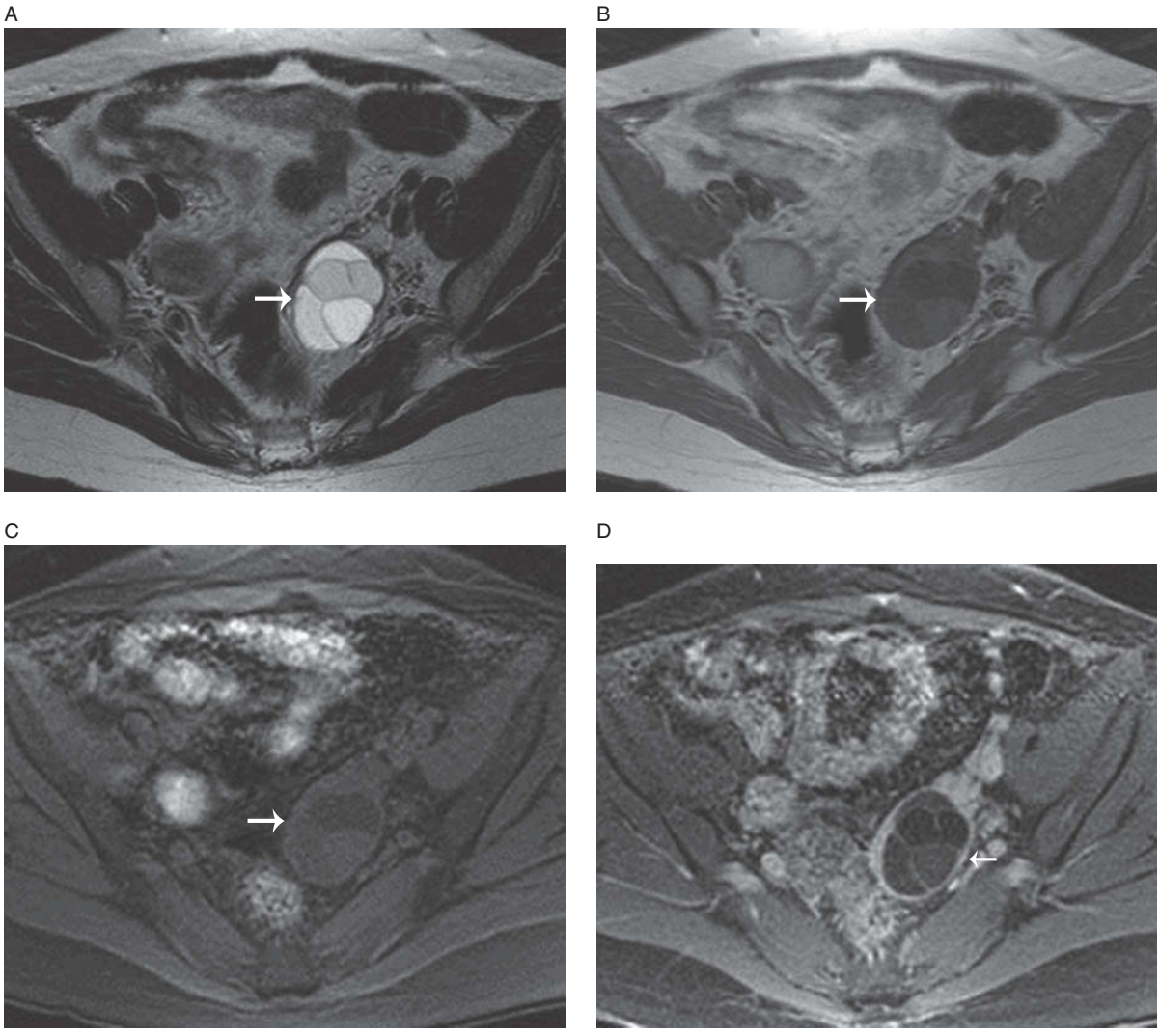

Figure 8.9. Mucinous cystadenoma in a 46-year-old female. Axial T2-weighted image (A) demonstrates a variable high and intermediate T2 signal intensity lesion in the left ovary (arrow). This lesion also has corresponding variable low and intermediate signal intensity on the corresponding axial T1-weighted images without (B) and with (C) fat saturation (arrow) due to mucinous contents. The post-contrast axial T1-weighted image with fat saturation (D) demonstrates enhancement of thin internal septations (arrow).

of malignancy is metastatic disease. Ovarian metastases are more likely to be bilateral and typically allow for preservation of the ovoid ovarian shape compared to other solid primary ovarian neoplasms. It can be difficult to differentiate between many of the solid primary malignant ovarian neoplasms, which are more often of germ cell or sex cord-stromal cell origin. These solid ovarian malignancies are typically larger and more aggressive appearing, with more variable T1 and T2 signal, than fibromas and fibrothecomas. Similar to the malignant appearing cystic ovarian lesions, suggesting a specific pathologic diagnosis for a solid ovarian malignancy on MRI is nice, but not required.

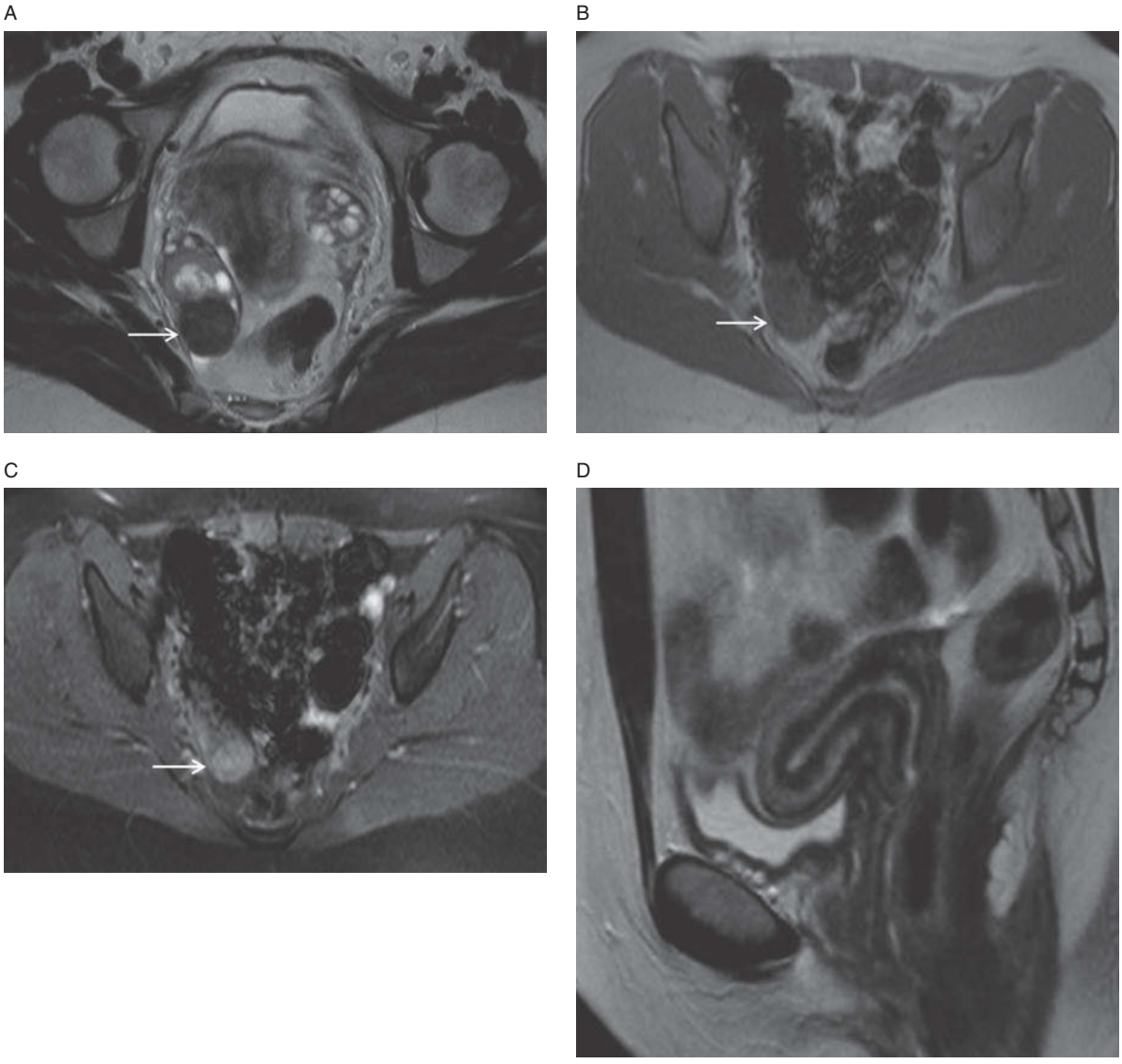

Figure 8.10. Fibroma in a 65-year-old female. Axial T2-weighted image (A) demonstrates a low T2 signal intensity mass in the right ovary (arrow). The mass is uniformly intermediate in signal intensity (arrow) on the corresponding axial T1-weighted image (B). The post-contrast axial T1-weighted image with fat saturation (C) shows uniform low level enhancement of the mass (arrow). The sagittal T2-weighted image through the uterus (D) demonstrates the absence of endometrial thickening. A normal thickness endometrium in this post-menopausal patient favors that the mass is a fibroma rather than a hormonally active fibrothecoma.

Pearls and pitfalls

(1) T1 weighted images without and with fat saturation are critical to characterize fat us blood products
(2) MRI features suggestive of malignancy in cystic ovarian lesions.
   (a) Mural nodules
   (b) Thick wall, septations
   (c) Size >5 cm
   (d) Ascites
   (e) Peritoneal implants

Chapter 9

Female urethra

Female urethra protocol

Indications

This protocol is primarily used to evaluate urethral diverticula. It is also used to evaluate indeterminate urethral and vaginal masses.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- Have the patient void prior to the start of the study
- Give patient 2 L nasal oxygen
- Start IV with at least 24-gauge needle; connect to power injector
- Cover from dome of bladder and uterine fundus through proximal thighs. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Sagittal T2 single-shot fast-spin echo – Evaluate coil placement, localize pathology for proper planning of T2 SSFSE sequences.
(2) Sagittal T2 fast-spin echo – Evaluate urethral anatomy. Identify T2-bright and T2-dark lesions.

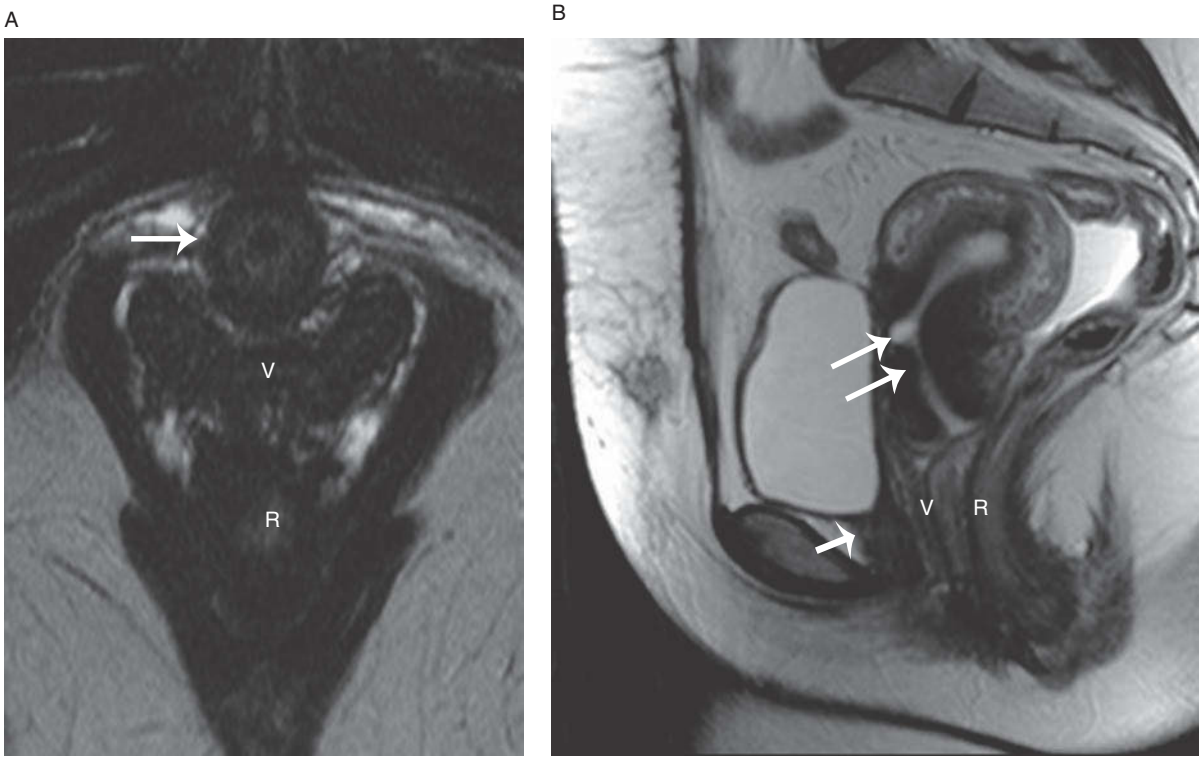

Figure 9.1. Normal urethra in a 39-year-old female. Axial (A) and sagittal (B) T2-weighted images demonstrate the normal concentric ring appearance of the urethra (arrow). Also note normal appearance of the vagina (V) and rectum (R). Also note incidectal C-section scar (B, double arrow)

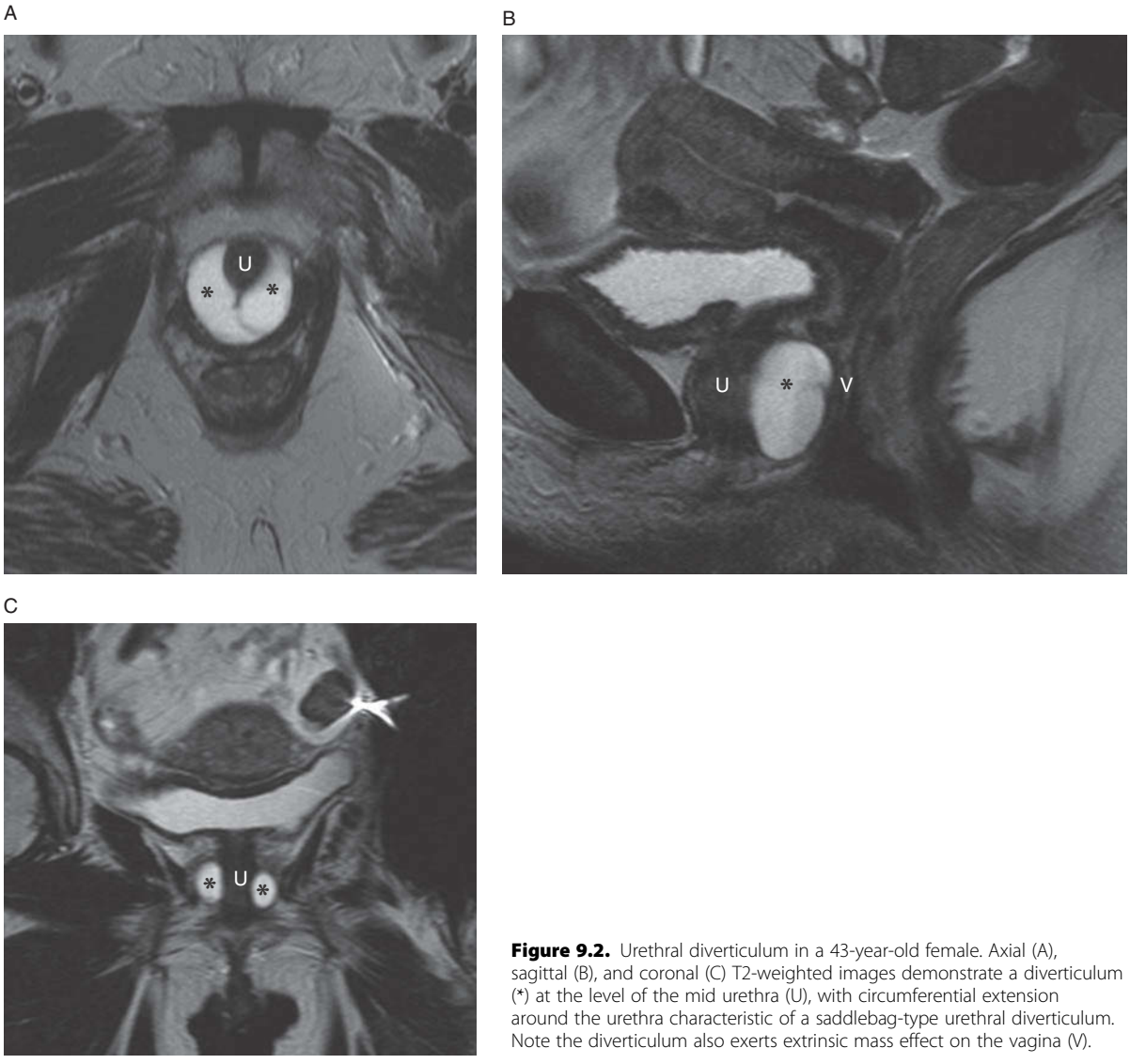

Figure 9.2. Urethral diverticulum in a 43-year-old female. Axial (A), sagittal (B), and coronal (C) T2-weighted images demonstrate a diverticulum (*) at the level of the mid urethra (U), with circumferential extension around the urethra characteristic of a saddlebag-type urethral diverticulum. Note the diverticulum also exerts extrinsic mass effect on the vagina (V).

(3) Axial T2 fast-spin echo – Identify T2-bright and T2-dark lesions.
(4) Coronal T2 FSE– Identify T2-bright and T2-dark lesions.
(5) Axial volume-interpolated gradient echo BH pre – Identify T1-bright lesions, blood products.
(6) Axial volume-interpolated gradient echo BH post at 70 seconds.
(7) Sagittal volume-interpolated gradient echo BH post to follow.
(8) Coronal volume-interpolated gradient echo BH post to follow.

Approach to MRI exam intepretation of female urethra

The high soft-tissue resolution of MRI allows terrific imaging of the urethra. Most commonly, MRI is requested for evaluation of a suspected urethral diverticulum which classically presents with post-void dribbling. The referring doctor has a high clinical index of suspicion for this diagnosis based on patient symptoms and physical exam. We can confirm the diagnosis and exclude others. But we don't always find what we think we're looking for so we need to

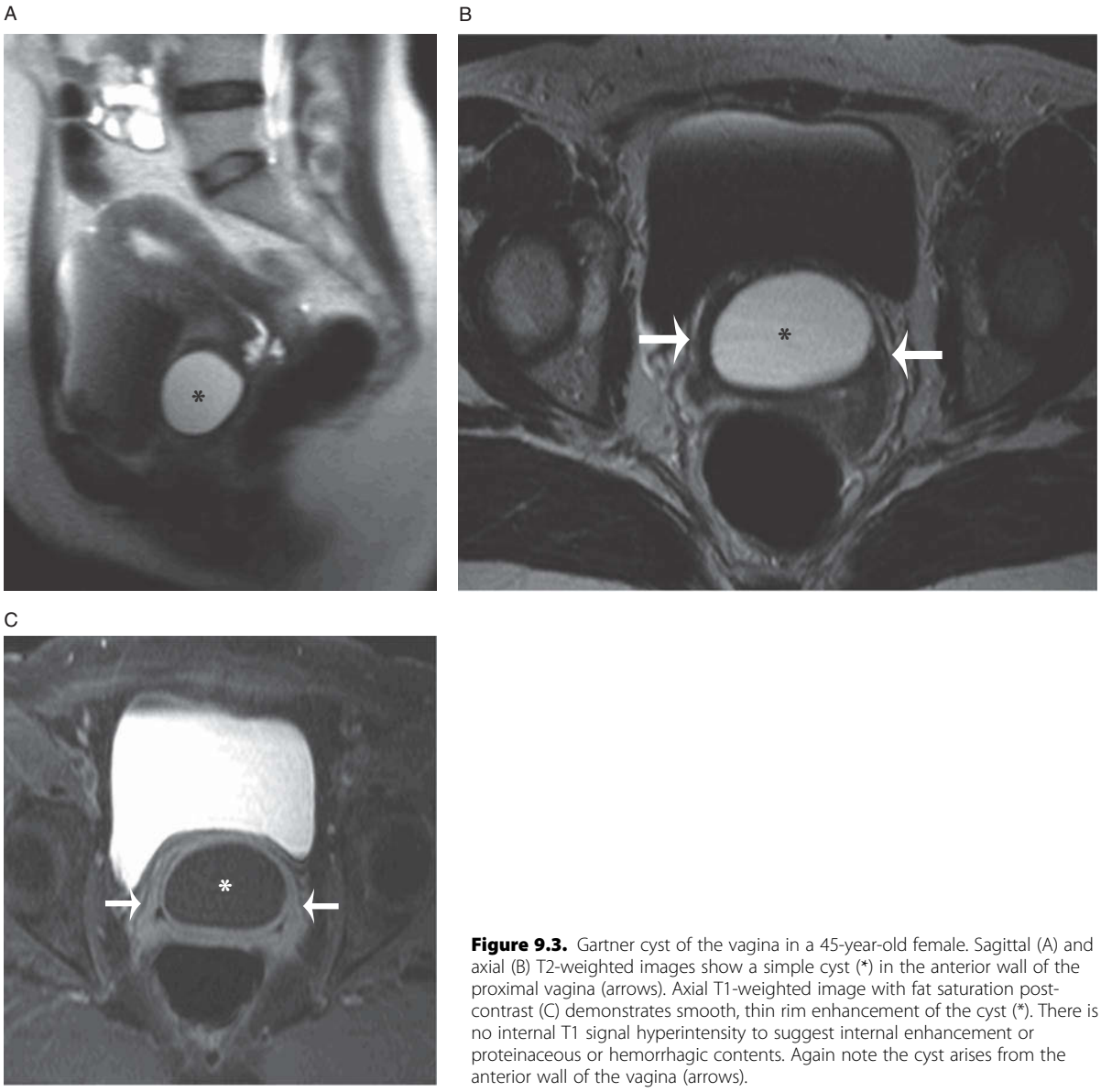

Figure 9.3. Gartner cyst of the vagina in a 45-year-old female. Sagittal (A) and axial (B) T2-weighted images show a simple cyst (*) in the anterior wall of the proximal vagina (arrows). Axial T1-weighted image with fat saturation post-contrast (C) demonstrates smooth, thin rim enhancement of the cyst (*). There is no internal T1 signal hyperintensity to suggest internal enhancement or proteinaceous or hemorrhagic contents. Again note the cyst arises from the anterior wall of the vagina (arrows).

be prepared to diagnose or exclude a solid urethral mass or a cystic vaginal mass. Treatment for a urethral diverticulum is surgical resection.

Approach

Sagittal single-shot fast-spin echo localization: T2-weighted images

This sequence is essentially a scout to ensure adequate surface coil coverage. The coil has to be placed lower than may be expected to cover the entire urethra.

Multiplanar T2 fast-spin echo: T2-weighted images

These are the key sequences for evaluating urethral and vaginal anatomy as well as the nature (cystic versus solid) and site of origin (urethra vs. vagina) of a suspected urethral lesion (Figure 9.1).

Urethral diverticula are very common. They are high signal intensity on T2-weighted images and typically arise from the posterior mid urethra (Figure 9.2). Stones may form within a urethral diverticulum are low T2 signal intensity (just like common bile duct stones).

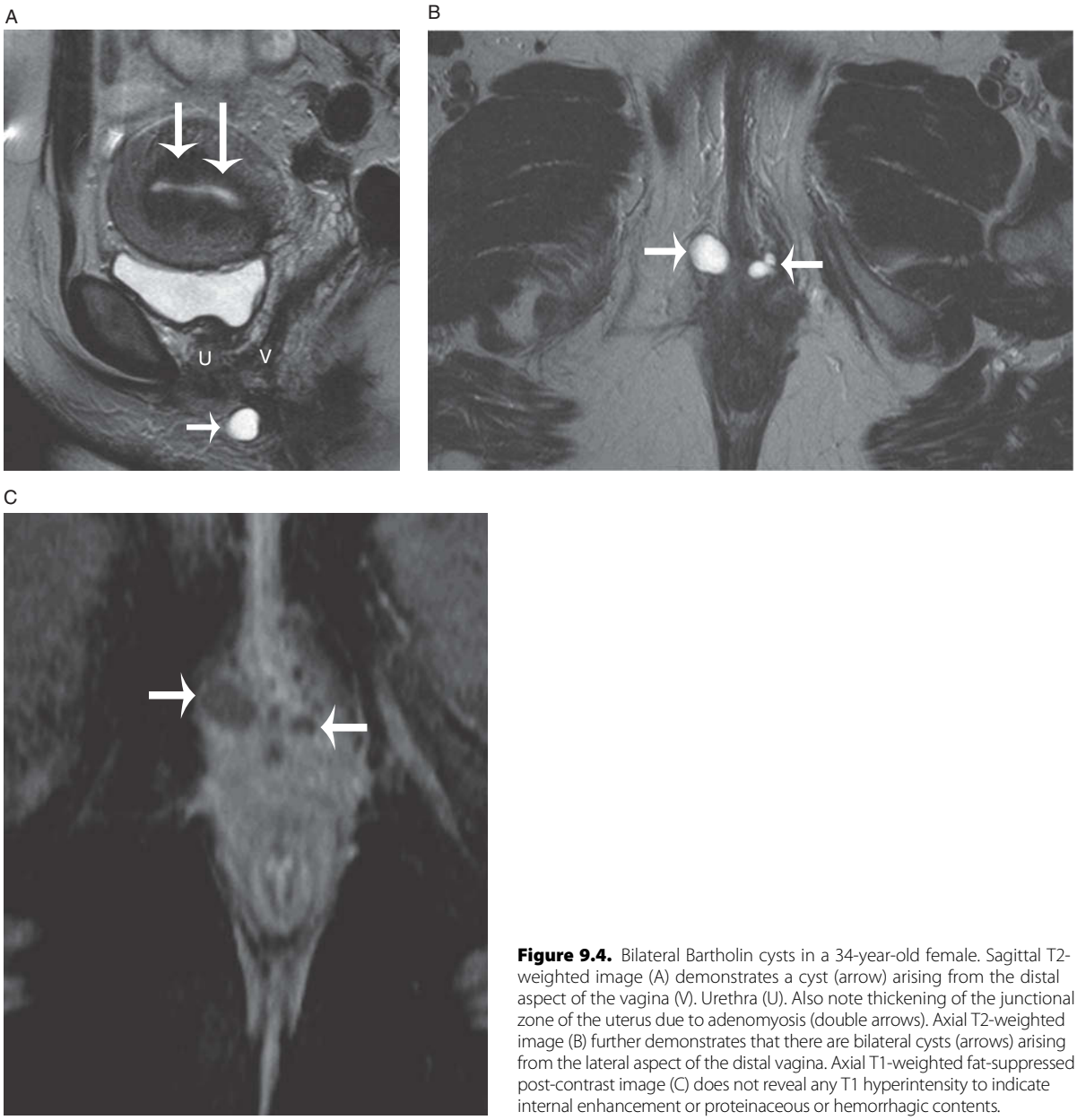

Figure 9.4. Bilateral Bartholin cysts in a 34-year-old female. Sagittal T2-weighted image (A) demonstrates a cyst (arrow) arising from the distal aspect of the vagina (V). Urethra (U). Also note thickening of the junctional zone of the uterus due to adenomyosis (double arrows). Axial T2-weighted image (B) further demonstrates that there are bilateral cysts (arrows) arising from the lateral aspect of the distal vagina. Axial T1-weighted fat-suppressed post-contrast image (C) does not reveal any T1 hyperintensity to indicate internal enhancement or proteinaceous or hemorrhagic contents.

Vaginal cysts are also bright on T2-weighted images. Gartner duct cysts arise from the proximal anterolateral vaginal wall, Bartholin gland cysts from the posterolateral distal vagina, and Skenes gland cysts arise from periurethral glands adjacent to the distal urethra (Figures 9.3–9.5).

Solid masses of the urethra and vagina, (including leiomyomas and carcinomas) are typically intermediate to high signal intensity on T2-weighted images (Figures 9.6 and 9.7).

Pre-contrast volume-interpolated gradient echo BH: T1-weighted images, fat saturation

This sequence is used to identify T1-bright signal intensity within urethral and vaginal lesions. Vaginal cysts often have hemorrhagic or proteinaceous contents. This sequence also serves as a comparison for

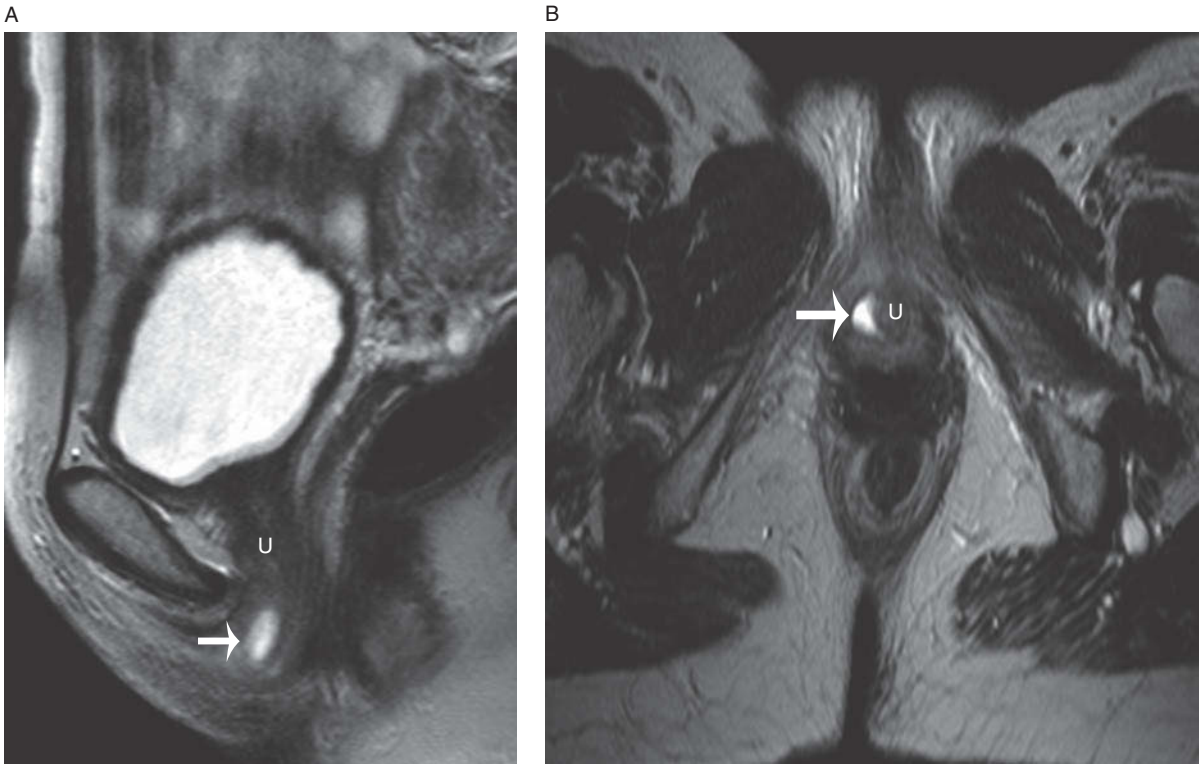

Figure 9.5. Skene's gland cyst in a 47-year-old female. Sagittal T2-weighted image (A) reveals a cyst (arrow) located distal to the urethra (U). Axial T2-weighted image (B) further reveals that the cyst (arrow) is lateral to the distal urethra (U).

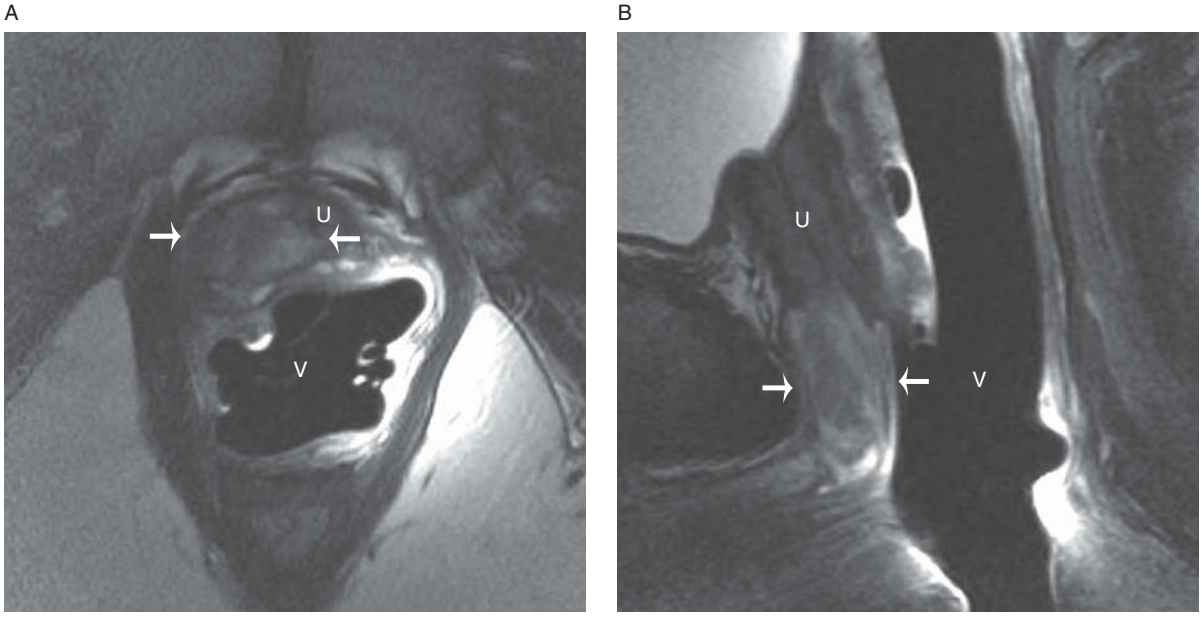

Figure 9.6. Squamous cell carcinoma of the distal urethra in a 52-year-old female. Axial (A) and sagittal (B) T2-weighted images illustrate a heterogeneous intermediate to high signal intensity mass (arrow) arising from the right lateral wall of the distal urethra (U). Note endoluminal coil in the vagina (V).

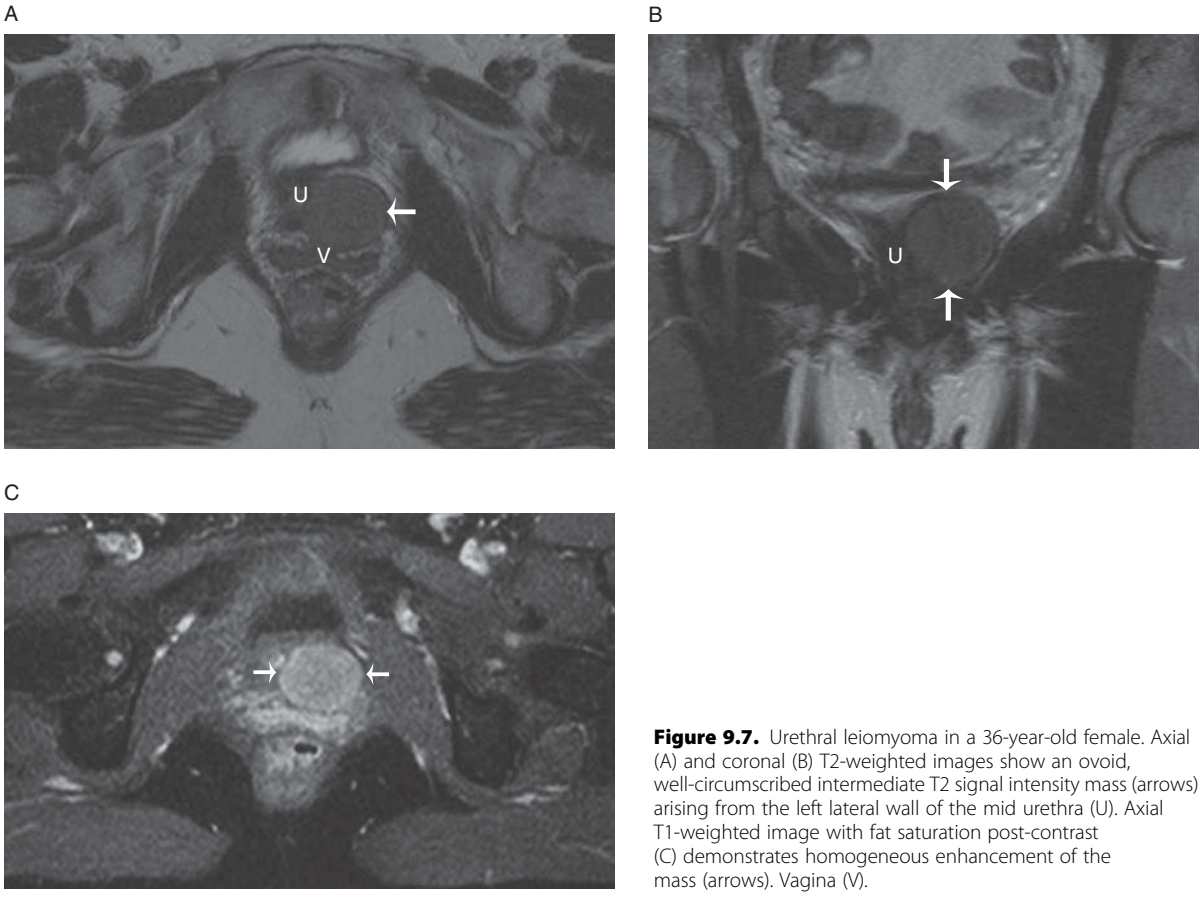

Figure 9.7. Urethral leiomyoma in a 36-year-old female. Axial (A) and coronal (B) T2-weighted images show an ovoid, well-circumscribed intermediate T2 signal intensity mass (arrows) arising from the left lateral wall of the mid urethra (U). Axial T1-weighted image with fat saturation post-contrast (C) demonstrates homogeneous enhancement of the mass (arrows). Vagina (V).

post contrast T1-weighted images. We don't want to mistake this T1-bright material for enhancement.

Post-contrast volume-interpolated gradient echo: T1-weighted images, fat saturation

This sequence is used to confirm the cystic versus solid nature of a urethral or vaginal mass. Solid masses have internal enhancement. (Figure 9.7) Cystic lesions do not. Contrast can also help localize the neck of a urethral diverticulum, which is important for planning surgical resection. Post-contrast images are performed after a standard 70-second delay. More delayed imaging post-void can also be performed in an attempt to fill a suspected diverticulum with intravenous contrast if the diagnosis remains uncertain.

Pearls and pitfalls

(1) Urethral diverticula most commonly arise from the posterior aspect of the mid urethra.
(2) Identifying the location of the neck of a urethral diverticulum is helpful for surgical planning. However, the neck can be difficult to identify even with MRI. Imaging after the administration of intravenous contrast material may help localize the neck.
(3) Gartner duct cysts arise from the anterolateral wall of the proximal vagina and Bartholin gland cysts originate in the posterolateral aspect of the distal vagina.
(4) Skene's gland cysts originate lateral to the external urethral meatus.

Chapter 10

Pelvic floor / prolapse

Pelvic prolapse protocol

Indications

This protocol is used to evaluate disorders of the pelvic floor including pelvic organ prolapse and defecatory function. Indications include pelvic organ prolapse, urinary and fecal incontinence or retention, and possible enteroceles, sigmoidoceles, and rectoceles.

Preparation

- **IV contrast agent**: None
- **Oral contrast agent**: None
- NPO for 4 hours prior to the exam
- Have the patient void prior to the start of the study
- 120 cc sonography gel placed per rectum using 60 cc Tume syringe
- Center patient on water-absorbent pad
- Place cushion underneath the patient's knees
- Center pelvic phased array coil slightly lower than usual on pelvis to cover the proximal thighs. Pelvic organs may descend during stress maneuvers.
- Ensure patient understands how to perform Kegel and Valsalva maneuvers and understands the importance of evacuating the rectal contrast during the sagittal dynamic True FISP sequence.

Exam sequences

(1) Sagittal T2 single-shot fast-spin echo (rest) – Measure bladder, uterine, and rectal points of reference to chosen reference line.
(2) Sagittal T2 single-shot fast-spin echo (Kegel) – Measure bladder, uterine, and rectal points of reference to chosen reference line.
(3) Sagittal True FISP (rest, Kegel, defecation, rest): Update image acquisition every 1 second for 60 seconds. If patient is unable to evacuate the rectal contrast with the first series, repeat × 1–2. Evaluate defecatory function. Measure bladder, uterine, and rectal points of reference to chosen reference line.
(4) Sagittal T2 single-shot fast-spin echo (Valsalva) – Measure bladder, uterine, and rectal points of reference to chosen reference line.
(5) Axial T2 fast-spin echo – Evaluate pelvic floor muscles and ligaments.
(6) Coronal T2 single-shot fast-spin echo (Valsalva) – Evaluate for lateral pelvic organ prolapse and lateral rectoceles.
(7) Coronal T2 fast-spin echo – Evaluate pelvic floor muscles and ligaments.

Approach to pelvic prolapse MRI exam interpretation

The unparalleled soft-tissue resolution, multiplanar, and dynamic imaging capabilities of MRI allow for comprehensive evaluation of pelvic organ prolapse, cul-de-sac defects, defecatory function, and pelvic-floor support in all three pelvic compartments with one (relatively) non-invasive examination. The most common indications for MRI of pelvic-floor disorders include:

(1) Symptoms involving multiple pelvic compartments (MRI can evaluate anterior, middle, and posterior pelvic compartments simultaneously)
(2) Defecatory dysfunction with posterior compartment abnormality
(3) Severe prolapse
(4) Prior surgical repair
(5) Poor correlation between physical exam and symptomatology.

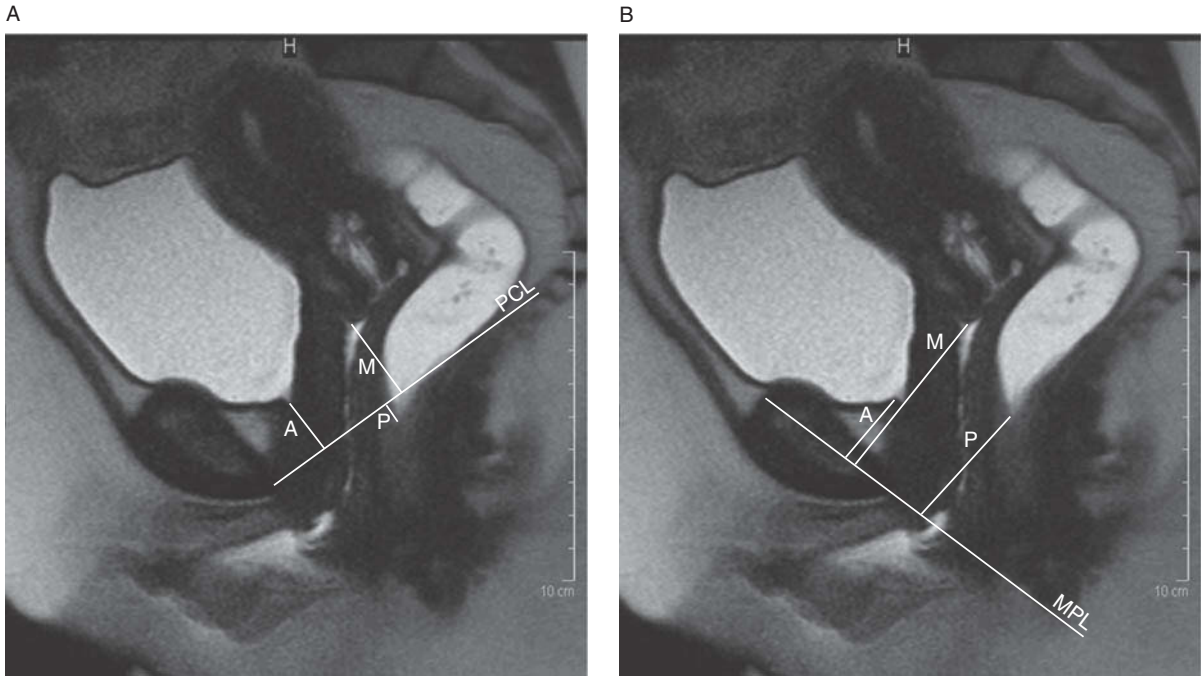

Figure 10.1. Pelvic floor reference lines. Midline sagittal T2-weighted SSFSE images (A, B) illustrate the pelvic floor reference lines. The pubococcygeal line (PCL) (long white line) extends from the inferior symphysis pubis to the last joint of the coccyx (A) and the midpubic line (MPL) (long white line) extends along the long axis of the symphysis pubis (B). Perpendicular measurements from anatomic reference points in the anterior (A), middle (M), and posterior (P) compartments (shorter white lines) are also depicted with respect to the PCL and MPL.

Approach

Sagittal T2 single-shot fast-spin echo: T2-weighted images

Coverage for this sequence extends laterally from femoral head to femoral head while the patient is imaged first at rest and then during Kegel and Valsalva maneuver*. These sequences are used to detect and stage pelvic organ prolapse based on the distance of the pelvic organs from a chosen fixed bony landmark or reference line for the pelvic floor.

The most commonly used reference lines are the *pubococcygeal line*, which extends from the inferior aspect of the symphysis pubis to the last joint of the coccyx, and *the midpubic line*, which extends along the long axis of the symphysis pubis and which approximates the level of the vaginal hymen. The pelvis is then divided into three compartments for purposes of reporting pelvic organ prolapse:

Anterior = bladder and urethra
Middle = uterus and vagina
Posterior = rectum and anal canal.

Measure and report the distance from the reference line (Figure 10.1) to the:

Posterior bladder base (anterior compartment)
Anterior cervical lip (in the middle compartment)
Anterior inferior anorectal junction (posterior compartment).

The largest measurement during strain or evacuation, defined as the measurement farthest below the reference line or closest to the reference line if located above the line, is used to stage the presence and degree of pelvic organ prolapse (Figures 10.2 and 10.3; Table 10.1 and Table 10.2).

Cul-de-sac defects which allow fat (peritoneocele), small bowel (enterocele), or large bowel (sigmoidocele) to extend between the posterior vaginal and anterior rectal walls are evaluated on the Valsalva images

* Kegel maneuver: Contraction of muscles used to stop flow of urine.
Valsalva maneuver: Attempt to exhale against closed glottis.

Table 10.1 Staging pelvic organ prolapse using pubococcygeal line (PCL)

Stage	Criterion[a]
Small prolapse	1 to <3 cm below PCL
Moderate prolapse	3 to 6 cm below PCL
Large prolapse	>6 cm below PCL

[a] Distance of inferior bladder base, anterior cervical lip, anterior anorectal junction from PCL.

Table 10.2 Staging pelvic organ prolapse using midpubic line (MPL)

Stage	Criterion[a]
0	>3 cm to (TVL[b] – 2 cm) above MPL
1	Does not meet stage 0, but >1 cm above MPL
2	≤1 cm above or below MPL
3	>1 cm below MPL
4	Complete organ eversion

[a] Distance of inferior bladder base, anterior cervical lip, and anterior anorectal junction from MPL.
[b] On sagittal MRI images, total vaginal length (TVL) is the greatest vertical vaginal measurement in centimeters from the posterior vaginal fornix to the level of the introitus in patients with a cervix and from the most superior aspect of the vaginal cuff to the level of the introitus in patients without a cervix.

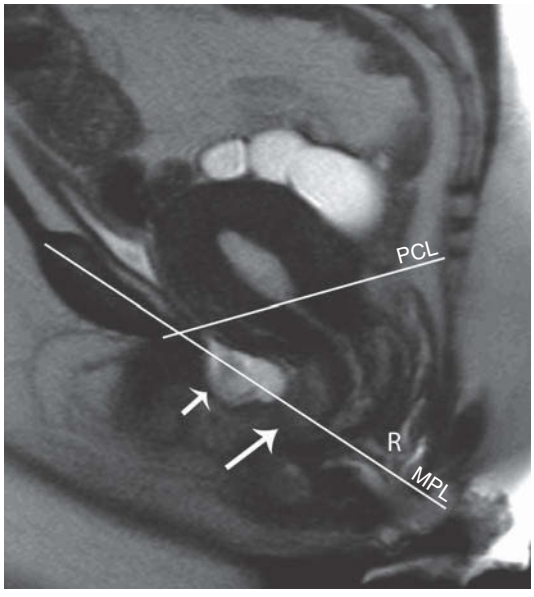

Figure 10.2. Global pelvic prolapse in a 46-year-old female. Sagittal T2-weighted SSFSE image obtained during Valsalva maneuver reveals descent of the bladder (short arrow) and the uterus (long arrow) below both the midpubic line (MPL) and the pubococcygeal line (PCL). The bladder base is 1.5 cm below the MPL and 4.5 cm below the PCL corresponding to Stage 3 or moderate bladder prolapse. The anterior cervical lip is 1.3 cm below the MPL and 5.5 cm below the PCL corresponding to Stage 3 or moderate uterine prolapse. Also note rectal descent (R), although evaluation of the rectum is limited on this image as the rectal contrast material has already been evacuated.

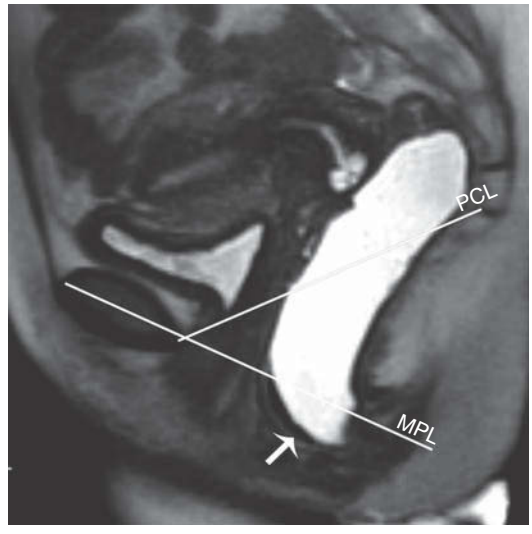

Figure 10.3. Rectal prolapse in a 53-year-old female. Sagittal T2-weighted SSFSE image obtained during Valsalva maneuver reveals descent of the rectum (arrow) below both the midpubic line (MPL) and the pubococcygeal line (PCL). The anterior inferior anorectal junction is 1.6 cm below the MPL and 5.2 cm below the PCL corresponding to Stage 3 or moderate rectal descent.

(Figure 10.4). If such a defect is present, the length to which fat or bowel extends between the vagina and rectum is measured (>2 cm is considered significant).

Rectoceles, rectal wall bulges due to loss of rectal fascial support, are usually anterior in location, and are also best evaluated on the sagittal Valsalva images (Figure 10.5). A rectal bulge extending more than 2 cm beyond the expected location of the rectal wall is typically considered reportable (Table 10.3).

Coronal T2 SSFSE: T2-weighted images

Performed during Valsalva to detect lateral pelvic organ prolapse and lateral rectoceles.

Sagittal True FISP: steady-state free-precession images

This is a very fast sequence which allows for real-time imaging of the pelvis during defecation (Isn't technology terrific? What a great time to be alive!). Images can be viewed in cine mode.

Table 10.3 Staging rectoceles

Stage	Criterion[a]
Small	2 to 4 cm
Moderate	4 to 6 cm
Large	>6 cm

[a] Distance of the rectal bulge from the anterior anal wall/ expected location of the anterior rectal wall.

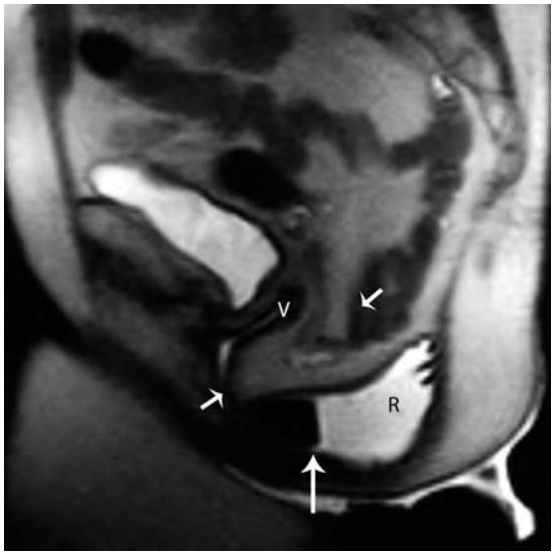

Figure 10.4. Cul-de-sac defect in a 74-year-old female. Sagittal T2-weighted SSFSE image obtained during Valsalva maneuver reveals descent of the sigmoid colon and peritoneal fat (short arrows) into the rectovaginal space. Also note an anterior rectocele (long arrow). V, vaginal canal; R, rectum.

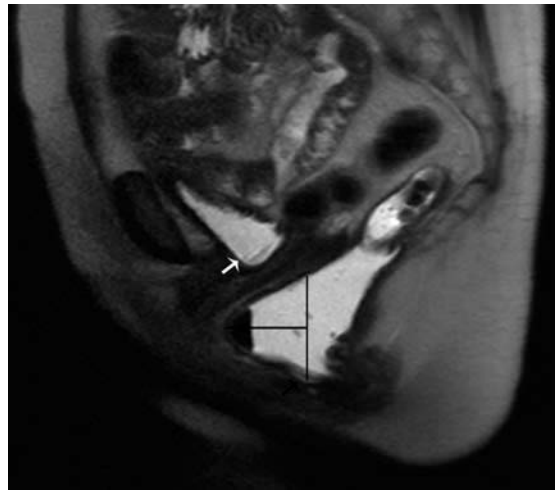

Figure 10.5. Anterior rectocele and rectal and bladder prolapse in a 66-year-old female. Sagittal T2-weighted SSFSE image obtained during Valsalva maneuver reveal an anterior rectocele (long black arrow). The anterior rectal bulge extends 3.5 cm anterior to the anterior anal wall/expected anterior rectal wall (straight black line). Also note descent of the bladder (white arrow) and rectum (short black arrow).

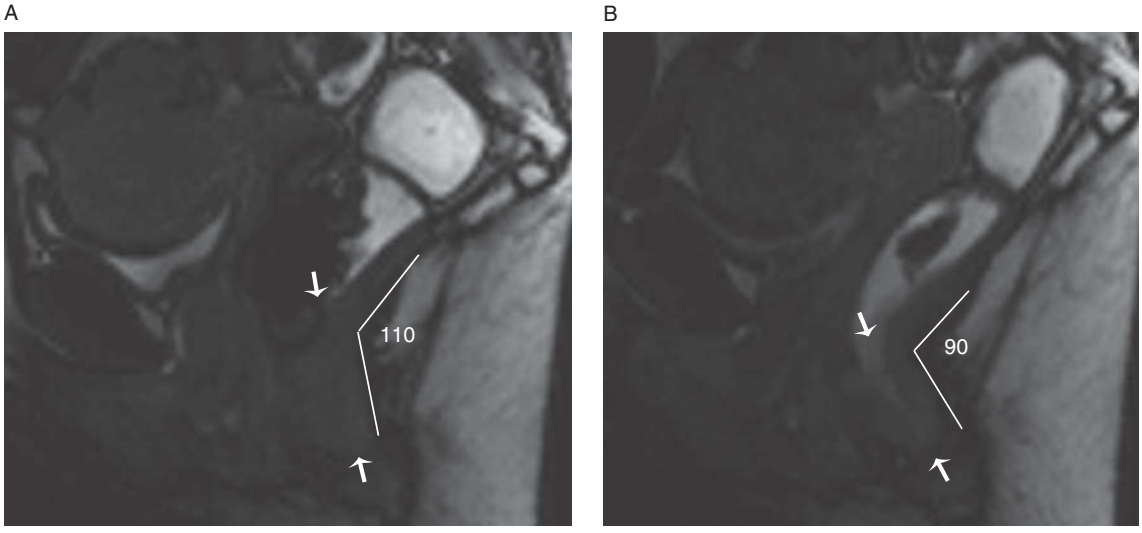

Figure 10.6. Anismus in a 35-year-old female. Sagittal true FISP images obtained with the patient at rest (A) and during attempted evacuation (B) show a more acute change in the anorectal angle with evacuation, 110° to 90°. Also, note the anal canal (arrows) does not shorten or widen during attempted evacuation. The patient was unable to evacuate the rectal contrast material despite three attempts.

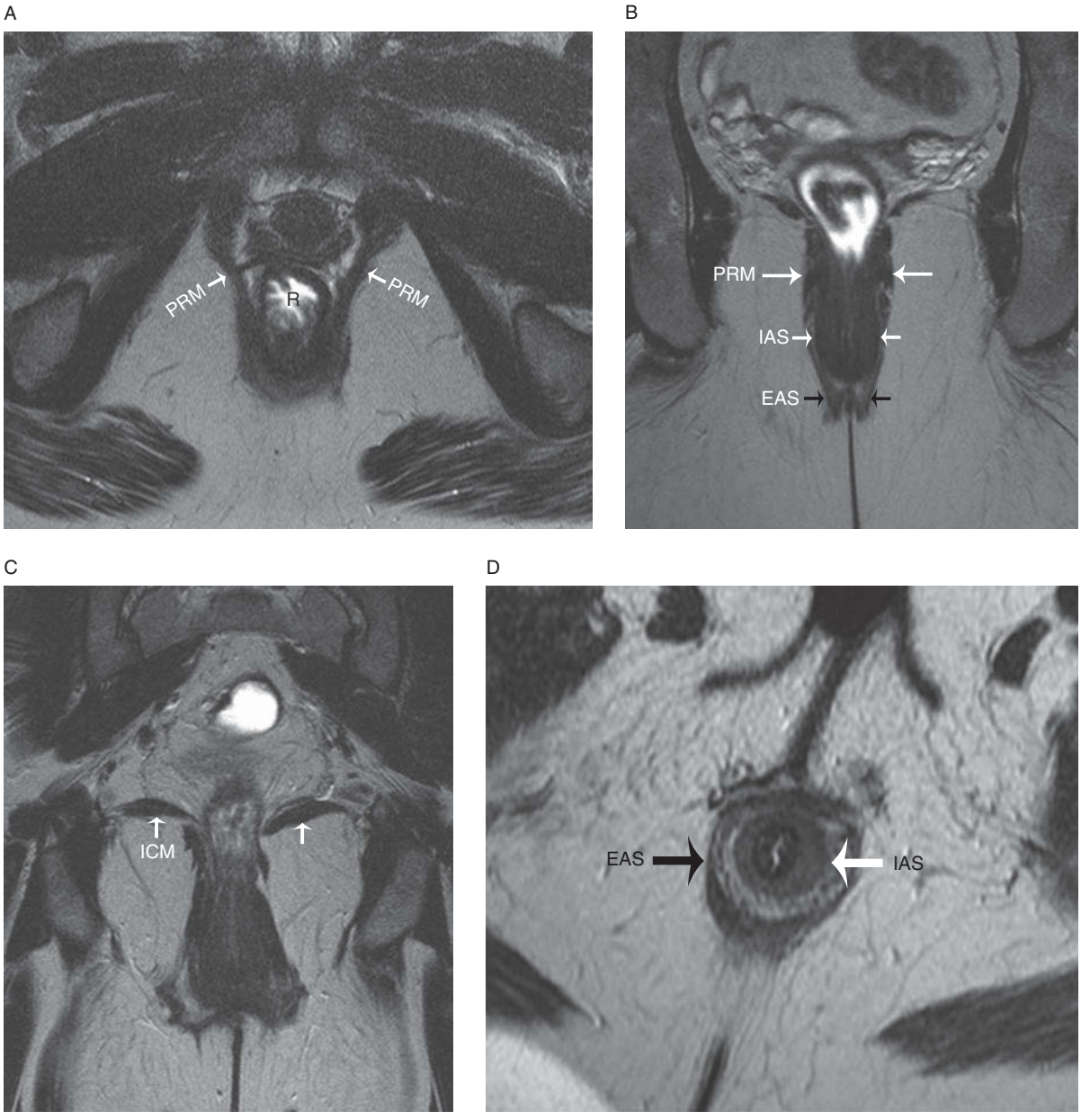

Figure 10.7. Normal levator ani and anal sphincter anatomy in a 47-year-old female. Axial T2-weighted FSE image (A) at the level of the anorectal junction shows an intact puborectalis muscle (arrows) forming a sling around the rectum (R). Coronal T2-weighted FSE image (B) at the level of the rectum shows the relationship between the internal anal sphincter (short white arrows), external anal sphincter (black arrows), and puborectalis muscles (long white arrows). Coronal T2-weighted FSE image (C) at the level of the rectum shows the normal iliococcygeus muscles (arrows) with upward convexity. Axial T2-weighted FSE image (D) at the level of the anal canal illustrates the normal internal (white arrow) and external (black arrow) anal sphincter muscles.

Normal defecation:

(1) the patient readily evacuates the rectal contrast material

(2) resting anorectal angle increases (becomes more obtuse) by 10–15° (normal 100° to 115°)

(3) the anal canal shortens and widens.

Paradoxical contraction of the puborectalis muscle (a.k.a. pelvic dyssynergia or anismus) (Figure 10.6):

(1) the patient is unable to evacuate the rectal contrast material

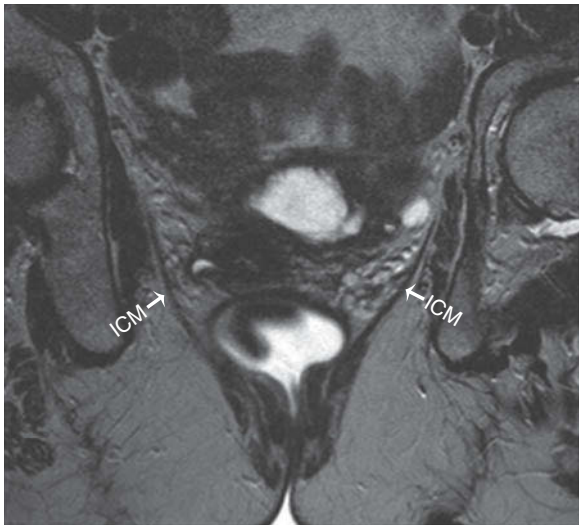

Figure 10.8. Atrophic iliococcygeus muscles in a 52-year-old female with global pelvic prolapse. Coronal T2-weighted FSE image at the level of the rectum reveals thinning and downward convexity of the iliococcygeus muscles (arrows).

(2) the anorectal angle does not change or becomes more acute
(3) the puborectalis muscle impression persists.

This is most commonly seen in young women with chronic constipation. Please note that the ability to evacuate rectal contrast trumps the anorectal angle. That is, if they can defecate, the exam is normal.

Rectal intussusception can also be evaluated with this sequence although MRI is not as sensitive as fluoroscopic defecography for detecting rectal intussusception.

T2 FSE: T2-weighted images

These high-resolution images are used to evaluate the pelvic floor muscles: the levator ani (iliococcygeus, puborectalis, pubococcygeus) and anal sphincter (external, internal) muscles (Figures 10.7). Any findings of muscular atrophy or muscle tears should be reported (Figures 10.8 and 10.9). The iliococcygeus muscle is best visualized in the coronal plane, the puborectalis muscle in the coronal and axial planes (the pubococcygeus is not separately visualized from the puborectalis muscle on MRI), and the external and internal anal sphincter muscles are visible in both the coronal and axial planes.

The supportive ligaments of the pelvic floor are not routinely visualized. Defects in the ligaments and fascia are inferred from the presence of pelvic organ prolapse.

An approach to interpreting pelvic prolapse examinations is outlined in Table 10.4.

Table 10.4 Summary algorithm for interpreting MRI prolapse examinations

Step 1: Draw chosen pelvic floor reference line, pubococcygeal or midpubic line, on midsagittal T2 SSFSE image.
Step 2: Measure distances from anterior, middle, and posterior compartment reference points to pelvic floor reference line on sagittal T2 SSFSE.
Step 3: Stage presence and degree of prolapse in each compartment on sagittal T2 SSFSE.
Step 4: Evaluate for cul-de-sac defect on sagittal T2 SSFSE. Measure length of extension of fat or bowel between vagina and rectum.
Step 5: Evaluate for rectocele on sagittal and coronal T2 SSFSE. Measure distance of rectal bulge from anal wall/expected location of rectal wall.
Step 6: Evaluate defecatory function from sagittal true FISP. Assess change in anorectal angle and ability to evacuate rectal contrast.
Step 7: Evaluate integrity of pelvic floor muscles from axial and coronal T2 FSE.

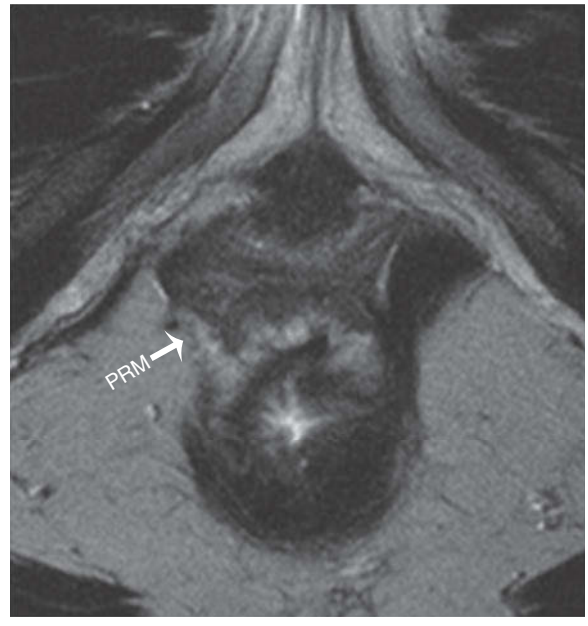

Figure 10.9. Tear of the right puborectalis muscle in a 48-year-old female with pelvic prolapse and incontinence. Axial T2-weighted FSE image at the level of the anorectal junctions shows thinning posteriorly (arrow) and discontinuity anteriorly of the puborectalis muscle on the right.

Pearls and pitfalls

(1) A comprehensive MRI examination of the pelvic floor will evaluate pelvic organ prolapse, defecatory function, and pelvic-floor support structures.
(2) The most reliable exams are obtained with maximal Valsalva maneuver and evacuation of rectal contrast material during real-time imaging.
(3) The patient should be given multiple (at least three) attempts to evacuate the rectal contrast material before considering the examination abnormal.

Chapter 11

Imaging of the pregnant patient

Pregnant patient pelvic imaging protocol

Indications

This protocol is used to evaluate adnexal masses and fibroids in pregnant patients. It can also be used to evaluate nonspecific pelvic pain during pregnancy and appendicitis.

Preparation

- **IV contrast agent**: None
- **Oral contrast agent**: None
- Have the patient void prior to the start of the study
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage
- All T2 single-shot fast-spin echo sequences can be run without breath-hold to continuously cover the pelvis and pathology as one series.

Exam sequences

(1) Coronal T2 single-shot fast-spin echo (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis. Identify T2-bright and T2-dark lesions.
(2) Sagittal T2 single-shot fast-spin echo – Identify T2-bright and T2-dark lesions.
(3) Axial T2 single-shot fast-spin echo – Identify T2-bright and T2-dark lesions.
(4) Axial T1 in- and out-of-phase BH – Identify T1-bright lesions and microscopic fat.
(5) Axial volume-interpolated gradient echo BH – Characterize T1-bright signal in lesions. Identify blood products.

Approach to interpretation of pelvic MRI in pregnant patients

MRI is an excellent modality for characterizing and determining the side and site of origin of adnexal masses in pregnant patients when ultrasound is limited or indeterminate.

The American College of Radiology White Paper on MRI safety from 2007 states "present data have not conclusively documented any deleterious effects of MRI exposure on the developing fetus, no special consideration is recommended for the first, versus any other trimester."

If MRI is able to characterize an adnexal mass as definitively benign (e.g. teratoma, hemorrhagic cyst, fibroid) or most likely benign (e.g. simple unilocular ovarian cyst) and the size of the mass is <5 cm, surgery will typically be deferred until after delivery. Larger masses (>5 cm) and masses with malignant features may be removed during pregnancy, preferably during the early to mid second trimester (14–18 weeks) when there is a lower risk of pregnancy complications. Therefore, delaying MRI of adnexal masses to the second trimester does not routinely delay patient care or surgical planning.

When imaging the pregnant abdomen or pelvis, the number and time of each sequence should be kept as short as possible. Fast T1 and T2 imaging is the key to limiting motion artifact from both the moving fetus and the amniotic fluid. Pregnant patients also often have difficulty performing long or multiple breath holds.

DO NOT administer intravenous contrast for MRI examinations performed during pregnancy. Ever. Just don't do it.

Approach

(1) Multiplanar T2 single-shot fast-spin echo – Evaluate side, size, and site of origin of pelvic pathology. Identify T2-bright and T2-dark lesions.
(2) T1 in/out of phase – Identify T1-bright and microscopic fat components within a lesion.
(3) Volume-interpolated gradient echo – Characterize T1-bright signal in lesions as fat or hemorrhage/protein.

Multiplanar single-shot fast-spin echo: T2-weighted images

Just like the non-pregnant female pelvis, the key sequences for characterizing and localizing pelvic pathology are multiplanar T2-weighted imaging (Figure 11.1). SSFSE is used in place of FSE during pregnancy as the shorter imaging acquisition time limits fetal and amniotic fluid motion and can be performed without breath-holding if necessary.

In/out of phase: chemical-shift images; T1-weighted images, no fat saturation

Primarily used for detection of fat and hemorrhagic/proteinaceous processes in the pelvis. The out-of-phase images allow for characterization of pelvic masses with microscopic fat (mature or immature ovarian teratoma). This sequence is also essential for evaluation of bone marrow (metastases) and adenopathy.

Volume-interpolated gradient echo: T1-weighted images, fat saturation

This sequence should be directly compared to the T1 in-phase images and is used to characterize T1-bright signal intensity in the pelvis as macroscopic fat (signal loss from T1 without to T1 with fat saturation) versus hemorrhage/protein (no signal loss from T1 without to T1 with fat saturation) (Figure 11.2).

Pearls and pitfalls

(1) MRI of the pregnant pelvis should be performed with fast sequences in order to minimize motion artifact related to the fetus and maternal breathing.
(2) T1-weighted imaging without and with fat saturation is essential for characterizing and differentiating between lesions containing fat versus hemorrhagic/proteinacous contents.
(3) Don't give gadolinium to pregnant patients.

i Pregnant appendicitis protocol

Indications

This protocol is used to evaluate abdominal pain in pregnant patients particularly when appendicitis is suspected. Before proceeding with MRI in this patient population, we routinely perform an ultrasound first, looking for appendicitis or ovarian pathology. If the ultrasound is indeterminant, MRI is our next test.

Preparation

- **IV contrast agent**: None
- **Oral contrast agent**: None
- Have the patient void prior to the start of the study
- Cover from mid liver through symphysis pubis
- All T2 SSFSE sequences can be run without breath-hold to continuously cover the abdomen and pelvis as one series.

Exam sequences

(1) Coronal T2 SSFSE – Identify the appendix and possible surrounding edema.
(2) Sagittal T2 SSFSE – Identify the appendix and possible surrounding edema.
(3) Axial T2 SSFSE – Identify the appendix and possible surrounding edema.
(4) Axial True FISP – Differentiate the appendix from adnexal varicosities.
(5) Axial T1 in and out of phase – Identify the appendix. Look for blooming artifact in the appendiceal lumen on T1 in phase.

Approach to interpretation of MRI of pregnant patients with suspected appendicitis

In the United States, MRI is now the preferred modality for evaluating acute appendicitis during pregnancy. To diagnose appendicitis, you have to be able to see the appendix and while they can consistently find the appendix on ultrasound in Europe, Americans are simply too fat large and inexperienced.

With MRI, we consistently visualize the appendix. To diagnose appendicitis we look for an enlarged, obstructed appendix with periappendiceal T2-bright signal indicative of inflammation.

But, with this single exam we can also evaluate for gallstones, choledocholithiasis, and even renal stones.

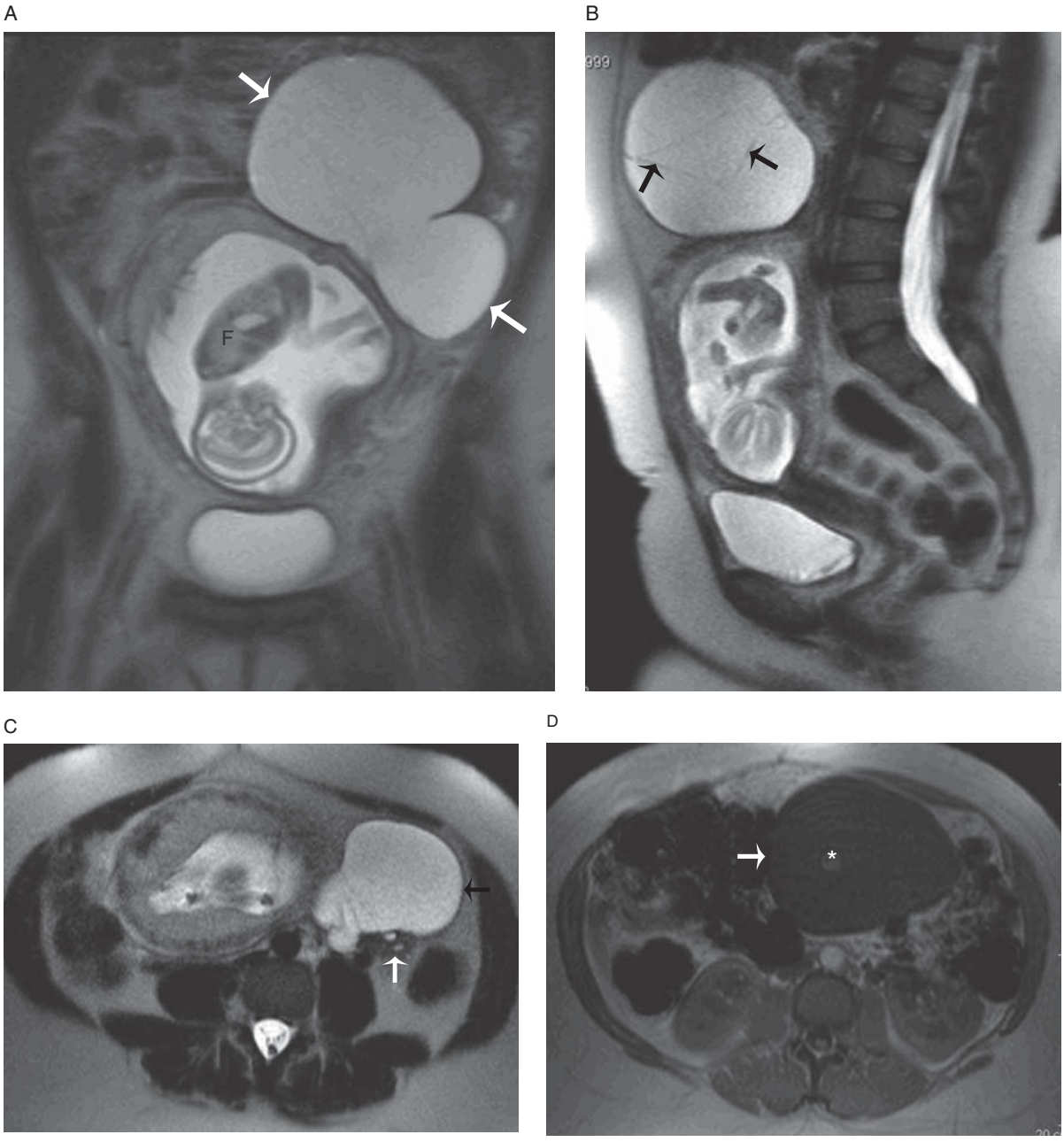

Figure 11.1. Mucinous cystadenoma in a 26-year-old pregnant female. Coronal T2-weighted image (A) demonstrates a 12 cm, high T2 signal intensity cystic lesion (arrows) in the left adnexa, extending superior to the gravid uterus. Note fetus (F). Sagittal T2-weighted image (B) further demonstrates that there are several thin internal septations in the cystic lesion (arrows). Axial T2-weighted image (C) confirms that this lesion (black arrow) arises from the left ovary; note normal ovarian tissue with a few subcentimeter follicles (white arrow) along the posterior aspect of the cyst. Axial T1-weighted image (D) without fat saturation reveals uniform intermediate T1 signal intensity in the cyst (arrow). Note aortic pulsation artifact in the cyst (*).

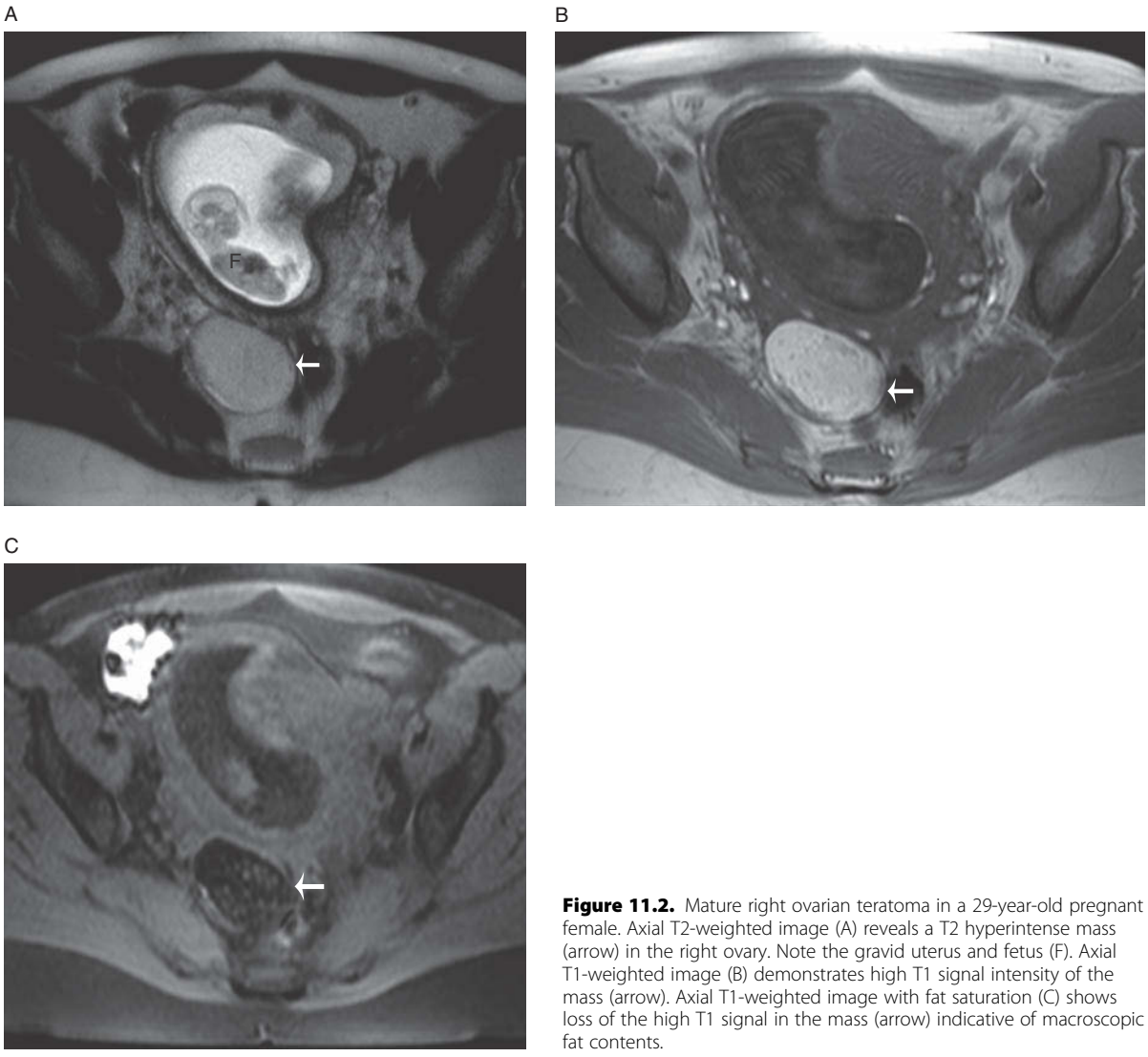

Figure 11.2. Mature right ovarian teratoma in a 29-year-old pregnant female. Axial T2-weighted image (A) reveals a T2 hyperintense mass (arrow) in the right ovary. Note the gravid uterus and fetus (F). Axial T1-weighted image (B) demonstrates high T1 signal intensity of the mass (arrow). Axial T1-weighted image with fat saturation (C) shows loss of the high T1 signal in the mass (arrow) indicative of macroscopic fat contents.

Initially, we used oral contrast to turn enteric contents dark on T2-weighted images. Theoretically, then, a patient with appendicitis would have dark bowel contents (due to oral contrast) except in the obstructed appendix (which would be bright). Nice in theory, but oral contrast adds an extra step and pregnant patients who feel sick rarely want to drink something that tastes terrible.

At its best, it worked inconsistently. So we stopped giving oral contrast. We have found it made no real difference in our ability to diagnose appendicitis.

Approach

Multiplanar SSFSE: T2-weighted images

These fast T2 sequences allow for imaging of the bowel without motion artifact. In pregnant patients, they may be performed without breath-holding.

The appendix can be found using the same principles as CT: first identify the terminal ileum and then look inferiorly for a blind-ending tubular structure arising from the cecal base (Figure 11.3). As pregnancy progresses there is gradual upward displacement of the cecum and appendix by the

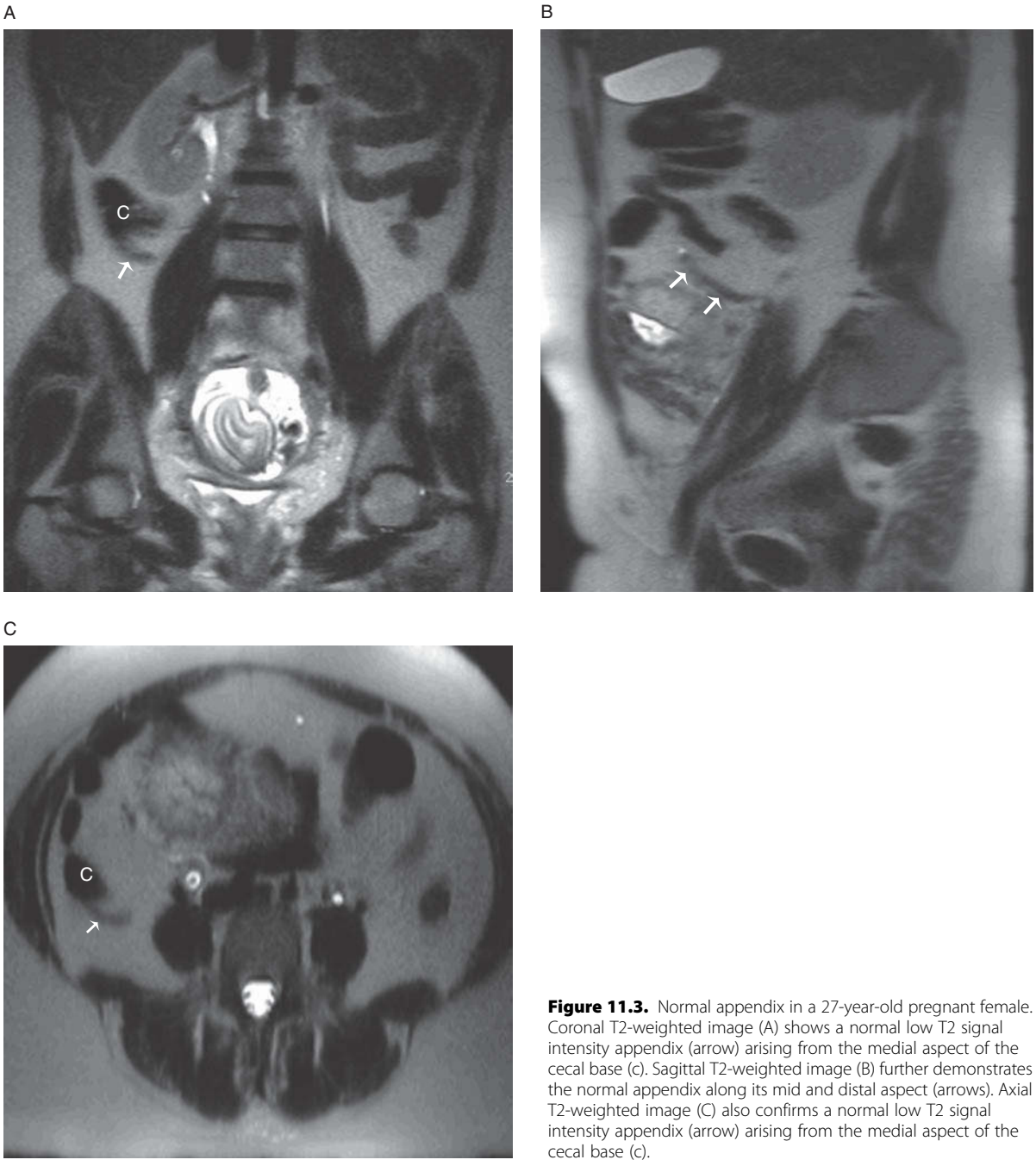

Figure 11.3. Normal appendix in a 27-year-old pregnant female. Coronal T2-weighted image (A) shows a normal low T2 signal intensity appendix (arrow) arising from the medial aspect of the cecal base (c). Sagittal T2-weighted image (B) further demonstrates the normal appendix along its mid and distal aspect (arrows). Axial T2-weighted image (C) also confirms a normal low T2 signal intensity appendix (arrow) arising from the medial aspect of the cecal base (c).

gravid uterus and the appendix can be located as high as the liver.

The inflamed appendiceal lumen and wall are high T2 signal intensity due to luminal fluid and wall edema. Appendicoliths are low signal intensity on T2-weighted images.

Most importantly, look for T2-bright periappendiceal inflammation. Search for free fluid and edema along the right side of the abdomen and pelvis. Identify the ovaries. *It is common to have a small amount of free fluid adjacent to the ovaries which should not be mistaken for fluid related to appendicitis* (Figure 11.4).

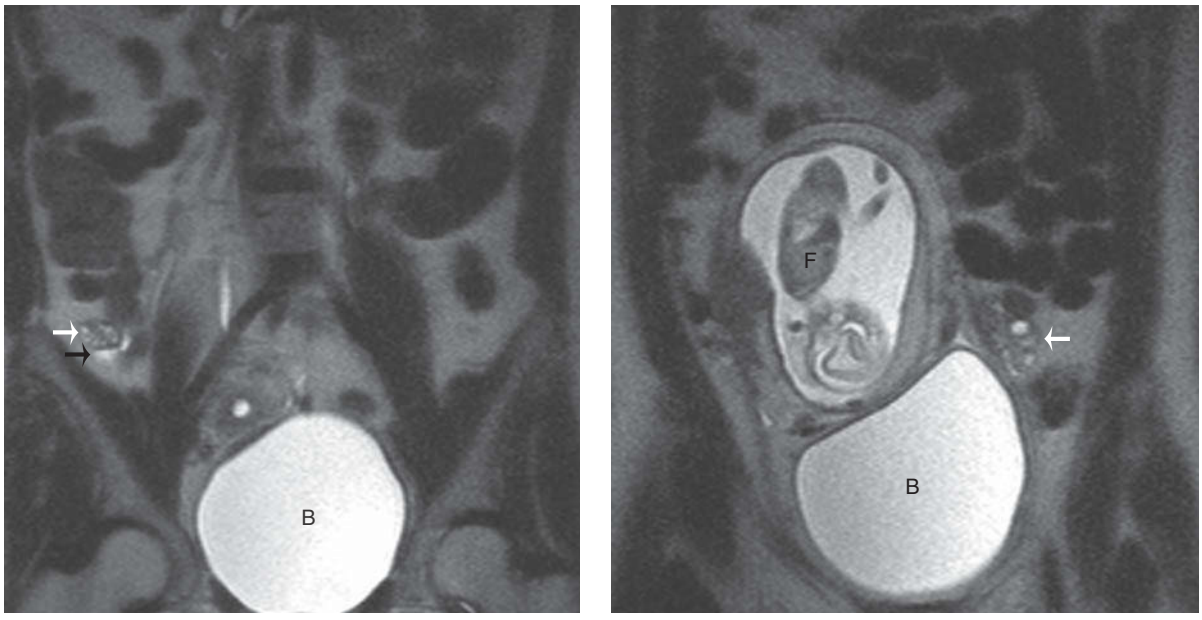

Figure 11.4. Normal ovaries in a 25-year-old pregnant female. Coronal T2-weighted image (A) demonstrates a normal right ovary (white arrow) with a small amount of physiologic free fluid (black arrow) adjacent to the ovary. More anterior coronal T2-weighted image (B) also demonstrates a normal left ovary (arrow) with subcentimeter follicles. Bladder (B). Also note gravid uterus with fetus (F).

Once you've evaluated the appendix, don't forget to look for ovarian pathology, hydronephrosis, cholelithiasis/cholecystitis, ovarian vein thrombosis and other bowel entities such as Crohn's disease and bowel obstruction.

True FISP: steady-state free-precession images

This sequence is helpful because flowing blood is bright which helps differentiate the normal appendix (low signal intensity) from right lower quadrant varicosities (high signal intensity) (Figure 11.5). This sequence is also used to screen for pelvic deep venous thrombosis and gonadal varicosities/ thrombosis which can also be a source of pain.

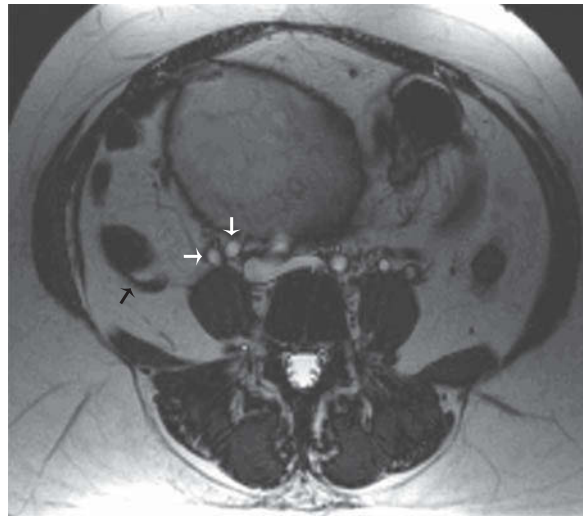

Figure 11.5. Normal appendix in a 32-year-old pregnant female. Axial True-FISP image demonstrates a normal low signal intensity appendix (black arrow) and normal high signal intensity (white arrows) right pelvic veins.

In/out phase: chemical-shift images; T1-weighted

Another opportunity to identify the appendix. Search for blooming artifact in the appendix. Blooming occurs on in-phase imaging (longer TE) compared to the out-of-phase imaging (shorter TE) due to susceptibility artifact from intraluminal air or oral contrast (just like the liver drops in signal on in-phase imaging compared to out-of-phase imaging if there is iron overload). Detection of in-phase blooming artifact in the appendix lumen helps confirm appendiceal patency (intraluminal air) (Figures 11.6).

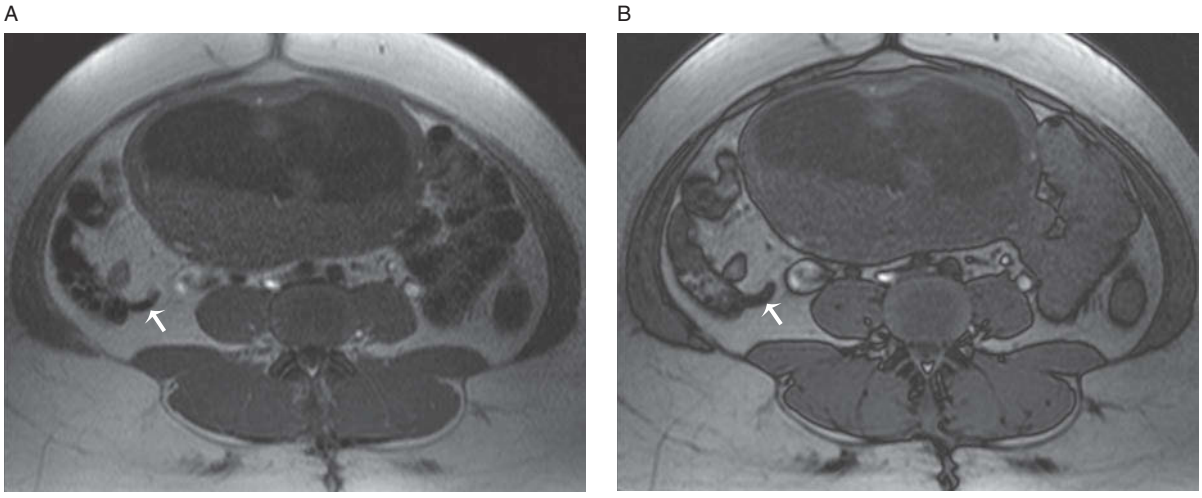

Figure 11.6. Normal appendix in a 26-year-old pregnant female. Axial T1-weighted image in phase (A) also demonstrates the normal-caliber appendix (arrow) as well as decreased luminal signal intensity on the in-phase image compared to the out-of-phase (B) image due to blooming artifact from air in the appendix, further supporting the diagnosis of a normal, non-obstructed appendix.

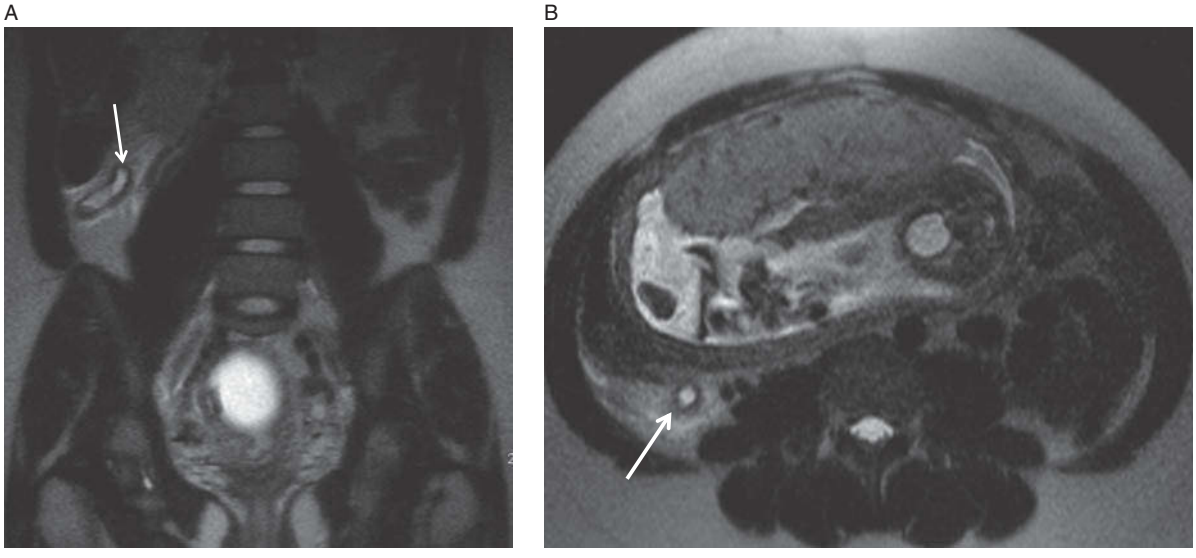

Figure 11.7. Acute appendicitis. Coronal and axial SSFSE (A, B) show a dilated, tubular, blind-ending structure (arrow) with high luminal and high surrounding T2 signal.

Appendiceal patency means the appendix is normal, appendicitis is due to appendiceal obstruction and secondary inflammation.

The appendix is also often highlighted on the out-of-phase images by etching artifact at the margin of the adjacent fat. The T1-weighted images also help identify hemorrhagic processes in the pelvis, such as subchorionic hemorrhage or a hemorrhagic ovarian cyst, which may account for the patient's pain.

MRI features of a normal appendix, indeterminate appendix for appendicitis, and acute appendicitis (Figures 11.6–11.8) are outlined in Table 11.1.

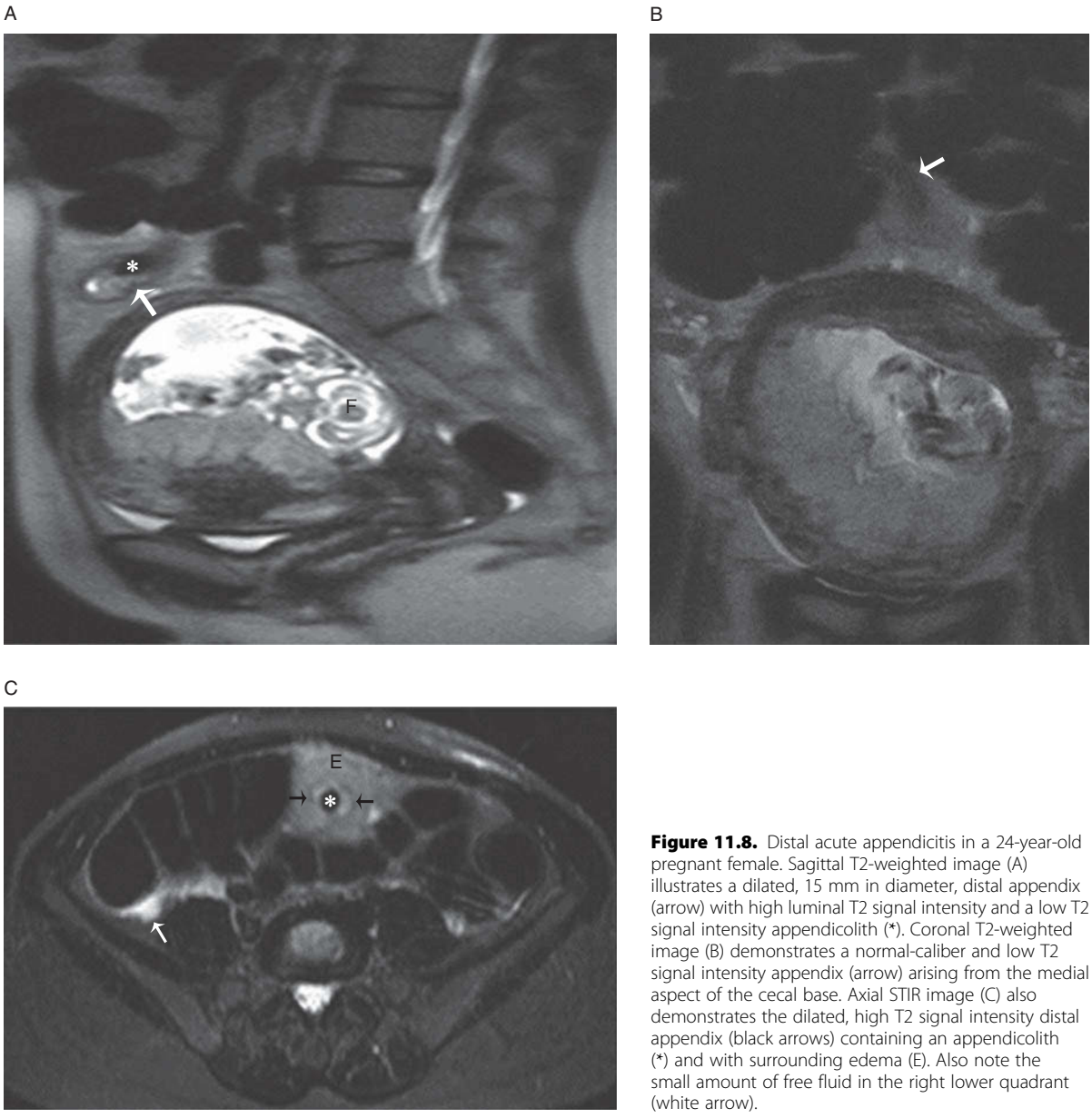

Figure 11.8. Distal acute appendicitis in a 24-year-old pregnant female. Sagittal T2-weighted image (A) illustrates a dilated, 15 mm in diameter, distal appendix (arrow) with high luminal T2 signal intensity and a low T2 signal intensity appendicolith (*). Coronal T2-weighted image (B) demonstrates a normal-caliber and low T2 signal intensity appendix (arrow) arising from the medial aspect of the cecal base. Axial STIR image (C) also demonstrates the dilated, high T2 signal intensity distal appendix (black arrows) containing an appendicolith (*) and with surrounding edema (E). Also note the small amount of free fluid in the right lower quadrant (white arrow).

Table 11.1 MRI appendix criteria

	Normal appendix	Indeterminate appendix	Acute appendicitis
Appendix diameter (mm)	≤ 6	6–7	>7
Appendix lumen	Low T2	High T2	High T2
Appendix wall (mm)	< 2	< 2	>2
Right lower quadrant inflammation	N	N	Y
T2 signal	↓	↔	↑
T1 blooming on in-phase imaging	↑	↔	↓

Pearls and pitfalls

(1) MRI is the preferred modality for evaluating acute appendicitis during pregnancy due to its high soft-tissue resolution, multiplanar imaging capabilities, and lack of ionizing radiation.
(2) Features of acute appendicitis on MRI are a dilated (>7 mm in diamter) appendix with high T2 luminal signal intensity, appendiceal wall thickening (>2 mm), and periappendiceal fat stranding and fluid.
(3) A small amount of physiologic free fluid adjacent to the ovaries is a normal finding and should not be mistaken for periappendiceal free fluid due to appendicitis.

ii Pregnant MRI urography

Pregnant MRI urography protocol

Indications

This protocol can be used to evaluate hematuria and flank pain during pregnancy.

Preparation

- **IV contrast agent**: None
- **Oral contrast agent**: None
- All T2 SSFSE sequences can be run without breath-hold to continuously cover the abdomen and pelvis as one series
- Field of view should be from adrenal glands through symphysis pubis.

Exam sequences

(1) Coronal SSFSE – Evaluate kidneys, ureters, retroperitoneum, T2-bright lesions.
(2) Axial SSFSE – Evaluate kidneys, ureters, retroperitoneum, T2-bright lesions.
(3) Sagittal SSFSE – Evaluate kidneys, ureters, retroperitoneum, T2-bright lesions.
(4) Axial T1 in- and out-of-phase BH – Extend through adrenal glands and kidneys.
(5) Coronal SSFSE thick slab – Angle parallel to renal collecting systems and ureters. Avoid including amniotic fluid.
(6) Coronal SSFSE thin – Evaluate for urinary tract calculi.
(7) Axial SSFSE thin – Evaluate for urinary tract calculi.

Approach to interpretation of MRI urography exams

Ultrasound is the initial imaging modality of choice for evaluating hematuria and flank pain in the pregnant patient. Ultrasound can readily demonstrate the presence or absence of hydronephrosis and ureteral jets. However, it is unable to visualize the ureters in their entirety and can miss small (<5 mm) renal calculi. MRI is not as sensitive as CT for detection of urinary tract calculi. However, it can fully depict the course and caliber of the ureters as well as readily demonstrate perinephric and peri-ureteral fluid.

Physiologic hydronephrosis, due to a combination of hormone-related relaxation of the ureters and extrinsic compression of the ureters by the growing uterus, is common during pregnancy. Differentiating between physiologic hydronephrosis of pregnancy and obstructive hydronephrosis, both of which can be a source of flank pain, can be difficult with ultrasound. Even if an obstructing stone is not visible on MRI, the course and caliber of the ureter as well as the presence or absence of perinephric fat stranding can help

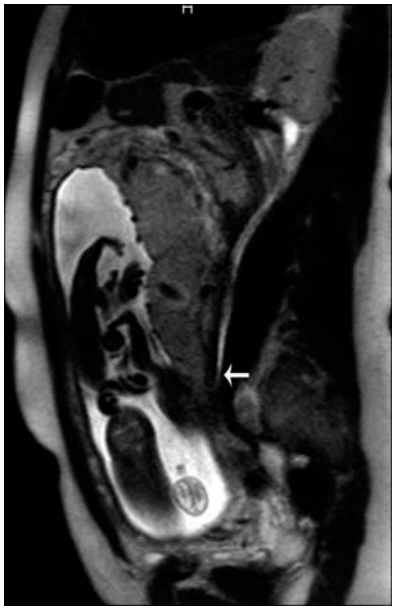

Figure 11.9. Physiologic hydronephrosis in a 22-year-old pregnant female. Sagittal T2-weighted SSFSE MRI image through the abdomen and pelvis demonstrates mild dilatation of the right ureter with gradual, smooth tapering of the distal ureter (arrow) between the posterior gravid uterus and iliopsoas muscle. These findings are consistent with physiologic hydronephrosis, a normal finding of pregnancy that can cause abdominal pain and be confused with obstructive hydronephrosis. (Image previously published in AJR Integrative Imaging: Woodfield CA, Lazarus E, Chen KC, Mayo-Smith WW. Abdominal pain in pregnancy: diagnoses and imaging unique to pregnancy – review. *AJR* 2010; **194**(6 Suppl):WS14–30.) Reprinted with permission.

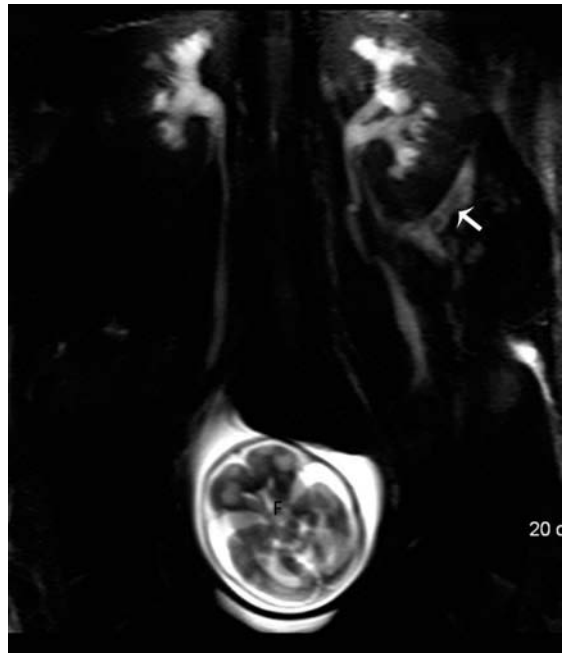

Figure 11.10. Physiologic hydronephrosis in a 33-year-old pregnant female with a triplet gestation. Coronal thick slab T2-weighted image demonstrates mild bilateral hydronephrosis and hydroureter to the level of the gravid uterus. Also note a small amount of left perinephric fluid (arrow) and one of the fetal heads (F). Perinephric fluid is more commonly associated with obstructive hydronephrosis, but can less commonly be seen with physiologic hydronephrosis.

differentiate between physiologic and obstructive hydronephrosis.

On MRI, physiologic hydronephrosis is characterized by gradual, smooth tapering of the distal ureter due to compression between the posterior mid uterus and iliopsoas muscle (Figure 11.9). Extrinsic compression is typically greater on the right than the left, which is thought to be due to the sigmoid colon protecting the left ureter from some degree of extrinsic compression by the gravid uterus. In contrast, ureteral dilation with an abrupt change in caliber above or below the posterior mid uterus is suggestive of obstructive hydronephrosis. Calyceal rupture with resulting perinephric and periureteral fluid is also much more common with obstructive rather than physiologic hydronephrosis (Figure 11.10). MRI readily depicts retroperitoneal and pelvic fluid.

Detection of stones is important not only because it reassures the clinician that the source of the patient's pain is not life-threatening, but also because urologists tend to be more aggressive about intervening in pregnant patients rather than relying on conservative management.

Approach

Multiplanar SSFSE: T2-weighted images

These fast T2-weighted images allow for assessment of hydronephrosis, hydroureter, perinephric stranding, and retroperitoneal fluid. Flow voids in the renal collecting systems and ureters are common, particularly in dilated systems. Multiplanar imaging helps determine whether or not a filling defect in the collecting system or ureter is a flow void (centrally located) or true filling defect due to a stone (dependent). If a stone, the filling defect should also be present in more than one plane.

The thick coronal slab gives a nice overview of the course and caliber of the renal collecting systems and ureters (Figure 11.11). Thinner section axial and coronal images similar to MRCP aid in the detection of urinary-tract calculi.

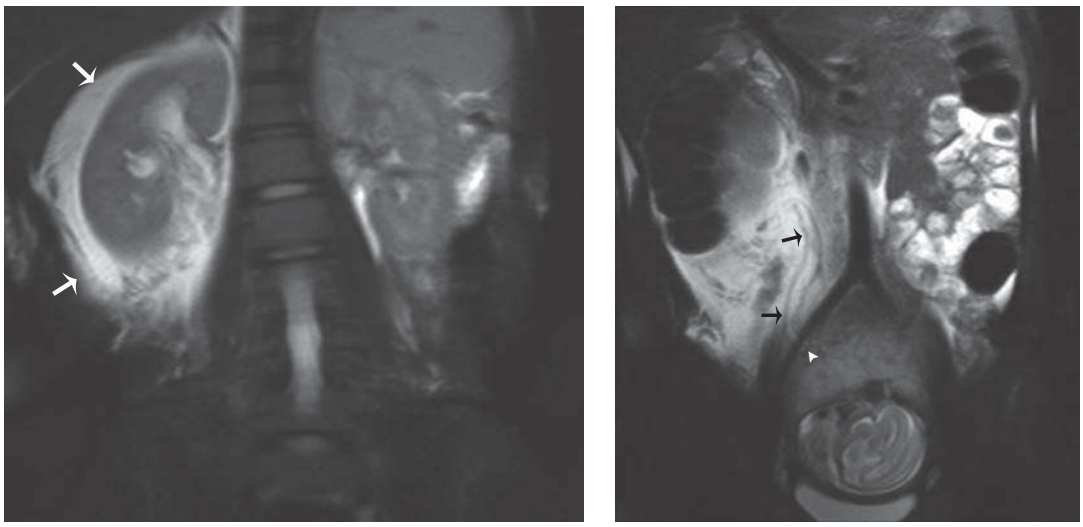

Figure 11.11. Obstructive hydronephrosis in a 31-year-old pregnant female with right flank pain. Coronal T2-weighted SSFSE MRI images (A, B) through the abdomen illustrate moderate right perinephric fluid (white arrows) and mild right hydroureteronephrosis (black arrows) with abrupt termination of the dilated right ureter at the level of the pelvic rim (arrowhead) suggestive of obstructive right hydroureteronephrosis. A ureteral calculus was not visible on the MRI examination. The patient subsequently passed a 3-mm calculus. (Image previously published in AJR Integrative Imaging: Woodfield CA, Lazarus E, Chen KC, Mayo-Smith WW. Abdominal pain in pregnancy: diagnoses and imaging unique to pregnancy – review. *AJR* 2010; **194**(6 Suppl):WS14–30). Reprinted with permission.

In/out of phase: T1-weighted images

Allows for evaluation of microscopic fat containing lesions in the adrenal glands (adenomas) and kidneys (usually either lipid-poor angiomyolipomas or clear all renal carcinomas) as well as hemorrhagic/proteinaceous lesions of the kidneys (Figure 11.12).

Pearls and pitfalls

(1) MRI can readily demonstrate hydronephrosis and hydroureter during pregnancy on T2-weighted images. However, urinary-tract calculi are not reliably depicted with MRI.
(2) Physiologic hydronephrosis is common during pregnancy and is characterized on MRI by gradual, smooth tapering of the distal ureter.
(3) Features of obstructive hydronephrosis on MRI are:
    (a) Abrupt change in the caliber of the ureter above or below the uterus
    (b) Renal enlargement
    (c) Perinephric fluid.

iii MRI of the placenta

Placenta protocol

Indications

This protocol is used for evaluation of placenta accreta.

Preparation

- **IV contrast agent**: None
- **Oral contrast agent**: None
- NPO for 4 hours prior to the exam (if possible)
- Cover entire uterus
- Patient should be imaged with a partially full bladder. Do not have the patient void prior to the start of the study
- All T2 SSFSE sequences can be run without breath-hold to continuously cover the uterus as one series.

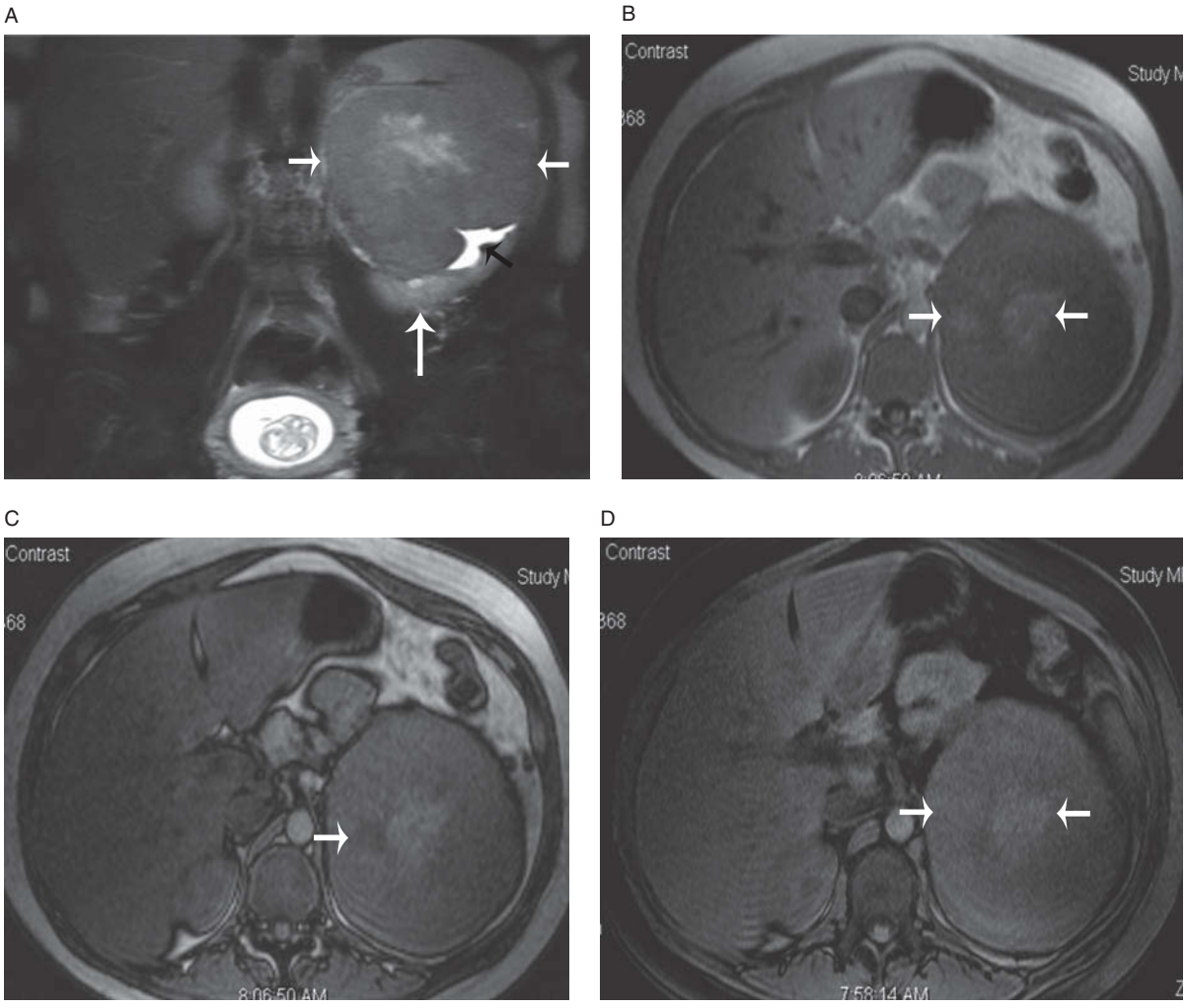

Figure 11.12. Clear cell renal carcinoma in a 36-year-old pregnant female with an indeterminate left renal mass on ultrasound. Coronal T2-weighted SSFSE image (A) demonstrates an 8-cm predominantly intermediate T2 signal intensity mass (short white arrows) arising from the upper pole of the left kidney. Note adjacent left renal parenchyma (long white arrow) and mild left lower pole hydronephrosis (black arrow) due to partial obstruction of the collecting system by the mass. Axial T1-weighted in-phase image (B) reveals two areas of high T1 signal intensity in the mass (arrows). Axial T1-weighted out-of-phase image (C) demonstrates corresponding loss of signal (arrow) in one of the areas of high signal intensity on the in-phase image, indicative of the presence of microscopic fat in the mass. Microscopic fat can be seen in the clear cell subtype of renal cell carcinomas. Also note in this patient that an axial T1-weighted 3D SPGR image with fat saturation (D) does not demonstrate definitive loss of signal in the high T1 signal areas on the T1 in-phase image, indicating that the mass does not contain macroscopic fat as would be expected with a renal angiomyolipoma.

Exam sequence

(1) Coronal T2 single-shot fast-spin echo – Evaluate placental location, extension into or through myometrium.
(2) Sagittal T2 single-shot fast-spin echo – Evaluate placental location, extension into or through myometrium.
(3) Axial T2 single-shot fast-spin echo – Evaluate placental location, extension into or through myometrium.
(4) Sagittal True FISP – Evaluate placental extension into or through myometrium.
(5) Axial volume-interpolated gradient echo BH – Identify pre-placental, placental, and retroplacental blood.

Approach to interpretation of MRI exams of the placenta

When ultrasound is limited or indeterminate, MRI imaging can be used to evaluate placental location and for placenta accreta. When asked to evaluate for placenta accreta, most radiologists run out of the room. Why? Accreta is the ultimate radiologic trifecta of torture:

(1) It's rare.
(2) The studies are hard to interpret.
(3) The implications are significant.

Don't run. It's not so bad . . . First, some definitions. The word accreta is an umbrella term for the three types of abnormal placental implantation (accreta, increta, percreta), distinguished by the depth of myometrial invasion.

Placenta accreta =	Placenta is adherent to the myometrium.
Placenta increta =	Placenta invades into the myometrium but not through the serosa.
Placenta accreta =	Placenta invades all the way through the myometrium into the serosa and may attach/invade other organs.

The major risk factors for placenta accreta are a history of prior cesarean section, prior myomectomy and an anterior complete placenta previa. Placenta accreta and increta can be especially difficult to visualize and diagnose on MRI, so special attention should be given for any secondary signs of abnormal placental implantation on MRI. Anterior placenta accreta is usually well visualized by ultrasound; however, diagnosing posterior placenta accreta may require MRI. Posterior placenta accreta most commonly occurs at myomectomy sites.

Placenta accreta is best evaluated in the second trimester when the myometrium is at its thickest. As pregnancy progresses, and the myometrium thins, evaluation of placental extension is more challenging.

The MRI features of placenta accreta include:
(1) placental extension into or through the myometrium
(2) placental extension up to/into the bladder wall
(3) low T2 signal intensity bands in the placenta
(4) placental bulging.

Obstetricians suspect placenta accreta based on a prior history of cesarean section and an anterior complete placenta previa without definitive contiguous overlying myometrium on ultrasound. The diagnosis is critical, because patients with placenta accreta will require a cesarean section and likely hysterectomy to prevent life-threatening hemorrhage at the time of delivery.

Approach

Multiplanar SSFSE: T2-weighted images

Multiplanar T2 imaging is required to completely evaluate the placental/myometrial interface. Both the placenta and the myometrium are intermediate signal intensity on T2-weighted images while the bladder wall is low in T2 signal intensity (Figure 11.13).

The sagittal images are best for evaluating for placental extension up to or into the bladder wall. A partially filled bladder aids in evaluating the relationship between the bladder wall, myometrium, and placenta (Figure 11.14). Placental extension up to, across, or into the cervix is also best visualized in the sagittal plane (Figure 11.15).

Volume-interpolated gradient echo: T1-weighted images

This sequence is used to highlight any blood products associated with the placenta such as retro- or preplacental hematomas, or bleeding associated with placenta previa (Figure 11.16).

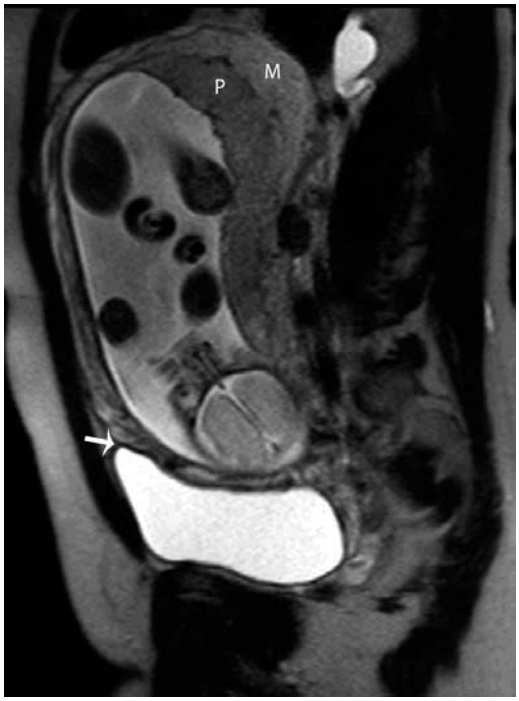

Figure 11.13. Normal placenta in a 25-year-old pregnant female at 24 weeks gestation. Sagittal T2-weighted SSFSE image illustrates the normal appearance of the placenta and myometrium in the second trimester. Both the placenta (P) and the myometrium (M) are intermediate in T2 intensity, while bladder wall has low T2 signal intensity (arrow).

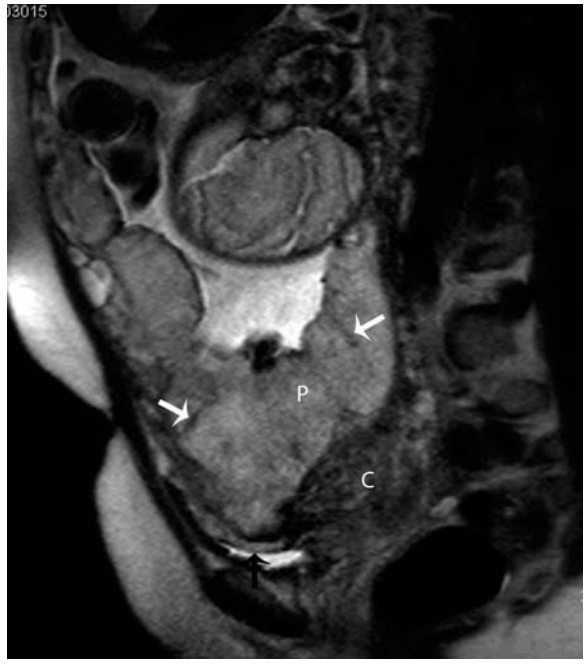

Figure 11.14. Placenta percreta in a 38-year-old pregnant female. Sagittal T2-weighted SSFSE image reveals a complete anterior placenta previa (P), extending across the cervix (C), and into and replacing the normal low T2 signal intensity bladder wall (black arrow) indicative of placental invasion of the bladder. Also note the bulging appearance of the placenta and low T2 signal intensity bands in the placenta (white arrows), additional supportive findings of placenta percreta.

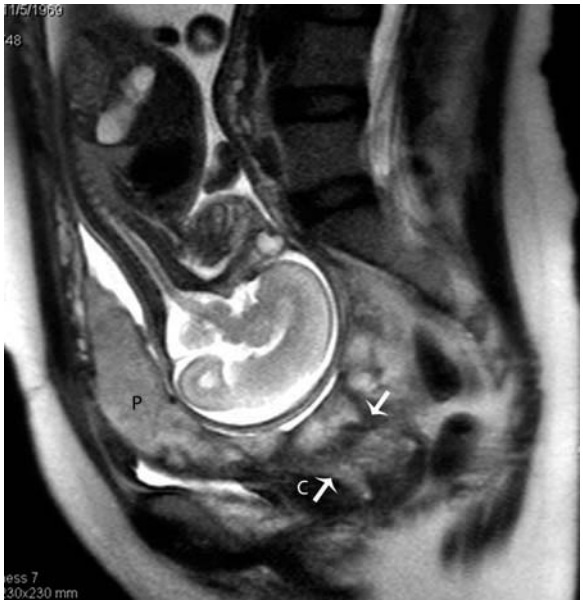

Figure 11.15. Placental invasion of the cervix in a 35-year-old pregnant female. Sagittal T2-weighted image demonstrates a complete placenta previa (P) with placental invasion (arrows) of the cervix (C).

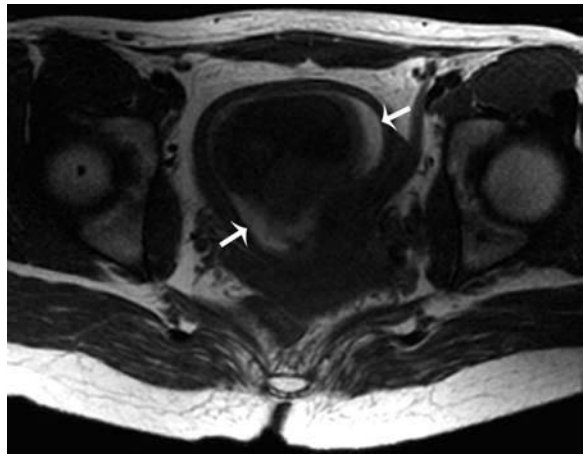

Figure 11.16. Subchorionic hematoma in a 27-year-old pregnant female. Axial T1-weighted 2D-SPGR image reveals high T1 signal intensity material (arrows) in the subchorionic location.

Pearls and pitfalls

(1) Ultrasound is the imaging modality of choice for evaluating placenta percreta. MRI is reserved for limited or indeterminate ultrasounds.
(2) The MRI features of placenta accreta are placental extension through the myometrium, placental extension up to/into the bladder wall, placental bulging, and low T2 signal intensity bands in the placenta.
(3) MRI with a partially distended bladder can help to better distinguish the boundaries between the bladder wall, myometrium, and placenta.
(4) Placenta accreta occurs most frequently at sites of prior myomectomy or cesarean sections so accurate patient history can be helpful with this diagnosis.

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Chapter 12

MRI of male pelvis

i Male pelvis protocol: scrotum

Indications

This protocol is used for generalized screening of pelvic pathology, i.e. pain, metastatic disease, masses. It can also be adapted for evaluation of scrotal and penile pathology (see below).

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- At least 24-gauge IV; connect to power injector
- Have patient void just prior to start of the exam

Exam sequences

(1) Coronal T2 single-shot fast-spin echo BH – Identify T2-bright and T2-dark lesions.
(2) Sagittal T2 fast-spin echo – Identify T2-bright and T2-dark lesions.
(3) Axial T2 fast-spin echo – Identify T2-bright and T2-dark lesions.
(4) Coronal T2 fast-spin echo FS – Identify T2-bright and T2-dark lesions. Evaluate for pelvic fluid and T2-bright osseous lesions.
(5) Axial volume-interpolated gradient echo BH – If using surface coil for scrotal imaging, perform this sequence with larger field of view pelvic phased-array coil to image the pelvis from the iliac crests through the symphysis pubis.
(6) Axial volume-interpolated gradient echo BH pre-contrast.
(7) Axial volume-interpolated gradient echo BH post IV administration of contrast at 70 seconds.
(8) Sagittal volume-interpolated gradient echo BH post IV administration of contrast at ~2 min.
(9) Coronal volume-interpolated gradient echo BH post IV administration of contrast at ~3 min.

NOTE

To adapt the protocol for imaging the scrotum: elevate scrotum with towels and cover with towels. Place surface coil directly over scrotum. For penile imaging, tape dorsiflexed penis to lower abdomen, cover with towel, use surface coil.

Approach to interpretation of male pelvic MRI

Approach and sequences

The fact is, we don't do much MRI of the male pelvis (aside from the prostate which gets its own section). But, once in a while a clinician will ask for it, so, like a Boy Scout, we are prepared.

Normal scrotum

The testicles are homogeneous intermediate signal intensity on T1 and intermediate to high signal intensity on T2-weighted images due to high fluid content. The epididymis is similar in T1 signal intensity, but lower in T2 signal intensity than the testicle (Figure 12.1). The mediastinum testis and tunica albuginea are both low signal intensity on T1- and T2-weighted images due to fibrous tissue.

The penile corporal bodies are intermediate T1 and high T2 signal intensity due to high fluid content and surrounded by low T1 and T2 signal intensity tunica albuginea (Figure 12.2). Pathology in the testicles and penile corporal bodies is usually lower in T2 signal intensity than the normal testicle and corporal body (Figures 12.3 and 12.4).

Pearls and pitfalls

(1) Ultrasound is the first imaging modality of choice for evaluating the scrotum, but MRI can be used for problem-solving, e.g. further differentiating between an intratesticular versus extratesticular

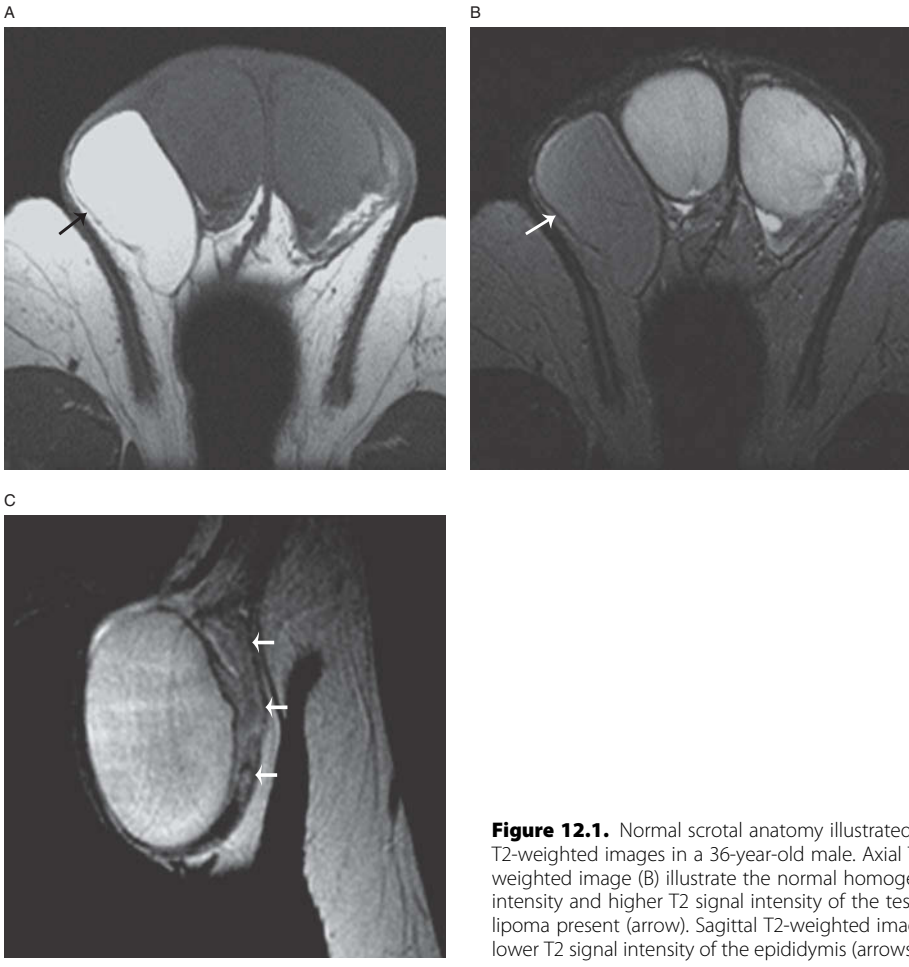

Figure 12.1. Normal scrotal anatomy illustrated on T1- and T2-weighted images in a 36-year-old male. Axial T1-weight image (A) and axial T2-weighted image (B) illustrate the normal homogeneous intermediate T1 signal intensity and higher T2 signal intensity of the testicles. There is also a right scrotal lipoma present (arrow). Sagittal T2-weighted image (C) also illustrates the normal lower T2 signal intensity of the epididymis (arrows) compared to the normal testicle.

mass, differentiating between benign tumor pathology such as a hematoma versus a solid enhancing mass.

(2) In the setting of trauma, MRI can also be used to evaluate for possible penile fracture or thrombosis.

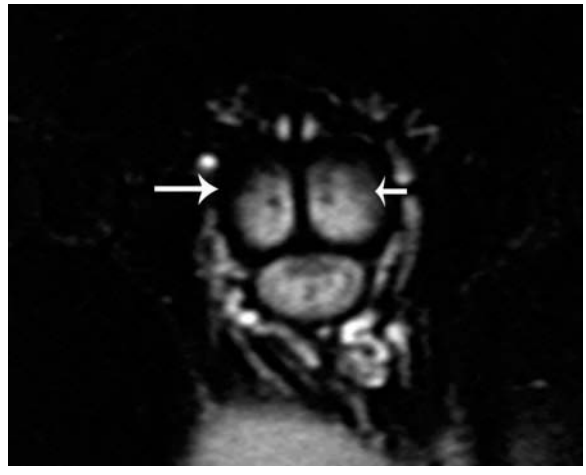

Figure 12.2. Normal penile anatomy in a 29-year-old male. Coronal T2-weighted image demonstrates the normal high T2 signal intensity of the penile corporal bodies (short arrow) and the normal lower T2 signal intensity of the surrounding tunica albuginea (long arrow).

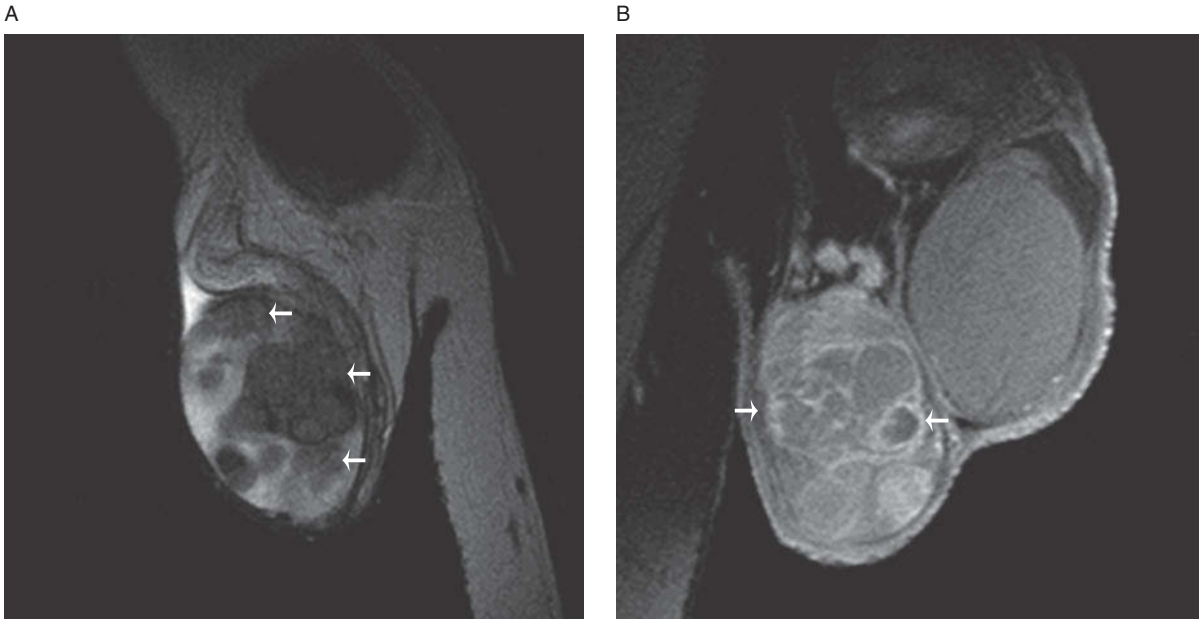

Figure 12.3. Multifocal right testicular seminoma in a 32-year-old male. Sagittal T2-weighted image (A) reveals multifocal low T2 signal intensity masses (arrows) in the right testicle. Coronal contrast-enhanced T1-weighted image with fat saturation (B) demonstrates heterogeneous enhancement of the dominant right testicular mass with thick internal septations (arrows).

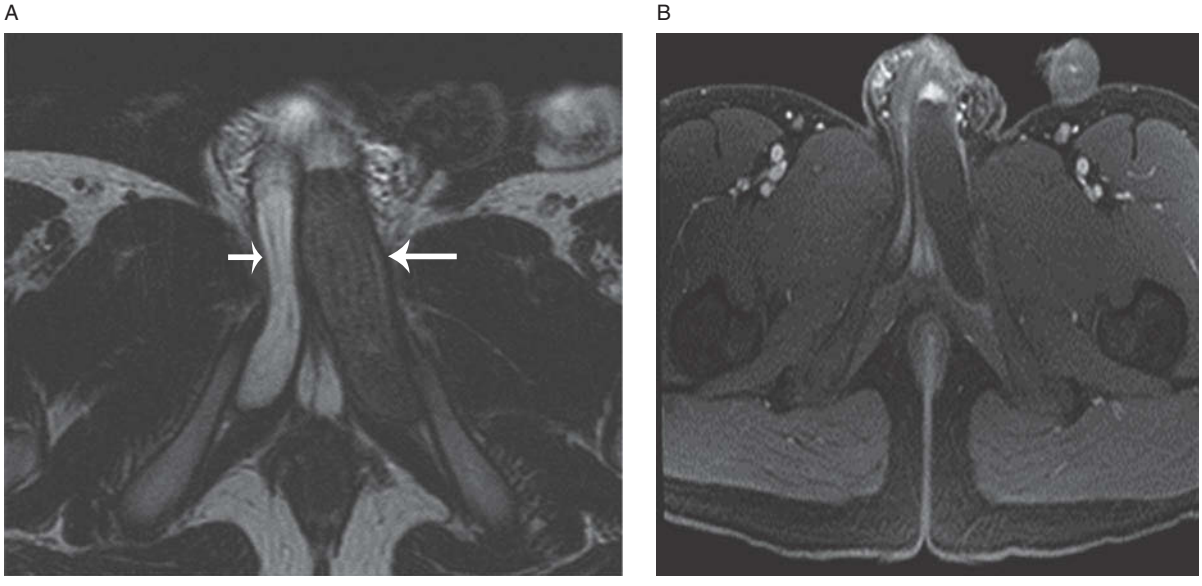

Figure 12.4. Cavernosal thrombosis in a 25-year-old male. Axial T2-weighted image (A) shows diffuse decreased signal intensity and enlargement of the left corpora cavernosa (long arrow) compared to the normal right corpora cavernosa (short arrow). Axial contrast-enhanced T1-weighted image with fat saturation (B) shows corresponding lack of enhancement of the left corpora cavernosa. There is normal enhancement of the right corpora cavernosa.

ii MRI prostate

Prostate protocol

Indications

Indications for MRI of the prostate include:

(1) staging intermediate to high-risk prostate carcinoma,
(2) elevated prostate-specific antigen (PSA) with negative digital rectal exam and negative biopsy
(3) rising PSA post treatment for prostate carcinoma
(4) recurrent urinary tract infections/prostatitis
(5) hematospermia
(6) infertility.

- **IV contrast agent**: None
- **Oral contrast agent**: None
- Have patient void just prior to start of the exam
- Exam performed with an endorectal as well as a pelvic phased-array coil.

Exam sequences

(1) Coronal T2 single-shot fast-spin echo BH, large field of view – Evaluate coil placement, localize the prostate and pathology for proper planning of T2 FSE sequences.
(2) Axial T2 fast-spin echo, small field of view – Evaluate prostate anatomy and identify T2-bright and T2-dark pathology.
(3) Sagittal T2 fast-spin echo, small field of view – Evaluate prostate anatomy and identify T2-bright and T2-dark pathology.
(4) Coronal T2 fast-spin echo, small field of view – Evaluate prostate anatomy and identify T2-bright and T2-dark pathology.
(5) Axial volume-interpolated gradient echo, small field of view – T1-bright material is blood not tumor.
(6) Axial volume-interpolated gradient echo, large field of view (from the aortic bifurcation through the symphysis pubis) – Identify lymphadenopathy and osseous lesions.
(7) Axial DWI b0, b1000, small and large field of view – Identify prostate carcinoma, lymphadenopathy, osseous metastasis.

Approach to interpretation of prostate MRI

The use of MRI for detection and staging of prostate cancer varies across institutions and is largely dependent on the practice of the referring urologists. However, there are many indications for prostate imaging, including:

(1) Identifying extracapsular extension in patients with known prostate cancer. Extracapsular extension typically indicates advanced-stage disease which may be treated with radiation therapy rather than surgical resection of the prostate.
(2) Searching for local tumor recurrence in patients with an elevated or rising PSA following treatment for prostate cancer.
(3) Evaluating patients with an elevated PSA but a negative digital rectal examination and negative prostate biopsies in search of a tumor that is presumed to exist. It can be used to guide subsequent biopsies.
(4) Non-cancerous conditions such as recurrent urinary tract infections/prostatitis, hematospermia, and infertility.

MRI of the prostate should be performed at least 3 weeks following any prostate biopsy. This allows some resorption of blood products within the gland which could obscure pathology. It also helps gives the patient time to recover before you place an endorectal coil.

The patient should not use an enema for 24 hours prior to prostate MRI in order to avoid potential motion artifact due to rectal spasm. With use of a 1.5-T magnet, the study should be performed with an endorectal coil in order to ensure high-quality images. An endorectal coil may not be required with a 3.0-T magnet, but this is still under investigation.

Contraindications to the endorectal coil include latex allergy, surgery or radiation to the rectum within the previous 8 weeks, brachytherapy seed placement within the last 12 weeks, anorectal fistula or stricture, severe hemorrhoids, and inflammatory bowel disease involving the rectum.

The key elements of any prostate MRI are high-resolution, multiplanar T2-weighted sequences in order to localize and stage prostate cancer. The prostate is the rare organ where pathology is T2-dark. When staging known prostate cancer the examination should be interpreted with knowledge of the patient's PSA and prostate biopsy results.

Sequences and approach

Sagittal SSFSE: T2-weighted images

This sequence is used to evaluate the position of the endorectal coil. The endorectal coil should be

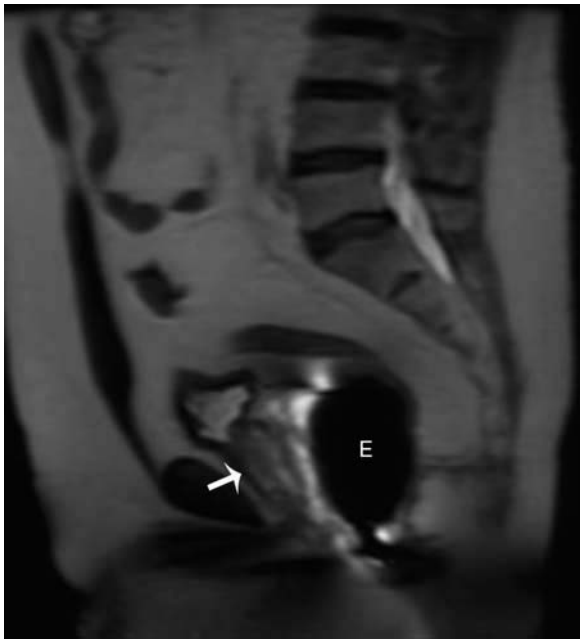

Figure 12.5. Endorectal coil placement in a 57-year-old male. Sagittal T2-weighted SSFSE image demonstrates correct placement of the endorectal coil (E) directly posterior to the prostate gland (arrow).

centered posterior to the prostate gland (Figure 12.5). If the coil is not centered on the prostate, it should be adjusted prior to proceeding to the smaller field of view, high-resolution T2- and T1-weighted images. The larger field of view of this sequence also allows for survey of the lumbar spine and retroperitoneum for pathology/metastatic disease.

Multiplanar FSE: T2-weighted images

These small field of view, high-resolution T2-weighted images provide detailed evaluation of prostate anatomy and cancer detection and staging. The normal peripheral zone of the prostate gland is high in T2 signal intensity due to increased fluid content and the central gland is usually variable in T2 signal intensity due to benign prostatic hypertrophy (BPH) (Figure 12.6). In contrast, prostate carcinoma is low in T2 signal intensity, making it easier to identify in the peripheral zone (Figure 12.7).

Once tumor is localized within the prostate gland, its size and location should be reported, as well as the MRI stage of the carcinoma. In order to locally stage prostate carcinoma, the seminal vesicles, periprostatic tissue, including the paired neurovascular bundles along the posterolateral aspect of the prostate gland, and adjacent organs (bladder, rectum) should

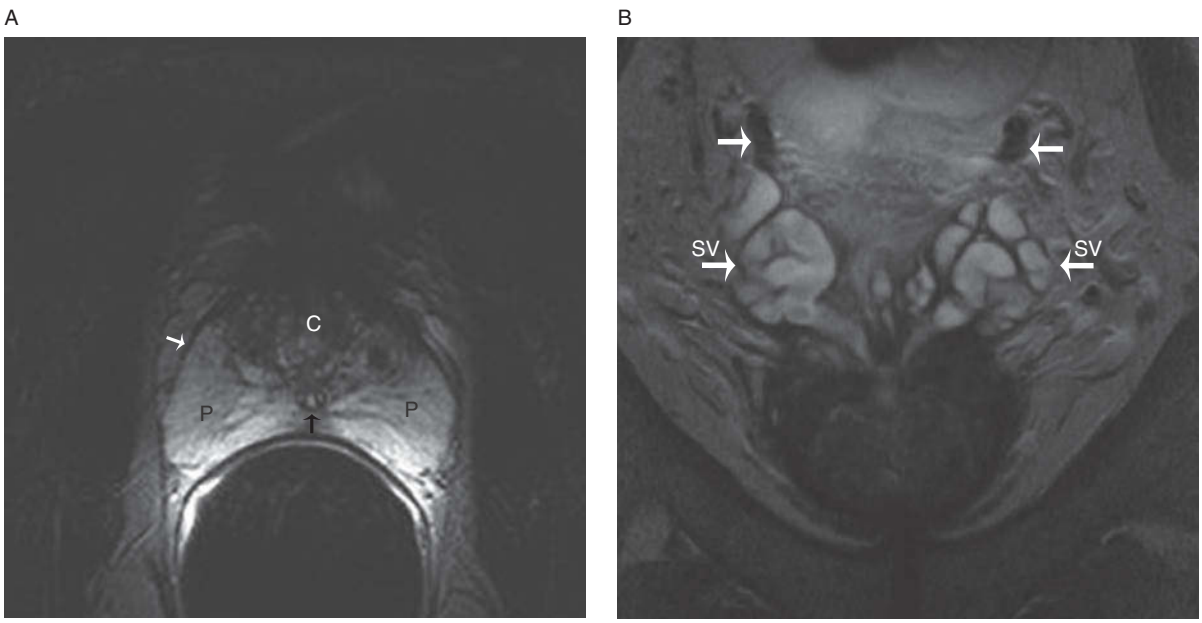

Figure 12.6. Normal prostate anatomy in a 52-year-old male. Axial T2-weighted FSE image (A) demonstrates the normal high signal intensity of the peripheral zone (P), variable lower signal intensity central gland (C), low signal intensity prostatic capsule (short white arrow), and high signal intensity urethra (black arrow). Coronal T2-weighted FSE image (B) further illustrates the normal high signal intensity of the seminal vesicles (white arrows) (SV) and the normal, low signal intensity, thick walled vas deferens (white arrows).

Table 12.1 TNM system of staging prostate cancer

T1 Occult tumor based on digital rectal examination (DRE) and imaging
T2 Palpable tumor on DRE
T2a Tumor present on ≤1/2 of 1 lobe
T2b Tumor present on ≥1/2 of 1 lobe
T2c Tumor present on both sides of the gland
T3 Tumor not confined to the prostate
T3a Tumor extension beyond the prostate without seminal vesicle involvement
T3b Tumor extension to seminal vesicles
T4 Tumor extension to bladder, rectum, or pelvic sidewall

be evaluated. The tumor–node–metastases (TNM) staging of prostate cancer is outlined in Table 12.1.

Our goal is to differentiate prostate-confined disease from extracapsular extension, T3 or greater. Most of our patients already know they have cancer.

MRI findings of extracapsular extension include asymmetry of the neurovascular bundles, blunting of the rectoprostatic angle, angulation and irregularity of the prostate capsule contour, and direct tumor extension beyond the prostate capsule into the periprostatic fat (Figure 12.8). MRI findings of seminal vesicle invasion include non-visualization of the

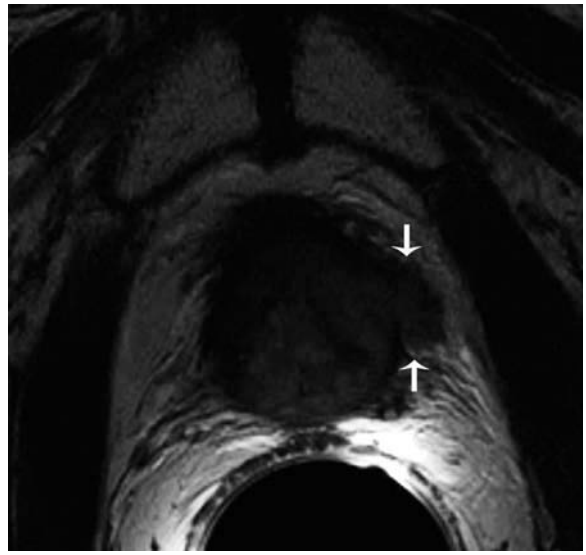

Figure 12.8. Extracapsular extension of prostate carcinoma in a 66-year-old male. Axial T2-weighted image at the level of the midgland demonstrates diffuse low signal intensity tumor with extension into the left periprostatic fat (arrows).

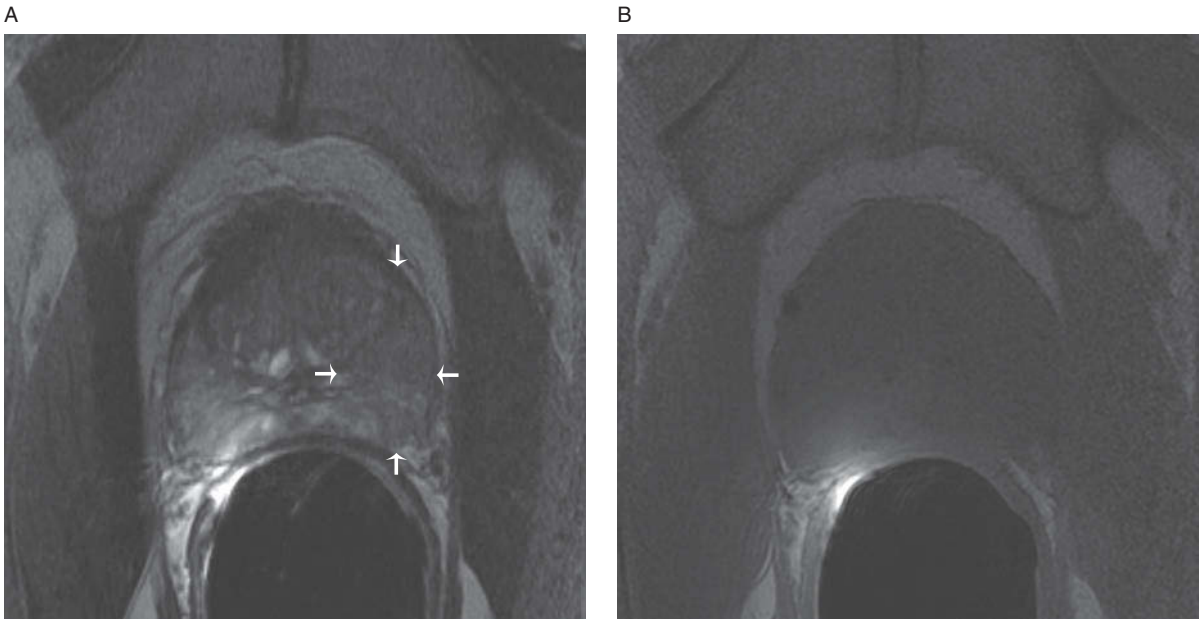

Figure 12.7. Prostate carcinoma in a 68-year-old male. Axial T2-weighted image (A) at the level of the midgland shows diffuse decreased signal intensity in the left peripheral zone (arrows). Corresponding axial T1-weighted image (B) reveals uniform hypointensity of the prostate gland, indicating that the low T2 signal intensity in the left peripheral zone is due to prostate carcinoma rather than blood products from recent biopsy.

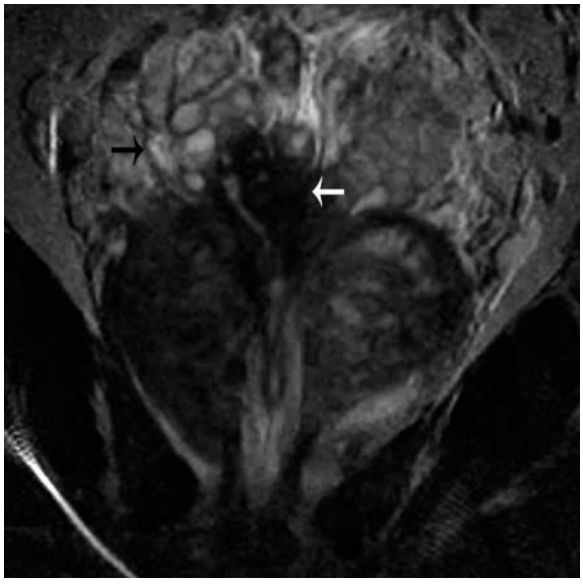

Figure 12.9. Seminal vesicle invasion of prostate carcinoma in a 72-year-old male. Coronal T2-weighted image reveals diffuse low signal intensity tumor throughout the right side of the gland with extension into the right seminal vesicle (white arrow). A more superior uninvolved portion of the right seminal vesicle is also noted on the right (black arrow).

ejaculatory ducts, thickening of the seminal vesicle wall, and low T1 and low T2 signal intensity of the normal high T2 signal intensity seminal vesicles (Figure 12.9). In particular, low T1 and low T2 signal intensity of the seminal vesicles with adjacent tumor at the prostatic base is highly predictive of seminal vesicle invasion.

Local recurrence of prostate carcinoma status post prostatectomy or radiation therapy will also manifest as a low T2 signal intensity nodule (Figure 12.10). However, such recurrence can be difficult to detect in the radiated prostate gland as radiation also results in low T2 signal intensity change of the prostate gland due to fibrosis.

Beyond prostate cancer, the T2-weighted images can depict a wide range of prostatic and periprostatic pathology and anomalies such as prostatitis, seminal vesicle cysts, and müllerian duplications cysts (Figure 12.11).

Axial volume-interpolated gradient echo, small field of view: T1-weighted images

These small field of view T1-weighted images should exactly match the field of view and slice level of the small field of view T2-weighted images. Prostate cancer should be T1 and T2 dark. T1-bright material can look mass-like, but is blood, not cancer (Figure 12.12).

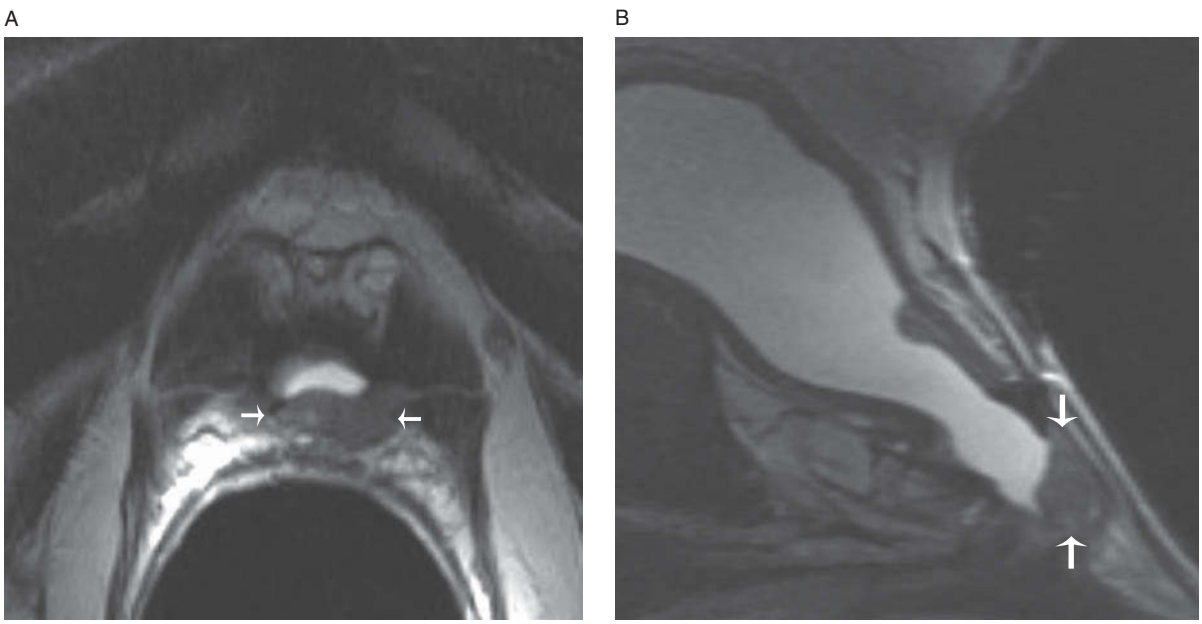

Figure 12.10. Local recurrence of prostate carcinoma in a 69-year-old male. Axial (A) and sagittal (B) T2-weighted images illustrate an intermediate signal intensity mass due to locally recurrent prostate carcinoma (arrows) in the prostatectomy bed, posterior to the bladder base.

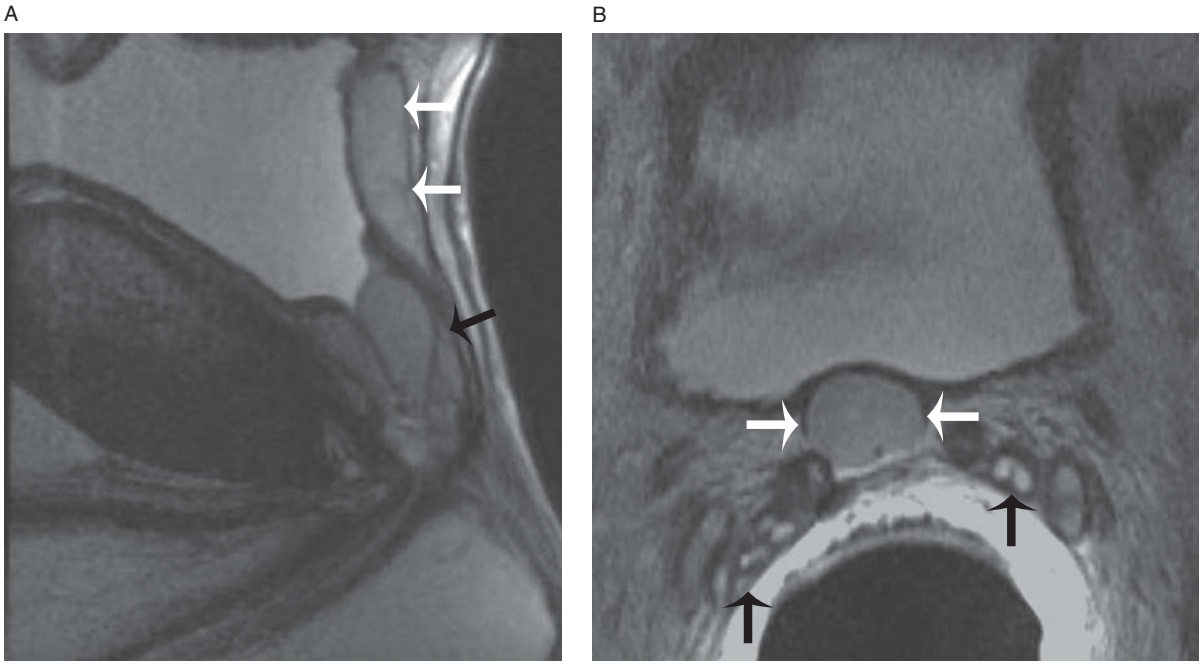

Figure 12.11. Müllerian duct cyst in a 62-year-old male status post prostatectomy. Sagittal T2-weighted image (A) shows an elongated high T2 signal intensity structure extending posterior to the bladder (white arrows). Also note intermediate signal intensity material at the bladder base (black arrow) due to prior collagen injections for urinary incontinence status post prostatectomy. Axial T2-weighted image (B) further demonstrates that the high T2 signal intensity structure posterior to the bladder is midline. The midline location and extension posterior to the bladder of this cystic structure is characteristic of a müllerian duct cyst. Also note residual seminal vesicles on the axial image (black arrows).

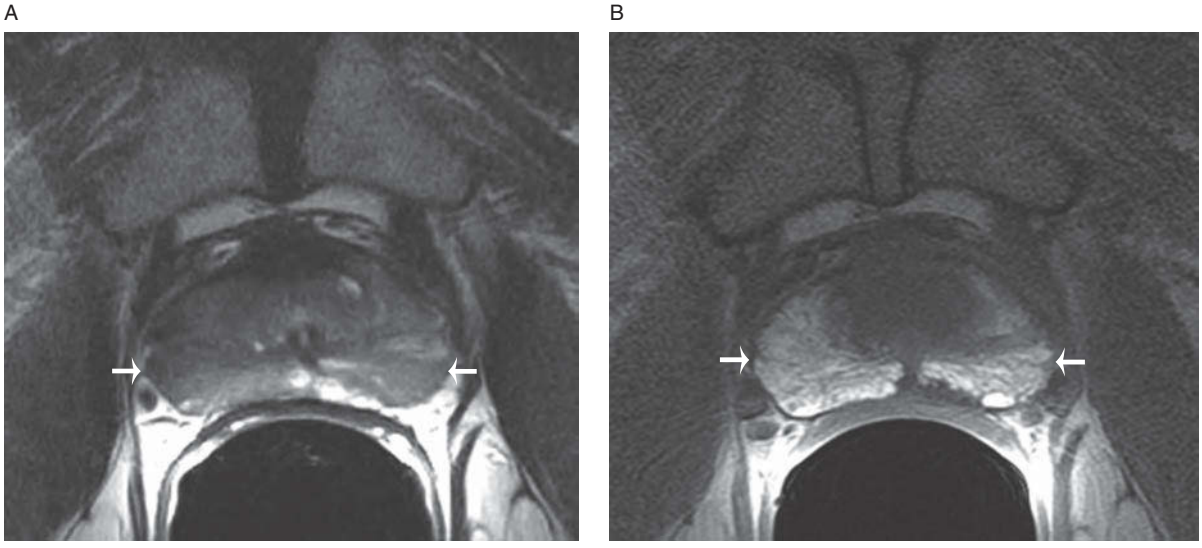

Figure 12.12. Post-biopsy hemorrhage in the peripheral zone of the prostate in a 55-year-old male. Axial T2-weighted image (A) reveals diffuse low signal intensity throughout the peripheral zone, right greater than left (arrows). Corresponding axial T1-weighted image (B) shows that the areas of low signal intensity on the T2-weighted image correspond to areas of high signal intensity blood products (arrows). No discrete tumor is identified on these MRI images.

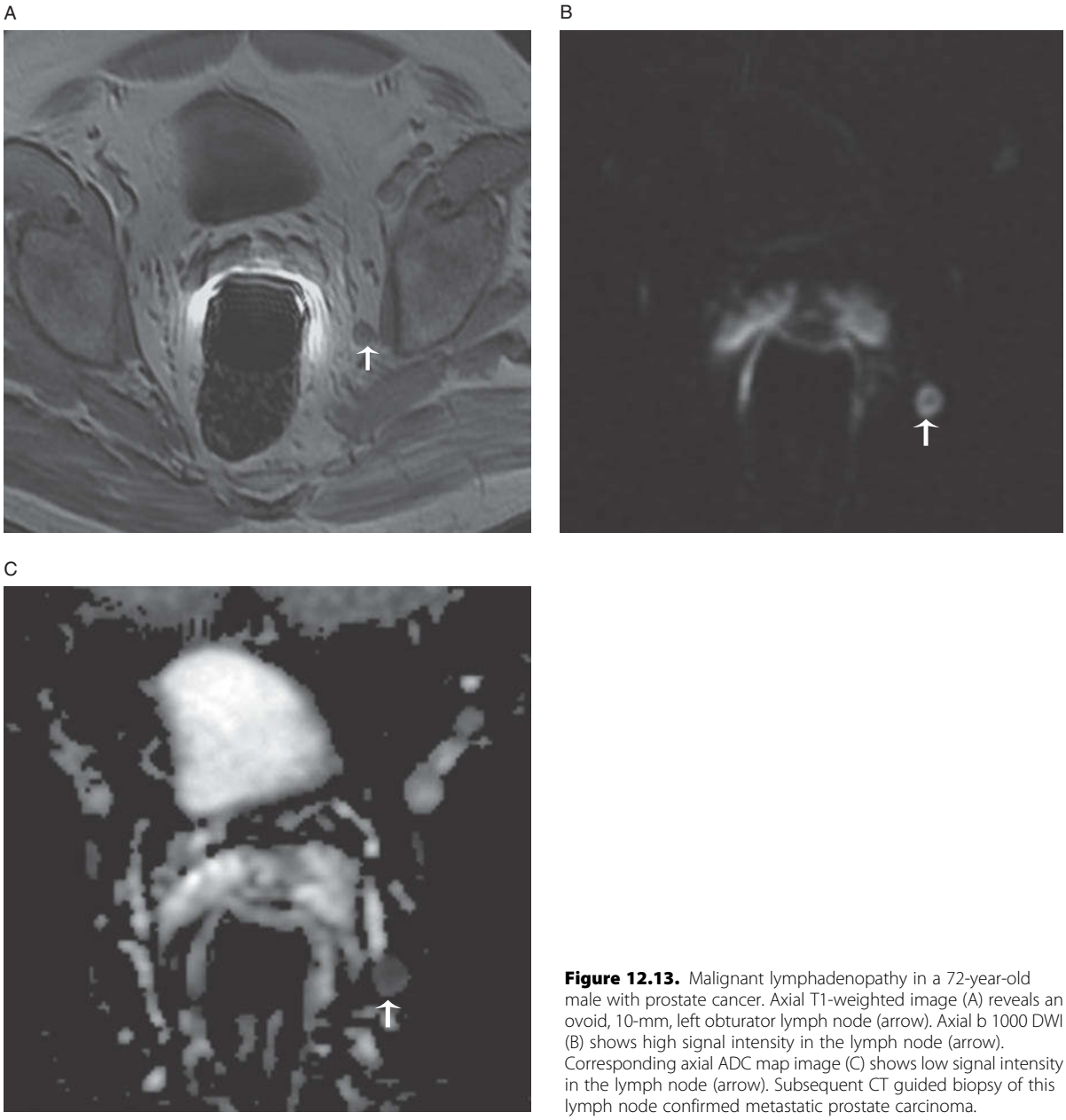

Figure 12.13. Malignant lymphadenopathy in a 72-year-old male with prostate cancer. Axial T1-weighted image (A) reveals an ovoid, 10-mm, left obturator lymph node (arrow). Axial b 1000 DWI (B) shows high signal intensity in the lymph node (arrow). Corresponding axial ADC map image (C) shows low signal intensity in the lymph node (arrow). Subsequent CT guided biopsy of this lymph node confirmed metastatic prostate carcinoma.

Axial volume-interpolated gradient echo, large field of view: T1-weighted images

These larger field of view T1-weighted images screen the pelvis for regional (obturator, iliac, presacral) and more distant (common iliac, paraortic) lymphadenopathy, as well as pelvic osseous metastasis. In general, oval lymph nodes greater than 10 mm in short axis and round lymph greater than 8 mm in short axis are suspect for metastatic disease (Figure 12.13).

Axial DWI b0, b1000, small and large field of view

At this time, DWI of the prostate is still in the research phase of development, but may supplement the small field of view, high-resolution T2-weighted images by helping to confirm or better localize prostate carcinoma as well as further highlight suspect adenopathy or osseous metastasis. In general, approach DWI of the prostate in the same manner in which you would approach DWI of the abdomen (Figures 12.13, 12.14):

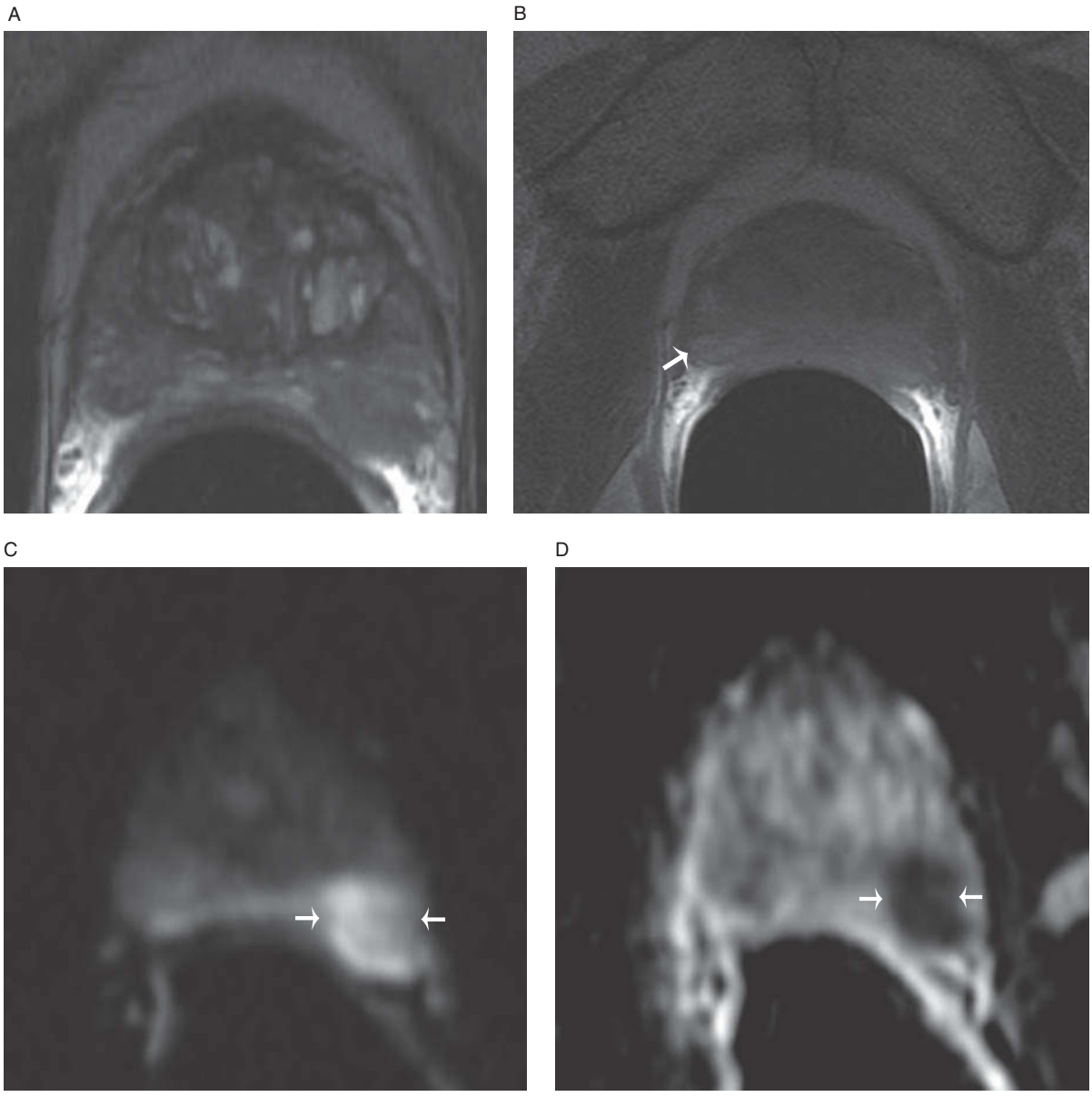

Figure 12.14. Prostate carcinoma with restricted diffusion in a 58-year-old male. Axial T2-weighted image (A) demonstrates diffuse low signal intensity in the peripheral zone of the prostate gland. Corresponding axial T1-weighted image (B) shows that some of the low signal intensity in the peripheral zone on the right is due to high signal intensity blood products (arrow). A discrete measurable tumor nodule is difficult to define on the T2- and T1-weighted images alone. However, axial DWI (C) illustrates a more defined high signal intensity area in the peripheral zone of the left midgland (arrows) with corresponding low signal intensity on the ADC map image (D, arrows) due to tumor. Prostate biopsy results described Gleason 7 prostate carcinoma in the left midgland.

(1) Identify bright areas in the prostate on b0. Areas that remain bright on b1000 image are more likely malignant and lesions that drop in signal on b1000 are more likely to be benign. Correlate with ADC.
   (a) DWI bright, ADC dark = Restricted diffusion, suggests a malignant finding.
   (b) DWI bright, ADC bright = T2 "shine-though," suggests a benign finding.

Pearls and pitfalls

(1) Indications for prostate MRI are:
   (a) Evaluation for extracapsular extension of known prostate cancer for staging.
   (b) Elevated PSA with negative blind biopsy to potentially guide further biopsies.
   (c) Elevated PSA status post prostatectomy looking for metastatic disease.
(2) T1-bright material is blood, not tumor. Always correlate dark signal on the T2s with the T1s to make sure you're not calling post-biopsy blood, tumor.
(3) Our main job is to find extracapsular extension. This is important because it may triage some patients to radiation rather than surgical therapy.

Further reading

Male pelvis

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Chapter 13

Rectal MRI

Anorectal fistula protocol

Indications

This protocol is used for evaluation of suspected or known anal and rectal fistulas.

Preparation

- Patients should not take an enema for 24 hours before the study. They can cause rectal spasm and motion artifact
- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- **Rectal contrast agent**: 60 cc ultrasound gel if evaluating for a rectovaginal fistula (Figure 13.1); otherwise, no rectal contrast

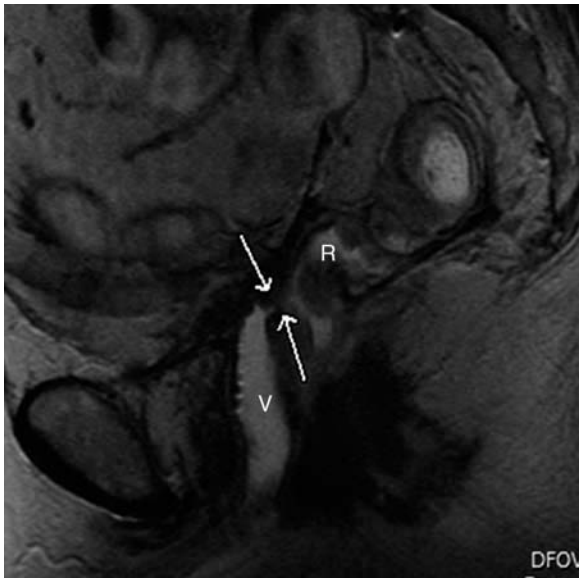

Figure 13.1. Rectovaginal fistula in a 67-year-old female. Sagittal T2-weighted image demonstrates high signal intensity gel in the rectum (R) and extending via a rectovaginal fistula (arrows) into the vaginal canal (V).

- Have the patient void prior to the start of the study
- The radiologist should be consulted for best plane to visualize pathology

Exam sequences

(1) Sagittal T2 FSE – Use to plan axial and coronal oblique sequences. Make sure that imaging is performed through the entire perineum to ensure complete visualization of fistula tracks.
(2) Axial oblique T2 FSE – Angled perpendicular to rectum or anal canal.
(3) Coronal oblique T2 FSE FS – Angled parallel to rectum or anal canal. T2-weighted images are used to identify fluid within fistulous tracks and abscesses.
(4) Axial oblique volume-interpolated gradient echo BH pre – Angled perpendicular to rectum or anal canal.
(5) Axial oblique volume-interpolated gradient echo BH post IV administration of contrast gadolinium injection at 70 seconds – Angled perpendicular to rectum or anal canal.
(6) Coronal oblique volume-interpolated gradient echo BH post IV administration of contrast at ~2 min. – Angled parallel to rectum or anal canal.
(7) Sagittal volume-interpolated gradient echo BH post IV administration of contrast at ~3 min.

Approach to interpretation of MRI exams of anorectal fistulas

Fistulas are typically first diagnosed clinically in patients who present with local pain and discharge. MRI is primarily used to evaluate patients with complex fistulas at clinical examination, recurrent fistulas, fistulas in patients with Crohn's disease (often complex), and to follow up medically managed fistulas. The referring surgeon often knows that a fistula exists but can't tell how deep it goes or what other structures it involves. We can.

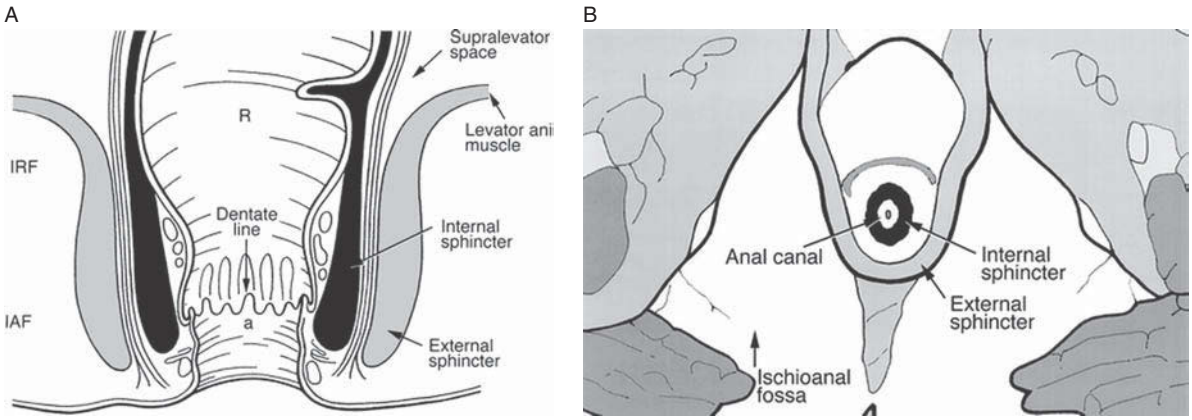

Figure 13.2. Normal anatomy of anal sphincters. (A) Line diagram shows the normal anatomy of the perianal region in the coronal plane. *a* = anal canal, *IAF* = ischioanal fossa, *IRF* = ischiorectal fossa, *R* = rectum. (B) Line diagram shows the normal anatomy of the perianal region in the axial plane. Reprinted with permission: *Morris et al. MRI classification of perianal fistuals and its implications for patient management. March 2000 RadioGraphics, 20, 623-635.*

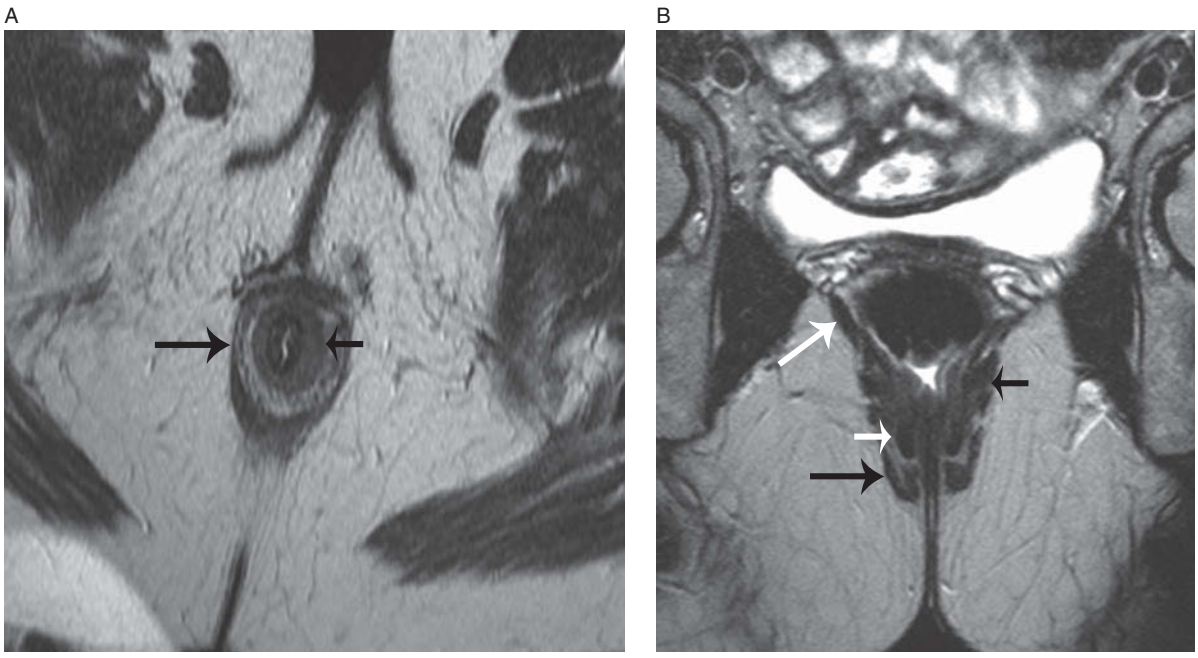

Figure 13.3. Normal anatomy of sphincter complex. Axial T2-weighted image (A), long arrow on the external anal sphincter (EAS) and short arrow on the internal anal sphincter (IAS). Coronal T2-weighted image (B), long arrow on EAS, short white arrow on IAS, short black arrow on puborectalis (PR), and long white arrow on iliococcygeus (IC) muscle.

The excellent soft-tissue resolution of MRI allows for differentiation of supralevator and infralevator disease, secondary tracks or ramifications, and differentiation of active disease versus fibrosis. Important questions to answer are the relationship between the fistula and anal sphincter and levator ani muscles and the presence of any unsuspected primary tract extensions that also require treatment to prevent recurrence.

Anal fistulas are classified as intersphincteric (between the internal and external anal sphincter muscles), transsphincteric (extending through both the internal and external anal sphincter muscles), suprasphincteric (extending above the level of the anal sphincter muscles), or extrasphincteric (extending from the rectum without extension through the anal sphincter muscles) (Figures 13.2–13.6).

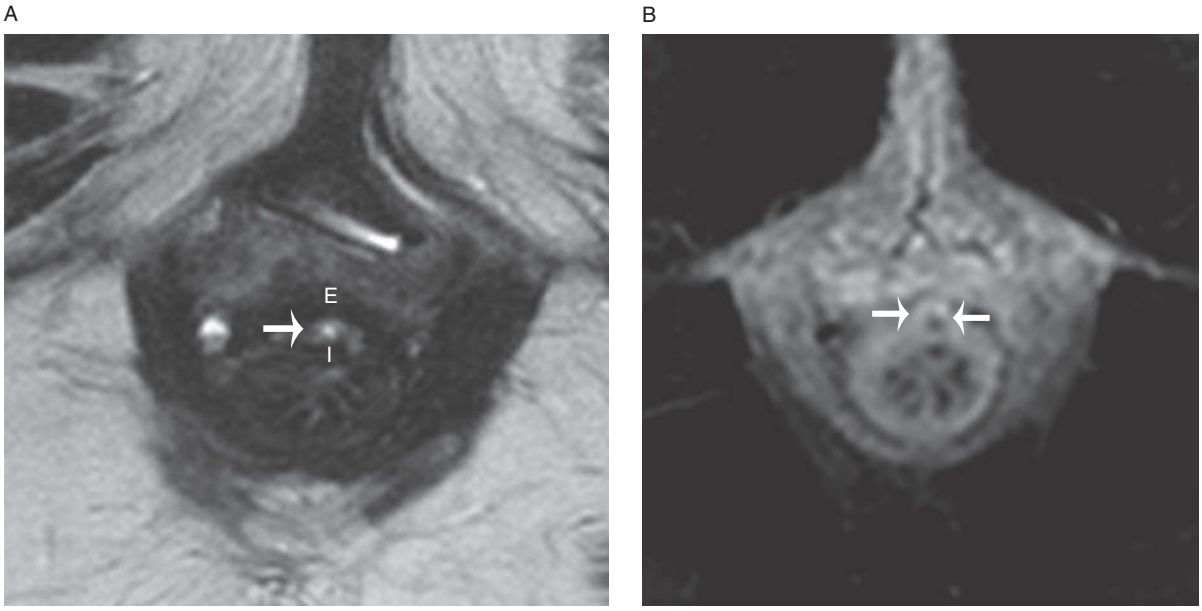

Figure 13.4. Active intersphincteric fistula in a 42-year-old female. Axial T2-weighted image (A) shows a high T2 signal intensity fistula (arrow) between the internal (I) and external (E) anal sphincter muscles. Axial contrast-enhanced T1-weighted image with fat saturation (B) demonstrates fistula wall enhancement (arrows).

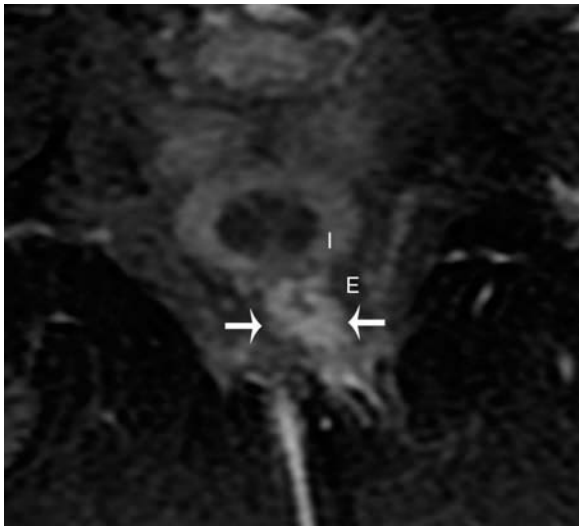

Figure 13.5. Active transsphincteric fistula in a 36-year-old male. Axial contrast-enhanced T1-weighted image with fat saturation demonstrates thick wall enhancement of a fistula (arrows) extending through both the internal (I) and external (E) anal sphincter muscles.

An endoanal coil would likely provide beautiful images of fistula, but it's cruel to try to insert one. Pelvic surface coil is fine.

Sequences and approach

Multiplanar FSE (TSE): T2-weighted images

T2-weighted images are useful for identifying active fistulas (high T2 luminal signal intensity) (Figures 13.4 and 13.6) and extensions of secondary tracks into the adjacent ischioanal fossa, sphincter, and levator ani muscles (Figures 13.6 and 13.7). Abscesses can also be identified by their high T2 signal intensity contents.

Oblique imaging along the true axial and true coronal planes of the rectum or anal canal is essential for accurately characterizing the type and extent of fistulas. The addition of fat saturation in at least one plane (typically coronal) helps to further highlight the high luminal T2 signal intensity of active tracks. Inactive tracks have low T2 signal intensity due to fibrosis (Figure 13.8).

Contrast-enhanced images: T1-weighted fat-saturated images

Often times, fistulas and fistulous tracks will best be identified on the post-contrast images. Track enhancement is indicative of an active fistula (Figures 13.4–13.7). Rim enhancement can also further highlight associated abscess formation.

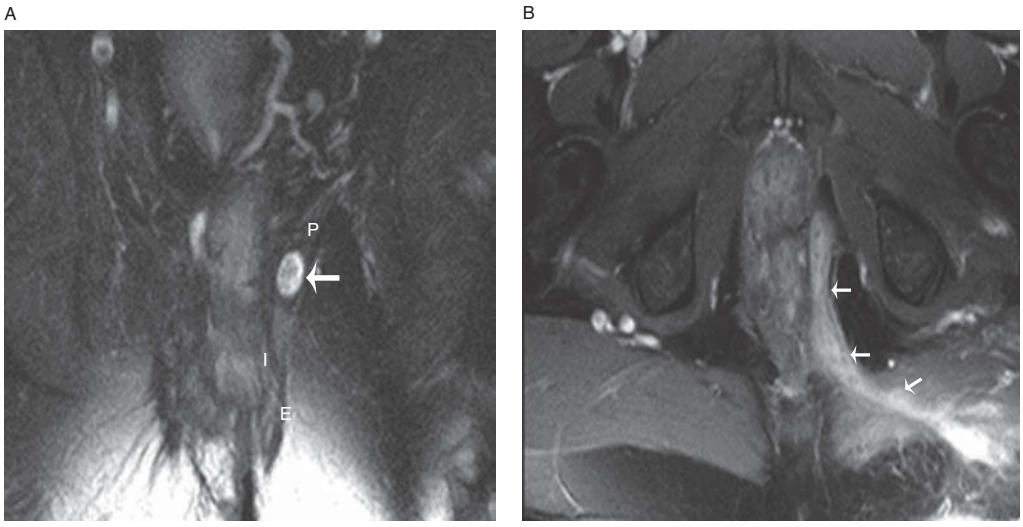

Figure 13.6. Active suprasphincteric fistula in a 40-year-old male. Coronal T2-weighted image with fat saturation (A) reveals fistula (arrow) extension above the level of the anal sphincter muscles (I: internal and E: external anal sphincters) into the left puborectalis muscle (P). Axial contrast-enhanced T1-weighted image with fat saturation (B) also demonstrates extension of the fistula (arrows) into the left ischiorectal fossa.

Pearls and pitfalls

(1) MRI of anal fistulas is primarily reserved for patients with complex or recurrent fistulas.
(2) MRI can readily characterize fistulas as intersphincteric, transsphincteric, suprasphincteric, or extrasphincteric.
(3) The MRI examination is performed without an endoanal coil and without an enema preparation within 24 hours of the examination.

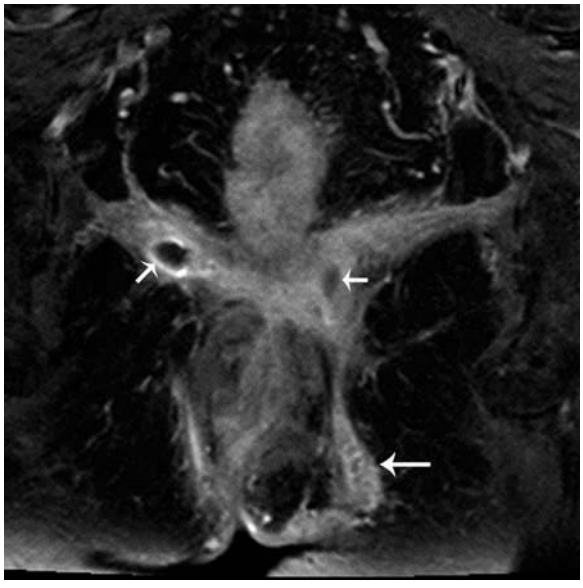

Figure 13.7. Active complex suprasphincteric and extrasphincteric fistulae in a 54-year-old male. Coronal contrast-enhanced T1-weighted image with fat illustrates active rim enhancing fistula tracks into the right and left levator ani muscles (short arrows) and chronic enhancing fistula track extending into the left ischiorectal fossa (long arrow).

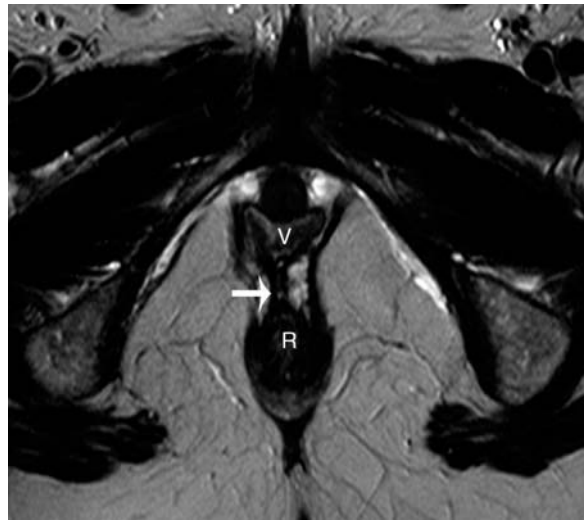

Figure 13.8. Chronic rectovaginal fistula in a 45-year-old female. Axial T2-weighted image shows a low T2 signal intensity track (arrow) between the rectum (R) and vagina (V) due to fibrosis.

Rectal cancer protocol

Indications

This protocol is used to stage known rectal carcinoma.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- At least 24 gauge-IV; connect to power injector

- Have patient void just prior to start of the exam
- Consult radiologist for best plane to visualize pathology

Exam sequences

(1) Diffusion-weighted imaging b50, 500/ADC – Excellent for mass detection.
(2) Sagittal T2 fast-spin echo, small field of view – Use to plan oblique planes.
(3) Axial oblique T2 fast-spin echo, small field of view – Angled perpendicular to rectal mass. T2-weighted images are used to identify the rectal mass.
(4) Coronal oblique T2 fast-spin echo, small field of view – Angled parallel to rectal mass.
(5) Axial oblique volume-interpolated gradient echo BH, small field of view – Angled perpendicular to rectal mass.
(6) Axial volume-interpolated gradient echo BH, large field of view – Screen pelvis for lymph nodes and osseous metastasis.
(7) Axial oblique volume-interpolated gradient echo BH, small field of view, post IV administration of contrast at 70 seconds.

NOTES

Patients should not have an enema 24 hours before the study. It can cause rectal spasm and motion artifact.

Small field of view: 180 cm

Large field of view: Aortic bifurcation through symphysis pubis.

Approach to interpretation of MRI exams for rectal masses

Don't let the name fool you, colorectal cancer is not one disease. Colon cancer and rectal cancer behave differently. Rectal cancer has a poorer prognosis and preferentially metastasizes to the lung rather than the liver due to different venous and lymphatic drainage.

When you see a low cancer, try to distinguish between rectal and colon. Don't just report that it's "rectosigmoid." In addition to different patterns of spread and prognosis, the treatment is different too. Locally advanced rectal cancers are often treated with pre-operative neoadjuvant chemotherapy and radiation; colon cancer is not.

Table 13.1 MRI staging of rectal cancer

T1	Tumor limited to mucosa/submucosa
T2	Tumor invades, but does not extend through muscularis propria
T3	Tumor extends through muscularis propria into the perirectal fat; a broad-based bulge or nodular extension. Fine linear extension is often due to desmoplastic reaction
T4	Tumor invades adjacent organs
N0	No nodes
N1	1 to 3 regional nodes
N2	>4 regional nodes
N3	Central nodes (retroperitoneal)

Anatomy

The rectum is 15 cm long. On MRI, the exact delineation of sigmoid from rectum is challenging, so we use the *sacral promontory* as the level at which the rectum ends and the sigmoid colon begins. It is helpful to mentally divide the rectum into proximal, mid, and distal thirds and report the location of the tumor as such.

The anal sphincter has two components, internal and external. The internal sphincter is a continuation of the muscularis propria of the rectum and is surrounded by the external anal sphincter inferiorly and the levater ani muscles (puborectalis, pubococcygeus, iliococcygeus) more superiorly. The inferior border of the iliococcygeus muscle demarcates the anorectal junction.

Like most pelvic imaging, T2 is the workhorse sequence. Rectal cancer is intermediate signal on T2-weighted images (brighter than muscular layers, darker than fat) (Figure 13.9).

Reports should always include the size of the tumor and distance from the and sphincter. The distance from the sphincter is critical. If there is an adequate margin, a low anterior resection can be performed which spares the anal sphincter. If not, treatment is abdominal perineal resection (APR) which removes the rectum and anus, leaving the patient with a permanent colostomy.

As stated above, our job is typically to distinguish T2 from T3 disease which means we are looking for tumor extension through the rectal wall and into the perirectal fat (Figure 13.10). Although T2-weighted images are the workhorse, gadolinium-enhanced sequences may increase our sensitivity.

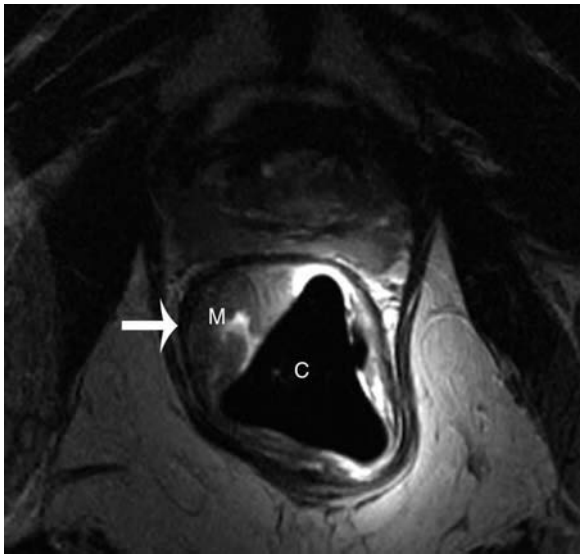

Figure 13.9. Stage T1 rectal carcinoma in a 65-year-old female. Axial T2-weighted image with an endorectal coil (C) reveals an intermediate T2 signal intensity mass (M) corresponding to the patient's biopsy-proven rectal carcinoma confined to the rectal mucosa. The lower T2 signal intensity rectal wall (arrow) is intact. (Please note: that while this is a beautiful image, we no longer routinely use endorectal coils for rectal cancer staging.)

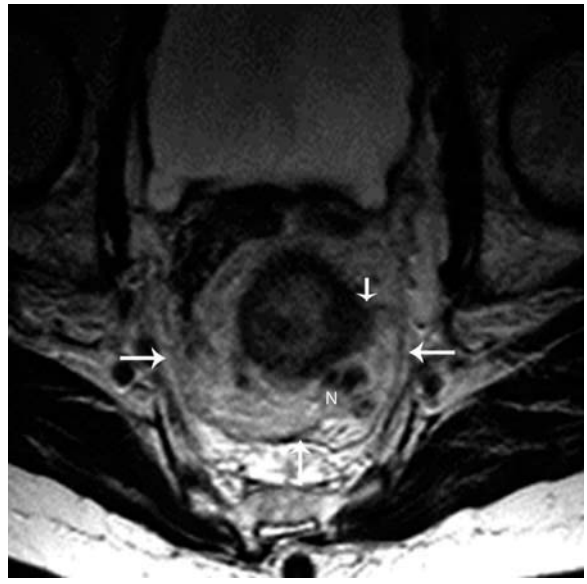

Figure 13.10. Stage T3 N1 rectal carcinoma in a 72-year-old male. Axial T2-weighted image demonstrates extension of the intermediate T2 signal intensity rectal carcinoma into the left perirectal fat (short arrow). Tumor does not breach the mesorectal fascia/circumferential resection margin (long arrows). Also note prominent regional perirectal lymph nodes (N).

The most common mistake made interpreting these studies is reporting minimal haziness of the rectal fat interface as tumor extension. Minimal haziness is OK and usually represents desmoplastic reaction, *but any nodularity is consistent with tumor extension*.

If the tumor extends through the rectal wall (T3 disease) be sure to report the smallest distance from the tumor margin to the mesorectal fascia. Tumor extension to within 5 mm of the mesorectal fascia increases the potential for inadequate surgical margin.

Next, look for perirectal nodes. In the setting of known rectal cancer, any nearby lymph node 6 mm or greater is suspicious (Figure 13.11).

Finally, don't forget to exclude T4 disease. Look for invasion of adjacent solid organs (prostate, seminal vesicles, vagina, bladder, pelvic sidewall) (Figure 13.12).

Rectal cancer staging

T1: Invades submucosa
T2: Invades muscularis propria
T3: Invades through rectal wall into perirectal fat
T4: Invades adjacent organs (prostate, seminal vesicles, bladder, vagina).

Most commonly, our job is distinguishing T2 from T3 disease. That is, our job is tell the surgeon whether or not the tumor extends through the rectal wall.

Technique and interpretation

In the past, we used endorectal coils. (Not the same coil as for prostate, which is also often referred to as an enodrectal coil, but a smaller version.) When placed perfectly, they provide exquisite images. Unfortunately, it's difficult to place them perfectly. They tend to be either too low, or too high, or they compress the

Pearls and pitfalls

(1) MRI is especially useful for staging high rectal carcinomas that may be difficult to visualize sonographically.
(2) The key staging feature to search for on MRI is tumor extension into the perirectal fat.
(3) Desmoplastic reaction can be mistaken for tumor extension into the perirectal fat. Desmoplastic reaction tends to manifest as finer, linear extensions and tumor as a larger, broad-based bulge or nodular extension.

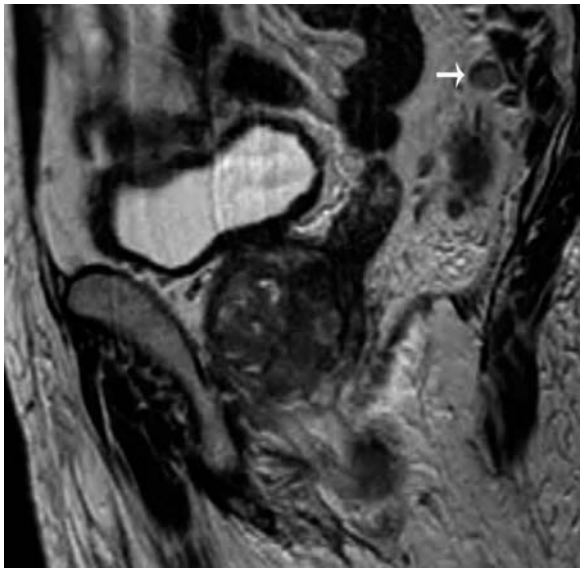

Figure 13.11. 62-year-old female with stage T3 rectal carcinoma. Sagittal T2-weighted image reveals a prominent, ovoid, 7-mm presacral lymph node (arrow).

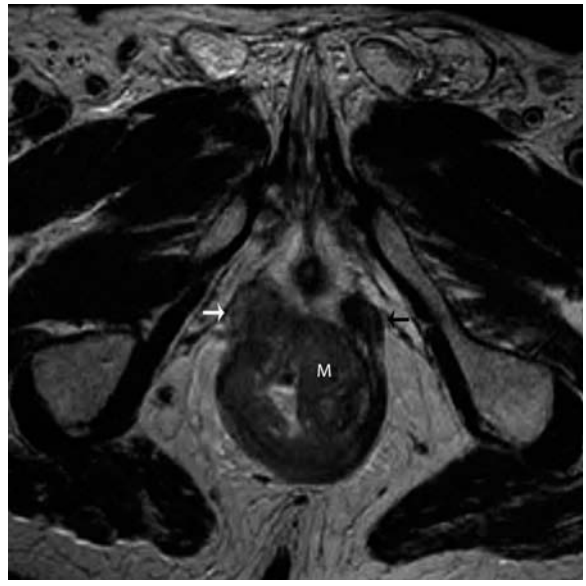

Figure 13.12. Stage T3 rectal carcinoma in a 55-year-old male. Axial T2-weighted image shows an intermediate T2 signal intensity rectal mass (M) with extension into the right puborectalis muscle (white arrow). Normal left puborectalis muscle (black arrow).

mass against the mucosa 'til you can't see it, or, worst of all, you encounter resistance trying to insert the coil and don't know if it's because you're pushing against tumor. We don't use them any more. No patient has complained. Use a pelvic phased-array coil. We do use glucagon.

Further reading

Anorectal fistula

Halligan S and Soker J. Imaging of fistula in ano. *Radiology* 2006 Apr; **239**(1): 18–33.

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Chapter 14

MRA introduction

Since the dawn of radiology, mankind has struggled to image the vasculature and conventional, or catheter-based, angiography has been the gold standard. Advantages of catheter-based angiography include: very high spatial resolution, temporal resolution (allowing the operator to follow the bolus of injected contrast from the artery through the vein), and the opportunity to intervene and treat a vascular abnormality. Many advances have been made in therapeutic interventional techniques particularly angioplasty, stenting, and embolization. However catheter angiography has several disadvantages including expense, imaging time, radiation exposure, and complications. Complications can be severe including hemorrhage, dissection, pseudoaneurysm, and inadvertent vascular occlusion.

Rapid advances in MRI, CT and ultrasound have led to the increasing use of these modalities to diagnose vascular abnormalities. The advantages of these modalities exactly address the disadvantages of catheter angiography including: decreased expense, imaging time, and, in the case of MRI and ultrasound, no ionizing radiation. Cross-sectional imaging has largely replaced catheter angiography for the *diagnosis* of vascular abnormalities. Catheter-based angiography is now used primarily for therapeutic *interventions*.

Comparing MRI angiography to catheter-based angiography is similar to comparing MRCP with ERCP. While it's true that ERCP can find and fix a problem at the same time, no one ever got pancreatitis from an MRCP. Similarly, MRI angiography can't repair a renal artery stenosis, but no one ever got a dissection or a retroperitoneal bleed from an MRA either.

MRI angiography techniques

Fundamentally, there are two categories of techniques used for MRA:

(1) Gadolinium enhanced

(2) Not gadolinium enhanced.

Gadolinium enhanced

Gadolinium-enhanced MRA remains the technique by which all others are judged. Essentially, it is a fast T1-weighted sequence which suppresses (turns black) most everything other than the vasculature which avidly enhances. We use a 3D-fat-suppressed spoiled gradient echo (SPGR). As you know, gadolinium shortens the T1 time of enhancing tissues giving bright signal on T1-weighted images. What else is bright on T1-weighted images? Fat (and a few other things).

Do we want to see bright fat on MRA sequences? No, so we use fat suppression to eliminate the signal from fat. Frankly, we would like to get rid of the signal from most everything else as well so we only see the vessels. So, we use sequences with relatively short TR intervals to saturate out any signal from stationary tissue. Short TR also enables rapid acquisition.

By using fat saturation and short TR T1-weighted imaging, the only thing we will see is the gadolinium-enhanced (with very short T1) blood flowing through the vessels.

How much gadolinium is necessary? We currently use 0.1 mmol/kg of gadobenate dimeglumine which boasts an increased T1 relaxivity over other gadolinium agents. The signal-to-noise ratio of contrast-enhanced MRA is very high so higher doses are not required.

The contrast is typically power-injected as a bolus (usually at 2 cc/s) followed by a saline chaser to flush it from the tubing. At what point one begins imaging following the bolus depends on two factors:

(1) What you are trying to image

(2) The patient's hemodynamics.

The first is easy to figure out. What you are trying to image is usually written on the order sheet. The second is a bit trickier.

Patient hemodynamics, such as cardiac output, are unfortunately variable. There are essentially three

ways to figure out the appropriate imaging delay following injection of contrast:

(1) *Fixed delay* This is the easiest way. Use the same delay every time. And if you're a gambler, then this technique is for you. It is also the worst. Adequate MRA is easy but excellent MRA requires some attention to detail. The point is, that there is enough variability in patient cardiac output and hemodynamics that using the same delay before imaging the aorta in a nervous 19-year-old professional cyclist who had three cups of coffee before the exam and a heavily sedated 99-year-old smoker with three-pillow orthopnea and an ejection fraction of 11% just doesn't make sense and won't produce very good (or reproducible) images.
(2) *Timing bolus* The technologist injects a small amount of contrast at the same rate as the MRA, starts a stopwatch, and then uses a very fast, time-resolved sequence (think of it as MRI-fluoroscopy) to watch the bolus reach the structure of interest and then stops the stopwatch. They now know the ideal timing of the actual, full-contrast bolus. This technique has been around the block and, simply put, it works so well that many institutions still swear by it. It's a bit complex for my taste. It requires two injections, a stopwatch, etc. . . .
(3) *Bolus tracking* This is basically the same as using a timing bolus but instead of injecting an initial small amount of contrast, the technologist injects the full bolus. Then, they watch using our MRI-fluoroscopy sequence until just before the structure of interest enhances at which point they trigger the MRA sequence. The nice thing about this technique is that it only requires a single injection, no stopwatch and no math. Of course, the technologist has to be familiar with the anatomy and trigger the MRA at the right time. There's really no second chance here. If you miss, you miss. But, with proper training, we have found this extremely useful and reproducible and now use it routinely not only for MRAs but also to more precisely time the arterial phase of our (non-MRA) abdominal exams.

It is interesting to note that bolus-tracking techniques are available on all modern CT scanners, but, at least at our institution, are not routinely used. In fact, for CT these techniques are even easier because the technologist can place a region of interest on a vascular structure (the main pulmonary artery for example) and the scanner can be set to trigger the CTA when a certain Hounsfield unit threshold is reached.

So, why do we swear by these techniques for MRA but ignore them for CTA?

(1) First, CT guys are to real radiologists the way orthopedists are to surgeons. They get the job done, but usually by a combination of luck and brute force.
(2) Second, MRA is less forgiving than CTA. The contrast bolus is much, much smaller in MRA. A typical 150-pound patient receives only 15 cc of gadolinium for an MRA while they would receive 130 cc of iodinated contrast for a CTA. It is much more difficult to "miss" the CT bolus (although the injection rate with MRA is only ½ to 1/3 that with CTA . . .).
(3) Third, the MRA sequences themselves take significantly longer than a CTA. A typical MRA sequence may require a full 20-second breath-hold. In that time, a modern CT scanner can image the whole emergency room.

Not gadolinium enhanced

In the 1980–90s body MRA was often performed without gadolinium using time-of-flight and phase-contrast techniques. The rapid advances in MRI hardware and software allowed the development of gadolinium-enhanced MRA which offered short imaging times, multiplanar imaging, and high spatial resolution. In the mid 1990s to mid 2000s gadolinium-enhanced MRA ruled the day and time-of-flight and phase-contrast MRA lost favor.

In the pre-NSF era, no one really cared much about non-contrast MRA – gadolinium-enhanced MRA worked too well and was considered (nearly) completely safe. NSF changed the paradigm and unfortunately, many of the patients who could benefit from vascular imaging are also those with low eGFR. The advent of NSF has led to the resurrection of non-contrast-enhanced techniques.

There are currently a wide variety of non-contrast MRA sequences available for routine use. Stay tuned . . . because of NSF this is a bustling area of research.

(1) *Time of flight (TOF)* –The classic neuroradiology MRI angiography sequence for the circle of Willis. By using a very short TR interval, it saturates out the signal of stationary tissues. The only tissue that

is not saturated is fully magnetized blood flowing into the imaging volume which has not "seen" the prior excitations. This works well for blood flowing perpendicular to the imaging plane, the carotids imaged in the axial plane for example. Blood flowing into a perpendicularly placed slice will be bright. A saturation band can be placed above or below the area of interest to selectively null the signal from blood in either arteries or veins. Unfortunately, it is too time-consuming to complete during a breath-hold so it is useless in the abdomen. Also, it only works well when flowing blood is directly perpendicular to the slice. *For body imaging, it is essentially only used for pelvic MRV*. (Remember that TOF images are T1-weighted.)

(2) *Phase contrast* (Warning. Some will consider the following explanation of how phase-contrast imaging works to be oversimplified.) Phase-contrast MRI works by using phase data to create an image. What do we mean by phase data? It turns out that MRI antennae can not only quantify the amount of signal coming from the patient, but also the phase of the signal. What do we mean by phase? Think of the RF energy as a sine wave which oscillates at a particular frequency. The MRI detector can tell where on the sine wave you are. For example, at the peak, the valley on the upslope, or on the downslope. Where on the sine wave you are is the phase of the signal. Phase-contrast is a regular MRI pulse sequence with the addition of two, opposing gradient pulses. For protons that aren't moving during the course of the acquisition, the pulses cancel. But . . . protons that move during the acquisition will experience a phase-shift proportional to their velocity. The scanner will then generate two sets of images: magnitude images and phase images. The magnitude images look like anatomic images. The phase images just show moving protons as black or white (depending on what direction they are moving).

Both sets of images are valuable and add to the power of phase-contrast imaging (Figure 14.1).

(a) Magnitude images – Use to examine anatomy. These are particularly valuable because turbulent flow such as at a stenosis will cause "de-phasing" of protons, resulting in signal loss (the area is black). This is a clue that a suspected stenosis is hemodynamically significant.

(b) Phase images – These images show swirls of black and white which correspond to different directions of blood flow. But that's not all. Using software built into the scanner we can actually quantify the blood flow. That's right. We can generate instantaneous velocity at each point (pixel) in a vessel for each time point in the cardiac cycle. By adding together data from each phase of the cardiac cycle on a pixel-wise basis, available software readily provides quantification of bulk flow through the vessel (per heart beat). In the same manner, we can determine the peak systolic velocities within stenotic vessels, which in turn are readily

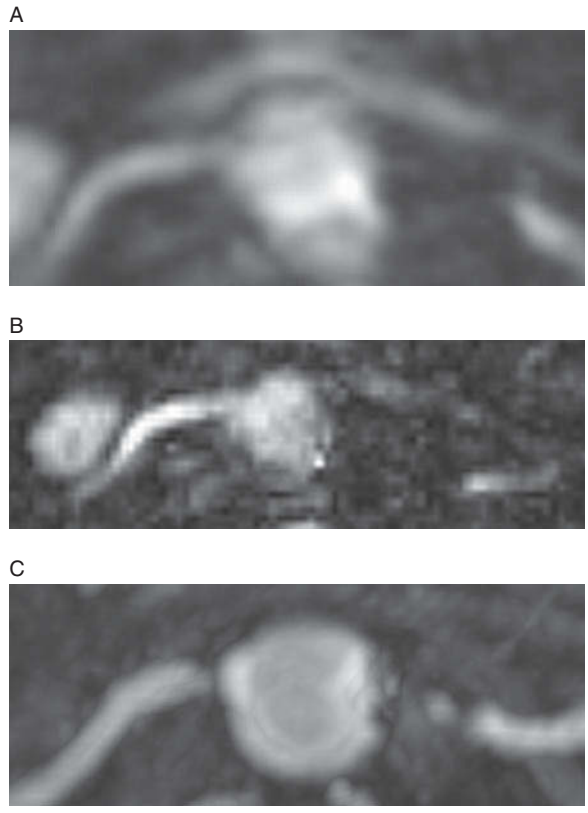

Figure 14.1. Phase-contrast images. Magnitude image (A) demonstrates flow within the right renal artery but absence of flow in the proximal left renal artery due to turbulence (de-phasing). Phase image (B) shows less anatomic detail but can be used for quantification. Gadolinium-enhanced MRA (C) shows extensive atherosclerotic plaque with severe narrowing of the origin of the left renal artery. Although there is obviously stenosis of the origin of the right renal artery, it's patency on the magnitude image (A) suggests that it is likely not hemodynamically significant.

converted to pressure gradients upon which treatment options are based. Quantification is most useful in cardiac application, but keep in mind that because of phase-contrast imaging MRI can be quantitative. CT cannot. Referring clinicians love numbers, particularly cardiologists.

Phase-contrast is typically performed during a breath-hold or free-breathing with multiple averages. But, recently, vendors have released respiratory triggered phase-contrast sequences which promise to further refine this technique. 2D and 3D techniques are available.

(3) *Double inversion recovery single-shot fast-spin echo* (black-blood images) – MRI is a marvel of modern physics. Once in a while, it is important to take a deep breath and appreciate how wondrous it is that we can obtain exquisite images of the inside of the body using a powerful magnet. Now add that physicists can turn flowing blood black, when it is naturally T1-bright, well, now . . . the wonders indeed never cease. Black-blood images are your best sequence for evaluation of vessel size and the vessel wall itself. The vessel wall should be nice and thin. Also, these images beautifully depict dissection flaps and can be used to exclude or characterize aortic dissection or hematoma without administering gadolinium.

(4) *Steady-state free precession (SSFP)* – Blood is inherently bright on these balanced images in both arteries and veins. However, newer techniques allow suppression of the signal from venous structures to provide crisp, clear visualization of arterial structures only. The most common clinical use of this sequence is to image the aorta and renal arteries.

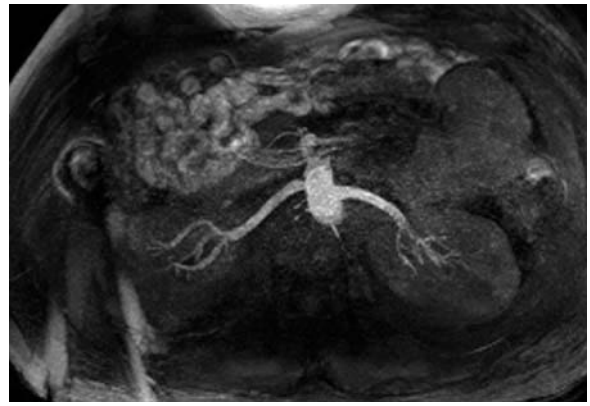

Figure 14.2. Non-contrast MRA. Axial MIP image from ECG-gated single-shot fast-spin echo demonstrating normal renal arteries bilaterally. No gadolinium necessary.

(5) *ECG-gated single-shot fast-spin echo (SSFSE)* – This technique exploits the changing signal characteristics of blood during systole and diastole. Venous blood, due to slow flow, will be bright during both systole and diastole. But, high flow in arteries during systole will create a flow void (i.e. will be dark) whereas it will be bright during diastole. Arterial angiographic images are then created by subtracting the systolic from the diastolic images. The veins were bright on both so will now be black. The arteries were bright on the diastolic images and dark on the systolic images, so will appear bright. Please note that while this technique has been around for years, it has not been in widespread clinical use and may not be available on all scanners (Figure 14.2).

Chapter 15

Chest MRA

Chest MRA protocol

Indications

This protocol is most commonly used for assessment of aortic size as well as for evaluation and follow-up of aortic dissection. It may also be used to evaluate for stenosis of the great vessels. If the thoracic aorta is not the primary vessel of interest, consider changing the plane of acquisition of the pre- and post-contrast SPGRs (although they are 3D, so they can be reformatted later in any plane).

Preparation

- **IV contrast agent:** gadobenate dimeglumine 0.1 mmol/kg at 2 cc/s
- **Oral contrast agent:** None
- 2 L nasal oxygen
- At least 22-gauge IV; connect to power injector
- Scan with patient's arms overhead, if patient can tolerate, for smallest field of view possible
- All sequences are cardiac-gated except the SPGRs.

Exam sequences and what we are looking for

(1) Axial double-inversion recovery SSFSE BH (dark blood) – Carefully evaluate the vessel wall and look for dissection flaps.
(2) Axial SSFP BH (bright blood; non cine) – Confirm vessel size and presence/absence of dissection flap.
(3) Coronal double-inversion recovery SSFSE BH – Examine aortic root, typically better seen in coronal plane.
(4) Sagittal oblique 3D-SPGR pre-contrast BH.
(5) Sagittal oblique 3D-SPGR post-contrast BH arterial – Best evaluation of great vessel anatomy and presence/absence of stenosis.
(6) Sagittal oblique 3D-SPGR post-contrast BH venous.
(7) Parasagittal cine SFFP 2D (candy-cane) – View the thoracic aorta in its natural plane. Lumen and wall can be evaluated. And as below dissection flaps may be identified.
(8) Axial SSFP cine (optional) if aortic dissection is known, suspected, or discovered. This essentially gives us 20–25 images at the same axial location. A flap can be detected, confirmed, and observed through the cardiac cycle.

Chapter 16

Abdomen-pelvis MRA

Chest/abdomen (aorta) MRA protocol

Indications

This protocol is used for routine evaluation of the aorta from the aortic root to the iliac bifurcation, typically to evaluate for aneurysm and/or dissection. The protocol is remarkably similar to that of the chest MRA, but requires two injections of contrast to obtain arterial phase imaging of both the thoracic and abdominal aorta. We inject 0.1 mmol/kg gadobenate dimeglumine for each.

Preparation

- **IV contrast agent:** Gadobenate dimeglumine 0.2 mmol/kg (total) at 2 cc/s
- **Oral contrast agent:** None
- 2 L nasal oxygen
- At least 22-gauge IV; connect to power injector
- Scan with patient's arms overhead, if patient can tolerate, for smallest field of view possible
- All sequences are cardiac-gated except the SPGRs.

Exam sequences and what we are looking for

(1) Axial double-inversion recovery SSFSE BH (dark blood) – Carefully evaluate the vessel wall and look for dissection flaps.
(2) Axial SSFP BH (bright blood) – Confirm vessel size and presence/absence of dissection flap.
(3) Coronal double-inversion recovery SSFSE BH – Examine aortic root, typically better seen in coronal plane.
(4) Parasagittal cine SFFP BH 2D – These images are acquired through the aortic root and allow prelimninary evaluation of the aortic valve and sinuses of Valsalva.
(5) Axial double-inversion recovery SSFSE BH (abdomen) – Carefully evaluate the vessel wall and look for dissection flaps.
(6) Axial SSFP BH (abdomen) – Confirm vessel size and presence/absence of dissection flap.
(7) Coronal oblique 3D-SPGR pre-contrast BH (abdomen).
(8) Coronal oblique 3D-SPGR BH arterial (abdomen) – Best evaluation of great vessel anatomy and presence/absence of stenosis.
(9) Coronal oblique 3D-SPGR BH venous (abdomen).
(10) Sagittal oblique 3D-SPGR pre-contrast BH (chest).
(11) Sagittal oblique 3D-SPGR BH arterial (chest) – Best evaluation of great vessel anatomy and presence/absence of stenosis.
(12) Sagittal oblique 3D-SPGR BH venous (chest).

Renal/mesenteric MRA protocol

Indications

This protocol is essentially the same as the abdominal portion of the aortic protocol. Renal MRA is performed in hopes of finding a correctable cause of hypertension such as renal artery stenosis or fibromuscular dysplasia. Because all of these patients will have hypertension, an SSFSE sequence is added to look for an adrenal mass (pheochromocytoma). Renal MRA can also be performed to evaluate for vascular anatomy in renal transplant donors. Mesenteric MRA is performed to evaluate for stenosis of the celiac, superior mesenteric artery (SMA), or inferior mesenteric artery (IMA). If the patient has a decreased eGFR, substitute a non-contrast MRA sequence for the SPGRs (see introduction).

Preparation

- **IV contrast agent:** gadobenate dimeglumine 0.1 mmol/kg (total) at 2 cc/s
- **Oral contrast agent:** None
- 2 L nasal oxygen
- At least 22-gauge IV; connect to power injector

- Scan with patient's arms overhead, if patient can tolerate, for smallest field of view possible
- All sequences are cardiac-gated except the SPGRs.

Exam sequences and what we are looking for

(1) Axial SSFSE BH – Localizer, although also useful to assess vessels.
(2) Coronal SSFSE BH – Evaluate for adrenal mass.
(3) Coronal oblique 3D-SPGR pre-contrast BH.
(4) Coronal oblique 3D-SPGR post-contrast BH arterial.
(5) Coronal oblique 3D-SPGR post-contrast BH venous.
(6) Coronal oblique 3D-SPGR post-contrast BH delayed.

An approach to interpretation of MRI angiography

MRI angiography is an excellent tool which rapidly and accurately depicts the anatomy of any vascular system within the body. Do not be intimidated. These are some of the simplest exams you will interpret. Let's start from the chest and work our way down into the abdomen.

Chest MRA

I follow five steps whenever interpreting chest MRA:

(1) Measure the aorta. On the axial, black-blood images, measure (and report) the diameter of the ascending aorta at the level of the main pulmonary artery. On the coronal, black-blood images, measure (and report) the diameter of the aorta at the sinuses of Valsalva (Figure 16.1).
(2) On the axial, black-blood images, look for dissection. What does a dissection look like on MRI? It looks the same as it does on CT. Look for a flap. **If there is a flap, the blood within the false lumen will often be of different (brighter) signal intensity due to slow flow**.
(3) On the axial, black-blood images evaluate the vessel wall. Make sure it's nice and thin. If it isn't, you must consider atherosclerotic plaque, vasculitis, or even intramural hematoma.
(4) On the axial, black-blood images, look for ancillary findings such as adenopathy within the mediastinum.
(5) Use the contrast-enhanced MRA to evaluate the origins of the great vessels and exclude stenosis. (For more on evaluating a potential stenosis, see abdominal MRA section below.)

Abdominal MRA

Interpretation is eerily similar to that of chest MRA.

Measure the aorta. If there's an aneurysm be sure to report its relationship to the vessels. In this era of graft placement, be specific. Give measurements regarding the aneurysm's proximity to the renal arteries (for example). Don't forget to include the size of the common iliacs as well.

Exclude dissection. If dissection is present, comment on which vessels arise from the true and which vessels arise from the false lumen.

Evaluate vessel wall. Be sure it's nice and thin. If it isn't, consider plaque, intramural hematoma, and vasculitis.

Look for incidental findings such as retroperitoneal adenopathy.

Use contrast-enhanced MRA to evaluate for stenosis and thrombosis. Specifically, one should mention the origins of the celiac trunk, the superior mesenteric artery, inferior mesenteric artery, and all renal arteries. Remember that accessory renal arteries are very common and may originate as inferior as the common iliac arteries. Generally speaking, a stenosis greater than 50% is considered hemodynamically significant (Figure 16.2). How does one judge whether the vessel lumen is narrowed greater than 50% of its expected diameter? The easiest way to quantify a vessel narrowing is to look at the segment of vessel just distal to the stenosis and assume that the diameter is the normal expected diameter of the segment just proximal to it. One then simply has to judge the degree of stenosis. Some will attempt to do this with a ruler. I believe this can be done just as well with your well-trained eye. Besides, does it matter if a vessel is 48% stenosed rather than 52% stenosed? Finally, if you look, you'll be surprised how often you'll see vascular thrombosis (Figure 16.3).

Don't forget fibromuscular dysplasia (FMD). Classically, FMD involves the renal arteries of young women and has an irregular, beaded appearance (Figure 16.4). The vessels look like the bile ducts of patients with primary sclerosing cholangitis. This is an important diagnosis to make because FMD responds very well to angioplasty without stent placement.

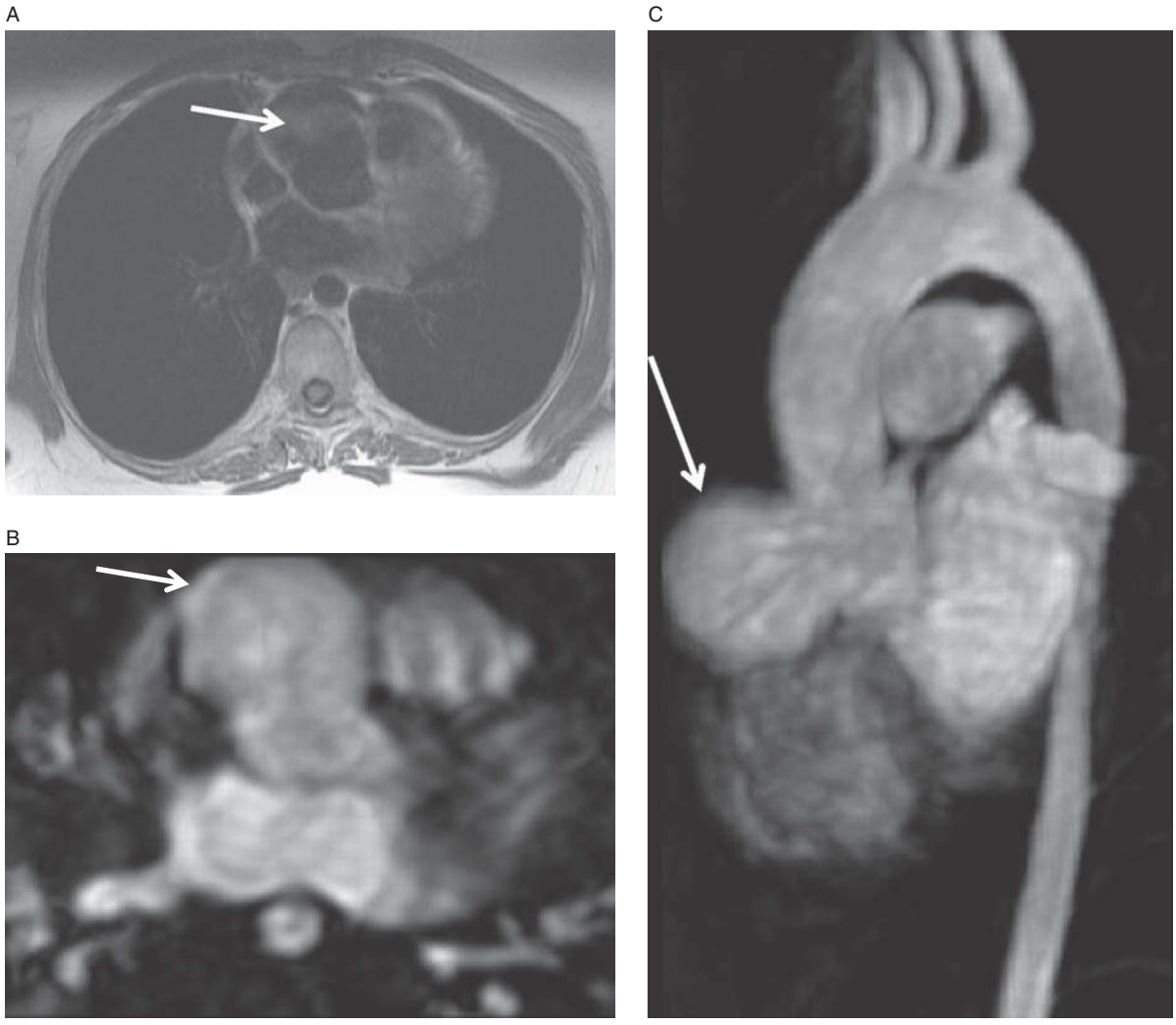

Figure 16.1. Sinus of Valsalva aneurysm. Axial dark-blood image (A) and corresponding axial gadolinium-enhanced image (B) demonstrate aneurysmal dilatation of the sinuses of Valsalva (arrows). Image C is a corresponding multiplanar reconstruction from the gadolinium-enhanced dataset.

MRA pathology (in a nutshell)

Aneurysm

Strictly speaking, an aneurysm is dilatation of a vessel greater than 1.5× its normal diameter. For the most part, you know it when you see it.

Aortic aneurysm

Aortic aneurysms are extremely common. They come in three varieties:

(1) *Atherosclerotic* By far the most common. If you attribute every aneurysm you see in your career to atherosclerosis, you will nearly always be right. They are usually fusiform and smooth, though may contain variable amounts of thrombus. They are less commonly saccular.

(2) *Mycotic* Mycotic aortic aneurysms are caused by infection rather than atherosclerosis. Clinically, a mycotic aortic aneurysm should be considered in patients with positive blood cultures and, commonly, a history of intravenous drug use. Radiologically, consider mycotic aneurysm whenever you see an eccentric, saccular, lobulated, weird-looking aneurysm (not smooth or fusiform) in the appropriate clinical context.

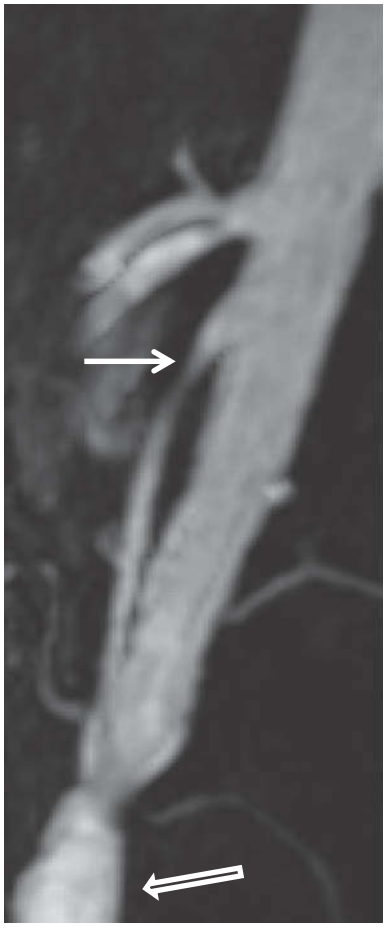

Figure 16.2. SMA stenosis. Maximum intensity projection reconstruction from contrast-enhanced MRA demonstrates narrowing of the SMA (solid arrow). Also note partially visualized abdominal aortic aneurysm (open arrow). Atherosclerosis is a systemic disease.

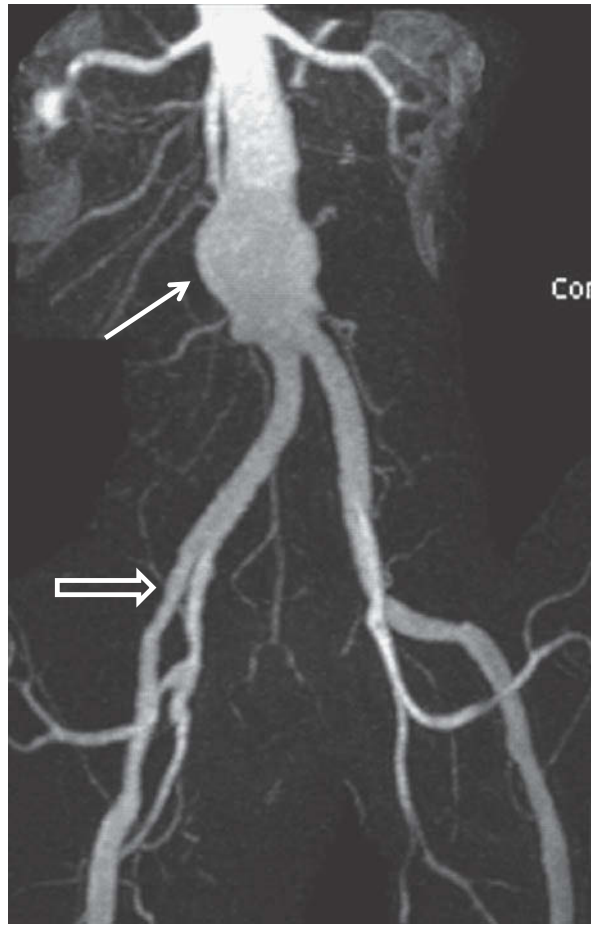

Figure 16.4. Abdominal aortic aneurysm. Maximum intensity projection reconstruction of abdominal MRA demonstrates typical smooth (minimally lobulated) appearance of infrarenal, atherosclerotic, abdominal aortic aneurysm (solid arrow). Note the irregularity of the right common external iliac artery (open arrow) – atherosclerosis is a systemic disease.

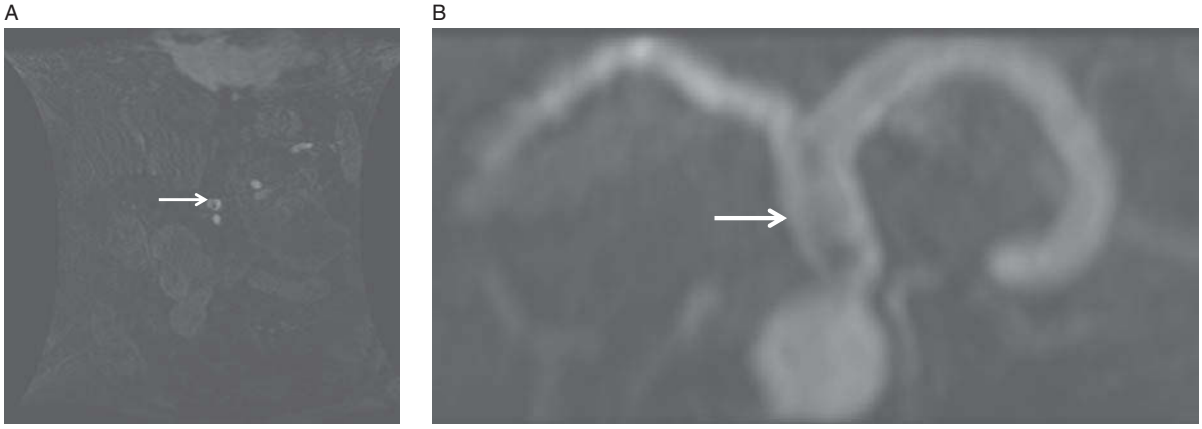

Figure 16.3. Celiac artery thrombus. Coronal gadolinium-enhanced MRA image (A) demonstrates thrombus within the celiac artery (arrow) which is also depicted nicely on reformatted maximum intensity projection (B) (arrow).

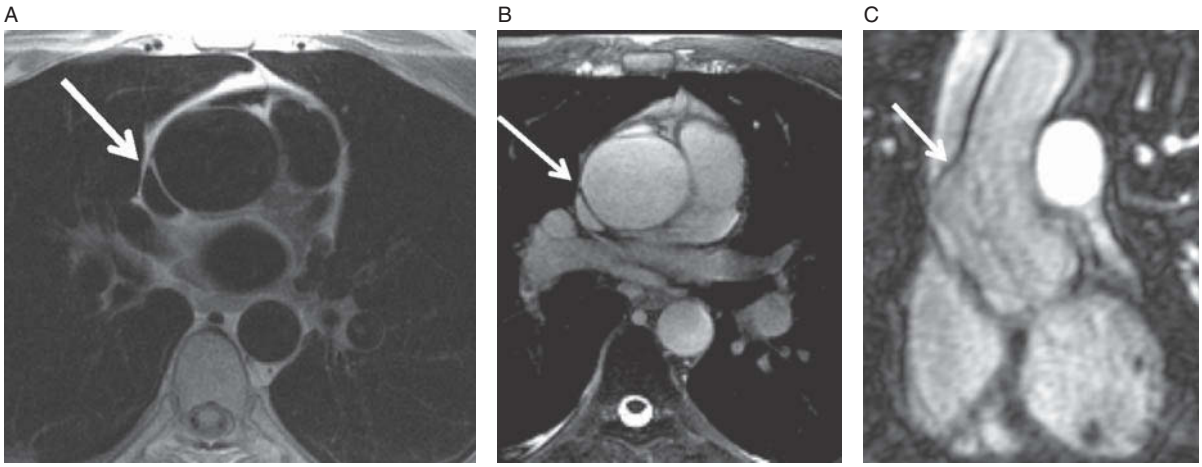

Figure 16.5. Type A aortic dissection. Axial dark-blood image (A) and bright-blood image (B) nicely depict the intimal flap within the ascending aorta (arrows). This is also well demonstrated on reformatted post-gadolinium image (C, arrow).

(3) *Inflammatory* Inflammatory aneurysms are rare. Clinically, patients are typically symptomatic and complain of abdominal or back pain. Radiologically, inflammatory aortic aneurysms are characterized by a retroperitoneal inflammatory response which mimics retroperitoneal fibrosis, except that the aorta is big. Although you may never see one, keep it in mind because inflammatory aortic aneurysms may respond to immunosuppressive therapy such as corticosteroids.

Aortic dissection

Aortic dissections occur due to a tear in the aortic intima which allows blood to flow in between the layers of the vessel wall and, potentially, to rupture the vessel itself. The most important radiologic feature is whether the dissection involves the ascending aorta (proximal to the innominate artery).[1] The Stanford classification is the most widely used system for categorization.

Stanford A Aortic dissection involves the ascending aorta +/− the descending aorta (Figure 16.5). Involvement of the ascending aorta (proximal to the origin of the innominate) is the key because of the risk of retrograde dissection into the coronary arteries or pericardium. Typically, because of these risks, emergency surgery is recommended.

Stanford B This does not involve the ascending aorta (Figure 16.6). Type B dissections are much more common and may be managed medically (antihypertensive therapy) or in some circumstances with stent grafts.

A few words on intramural hematoma

Intramural hematoma is diagnosed when blood is noted within the aortic wall, but a flap (or intimal tear) is not visualized (Figure 16.7). (Remember that in this case blood in the vessel wall will usually be bright on the dark-blood images but may not be seen on the post-contrast images because there is no communication with the lumen.) Most commonly, this is believed to occur secondary to bleeding of the vasa vasorum but can occur from fracture of an atherosclerotic plaque. These lesions may spontaneously resolve; however, they may progress to aortic dissection and are therefore probably best considered dissection precursors.

Management of intramural hematoma is evolving, however because of the risk of impending dissection the Stanford criteria are typically applied. That is: medical management for intramural hematoma of the descending aorta and consideration of surgical treatment for intramural hematoma within the ascending aorta.

[1] That's right. I said the innominate, not the left subclavian. There is a lot of confusion on this topic among MDs of all specialties. I will write it again: Stanford A involves the ascending aorta (up to the innominate). If it does not, it is Stanford B.

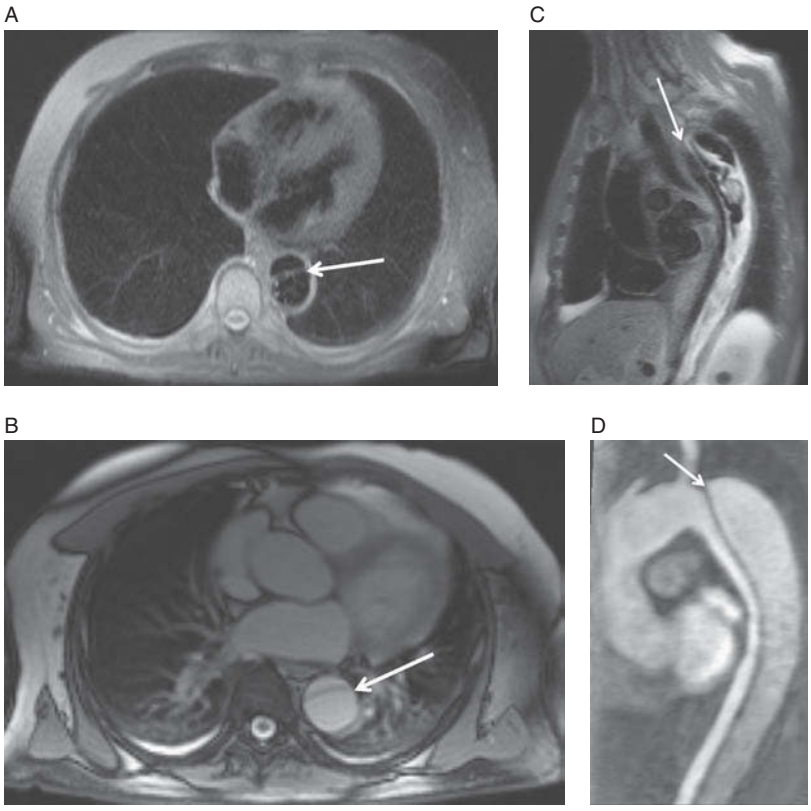

Figure 16.6. Type B aortic dissection. Axial dark-blood image (A) and cervical bright-blood image (B) demonstrate intimal flap within descending thoracic aorta indicative of aortic dissection (arrows). Note the dark true lumen and bright false lumen in sagittal dark-blood image C (arrow). This is due to slow flow within the false lumen. The flap and slow flow within the false lumen are also nicely shown in sagittal T1-weighted gadolinium-enhanced image (D) where the false lumen is not as bright as the true lumen.

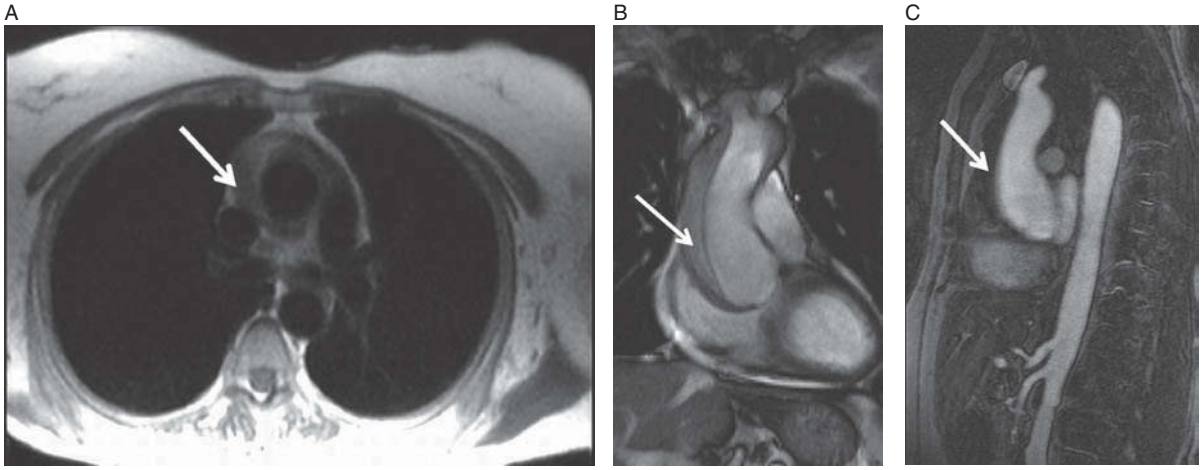

Figure 16.7. Intramural hematoma. Axial dark-blood image (A) and coronal bright-blood image (B) demonstrate bright blood products within the wall of the ascending aorta (arrow); however, no discrete flap is seen. T1-weighted, fat-saturated Post-contrast image (C) shows no enhancement of the wall confirming that there is no communication with the lumen.

Vasculitis

As the name suggests, vasculitis is inflammation of a blood vessel. For practical purposes, there are three types to know about:

(1) Takayasu's arteritis – Involves large vessels including the aorta itself.
(2) Polyarteritis nodosa – Involves medium to small vessels.

(3) Kawasaki's disease – Included here because it is often discussed. May lead to aneurysms of the coronary arteries (Figure 16.8).

On MRA, the key to the diagnosis is to see a thickened vessel wall which will typically enhance following administration of gadolinium. Enhancement is often most conspicuous on delayed phases as the contrast washed out of the vessel lumen "sticks" within the inflammatory response (Figure 16.9).

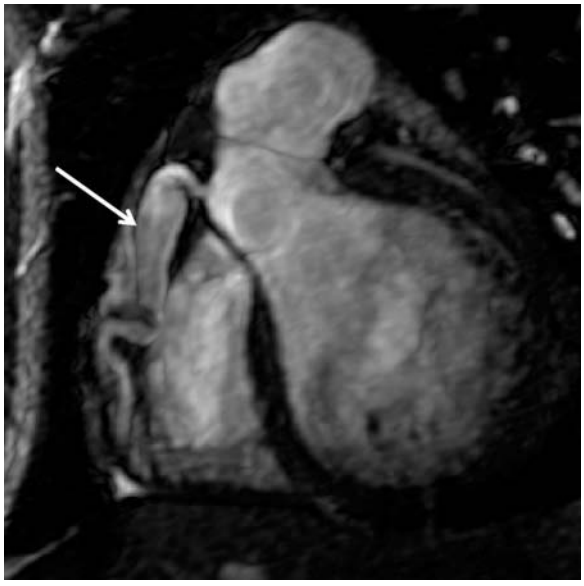

Figure 16.8. Kawasaki's disease. Oblique reformatted image from gadolinium-enhanced MRA demonstrates aneurysm involving a long segment of the right coronary artery.

MRI–MRA uterus protocol

Indications

This protocol is most commonly performed to plan uterine artery embolization.

Preparation

- **IV contrast agent**: 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent**: None
- NPO for 4 hours prior to the exam
- Have the patient void prior to the start of the study
- Give patient 2 L nasal oxygen
- Start IV with at least 24-gauge needle; connect to power injector
- Be sure to subtract coronal pre-contrast images from the post-contrast images
- Cover from iliac crests through symphysis pubis. If pathology extends above or below these levels, increase coverage.

Exam sequences and what we are looking for

(1) Diffusion-weighted imaging b50, 500/ADC – Excellent for lesion detection.

(2) Coronal T2 single-shot fast-spin echo BH (large field of view to cover at least ½ kidneys) – Assess presence and location of kidneys. Evaluate for hydronephrosis.

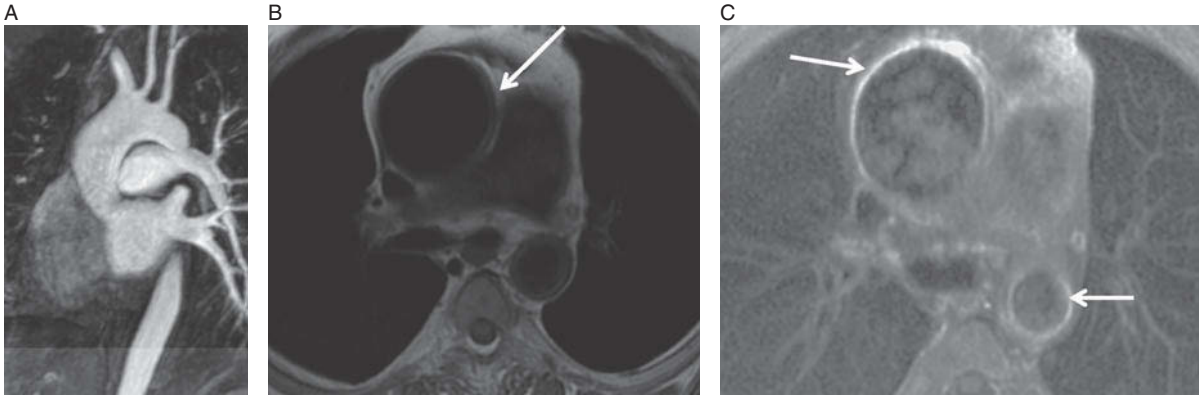

Figure 16.9. Takayasu's arteritis. Sagittal-oblique reformatted image from MRA (A) demonstrates what appears to be a normal aorta, but, remember, this is just the lumen-ogram. Dark-blood image (B) demonstrates aortic wall thickening with enhancement on T1-weighted post-contrast image (C). These cross-sectional images are critical for imaging vasculitis.

(3) Sagittal T2 FSE – Evaluate uterine anatomy. Identify T2-bright and T2-dark lesions.
(4) Axial T2 FSE – Identify T2-bright and T2-dark lesions.
(5) Coronal T2 FSE FS – Identify T2-bright and T2-dark lesions. Evaluate for pelvic fluid and T2-bright osseous lesions.
(6) Axial T1 in and out of phase (IP/OOP) – Identify T1-bright lesions, blood products, microscopic fat.
(7) Coronal volume-interpolated gradient echo BH pre-contrast.
(8) Coronal volume-interpolated gradient echo BH post-contrast at 20 seconds, 1 minute, and 2 minutes.
(9) Axial volume-interpolated gradient echo BH post to follow.

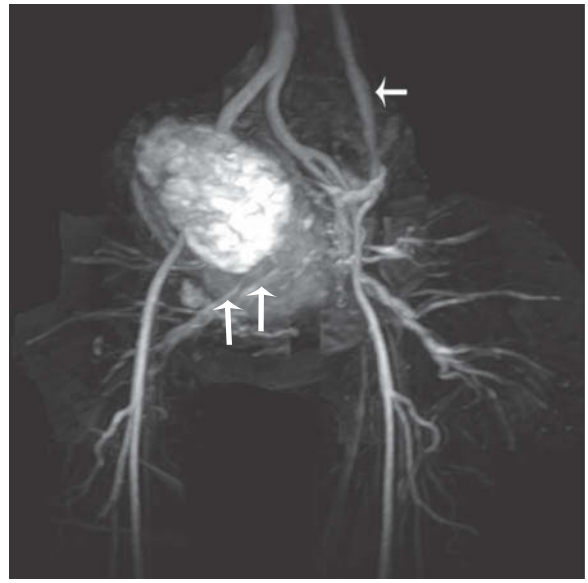

Figure 16.10. Myomatous uterus in a 35-year-old female. Coronal maximum intensity projection from a contrast-enhanced pelvic MRA examination reveals a prominent left ovarian artery (arrow) which is also supplying the enlarged myomatous uterus (double arrows).

An approach to interpretation of pelvic MRA examinations

Pelvic MRA is most commonly performed to evaluate the blood supply to a myomatous uterus prior to uterine artery embolization (Figure 16.10) Occasionally, we are also asked to evaluate for uterine arterio-venous malformations which can be seen as large flow voids (dark) on T2-weighted images with corresponding enhancement on post-gadolinium enhanced images.

Chapter 17

Body MRV

Pelvic MRV protocol

Indications

This protocol is used for evaluating venous flow through the pelvis.

Preparation

- **IV contrast agent:** 1 mmol/kg gadopentetate dimeglumine at 2 cc/s
- **Oral contrast agent:** None
- No IV contrast if the patient is pregnant
- Utilize a superior saturation pulse to saturate arterial flow
- Cover from above aortic bifurcation (approximately L3) through iliacs
- If flow is absent on one or both sides, add repeat axial SSFP in the decubitus position with the affected side up. This is done to determine if the void is due to a compression rather than a blockage.

Exam sequences and what we are looking for

(1) Coronal T2 single-shot fast-spin echo – Evaluate coil placement.
(2) Axial 2D time of flight (TOF) – Cover from aorta bifurcation (approx. L3) through iliacs. Evaluate for filling defects within pelvic veins.
(3) Axial SSFP – Cover from aorta bifurcation (approx. L3) through iliacs. Evaluate for filling defects within pelvic veins.
(4) Coronal SSFP – Evaluate for filling defects within pelvic veins.
(5) Axial T1-weighted volume-interpolated gradient echo BH pre.
(6) Axial T1-weighted volume-interpolated gradient echo BH post IV administration of contrast at 2 minutes.

An approach to interpretation of pelvic MRV exams

This protocol is most commonly used to evaluate the distal IVC and pelvic veins for thrombus in patients with lower-extremity swelling and negative lower-extremity venous Doppler ultrasound.

Sequences

Coronal SSFSE (HASTE): T2-weighted images

Useful primarily for ensuring adequate coil coverage prior to proceeding. Evaluate for soft-tissue edema, flow voids within patent veins.

Time of flight (TOF)

Remember that TOF imaging is based on flow-related enhancement. A saturation band is applied above each slice to eliminate signal from vessels flowing in the opposite direction (arteries). Flow within patent vessels is bright (Figures 17.1 and 17.2).

Multiplanar steady-state free precession

Fast sequence which depicts flowing blood within vessels as high signal intensity ("bright-blood" MRI). Allows for evaluation of the distal IVC and pelvic veins without the use of intravenous contrast material. Faster sequence than TOF. Therefore, if imaging needs to be repeated in one or both decubitus positions, this is the sequence of choice. During pregnancy, it is common for the gravid uterus to compress the distal IVC, and the common and external iliacs, limiting their evaluation. Imaging in the contralateral decubitus position (i.e. left side up if the left pelvic veins are compressed) helps relieve this extrinsic mass effect and improves venous evaluation.

Contrast-enhanced images: T1-weighted volume-interpolated gradient echo

For patients who can receive gadolinium, contrast-enhanced MRI provides the best assessment of venous

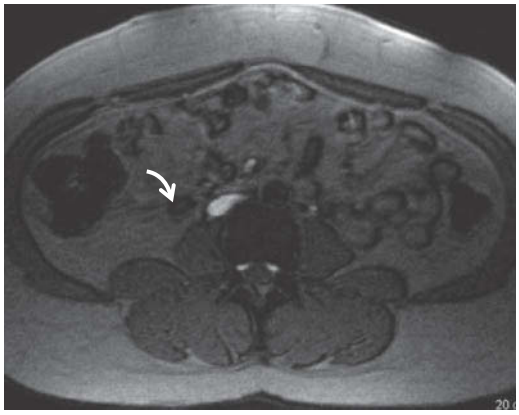

Figure 17.1. Gonadal vein thrombosis. Axial, time-of-flight MRV. Arrow indicates expansion of the right gonadal vein with uniformly dark signal compatible with gonadal vein thrombosis in this recently post-partum patient. Note normal flow within IVC (curved arrow).

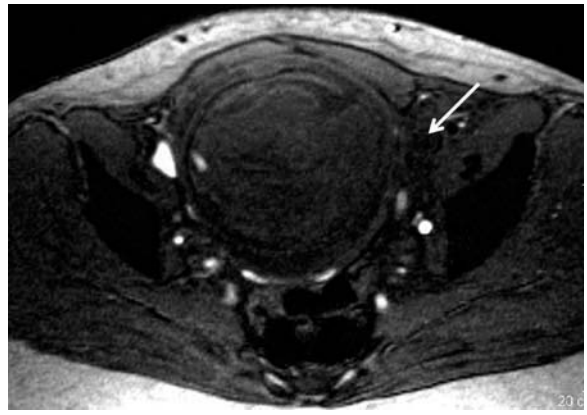

Figure 17.2. Deep venous thrombosis (DVT). Axial, time-of-flight MRV. Arrow denotes expansion and lack of flow-related enhancement within the left external iliac vein compatible with acute DVT (arrow) in this pregnant patient.

patency. Contrast-enhanced MRI eliminates the often confusing flow-related artifacts that are common with the unenhanced SSFP and TOF sequences. Contrast-enhanced MRI is also less prone to flow-related artifacts than contrast-enhanced CT.

Compression of the distal IVC and pelvic veins should be noted as venous compression alone can account for unilateral or bilateral lower-extremity swelling even in the absence of venous thrombus.

Pearls and pitfalls

(1) Atherosclerosis is extremely common, but not all that narrows is atherosclerosis. Don't forget FMD and vasculitis.
(2) Blood within the wall of the aorta (due to dissection or intramural hematoma) will typically have different signal characteristics than blood in the lumen (due to different velocity).
(3) Non-contrast MRA techniques continue to improve, stay tuned . . .

Further reading

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