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Renal Symptoms

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This chapter reviews commonly encountered renal symptoms. The four main points of this chapter are:

1. Computed tomography, performed both without and with contrast material, is the imaging study of choice for hematuria.
2. Computed tomography performed without contrast (CT-KUB) is the initial imaging study of choice for evaluating acute flank pain.
3. CT-angiography, MR-angiography, and nuclear medicine studies may be used for evaluating suspected renovascular hypertension.
4. Ultrasound is the study of choice for evaluating new onset renal failure.

COMPUTED TOMOGRAPHY IS THE IMAGING STUDY OF CHOICE FOR HEMATURIA

Computed tomography (CT), performed both without and with contrast material, is the imaging study of choice for the evaluation of hematuria[1]. In addition to CT, patients will also need to undergo cystoscopy, because mucosal bladder processes invisible on CT frequently cause bleeding. Prior to discussing and illustrating CT, however, note that evaluation of hematuria generally follows an algorithm (for example, see the American Academy of Family Physician's web page[2]) that specifically *excludes* many patients with hematuria from imaging and cystoscopy.

The algorithms exclude many patients from imaging and cystoscopy because hematuria is a common problem which is frequently transient. Froom et al[3] found microscopic hematuria on at least one occasion in 39% of 1,000 young men who had annual urinalyses between the ages of 18 and 33; 16% had hematuria on two or more occasions. In a different study, Mohr et al[4] found hematuria in 13% of men and postmenopausal women.

Most of the algorithms in evaluation of hematuria sequentially identify and exclude those who do not need imaging, typically those with exercise induced hematuria, bleeding associated with urinary tract infections, medication induced hematuria, hematuria associated with dehydration, myocardial infarction, or hypertension, and glomerular hypertension.

A variety of sports (both contact and noncontact) may result in exercise induced hematuria, with the degree of hematuria related to the intensity and duration of exercise[5]. Exercise induced hematuria is typically microscopic, asymptomatic, and almost always resolves within 72 hours, but if hematuria persists, further evaluation may be necessary.

Urinary tract infections may be asymptomatic and still cause hematuria. Most algorithms call for clearing a urinary tract infection and retesting for hematuria before proceeding with imaging.

Multiple medications cause hematuria by a variety of processes[5], including interstitial nephritis (e.g. captopril, furosemide, NSAIDs) papillary necrosis (e.g. aspirin, NSAIDs), hemorrhagic cystitis

(e.g. Cytoxan, Ifosfamide (Ifex), Mitotane (Lysodren)), and urolithiasis (e.g. indinavir, and NSAIDs once more). One notable exception to medications associated with hematuria: anticoagulation does *not* offer an adequate explanation for hematuria, and these patients typically need to be worked up[1].

In addition to infection and drugs, hematuria has also been related to a number of additional conditions, including dehydration, myocardial infarction, atrial fibrillation, and hypertension. Presence of any of these conditions may explain hematuria, and retesting for hematuria after treating the underlying condition should probably precede imaging.

Finally, patients with glomerular hematuria, diagnosed by the presence of red cell casts in the urine, do not need to undergo imaging, but likely need to be referred to a nephrologist[1]. Additional signs of glomerular hematuria include protein excretion exceeding 500 mg/day when there is no bleeding, a dysmorphic appearance of most red cells, and brown, cola-colored urine with gross hematuria[1].

After the exclusion of hematuria caused by exercise, infection, medications, other known medical conditions, and glomerular hematuria, further work-up should diagnose causative lesions of the kidneys, collecting system, and bladder. The likelihood of finding (or not finding) abnormalities has been studied several times, including a large study of 4,023 patients summarized in this table:

Cause	Microhematuria 1,950 patients	Macrohematuria 2,073 patients
Calculi	7.8% (153)	8.8% (183)
Renal cell cancer	1.0% (19)	2.0% (41)
Upper tract TCC	0.2% (3)	0.5% (10)
Bladder TCC	3.7% (73)	16.5% (342)
No cause identified	87.3% (1702)	72.2% (1497)

Table. Causes of hematuria. From: Edwards TJ, Dickinson AJ, Natale S et al. A prospective analysis of the diagnostic yield resulting from the attendance of 4,023 patients at a protocol-driven hematuria clinic. BJU Int 2006; 97:301-305. TCC = transitional cell carcinoma.

Given the diseases listed in the table, the evaluation of hematuria should target calculi and tumors of the kidneys and collecting system (including the bladder). As noted above, algorithms typically call for both CT, to evaluate for stone disease and renal and upper tract tumors, and cystoscopy, to evaluate for bladder mucosal lesions not visible with CT. CT is a good test, because it not only detects renal calculi which cause hematuria, but it also detects renal cell cancer (and other parenchymal tumors) as well as rare causes of hematuria.

With respect to renal stone disease, note that patients may have hematuria, not have flank pain, and still have calculi as the cause of their disease (Figure 1). The widespread use of CT in the evaluation of patients with hematuria has resulted in a greater recognition of this situation[6]. Also note that a combined unenhanced/enhanced study is necessary to study patients with hematuria, because the contrast material may obscure nonobstructing stones (Figure 2).

One of the main reasons to perform imaging in patients with hematuria is to discover otherwise asymptomatic renal cell cancer (Figure 3). Renal cell cancer, seen more frequently with increasing age, usually manifests as an exophytic contrast enhancing mass. Such lesions are usually not biopsied, but taken directly to surgery where the urologist will typically remove at least a portion of the kidney, and often the entire kidney. In elderly patients or those with multiple medical problems who cannot tolerate surgery, percutaneous thermal ablation forms an alternative therapy.

In some instances, CT may allow a specific histologic diagnosis of a renal tumor other than renal cell cancer. The presence of at least some fat in the lesion indicates an angiomyolipoma (Figure 4), a lesion that does not need to be biopsied. Some authorities advocate removing angiomyolipomas larger than 4 cm on the grounds that they may hemorrhage.

CT allows detection of several additional, rare causes of hematuria including a retroaortic renal vein, renal arteriovenous malformations, and renal artery aneurysms[7].

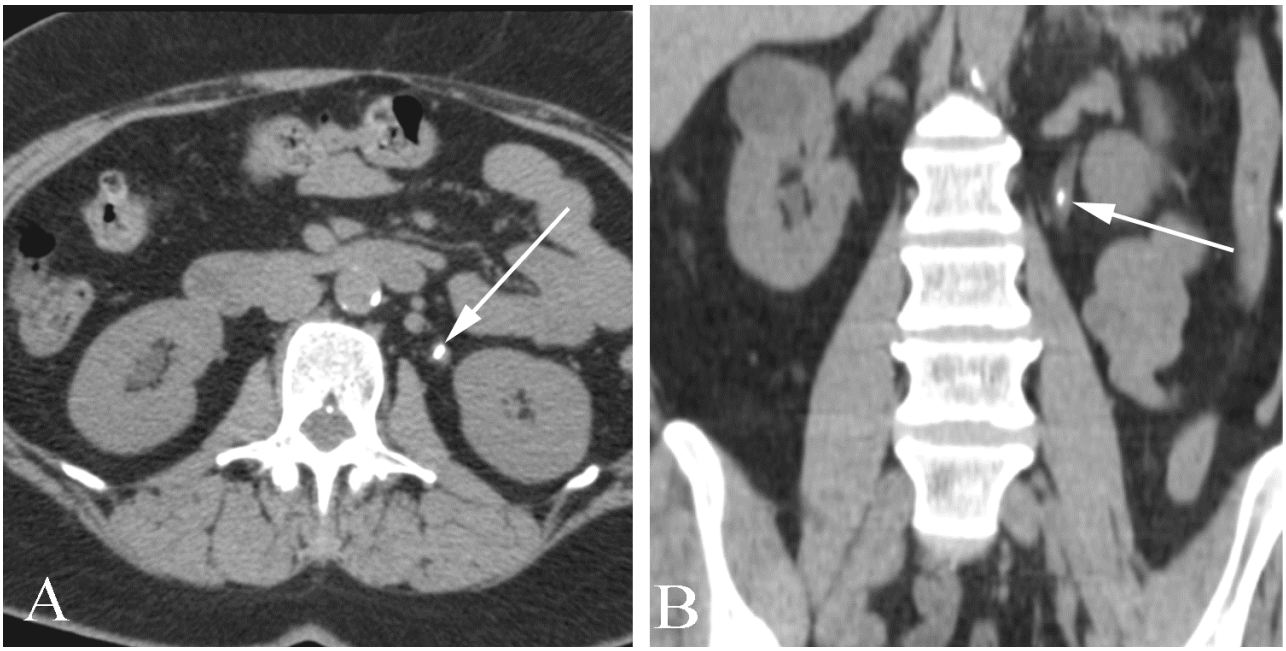

Figure 1. Renal stone in a 51 year old woman with asymptomatic hematuria. A. Noncontrast axial CT study shows a stone (arrow) at the left ureteropelvic junction. B. Noncontrast reformatted coronal CT (right) also demonstrates the stone (arrow). Incidentally noted is a cyst of the contralateral kidney.

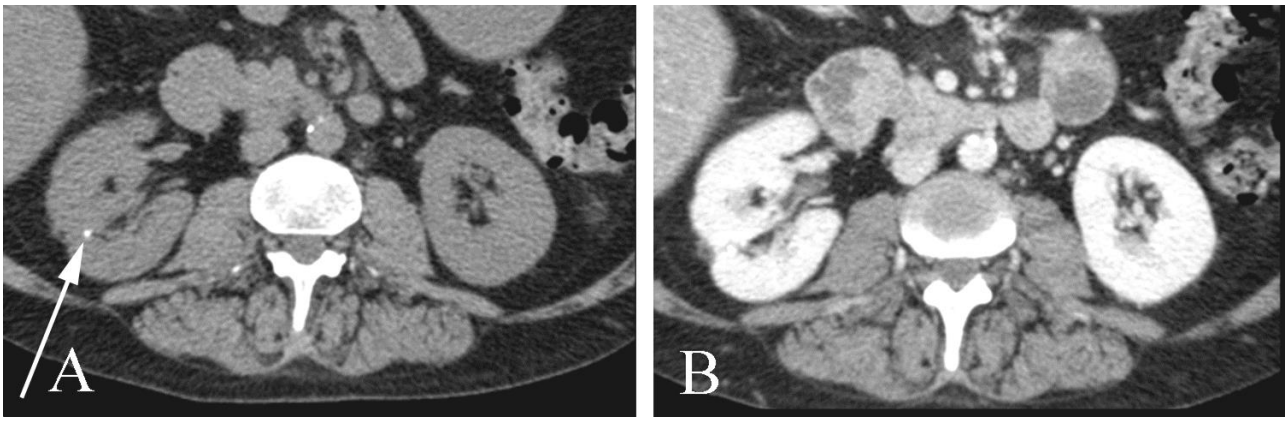

Figure 2. Renal stone associated in a 50 year old woman with hematuria. A. Precontrast (unenhanced) CT clearly shows a nonobstructing right renal stone (arrow). B. On the postcontrast CT, contrast material largely obscures the stone. If only the postcontrast CT had been performed, the stone would be very difficult (if not impossible) to recognize.

CT also demonstrates transitional cell tumors of the renal collecting system, including within the renal pelvis, within the ureter (figure 5), and within the bladder (Figure 6). Note that not all bladder lesions will be visible on CT, which is why CT *and* cystoscopy are recommended in the evaluation of patients with hematuria.

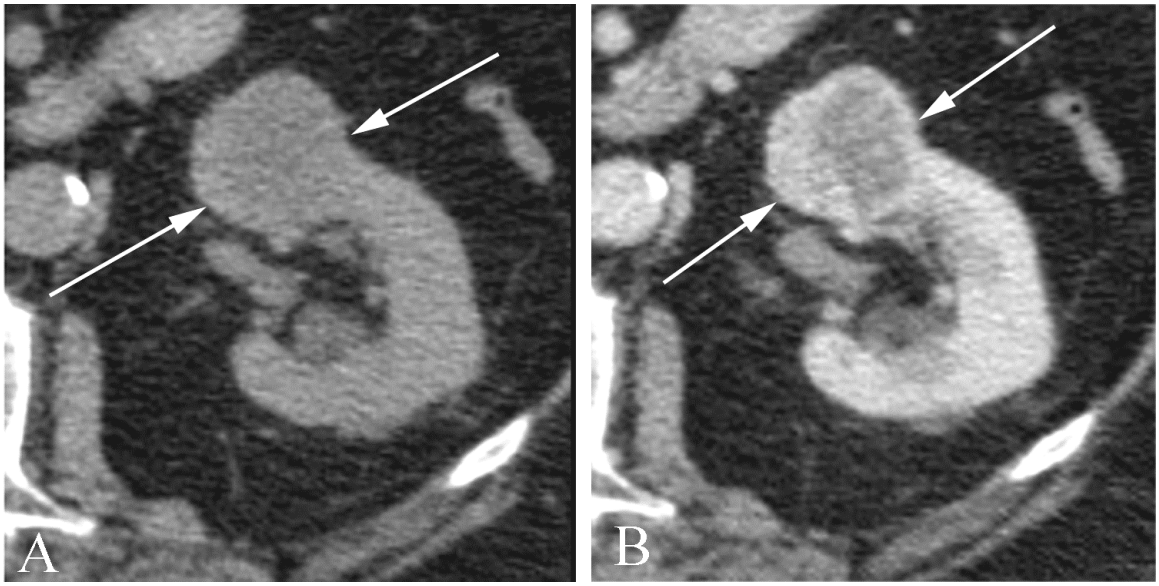

Figure 3. Renal cancer in a 76 year old man with hematuria. A. Unenhanced CT shows an exophytic mass (arrows) of the left kidney. B. Enhanced CT shows heterogeneous enhancement of the mass (arrow). Renal cell cancer was found at surgery.

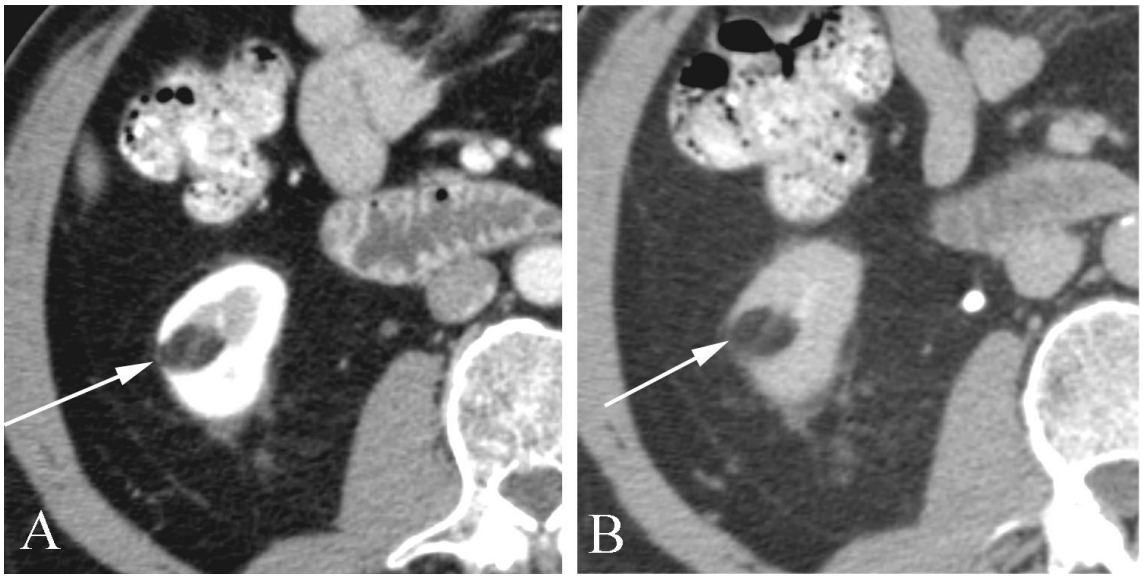

Figure 4. Renal angiomyolipoma in a 73 year old man with unrelated abdominal pain. A. Early, nephrographic phase CT shows a circumscribed, fat density mass of the right kidney (arrow), demonstrating the same density as the perinephric fat. B. Delayed examination shows the same lesion (arrow), which again demonstrates fat density. This lesion was unchanged five years later.

In the evaluation of hematuria, CT is superior to other methods of imaging. While, historically, intravenous pylograms (IVPs) were the imaging study of choice for evaluation of hematuria, these studies are virtually never performed when CT is available. Gray-Sears et al[8] found that CT had a sensitivity of 100% and a specificity of 97% in the evaluation of renal tumors, versus a sensitivity of 61% and a specificity of 91% for IVPs. Regarding ultrasound for evaluation of renal tumors, Warshauer et al[9] found US was only 60% sensitive.

Note, however, that CT should not be used in pregnant patients with hematuria. Ultrasound is the study of choice in these patients (Figure 7). If the ultrasound is normal, it is usually reasonable to wait until the pregnancy is completed and then, if hematuria persists, perform a CT at that time.

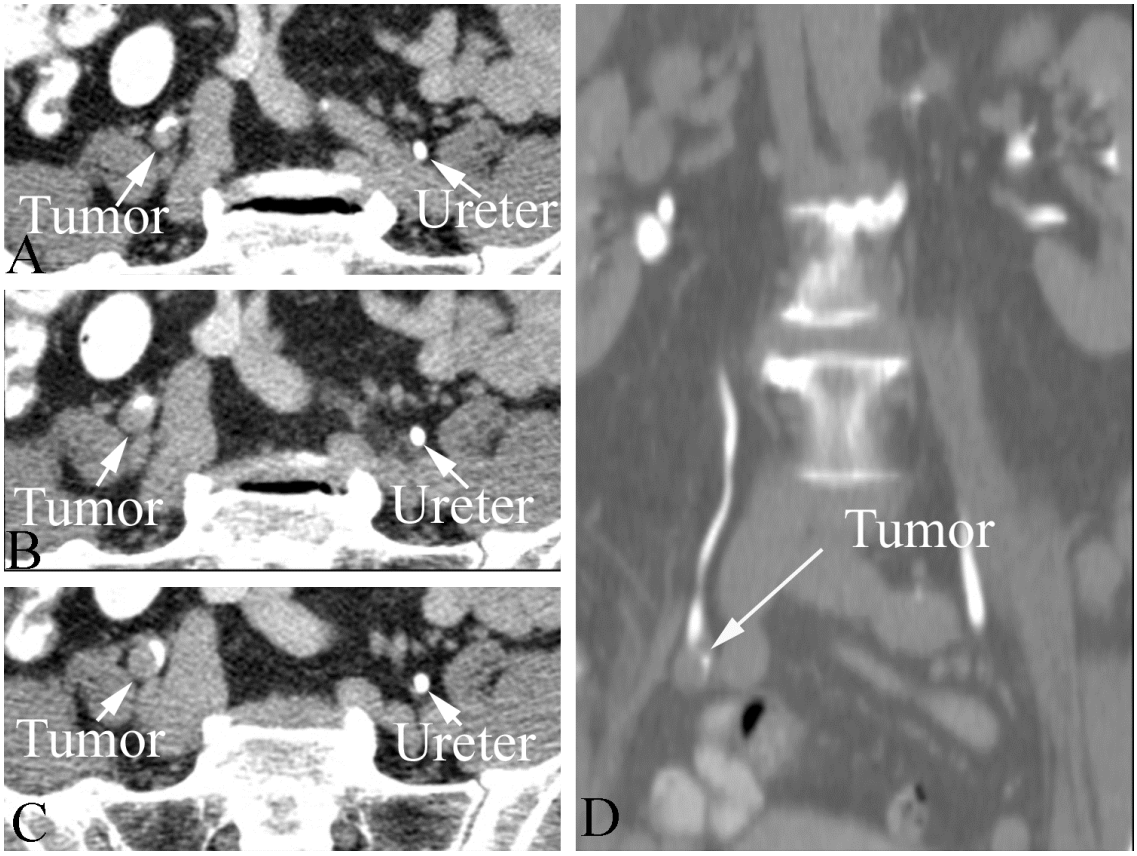

Figure 5. Transitional cell carcinoma of the ureter in a 78 year old man with hematuria. A, B, and C. Sequential axial delayed CT studies show a normal left ureter (in cross section, a small white circle) and an abnormal right ureter with a tumor distorting the ureter, which is peripherally displaced along the tumor margin. D. Delayed reformatted coronal CT shows the tumor (arrow) along the lower margin of the visualized right ureter.

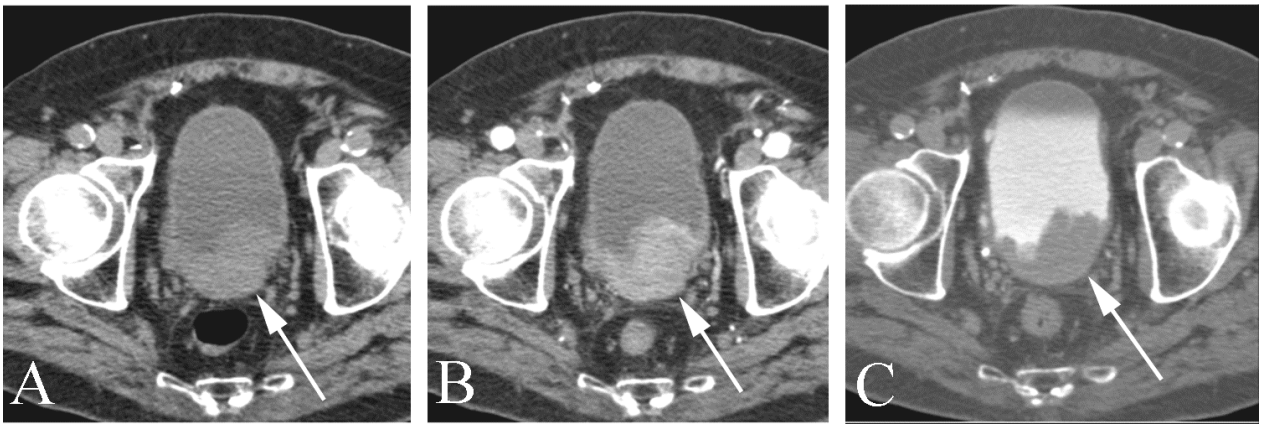

Figure 6. Transitional cell cancer of the bladder in a 79 year old man with hematuria. A. Unenhanced CT shows a subtle filling defect within the bladder (arrow). B. The portal venous phase image (taken two minutes following contrast injection) shows a contrast-enhancing lesion (arrow). C. On the delayed image (taken 10 minutes after contrast injection) the filling defect (arrow) is much more conspicuous against the background of the opacified urine. Less dense, unopacified urine is seen along the anterior bladder.

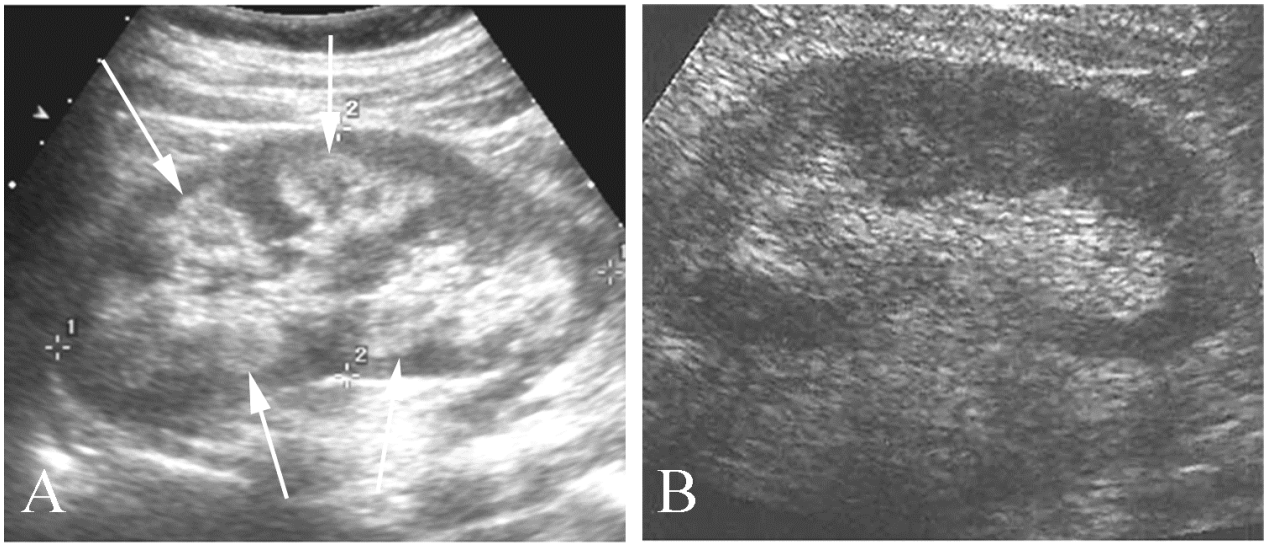

Figure 7. Medullary sponge kidney in a 30 year old pregnant woman with hematuria. A. Renal ultrasound of the patient shows marked increase in renal echogenicity extending through the renal cortex (arrows), indicating medullary sponge kidney. B. A normal kidney for comparison. Note the increased echoes are confined to the central portion of the kidney in the renal pelvis.

CT-KUB IS THE STUDY OF CHOICE FOR EVALUATING ACUTE FLANK PAIN

Decades ago, plain films of the abdomen showed stones of the "Kidneys, Ureter, and Bladder". Those films were therefore called "KUB" studies. The analogous unenhanced CT of the abdomen and pelvis has come to be called the "CT-KUB". In patients with a clinical history suspicious for renal stone disease, the CT-KUB has supplanted all other imaging modalities. CT-KUB allows detection not only of stone disease, but also other causes of abdominal or flank pain. Ha and MacDonald[10] found significant alternate pathology in 33% of patients with suspected nephrolithiasis. Overlap exists between this and the prior indication, as patients with stone disease (and other genitourinary diseases) may have either hematuria, flank pain or both. If the CT-KUB does not demonstrate a cause of flank pain, it makes sense to consider adding contrast-enhanced images to the exam.

Typical algorithms for evaluation of renal colic recommend CT-KUB, with the exception of pregnant patients (evaluated with ultrasound) and patients with known renal stone disease where prior plain films have documented the stones[11]. Certain indications, such as patients with urosepsis, acute renal failure, anuria, and/or unyielding pain, nausea, or vomiting, require urgent urologic consultation. The reason these algorithms support CT-KUB is that CT-KUB can demonstrate and characterize renal stones (Figure 8) as well as alternative causes of pain. Portis et al[6] found that stones less than 5 mm in diameter had a significantly higher likelihood of passage than stones 5 mm or greater, and that stones that had made it to the distal ureter by the time of diagnosis were more likely to pass without intervention than those discovered in the proximal ureter (74% versus 53%). This work was done in 1991 and was based on plain film or IVP data. Coll et al[12], using CT data, found a similar relationship between the likelihood of stone passage and stone size: 87% of stones 1 mm or less passed, 76% of stones 2 to 4 mm passed, 60% of 5 to 7 mm stones passed, 48% of 7 to 9 mm stones passed, and only 25% of stones > 9 mm passed. These findings support the general maxim that small stones will often pass, but large stones usually don't. CT-KUB is an excellent method of locating and sizing all stones. Furthermore, it can demonstrate which stones cause hydronephrosis. Renal distension, perinephric stranding (Figure 8) and hydronephrosis (Figure 9) indicate that the stone is obstructing the collecting system and likely symptomatic[6].

CT-KUB also allows diagnosis of any of several alternative causes in patients with acute flank pain with or without hematuria, including dissecting aortic aneurysm (see page 174), diverticulitis (Figure 10; see also pages 95-96), and appendicitis (see pages 94-95).

CT-KUB is superior to other methods of evaluation in patients with acute renal pain. When directly compared with plain films, CT-KUB shows greater sensitivity (95% - 100% versus 45% - 59%) and specificity (94% - 96% versus 71 – 77%)[6].

Plain films may represent a reasonable imaging study in patients who have a history of radio-opaque calculi (that may be seen on plain films) and acute flank pain that is similar to previous episodes. A CT-KUB will still likely be necessary if the plain film does not completely elucidate the size and location of the obstructing calculus[11].

CT-KUB has also supplanted IVPs in the evaluation of stone disease. Compared to IVPs, CT-KUB shows greater sensitivity (95 - 100% versus 64 – 87%) and specificity (94 - 96% versus 92 – 94%)[6]. Smith et[13] al studied 20 patients with acute flank pain, 12 of whom had stone disease, CT detected 11 and IVP 5. In addition to inferior diagnostic performance, IVPs require contrast injection, take longer to perform, and involve greater radiation exposure.

As in the case of plain films and IVP, CT-KUB outperforms US, which has a sensitivity of only 19% and a specificity of 97%[6]. As noted above, US is the study of choice in pregnant women (Figure 7).

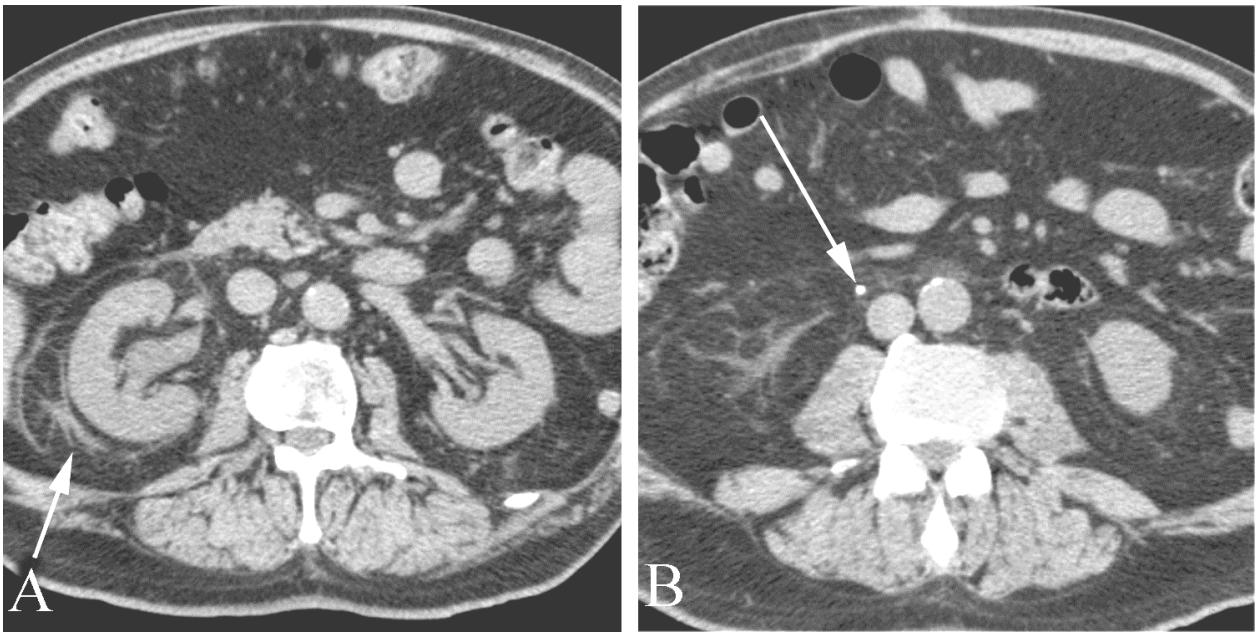

Figure 8. Renal stone disease in an 82 year old man with acute right flank pain. A. CT-KUB at the level of the kidneys shows perinephric stranding (arrow). B. CT-KUB at the level of the proximal ureter shows a calcified stone (arrow).

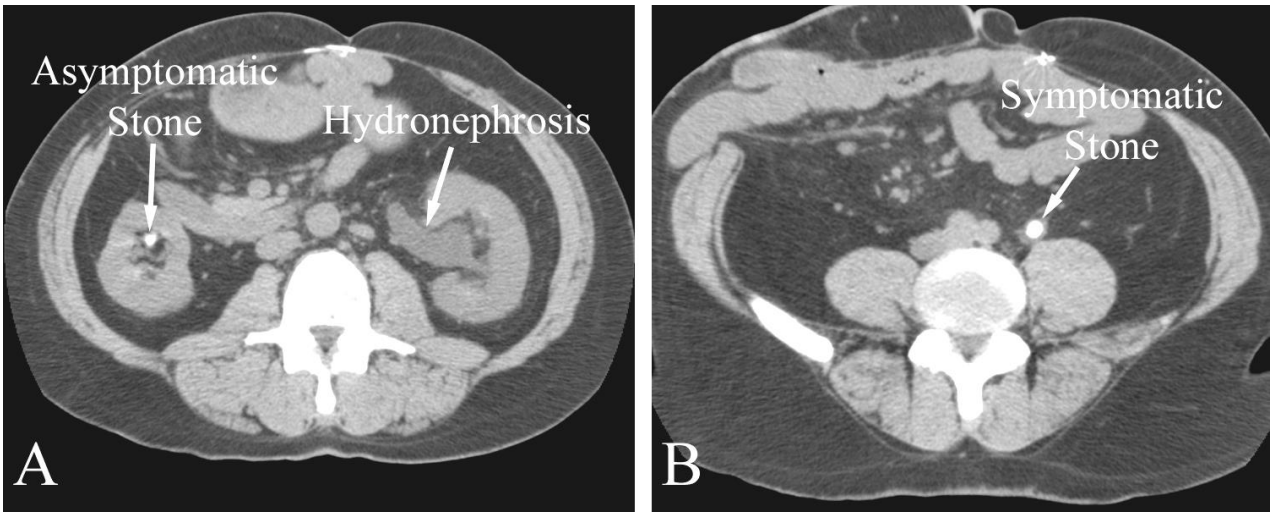

Figure 9. Renal stone disease and hydronephrosis in a 62 year old man with known stone disease and new onset left flank pain. A. CT-KUB at the level of the left collecting system shows hydronephrosis. Note contralateral, nonobstructing calculus. B. CT-KUB at the level of the uteropelvic junction shows an obstructing renal stone.

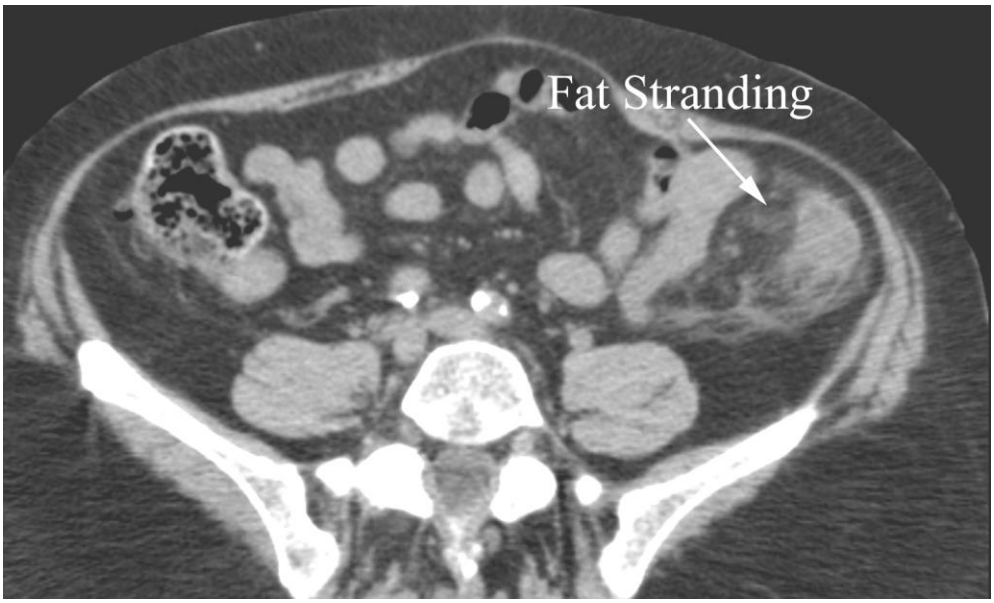

Figure 10. Diverticulitis in a 73 year old woman with flank pain and hematuria. CT-KUB shows typical fat stranding along the descending colon diagnostic of diverticulitis.

CT-ANGIOGRAPHY, MR-ANGIOGRAPHY, AND NUCLEAR MEDICINE STUDIES MAY BE USED FOR EVALUATING SUSPECTED RENOVASCULAR HYPERTENSION

Most cases of hypertension either have an obvious cause or are idiopathic, and respond to appropriate treatment. Imaging should be performed when there is a suspicion of renal artery stenosis, which arises in the following clinical scenarios:

1. Severe or refractory hypertension (e.g., not controlled with three drug regimen).
2. An acute rise in blood pressure over a previously stable value.
3. Proven age of onset before puberty.
4. Age less than 30 years in non-obese patients with a confirmed negative family history of hypertension[14].

CT angiography

CT angiography demonstrates the abdominal arterial tree including the renal arteries, and allows diagnosis of renal artery stenosis (Figure 12). It requires IV contrast material and therefore elevated creatinine or decreased creatinine clearance is a relative contraindication to the study (see pages 251-252).

MR angiography

MR angiography also demonstrates the abdominal arterial tree including the renal arteries, and allows diagnosis of renal artery stenosis. It may be done either with or without gadolinium containing IV contrast, although studies done without IV contrast are technically more demanding. The contrast given for magnetic resonance imaging was once thought to be considerably safer than the contrast material given for CT studies, and at one point MR angiography was the study of choice in patients with renal insufficiency. The discovery that MR contrast material may provoke nephrogenic systemic fibrosis in patients with renal insufficiency, however, has all but eliminated use of such contrast in these patients (see page 254). Nephrogenic systemic fibrosis is an uncommon side effect of MR contrast, which is occasionally fatal.

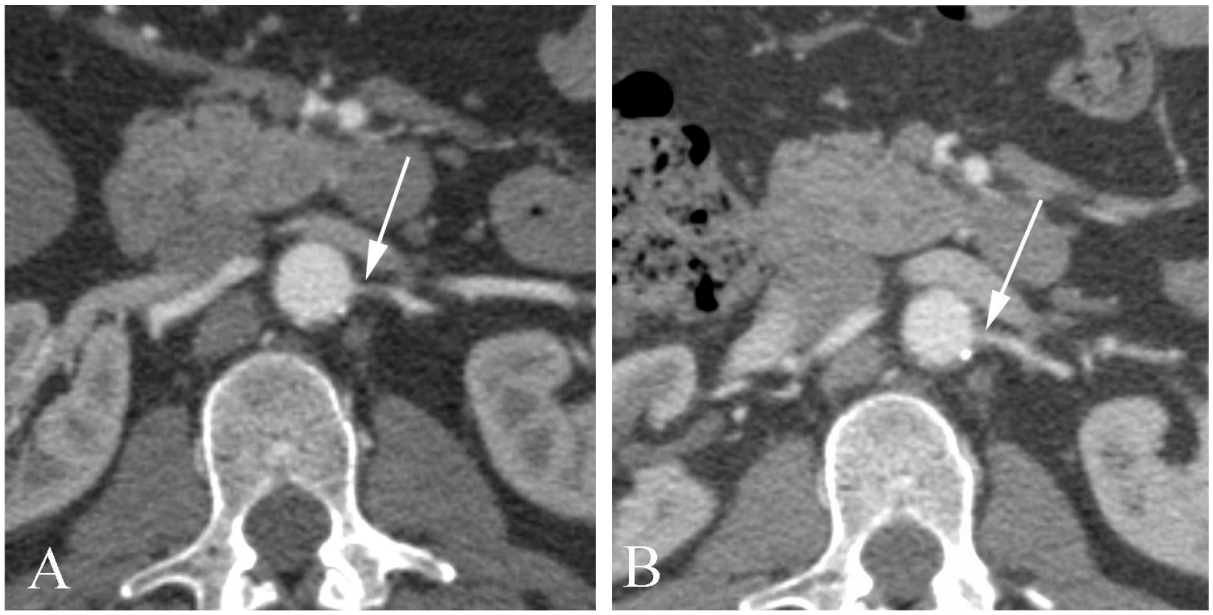

Figure 12. Renal artery stenosis treated with angioplasty in a 54 year old with hypertension. A. Initial CT angiogram (left) shows stenosis of the left renal artery (arrow). B. Follow-up CT following angioplasty shows decreased stenosis (arrow). The patient's hypertension was under better control with fewer medications following angioplasty.

Renal scintigraphy

Another method to screen patients with renal insufficiency and possible renovascular hypertension is with a nuclear medicine study. In these studies, an intravenous radiotracer (Tc-99m DTPA) is injected followed by scans of both kidneys. This usually yields a normal study with bilateral, symmetric function of the kidneys resulting in normal time-activity curves: the amount of radioactivity shows an early spike and then later declines. The test is then repeated with an angiotensin-I converting enzyme (ACE) inhibitor, typically 25 mg of oral captopril. The abnormal kidney (downstream from the level of renal artery stenosis) will show much less activity and a flattened peak on the time-activity curve compared to the normal kidney[15].

ULTRASOUND IS THE STUDY OF CHOICE FOR EVALUATING NEW ONSET RENAL FAILURE

Patients with renal failure may present with decreased urine, flank pain, edema, hypertension, weakness and fatigue, anorexia, vomiting, mental status changes, or fever, but many patients are asymptomatic and are simply found on a screening study to have an elevated plasma creatinine or an abnormal urinalysis (such as microscopic hematuria or proteinuria)[16]. Since obstruction of renal outflow is a treatable disease, and since ultrasound will make this diagnosis, these patients should undergo an ultrasound study. Of course, these patients by definition have renal insufficiency, a relative contraindication to contrast-enhanced CT and MRI (see page 252). Ultrasound is fast, relatively cheap, and involves no ionizing radiation or contrast material, all great advantages compared to other methods of evaluating acute renal insufficiency.

Renal obstruction will be accompanied by distention of the renal collecting system. Most frequently, this is secondary to either prostatic hypertrophy or tumors of the pelvis. These patients need referral to urology for drainage with expected return of renal function.

Ultrasound examination of patients with any of several "medical renal diseases" (for example, chronic glomerulonephritis), may show small, echogenic kidneys consistent with chronic disease, with the ultrasound appearance secondary to loss of renal parenchyma and increased interstitial fibrosis[16]. However, the ultrasound study may also be normal in chronic disease, and these patients typically require referral to a nephrologist for possible biopsy and diagnosis, with appropriate treatment and counseling.

Polycystic kidney disease may present with renal failure in an adult, and in these cases the ultrasound is usually diagnostic, demonstrating enlarged kidneys with multiple cysts (Figure 11).

SUMMARY

Imaging often plays a critical role in the evaluation of renal symptoms. In patients with hematuria, combined unenhanced/enhanced CT allows diagnosis of such common causes as renal stones, renal cell cancer and transitional cell carcinoma of the collecting system, ureters, and bladder. In patients with flank pain suspected to be of renal origin, CT-KUB can evaluate the size and position of obstructing stones as well as diagnose alternative causes of flank pain. In patients with suspected renovascular hypertension, CTA, MRI, and renal scintigraphy all offer noninvasive methods of screening for renal artery stenosis. In patients with acute renal failure US allows a rapid diagnosis of obstruction.

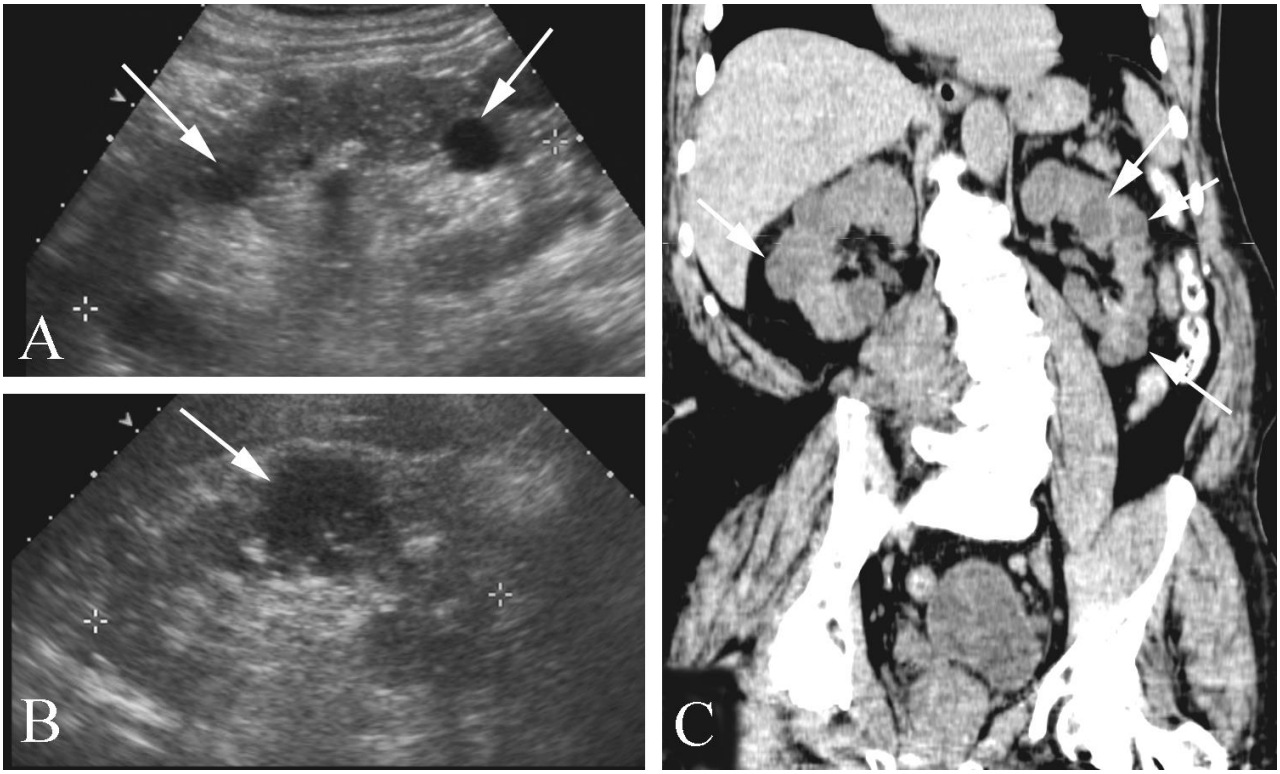

Figure 11. Polycystic kidney disease in a 63 year old with hematuria. A. Ultrasound study of the left kidney shows two cysts (arrows). B. Ultrasound study of the right kidney shows an additional cyst (arrow). C. Coronal reformatted image from a CT-KUB shows multiple variably sized cysts (arrows).

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Female Pelvis and Male Scrotum

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This chapter reviews the current recommendations for imaging the following commonly encountered clinical indications: women with abnormal uterine bleeding (either pre- or postmenopausal), women with pelvic pain, women with a pelvic mass, men with scrotal pain, and men with a scrotal mass. The main points of the chapter are:

1. Pelvic ultrasound is the imaging study of choice for the evaluation of abnormal uterine bleeding.
2. Pelvic ultrasound is the imaging study of choice for the evaluation of female pelvic pain and masses.
3. Scrotal ultrasound is the study of choice for evaluation of scrotal pain and masses.

The remainder of the chapter discusses the clinical indications, the disease processes leading to the clinical indications, and the rationale behind ordering the studies. The chapter also illustrates several of the causative diseases.

Before proceeding, a few words about ultrasound technology. A detailed -or even a superficial- discussion of the physics of ultrasound is beyond the scope of this chapter. It is of interest, however, that the technology of ultrasound (like CT and MR) continues to advance, with developments allowing prettier, and, of course, more diagnostic scans. These developments include processing software such as harmonic imaging, and hardware such as the development of smaller and smaller probes allowing, for example, endoscopic ultrasound. A technical development which may have a large impact on the practical, day-to-day business in the radiology department is the sector probe with associated software. This technology captures a volume of data. With the current widely deployed technology, the technologist performing the ultrasound study tries to align the two dimensional plane of the ultrasound beam in the exact orientation to optimally demonstrate the target anatomy. With three dimensional scanning, the ultrasound machine acquires a volume of data which may be manipulated on a workstation. This technology, if widely implemented, has the potential to substantially reduce patient scan time and improve patient service.

Regardless of the technology of ultrasound, it makes sense that patients are (at least somewhat) mentally prepared for the ultrasound exam. Female patients should be advised that, for most women, pelvic ultrasound examination consists of two parts. The first part is a transabdominal scan, performed with the scan probe on the abdominal wall just above the symphysis pubis, which requires a full bladder. The pressure of the probe against a full bladder is not typically painful but may be mildly uncomfortable. If the patient arrives in the department without a full bladder, it is usually necessary to ask the patient to drink a large volume of water and to wait until the bladder is full before proceeding, so if you send a patient for an immediate pelvic ultrasound make sure the patient does *not* stop at the restroom on the way to the radiology department. The second part of the female pelvic ultrasound examination is the

endovaginal (also known as transvaginal) exam, which involves the use of a specially designed probe which is placed in the vagina. This technology, obviously not used in women who have never been sexually active nor in those who do not consent to the procedure, allows much better evaluation of the interior of the uterus (particularly the endometrial stripe) and the adnexae. It is best that women know about this aspect of the exam beforehand so that they can be mentally prepared at the time of the examination.

Another method of uterine evaluation using sonography is the sonohysterogram. In this study, the cervix is cannulated with a catheter and water is instilled into the uterine cavity. This has the effect of demonstrating the endometrial lining with much greater clarity than studies done without the instilled water, particularly with respect to differentiation of focal versus diffuse endometrial lesions. Sonohysterography may be helpful on occasion, but many gynecologists forego its use. These gynecologists reason that if a relatively interventional study is required anyway, hysteroscopy allows not only direct inspection of the endometrial canal, but biopsy of any abnormality to determine its histology. Histologic characterization is generally necessary for definitive treatment.

Finally, note that in addition to "gray-scale" images, ultrasound studies may incorporate color (color Doppler examination) or graphs (with spectral Doppler examination) to depict flow.

PELVIC ULTRASOUND IS THE IMAGING STUDY OF CHOICE FOR THE EVALUATION OF ABNORMAL UTERINE BLEEDING

Pelvic ultrasound is the study of choice for both abnormal premenopausal and postmenopausal uterine bleeding, but the causes of bleeding in these two scenarios differ and therefore they will be discussed separately.

Postmenopausal bleeding

Postmenopausal bleeding may have any one of many causes (Table 1), and, while most are benign and self limiting, endometrial cancer accounts for approximately 10% of such cases[1]. Either endometrial biopsy or pelvic ultrasound may be used as the initial test for evaluation of the endometrium in women with postmenopausal bleeding, and often both tests will be used (see below). While the ultrasound examination will not provide an unequivocal histologic diagnosis, it is often helpful in directing further work-up. This section reviews the ultrasound results in the most common causes of uterine bleeding. Most of these processes will manifest with an abnormal, thickened endometrial stripe. The normal postmenopausal endometrial stripe measures less than 5 mm. Note that while the thickness of the endometrium is important, the stripe's appearance is also critical: the endometrial stripe should demonstrate uniform thickness and should be uniformly hyperechoic relative to the adjacent uterus[2].

Cause	%
Atrophy	59%
Polyps	12%
Endometrial cancer	10%
Endometrial hyperplasia	10%
Hormonal effect	7%
Cervical cancer	<1%

Table 1. Causes of postmenopausal bleeding in 1,139 patients. From: Karlsson B, Granberg S, Wikland M et al. Transvaginal ultrasonography of the endometrium in women with postmenopausal bleeding – a Nordic Multicenter study. Am J Obstet Gynecol 1995; 172:1488-1494.

Atrophy

Postmenopausal lack of estrogen causes atrophy of both the endometrium and vagina. Endometrial atrophy and the associated lack of lubricating fluid erode the endometrial lining[1]. Erosions of the atrophic endometrium may bleed. The ultrasound study in these patients may show either a normal or a thin endometrial stripe. The ultrasound study may also show blood (visualized as fluid) within the endometrial canal.

Endometrial polyps

Endometrial polyps are benign endometrial growths seen more frequently in women given

estrogen or the breast cancer drug tamoxifen[1]. Polyps may cause either diffuse or focal thickening of the endometrial stripe, and in this regard the transvaginal study is more accurate not only in measuring the exact stripe thickness but also in differentiating whether the stripe is diffusely or focally abnormal. Note that endometrial polyps may also cause bleeding in premenopausal patients (see below, Figure 2).

Endometrial hyperplasia

Endometrial hyperplasia may produce a thick stripe on pelvic ultrasound. This thick stripe cannot be distinguished from the thick stripe caused by endometrial cancer, so these women will typically undergo biopsy. Note that since postmenopausal women should be estrogen deficient, endometrial hyperplasia is abnormal; causes include endogenous estrogen production from ovarian or adrenal tumors or exogenous estrogen therapy[1].

Endometrial cancer

While approximately 90% of patients with postmenopausal bleeding will eventually be found to have a benign cause, and while many patients with a thick endometrial stripe may have a benign cause (such as hypertrophy or an endometrial polyp) for this finding, the combination of postmenopausal bleeding and a thick endometrial stripe needs to be regarded with great suspicion. Almost all these patients require biopsy, and on occasion, re-biopsy, for evaluation (Figure 1).

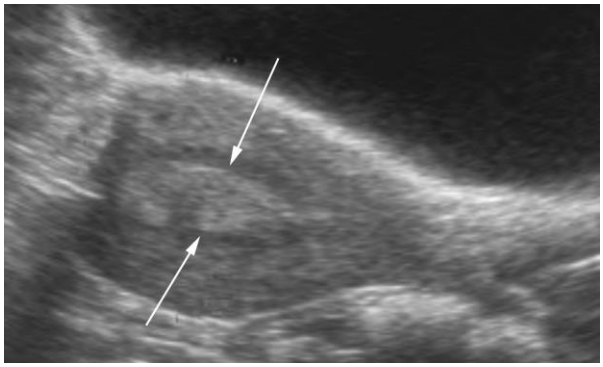

Figure 1. Endometrial cancer in a 60 year old woman with postmenopausal bleeding. This patient had a single episode of bright red blood followed by spotting. Endometrial biopsy resulted in a benign polyp and no malignancy. The ultrasound demonstrated a thick endometrial stripe, after which the patient had hysteroscopy and a D&C, with a diagnosis of endometrial cancer. This is an example of where the addition of an ultrasound study to endometrial biopsy resulted in improved patient management.

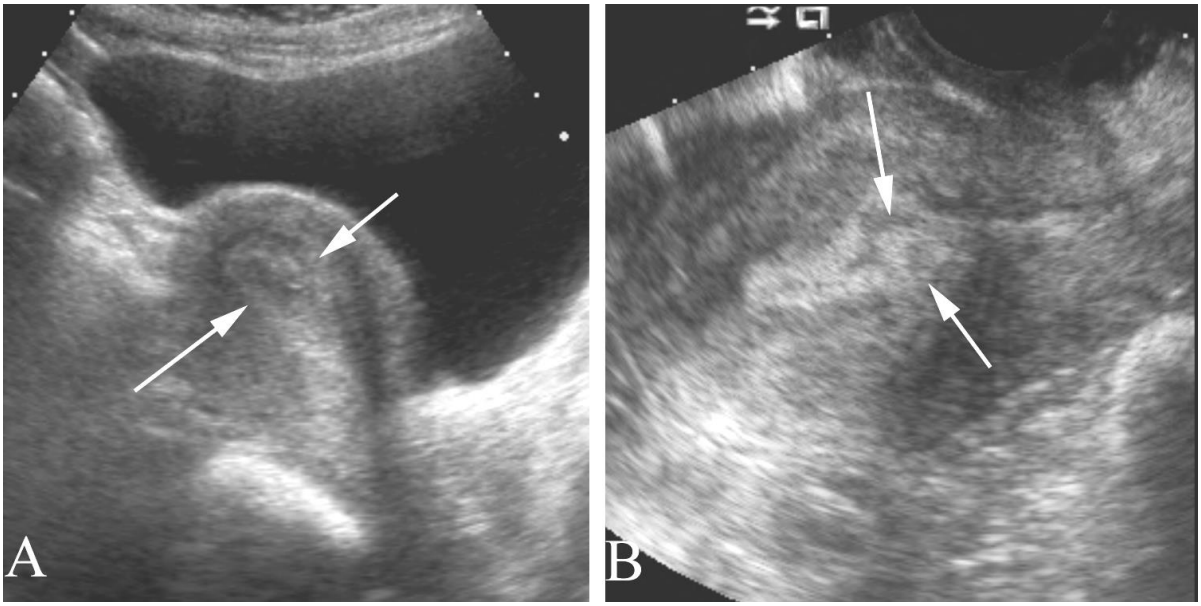

Figure 2. Endometrial polyp in a 50 year old with premenopausal bleeding. A. Transabdominal pelvic ultrasound study shows a diffusely abnormal thick endometrial stripe (arrows). B. Transvaginal pelvic ultrasound study demonstrates that there is a focal lesion along the mid to inferior aspect of the endometrial stripe. Biopsy demonstrated an endometrial polyp.

Abnormal premenopausal bleeding

As one source notes, "a confusing, inconsistent, and overlapping array of terms has evolved to describe abnormal frequency, duration, or volume of uterine bleeding". For this reason, the general term "abnormal uterine bleeding" is often used[3]. Pelvic ultrasound may be used in these patients to evaluate endometrial stripe thickness, because polyps (Figure 2), hyperplasia, and malignancy may also occur in premenopausal patients. For further discussion of these disease entities, see above.

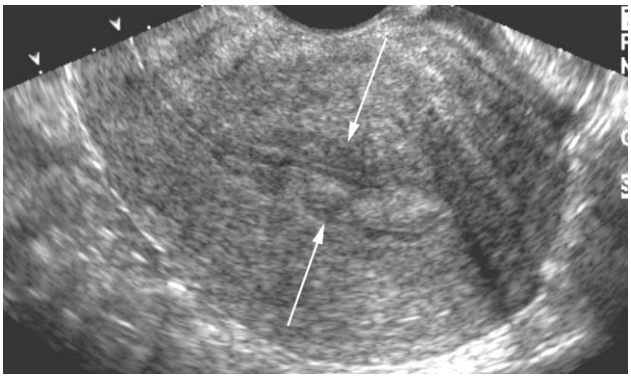

Figure 3. Secretory endometrium in a 45 year old with abnormal premenopausal bleeding. This patient had persistent abnormal uterine bleeding, and the ultrasound study shows a thick endometrial stripe (between the arrows). Secretory endometrium without hyperplasia or malignancy was found at biopsy.

Secretory endometrium may also cause a thick endometrial stripe (Figure 3), and for this reason it is far preferable to perform the ultrasound studies in the first few days after the cessation of menses (although this presumes a predictable pattern of bleeding, which these patients may not have). Submucosal fibroids may also cause bleeding in premenopausal patients, but are much less likely to cause postmenopausal bleeding.

PELVIC ULTRASOUND IS THE IMAGING STUDY OF CHOICE FOR THE EVALUATION OF FEMALE PELVIC PAIN AND MASSES

Women may present with pelvic masses, or pain in the adnexa, or painful masses, or pain in the adnexa with a mass found on ultrasound which may *not* be the cause of the pain. When imaging is necessary, as with abnormal bleeding, ultrasound is the study of choice.

Acute pelvic pain

Many diseases produce pelvic pain in women. This section discusses and illustrates gynecologic causes. Chapter 1 discusses renal causes and Chapters 7 and 8 discuss gastrointestinal causes. History and physical examination results may point to which organ system and which disease causes pelvic pain (Table 2), but features of the various diseases overlap. Ultrasound is usually the best first examination to perform because of the relatively low cost, absence of ionizing radiation, and availability. CT or MR may be done for further evaluation if necessary[4] (see page 101).

Pain Feature	Suggests
Missed period or positive pregnancy test	Ectopic pregnancy
New onset mid-cycle pain	Physiologic cyst
Pain following intercourse	Ruptured cyst
Dysmenorrhea and dysparunia	Endometriosis
Acute onset pain with nausea and vomiting	Ovarian torsion
Pain with fever	Pelvic inflammatory disease

Table 2. Pelvic pain features which suggest a specific diagnosis[5].

Ectopic pregnancy

Any woman of child-bearing age with acute pelvic pain needs to have a pregnancy test done, and if the pregnancy test is positive it is imperative to exclude an ectopic pregnancy. If there is a live, normal appearing intrauterine pregnancy with compatible beta HCG measurements, then other sources of pelvic pain should be sought, unless the patient is on fertility drugs (which greatly increase the changes of otherwise extremely rare simultaneous intra- and extra-uterine pregnancy). If there is no obvious, appropriately sized intrauterine

pregnancy, then ectopic pregnancy should be suspected. In some ectopic pregnancies, there is fluid in the endometrium which forms a so-called "pseudogestational sac" which may be impossible to distinguish from an early intrauterine pregnancy. Pelvic ultrasound studies may or may not show an adnexal mass, and will rarely demonstrate a genuine fetus (with heartbeat) outside the gestational sac.

Ovarian cyst - simple

A simple cyst is a cyst with no solid component. Cysts may cause pain because of expansion of the ovarian capsule, rupture (in which case there may be little remaining of the cyst but there may be free fluid in the pelvis) and hemorrhage (see below)[6]. Given the frequent appearance of cysts in asymptomatic patients, the causal connection between cysts and pelvic pain may be difficult to establish. For *premenopausal patients*, simple cysts smaller than 3 cm almost always represent dominant (Graafian) follicles, and some authors advocate the term "follicle" for such lesions rather than "cyst" (even though they are cysts), as a way to indicate that such small, simple cysts are probably best ignored, particularly if asymptomatic[6]. Simple cysts larger than 10 cm typically undergo surgical exploration, while cysts between 3 and 10 cm are followed with sequential ultrasound studies to document stable or decreased size. Recommendations regarding the timing of follow-up studies vary from a single study done following the next menstrual cycle to multiple studies done at 3 month intervals for up to two years. Most of these cysts decrease in size within 12 to 24 months[5]. For *postmenopausal patients*, size criteria for intervention move downward, with follow-up typically recommended for all cysts (or at least cysts over 20 mm) and surgical exploration for cysts over 5 cm, although again recommendations vary. In most cases, correlation with CA-125 measurements is advised, with surgical exploration in those patients with elevated levels.

Ovarian cyst - hemorrhagic

Hemorrhage into a simple cyst typically causes pain. The ultrasound appearance of hemorrhagic ovarian cysts is typically highly characteristic, allowing a presumptive diagnosis; a follow-up study is usually performed to confirm resolution of the abnormality (Figure 4).

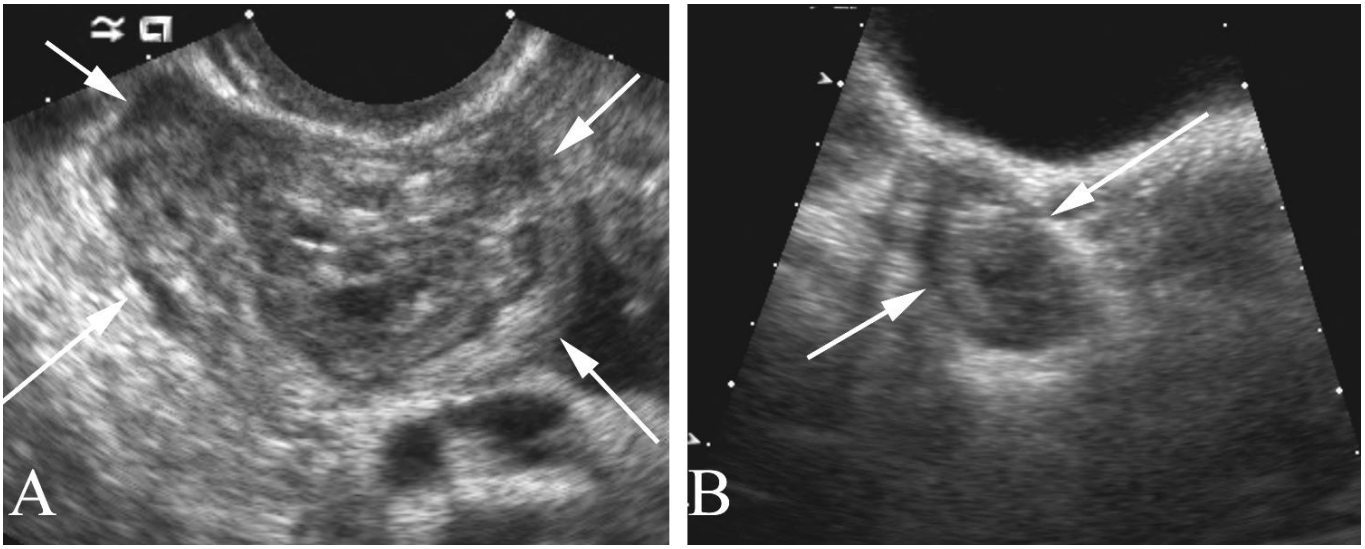

Figure 4. Hemorrhagic cyst in a 44 year old with sudden onset pelvic pain. A. Pelvic US done when the patient was in acute pain shows an enlarged heterogeneous ovary (arrows). B. Follow-up ultrasound done 10 weeks later following resolution of symptoms demonstrates that the ovary has returned to normal (arrows).

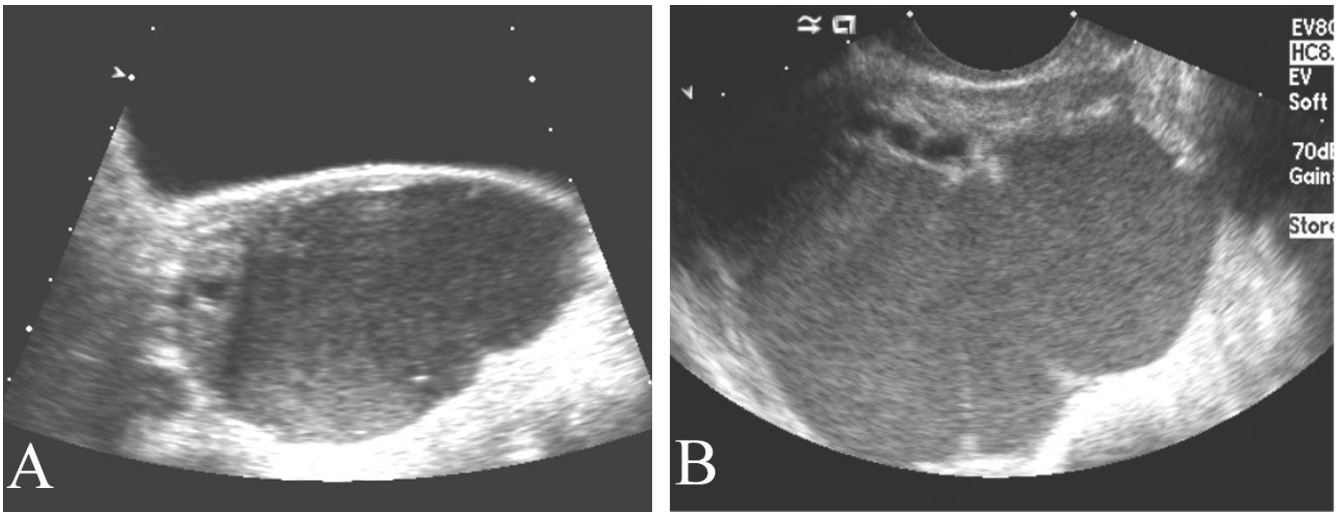

Figure 5. Endometriosis in a 32 year old with pelvic pain. A. Transabdominal ultrasound shows a large homogeneous pelvic mass. B. Transvaginal ultrasound also shows a large homogenous mass with somewhat better detail of the uniform coarse echotexture. Imaging features are characteristic of an endometrioma (as was found at surgery), but some hemorrhagic cysts have a similar appearance.

Endometriosis

While endometriosis typically causes chronic rather than acute pelvic pain, hemorrhage into an endometrioma may cause acute pelvic pain. The ultrasound study may occasionally demonstrate a typical appearance of a relatively homogenous, hypoechoic adnexal lesion (Figure 5), but the imaging features may also resemble those of a hemorrhagic cyst. Small endometrial implants may cause pain but be very difficult to identify on ultrasound. MR may be used to identify and characterize endometriomas[7].

Ovarian torsion

The ovary may twist on its pedicle, compromising blood flow and resulting in pelvic pain, nausea, and vomiting, clinical features shared by appendicitis. Ultrasound will typically demonstrate a swollen ovary, often accompanied by inflammatory free fluid in the pelvis. While color and spectral Doppler studies may be abnormal and characteristic with obvious diminished or absent flow within the ovary, these studies may show normal appearing flow *either* because of intermittent torsion *or* because arterial flow (demonstrated on the ultrasound study) is impeded only after venous flow (not demonstrated on the ultrasound study, but a source of symptoms when diminished)[8].

Pelvic inflammatory disease

Pelvic inflammatory disease (PID) may cause pelvic pain secondary to inflammation of mucosal surfaces. Most cases of PID result from ascending infection from a sexually transmitted disease which causes cervicitis, endometritis, and then infection of the fallopian tubes with associated pyosalpinx[8]. Sonography may be normal prior to development of pyosalpinx. See Figure 1 in Chapter 8, page 102.

Degenerating uterine fibroid

Degenerating uterine fibroids undergoing hemorrhage or infarction may cause pelvic pain. Ultrasound performs well in diagnosing uterine fibroids, although, as with ovarian cysts, fibroids are so commonly seen in asymptomatic patients that it may be difficult to establish the fibroid as a cause of pain. Sonographic features which may suggest degeneration include anechoic areas suggesting hemorrhage or color Doppler studies showing a lack of blood flow suggesting infarction. Magnetic resonance imaging may be helpful if differentiation between infarcted tissue and remaining vascularized tissue is necessary for surgical planning (Figure 6).

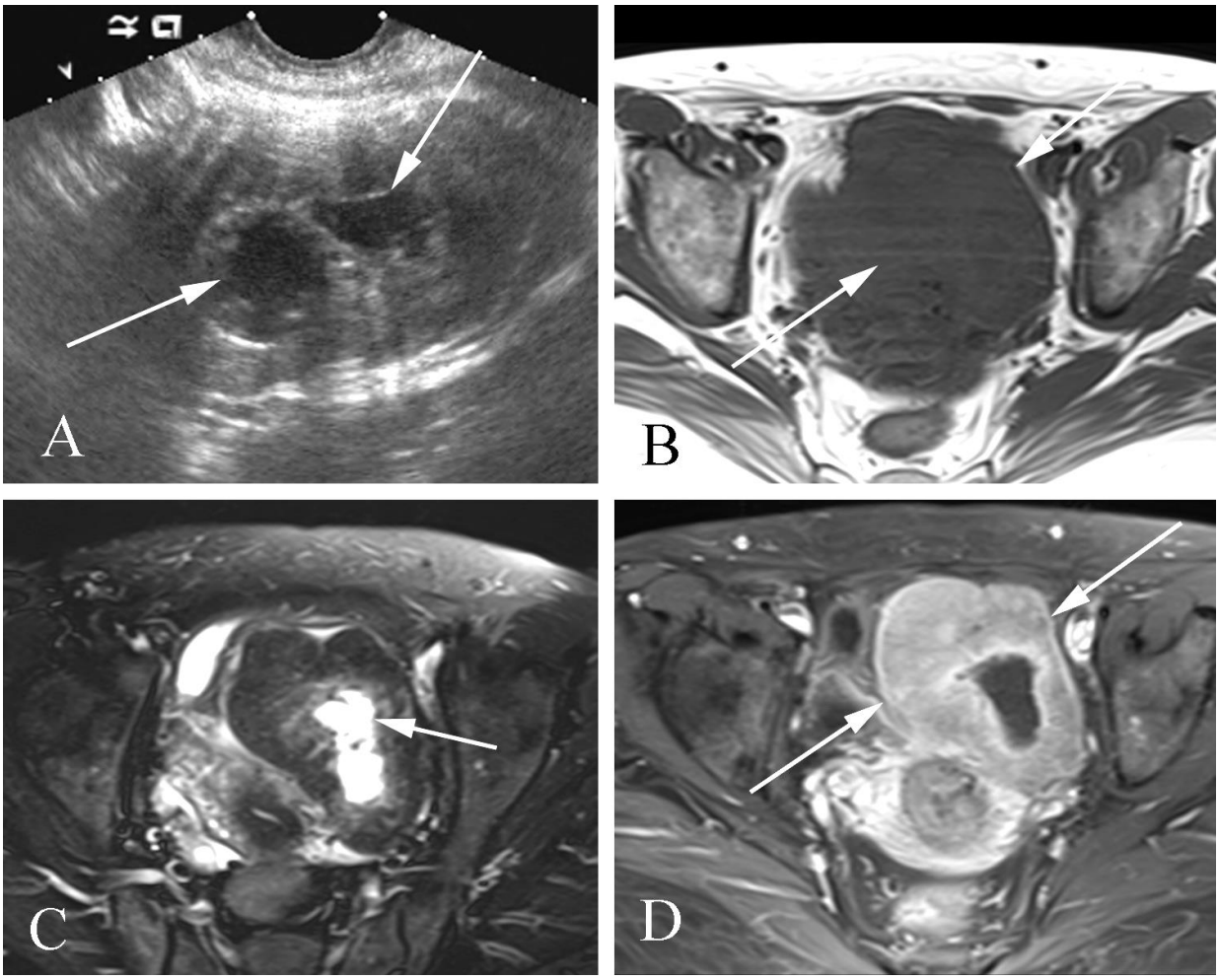

Figure 6. Degenerating (infarcting) fibroid in a 46 year old with pelvic pain. A. US shows hypoechoic areas within a fibroid (arrows) compatible with necrotic debris. B. T1 weighted MR done without contrast shows the exophytic fibroid (arrows). C. T2 weighted MR shows areas of T2 prolongation (bright signal, arrow) within the fibroid corresponding to the hypoechoic areas on the US study, representing fluid secondary to necrosis. D. Contrast-enhanced, fat-suppressed T1 weighted image demonstrates intense enhancement of the fibroid (arrows) except for the necrotic central portion.

Female pelvic mass

Multiple diseases may produce pelvic masses. Many of these disease processes will also cause pain, and these processes are discussed above. Painless pelvic masses of the uterus typically represent fibroids (see above). Painless adnexal masses may represent simple cysts (again, see above) or a complex mass, often arising in the ovary. Such complex masses generally require gynecologic or even gynecologic oncologist referral, particularly in the postmenopausal patient. While researchers have made multiple attempts to define ultrasound criteria to separate benign and malignant adnexal and ovarian masses based on various imaging features (size, complexity, vascular flow indices), no imaging feature or set of features is entirely accurate and most of these patients either need close follow-up or surgical exploration[8].

Complex (combined cystic and solid) adnexal lesions

In premenopausal patients, dermoid cysts (Figure 7) and hemorrhagic cysts (Figure 1) will often demonstrate a characteristic appearance, with surgery typically performed on the former and sequential follow-up studies performed on the latter to prove resolution. Absent a typical appearance of one of these entities, or in postmenopausal

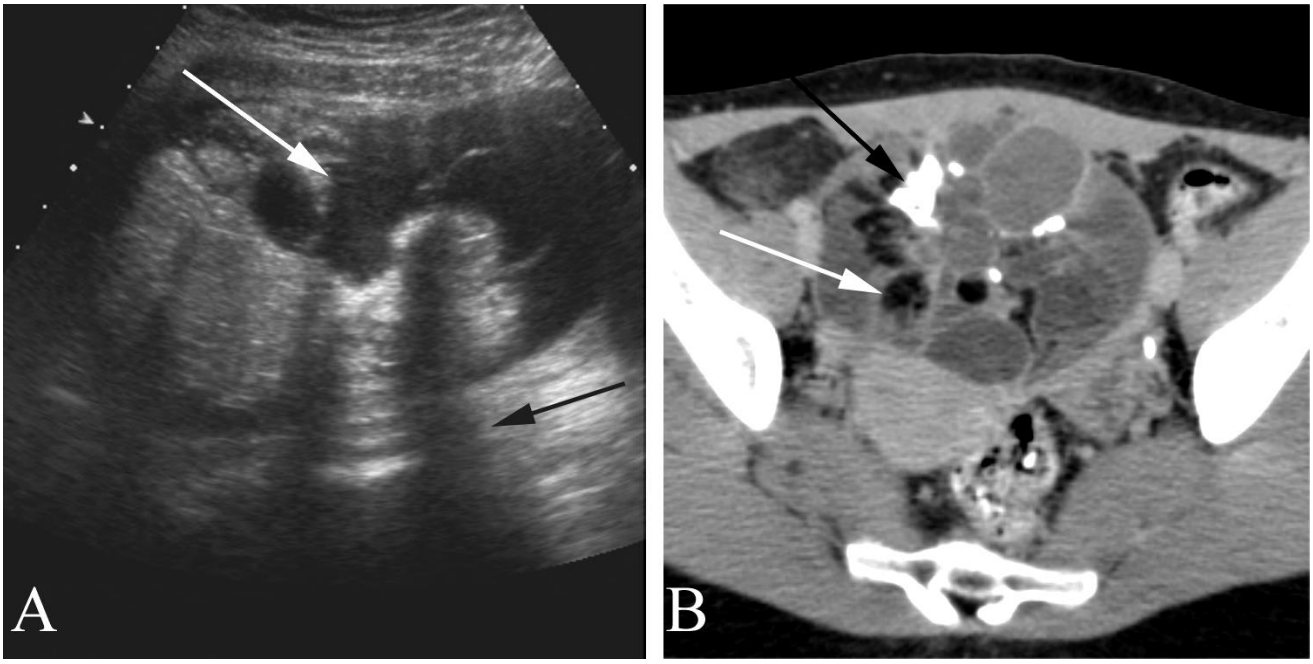

Figure 7. Pelvic (ovarian) dermoid in a 17 year old with a painless pelvic mass. A. Transvaginal ultrasound shows a cystic component (white arrow) as well as an intensely echogenic region which casts a shadow (black arrow). B. CT scan shows both calcified (black arrow) and fatty (white arrow) components in a large predominantly hypodense (cystic) lesion. Surgical resection was performed, yielding an ovarian dermoid.

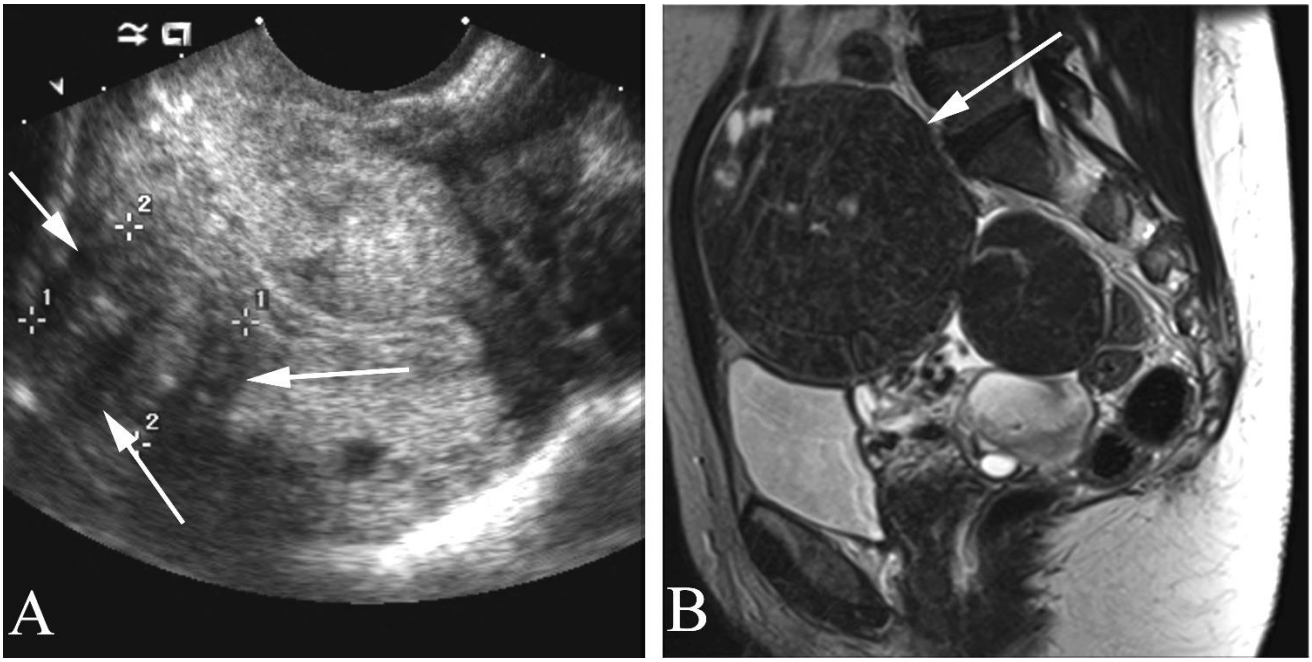

Figure 8. Uterine fibroids in a 40 year old with a painless pelvic mass. A. US study shows multiple fibroids within the uterus with typical "venetian blind" shadowing (white arrows). B. Sagittal T1 weighted MR (done for further characterization of multiple fibroids) demonstrates a huge exophytic fibroid (arrow) emanating from the superior margin of the uterus.

patients, referral to a gynecologic oncologist is usually appropriate because of the likelihood of malignancy.

Uterine fibroids

Fibroids make up the vast majority of uterine masses. US can demonstrate the size and location of uterine fibroids, which typically show a characteristic "venetian blind" type of shadowing (Figure 8).

SCROTAL ULTRASOUND IS THE STUDY OF CHOICE FOR EVALUATION OF SCROTAL PAIN AND MASSES

Scrotal symptoms may be divided by various methods, for example by how acute the symptoms are, whether the presentation is pain or a painless mass, or by the age of the patient. This chapter will address scrotal pathology in terms of how acute the symptoms are. Note that many of the acute patients may come to the emergency room rather than to a clinic. If imaging is required, regardless of how scrotal symptoms are divided, ultrasound is the imaging study of choice.

Scrotal US is the study of choice to evaluate acute scrotal pain

Disease	%
Testicular torsion	16%
Torsion of the testicular appendage	46%
Epididymitis	35%

Table 3. Distribution of 238 ER cases with acute scrotal pain. From: Lewis AG et al. Evaluation of acute scrotum in the emergency department. J Pediatr Surg 1995; 30:277.

Ultrasound performs admirably in the evaluation of scrotal pain. Ultrasound should be able to diagnose or at least suggest one of the three most common diseases accounting for scrotal pain: testicular torsion, torsion of the testicular appendix, and epididymitis (see Table 3). Less frequent causes include infections of the scrotal wall and testicular rupture from trauma. Note that epididymitis and torsion may occur with or without associated trauma.

Testicular torsion

Patients with testicular torsion present with pain, usually acute in onset and sometimes after vigorous physical activity or minor trauma[9]. Children may awake with pain in the scrotum. Torsion occurs when the testicle twists on its vascular pedicle, impeding blood flow both into and out of the testicle. Impaired blood flow causes testicular swelling and pain, and may progress to infarction relatively quickly, making prompt diagnosis imperative. The imaging features include swelling and abnormal color Doppler images (Figure 9), but because the torsion may be intermittent or not have progressed to the point where arterial flow has stopped, color Doppler imaging may still show flow and thus be misleading. Pulsed wave or spectral Doppler imaging is more sensitive and should be included in all scrotal examinations, but it, too, may be misleading, and the scan may show only testicular swelling or even be normal (especially in cases of intermittent torsion). Therefore, patients with severe intermittent or severe persistent pain should probably be referred to urology for evaluation regardless of the ultrasound results, unless the clinical features and ultrasound results are clearly those of epididimo-orchitis or torsion of a testicular appendage.

Torsion of the testicular appendage

The testicular appendage is a small, vestigial structure along the anterior, superior aspect of the testicle. These appendages may twist on their pedicles, impeding blood flow with subsequent testicle infarction and associated pain. The pain is usually less severe and of more gradual onset than testicular torsion, but the two processes may be quite difficult to distinguish clinically[9]. Ultrasound generally demonstrates a normal appearance of the testicles and epididymis in torsion of the testicular appendage; occasionally, ultrasound demonstrates the torsed appendage as a small, avascular structure adjacent to the testicle at the location of maximum pain.

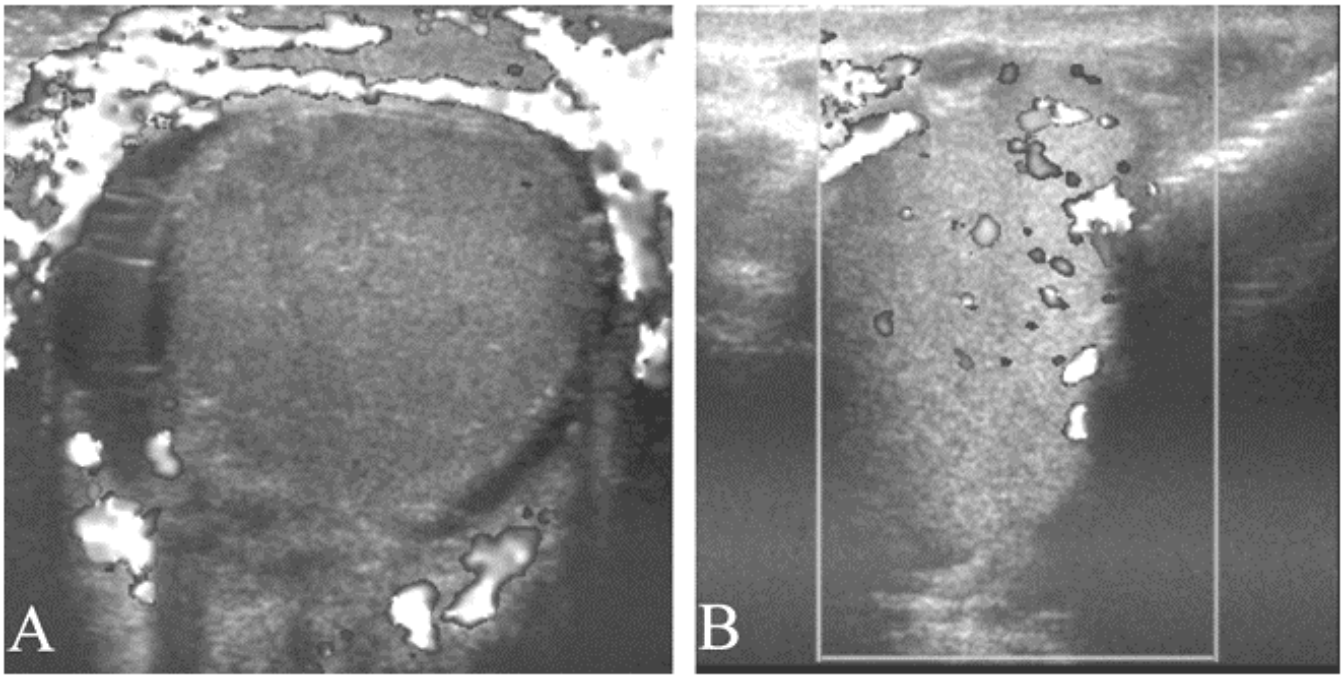

Figure 9. Testicular torsion with infarction in a 59 year old man with four days of scrotal pain. A. Scrotal ultrasound of the abnormal side shows a swollen testicle with no flow to the testicle but hyperemic tissue around the testicle, along with a reactive hydrocele. B. Ultrasound of the opposite testicle shows the normal, contralateral testicle with normal flow, as indicated by flow within the testicle.

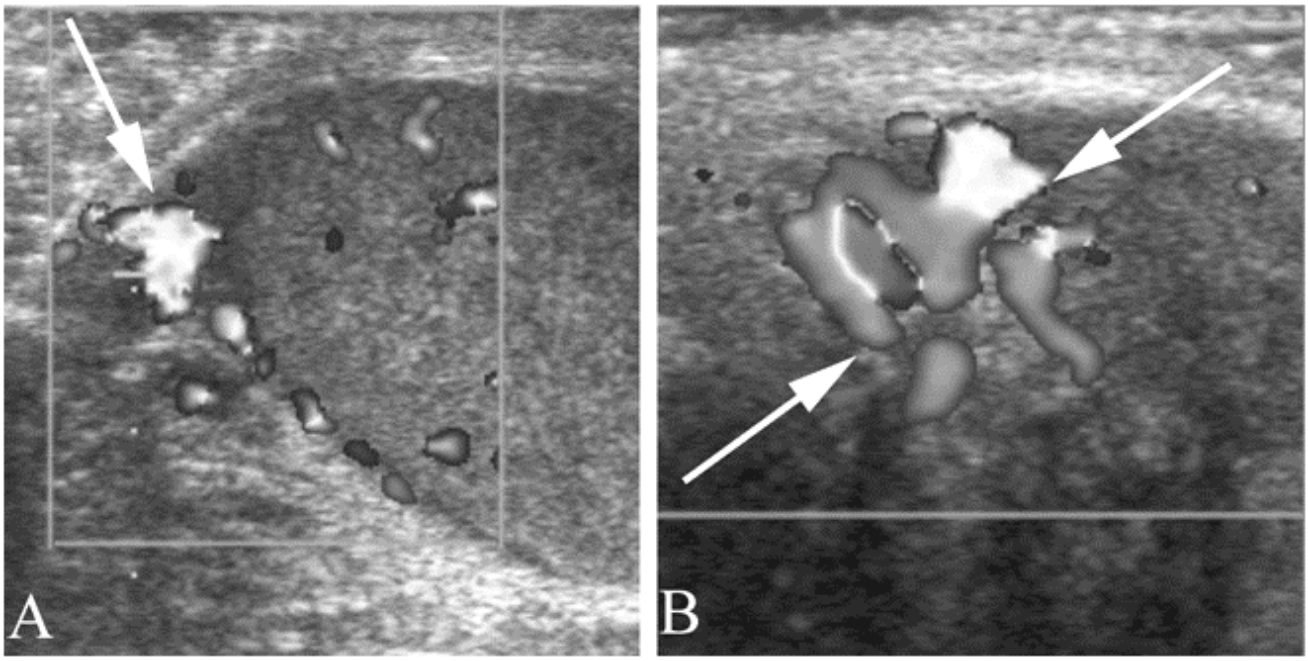

Figure 10. Epididimytis in a 14 year old with three days of scrotal pain. A. Ultrasound with color Doppler of the asymptomatic side shows a normal sized epididymis with normal flow (arrow). B. Ultrasound with color Doppler of the symptomatic side shows swelling and hyperemia of the epididymis, with increased flow (arrow).

Epididymitis

The epididymis connects the testicle to the vas deferens, and is a coiled tubular structure along the posterior, superior margin of the testicle. Inflammation of the epididymis may occur secondary to trauma, severe straining (particularly young men participating in the weight lifting exercise known as "squats"), bicycle and motorcycle riding, or sexually transmitted diseases. The epididymis is swollen and painful on clinical examination. Ultrasound demonstrates an enlarged epididymis with increased flow compared to the contralateral side on color Doppler imaging (Figure 10).

Other infections

Infections of the scrotal wall and perineum (Fornier's gangrene) may also be assessed with ultrasound, which will demonstrate extensive skin thickening and hyperemia with normal testicles deep to the abnormal superficial tissues (Figure 11). These infections require emergent referral and treatment.

Scrotal US is the study of choice to evaluate chronic scrotal conditions

Ultrasound is also the study of choice for the evaluation of chronic scrotal conditions, including an absent testicle (with suspected cryptorchidism) chronic scrotal pain (which may be caused by varicocele or chronic epididymitis), and a scrotal mass (which may be caused by a spermatocele, hydrocele, or testicular tumor).

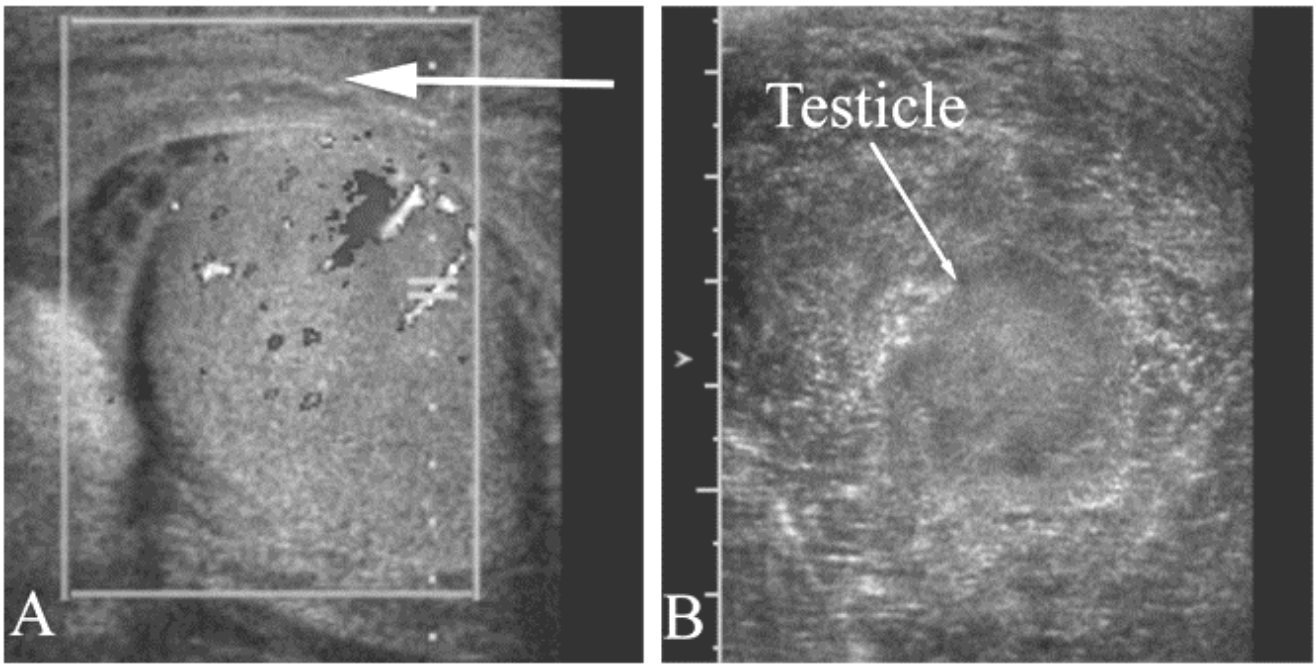

Figure 11. Fornier's gangrene in a 48 year old with severe pain and swelling of the scrotum for several days. A. Normal testicle with normal flow on color Doppler, but with thick surrounding tissue (arrow). B. The testicle from a different angle, with a larger field of view, demonstrates the testicle swimming in a sea of inflammatory tissue.

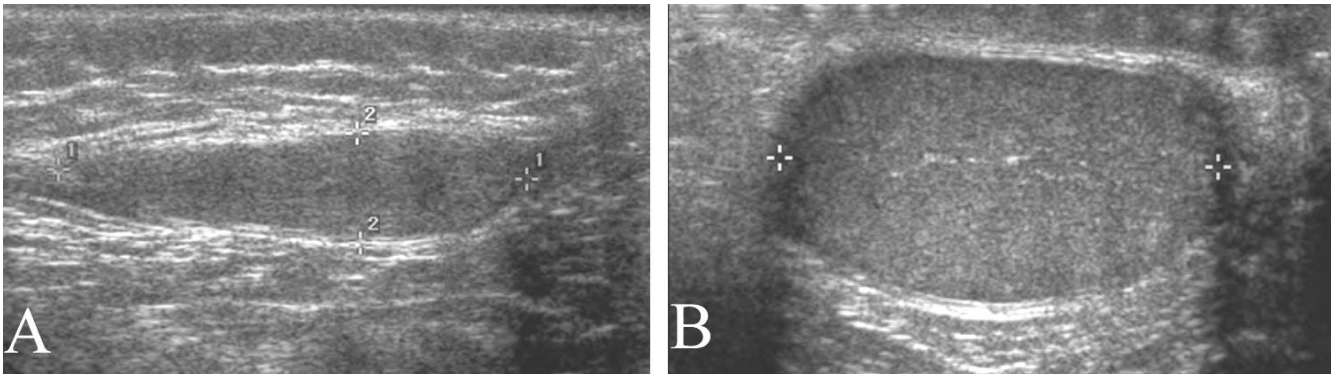

Figure 12. Undescended testicle in a 10 year old with a single testicle palpated in the scrotum. A. Ultrasound shows that the undescended testicle is in the inguinal canal and shows an abnormal, elongated appearance. B. The contralateral, normal, descended intrascrotal testicle.

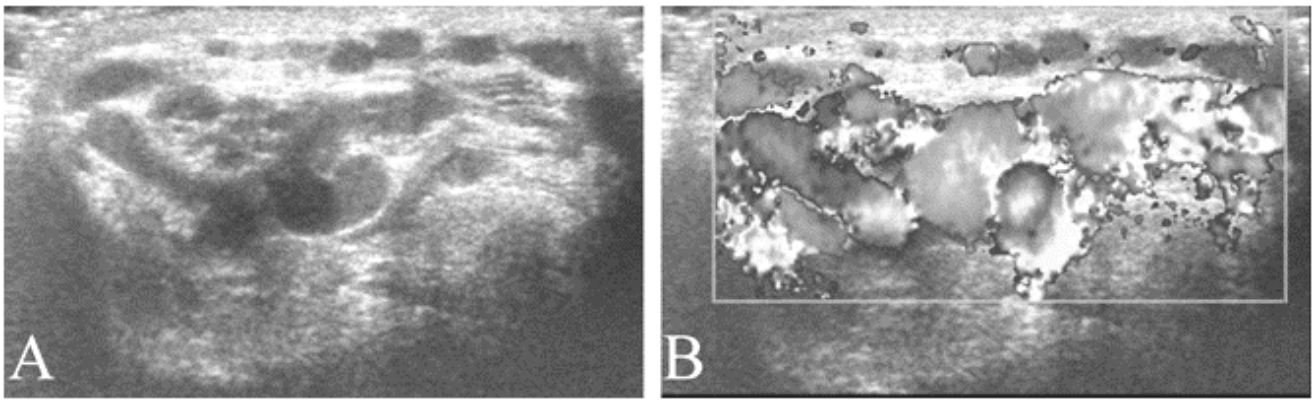

Figure 13. Varicocele in a 16 year old with a painless mass in the left scrotum. A. Gray-scale ultrasound of the mass in the scrotum shows the typical "bag of worms" appearance of a varicocele. B. Color Doppler exam demonstrates extensive flow through the lesion.

Undescended testicle

Undescended testicles present with an absence of one or both testicles in the scrotum. Undescended testicles do not produce sperm as well as testicles in the scrotum, and are prone to malignant degeneration[10]. Undescended testicles are usually in the inguinal canal and may be seen with ultrasound (Figure 12). Intra-abdominal undescended testicles may be evaluated with CT.

Varicocele

Varicoceles may cause pain or present as a painless mass. The root problem is venous drainage: on the left side, the testicular vein runs from the testicle to the left renal vein, which it enters at an approximately perpendicular angle, making the left side prone to reflux and varicocele formation. Ultrasound will demonstrate a "bag of worms" appearance adjacent to the testicle, with color flow imaging showing abundant flow in the varicocele (Figure 13).

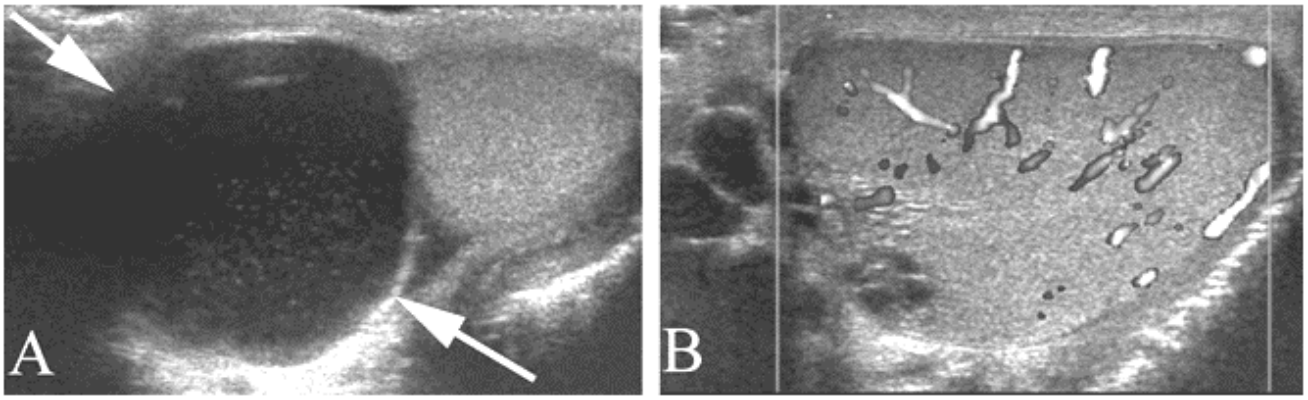

Figure 14. Epididymal cyst/spermatocele in a 46 year old with a painless mass in the left scrotum. A. Large spermatocele (arrow) adjacent to the (much smaller) testicle. B. Color Doppler of the normal adjacent ipsilateral testicle, with normal flow.

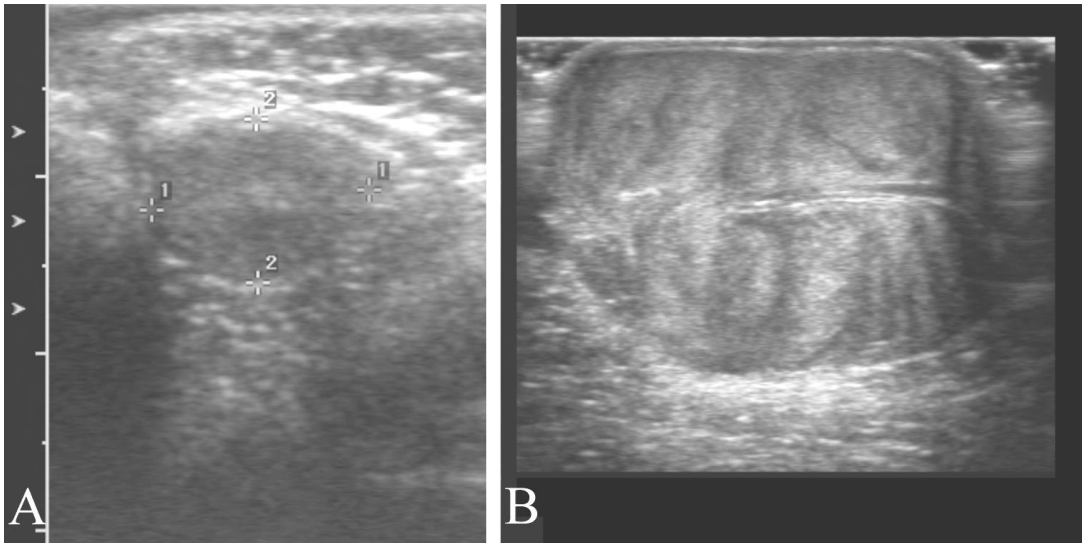

Figure 15. Solid scrotal tumor in a one year nine month old with swelling and prominence of his scrotum. A. Ultrasound of the normal side shows a normal size testicle with normal echogenicity. B. Ultrasound of the abnormal side shows a large heterogeneous mass. Orchiectomy followed, and the lesion was a malignant rhabdomyosarcoma of the peritesticular tissues which had engulfed the testicle.

Epididymal cyst

With respect to palpable lesions of the scrotum, most of the extratesticular lesions will be benign cysts, and represent either epididymal cysts (less than 2 cm) or spermatoceles (greater than 2 cm). These lesions demonstrate the classic ultrasound appearance of a cyst, showing an anechoic appearance with no internal echoes, a sharply defined wall, and posterior enhancement (because more of the ultrasound beam travels through the cyst than the adjacent soft tissues) (Figure 14). Typically, these lesions require no treatment and the course of action consists of reassuring the patient that all is well.

Testicular tumor

Intratesticular palpable lesions, unlike extratesticular lesions, are more frequently solid and such lesions are always worrisome (Figure 15). While there are some generalities regarding the appearance of the lesion and the ultimate pathologic diagnosis, distinguishing between the various cell types by ultrasound is not possible and basically a moot point anyway, since virtually all of these lesions result in orchiectomy and pathologic evaluation.

SUMMARY

Ultrasound examination is the study of choice for evaluation of female pelvic pain, abnormal uterine bleeding (both pre- and postmenopausal), and pelvic masses. Ultrasound allows evaluation of the endometrial stripe, which is helpful in the work-up of patients with possible endometrial cancer. It also allows differentiation of fibroids within the uterus from adnexal cysts and masses. Ultrasound examination is also the study of choice for scrotal pain and masses. It can usually identify and distinguish torsion of the testicular appendage and testicle. Ultrasound can also differentiate benign, extratesticular causes of masses (e.g., varicoceles and spermatoceles) from malignant, intratesticular lesions.

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Headache

Donald L. Renfrew, MD

This chapter discusses the diagnosis and imaging of headache. The chapter will follow the classification provided by the International Headache Society (IHS) (see www.i-h-s.org). The IHS uses the term "primary headache" to describe tension type, migraine, and cluster headaches, and "secondary headache" to describe headaches occurring secondary to another disorder. The three main points of this chapter are:

1. Most headaches represent one of three types of primary headaches, are diagnosed based on clinical features, and do not usually require imaging.
2. Secondary headaches may demonstrate "danger" signs which require immediate imaging and/or lumbar puncture.
3. Secondary headaches may rarely be insidious and mimic primary headaches.

MOST HEADACHES ARE PRIMARY HEADACHES, ARE DIAGNOSED BASED ON CLINICAL FEATURES, AND USUALLY DO NOT REQUIRE IMAGING

Primary care practitioners will see many patients with a chief complaint of headache. The diagnosis of primary headache, or headache which is not secondary to an anatomic cause, typically relies on clinical evaluation. Most primary headaches fall into one of three types: tension type headache, migraine headache, and cluster headache. Patients with chronic intermittent headaches usually do not require imaging, if there are no associated neurologic findings, if the pattern of headache is stable, and if the clinical features are characteristic of one of these types of headaches. Brief descriptions of the three common types of headache follow.

Tension type headache

To meet IHS criteria for tension type headache, the headache must last from 30 minutes to 7 days and have two of the following four characteristics: bilateral location, non-pulsating quality, mild to moderate intensity, and lack of aggravation by routine physical activity. The headache must also have no associated nausea or vomiting. The headaches may be associated with photophobia or phonophobia, but not both. Tension type headaches specifically lack auras, a feature of migraine headaches (see below).

Most patients with tension type headache do not seek medical care since they recognize the headache as temporary, self-limiting, and not particularly disabling[1]. Bendtsen and Jensen argue that infrequent episodic tension type headache is a normal phenomenon and not a disease per se[2]. Tension type headache apparently results from sensitized dorsal horn neurons misinterpreting innocuous stimuli as painful[3]. The diagnosis is based on the criteria listed above, and, absent any associated neurologic features or other unusual findings, imaging is not typically performed.

Migraine headache

To meet IHS criteria for migraine headache, the headache must last from 4 to 72 hours and have two of the following four characteristics: unilateral location, pulsating quality, moderate or severe intensity, and aggravation by routine activity. During the headache, the patient must have nausea and/or vomiting, or photophobia and/or phonophobia. Migraines may occur with or without auras; if without, 5 attacks meeting the above criteria

are necessary for diagnosis but if with auras, only 2 attacks are necessary. Auras consist of visual and/or sensory and/or speech symptoms characterized by gradual onset, duration of less than an hour, and complete reversibility[4]. Examples including seeing flickering lights or feeling a "pins and needles" sensation.

While migraine headaches are much less frequent than tension type headaches, patients are more likely to visit a primary care provider because of the severity of the headache. Because of the relative frequency and severity of migraine, almost all patients presenting to the primary care provider with severe episodic headaches are likely to have migraine headaches[5]. Severe migraines may be disabling and referral to a neurologist and/or headache specialist may be in order. Neurogenic inflammation causes migraine[5], and treatment is directed toward prevention or elimination of this inflammation. Patients meeting the above criteria for migraine headache do not typically require imaging unless there is a change of the headache pattern (Figure 1).

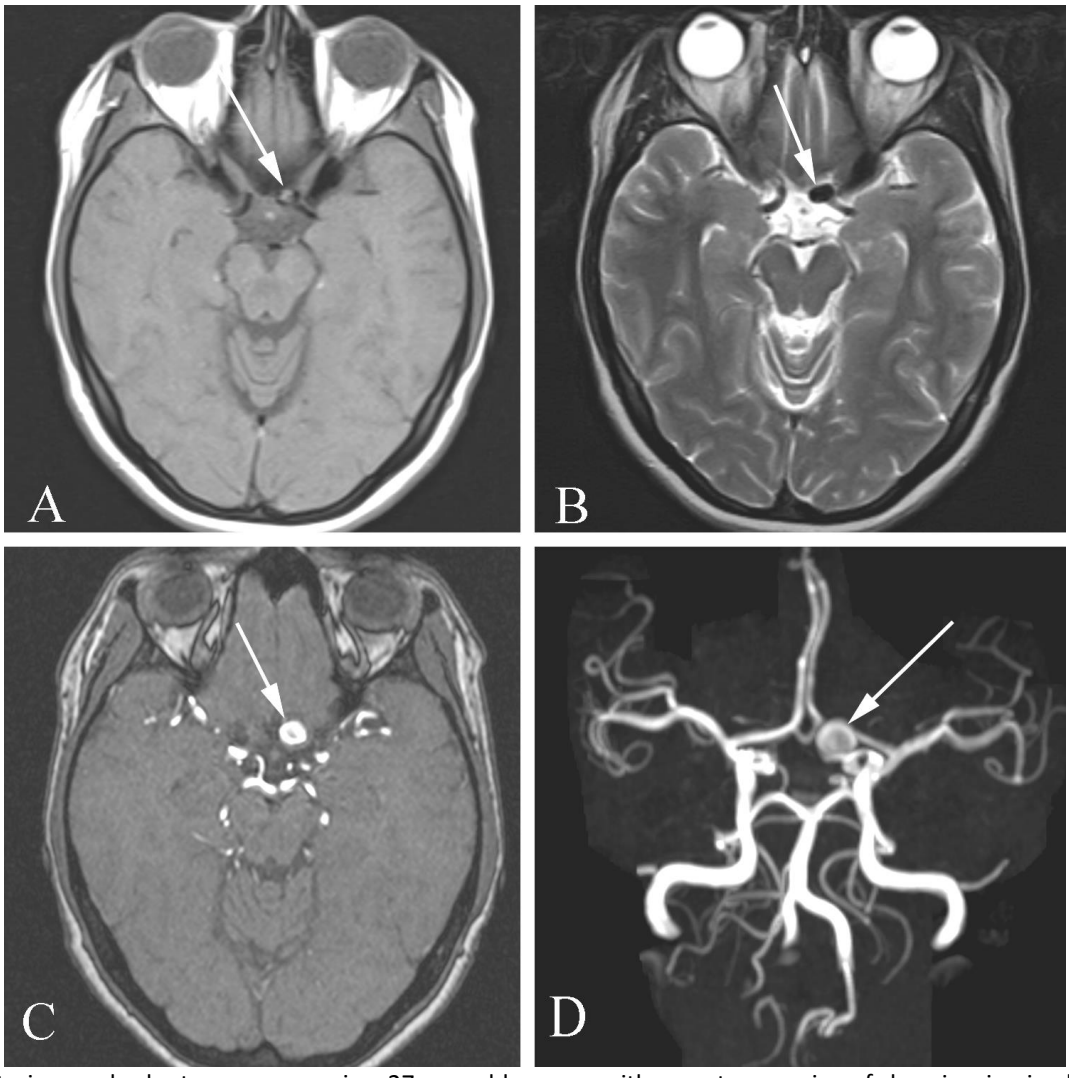

Figure 1. Anterior cerebral artery aneurysm in a 27 year old woman with recent worsening of chronic migraine headaches. A. Axial T1 weighted brain MR shows an aneurysm of the left anterior cerebral artery (arrow). B. Axial T2 weighted image shows the aneurysm as well (arrow). C. Axial source images from a magnetic resonance angiogram of the circle of Willis also show the aneurysm (arrow). D. 3D maximum intensity projection of the circle of Willis also shows the aneurysm (arrow).

Cluster headache

To meet IHS criteria for cluster headache, the headache must last from 15 to 180 minutes and have one of the following six characteristics: ipsilateral conjunctival injection and/or lacrimation; ipsilateral nasal congestion and/or rhinorrhea; ipsilateral eyelid edema; ipsilateral forehead and facial sweating; ipsilateral miosis and/or ptosis; or a sense of restlessness or agitation. The attacks must also have a frequency from one every other day to eight per day, with at least five such attacks in total (the term "cluster" refers to the tendency of the attacks to come in groups, frequently separated by longer periods without headache). Unlike patients with migraine headache who want to avoid movement, patients suffering with a cluster headache prefer to pace about[6].

Cluster headache is considerably less frequent than tension type headache and migraine headache, but is severe enough that most of these patients will seek medical care. Cluster headache is a subtype of a broader group of disorders known as trigeminal autonomic cephalgias with activation of the trigeminal-autonomic reflex causing the pain[6]. Unlike the situation with tension type headache and migraine headache, current recommendations are to perform imaging in patients with the initial diagnosis of cluster headache. This follows from the fact that a number of intracranial abnormalities have been reported to cause secondary cluster headache, and neuroimaging is necessary to exclude such causes as intracranial aneurysms, meningiomas, and pituitary tumors[7]. Magnetic resonance imaging of the brain performed without and with contrast, along with MRA of the circle of Willis, is the preferred imaging study in patients with the initial diagnosis of cluster headache.

SECONDARY HEADACHES MAY DEMONSTRATE "DANGER" SIGNS WHICH REQUIRE IMMEDIATE IMAGING AND/OR LUMBAR PUNCTURE FOR DIAGNOSIS

Most patients with "danger" signs requiring immediate diagnostic work-up (including CT and/or lumbar puncture) will come to the emergency room rather than the primary care provider's office. However, the evolution of health care has blurred the distinction between the ER and the office with the advent of urgent care walk-in clinics and facilitated same-day appointments, and primary care providers may find themselves dealing with patients who present with a new, acute severe headache. These patients typically require emergent CT scanning to evaluate for subarachnoid hemorrhage, followed by immediate neurosurgical referral if the CT scan is positive for subarachnoid hemorrhage and likely lumbar puncture if the CT scan is negative for subarachnoid hemorrhage and shows no mass effect. Because of the long list of danger signs, it is actually easier to remember who doesn't need to be imaged than who does need to be imaged.

Headache – who not to image

As noted in the previous section, patients with typical clinical features of primary headache from tension type headache or migraine headache do not usually require imaging, whereas patients meeting criteria for cluster headache do require imaging. Patients with no substantial change in their usual headache pattern, with no new concerning features (seizure, trauma, fever), and with no focal neurologic symptoms or abnormal neurologic exam findings do not require imaging, urgent or otherwise[8]. Conversely, patients with a change in their typical headache pattern *do* require imaging (Figure 1).

Headache – danger signs

A long list of headache features should provoke concern on the part of the clinician. Chief among these features is that the headache is the "worst or first"[5] and severe headaches with rapid onset, so-

called "thunderclap" headaches[9]. These features should provoke immediate transport to the emergency room or CT scanner. Other features accompanying the headache which should cause concern include a change in mental status or fluctuation in the level of consciousness (Figure 2), focal neurological symptoms (Figure 3), fever (Figure 4), rapid onset of pain during strenuous exercise, and headache spreading to the lower neck and between the shoulders[5]. Furthermore, new headache in a patient with cancer suggests metastasis (Figure 5), while headache during pregnancy or the post-partum period may signal any of several puerperal complications including cortical vein thrombosis, carotid dissection, and pituitary apoplexy[10]. Headache and neck pain following a round of golf or a visit to the chiropractor should arouse suspicion for carotid artery dissection.

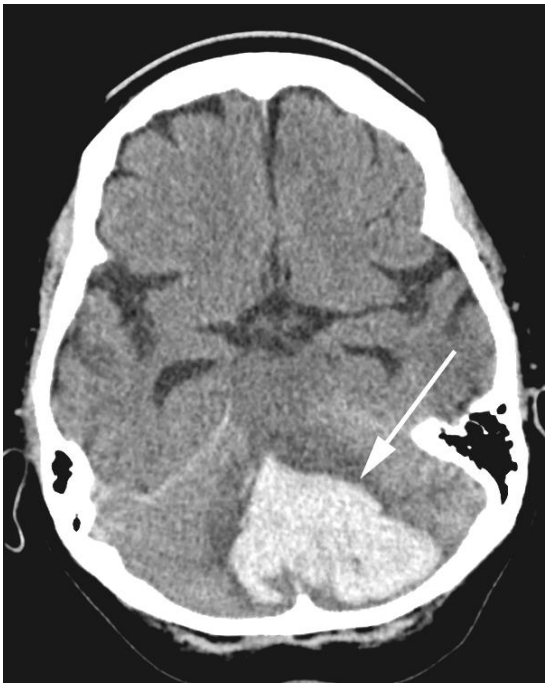

Figure 2. Hemorrhagic cerebellar infarction in a 61 year old woman with headache and altered mental status. The patient initially had headache, dizziness, and slurred speech, then became unresponsive. She did not survive. Axial unenhanced CT shows massive cerebellar hemorrhage (arrow).

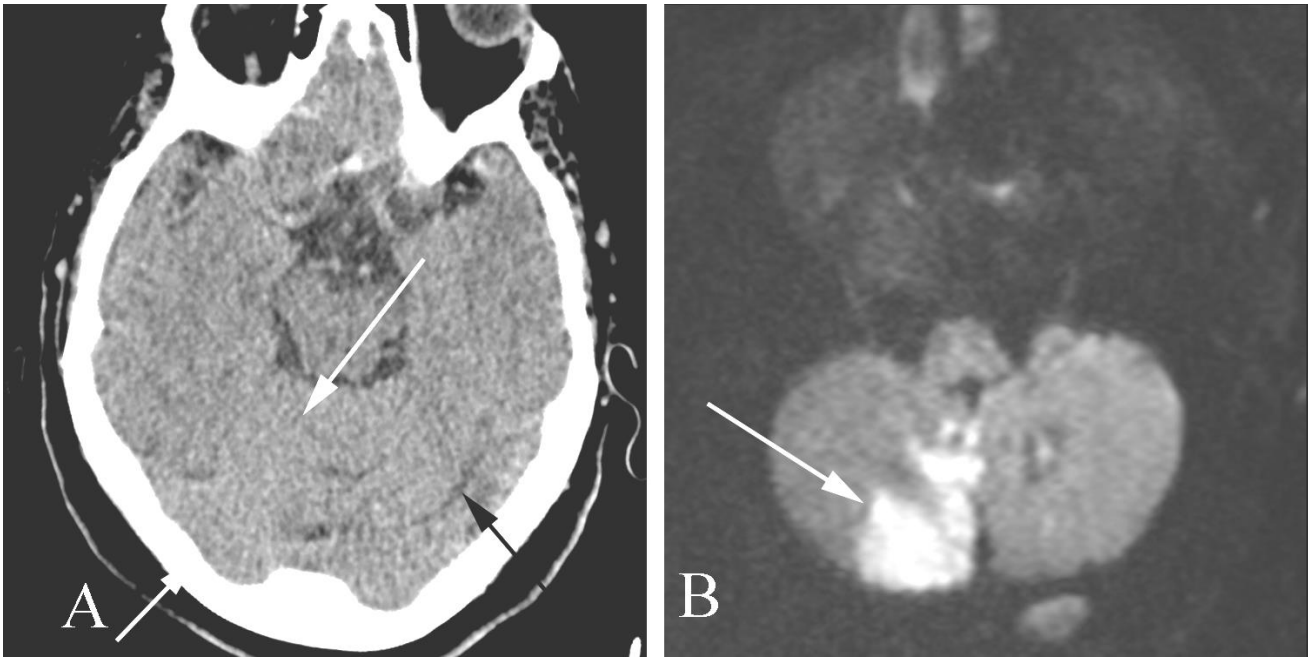

Figure 3. Nonhemorrhagic cerebellar infarction in a 65 year old man with headache who also had neurologic symptoms (dizziness and slurred speech). A. Axial unenhanced CT scan through the posterior fossa shows a broad area of effaced sulci indicating brain swelling (white arrows). Note the normal contralateral cerebellar hemisphere sulcus (black arrow). B. Diffusion weighted magnetic resonance imaging study shows restricted diffusion of the right cerebellar hemisphere, typical for stroke (arrow).

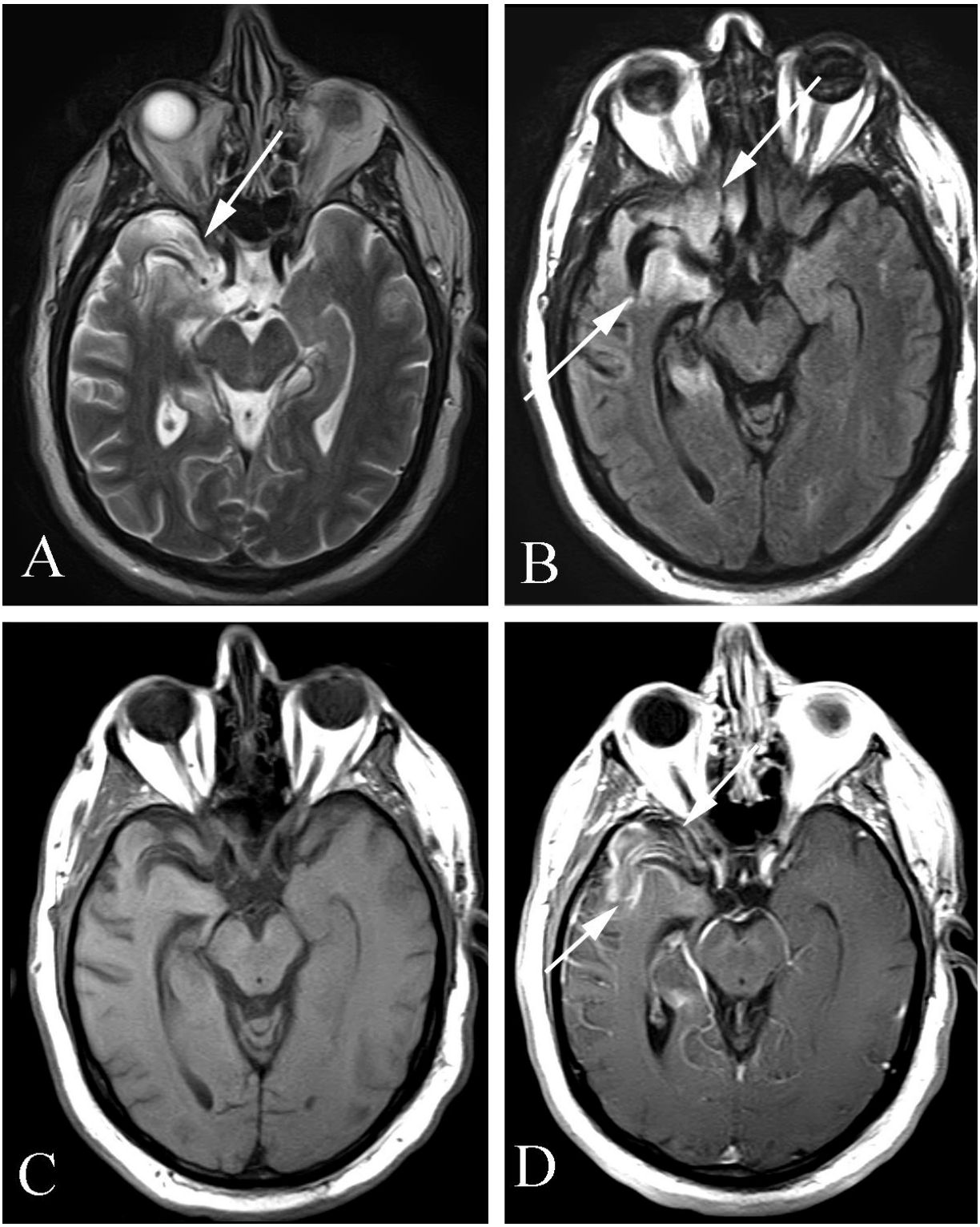

Figure 4. Herpes encephalitis in a 59 year old man with headache who also had fever. A. Axial T2 weighted MR image shows increased signal through the right temporal lobe (arrow). B. Axial FLAIR MR imaging also shows increased signal through the temporal lobe (arrows). C. Axial T1 weighted enhanced MR image shows accentuated sulci in the right temporal lobe. D. Axial T1 weighted postcontrast MR image demonstrates intense contrast enhancement of the abnormal brain parenchyma (arrows).

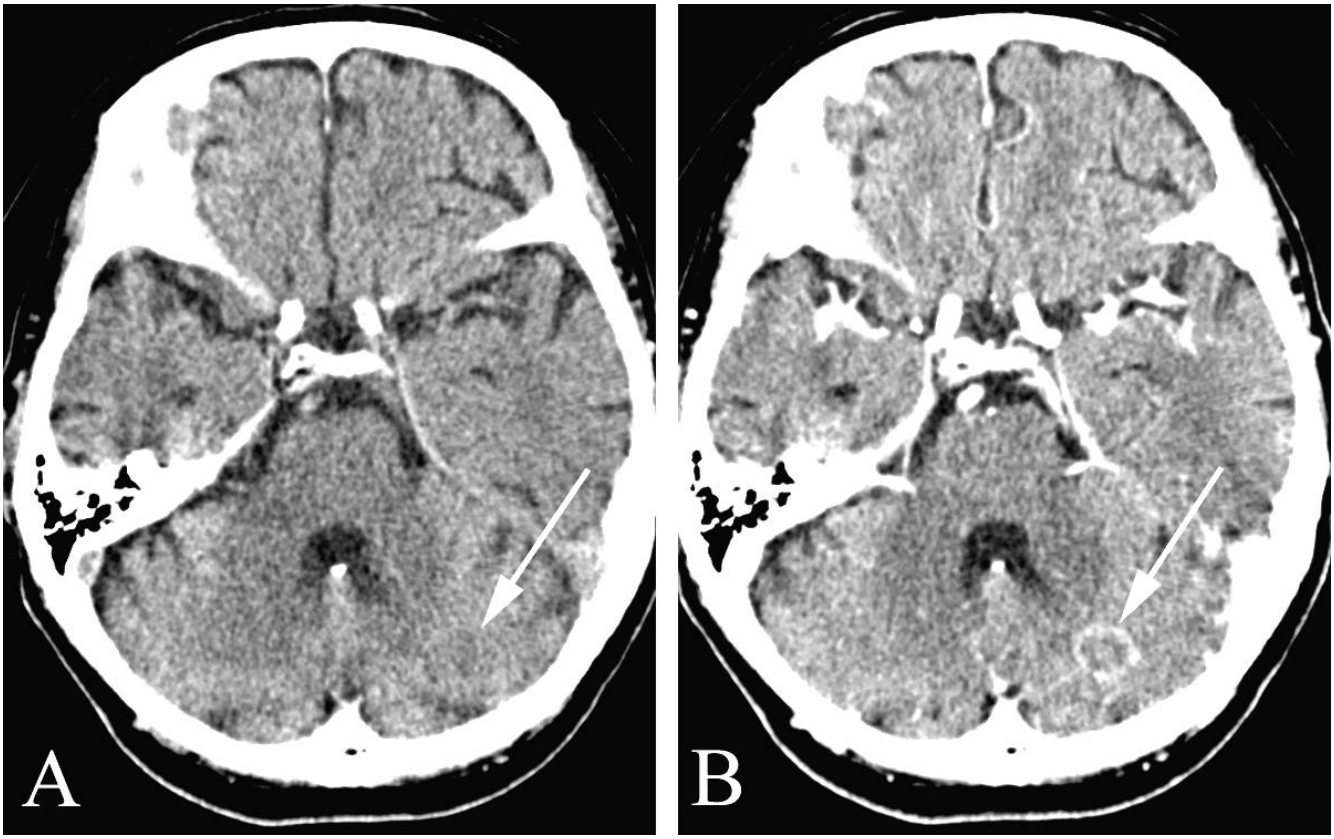

Figure 5. Metastatic disease in a 74 year old man with headache who had known lung cancer. A. Axial unenhanced CT study shows a subtle area of hypodensity in the left cerebellar fossa (arrow). B. Axial contrast enhanced CT shows a "rim enhancing lesion" (also called a "ring enhancing lesion") with peripheral contrast enhancement around a centrally isodense lesion compatible with metastatic disease (arrow). Note that such rim enhancing lesions may also be seen in primary brain tumors and abscesses.

Headache caused by subarachnoid hemorrhage

About 20% of patients who state that they are having "the worst headache of my life" will have a subarachnoid hemorrhage[11]. When a head CT demonstrates acute subarachnoid hemorrhage (Figure 6), the cause must be established as rapidly as possible because without treatment the likelihood of death in the next 30 days is greater than 50%[11]. This may be accomplished by immediate computed tomographic angiography (CTA) of the cerebral vascular tree, often the preferred method of imaging in perilously ill patients who require emergency craniotomy for evacuation of a large subarachnoid clot[11]. CTA performed on modern helical CT scanners is at least 90% accurate at identification of ruptured aneurysms[12]. On the other hand, if the patient is stable enough to undergo catheter angiography, this technique not only offers the gold standard in diagnosis, but also allows life saving percutaneous therapeutic treatment of leaking aneurysms (Figure 6). The decision of which specialist to use (neurosurgeon versus interventional neuroradiologist) and which technique is then employed for treatment (open repair versus percutaneous intervention) varies with local expertise.

When the CT demonstrates subarachnoid hemorrhage but CTA and subsequent catheter angiography fails to demonstrate a leaking aneurysm or other cause (e.g., vascular malformation, intracranial arterial dissection, or vasculitis), then MR (done without and with contrast) should be performed to search for alternative explanations of subarachnoid hemorrhage (e.g. angiographically occult vascular malformation, bleeding pituitary adenoma)[13]. If the CTA (when done), catheter angiography, and MRI

are all negative in a patient with a proven subarachnoid hemorrhage, angiography is repeated two weeks later since an aneurysm may be seen only on the delayed study[11].

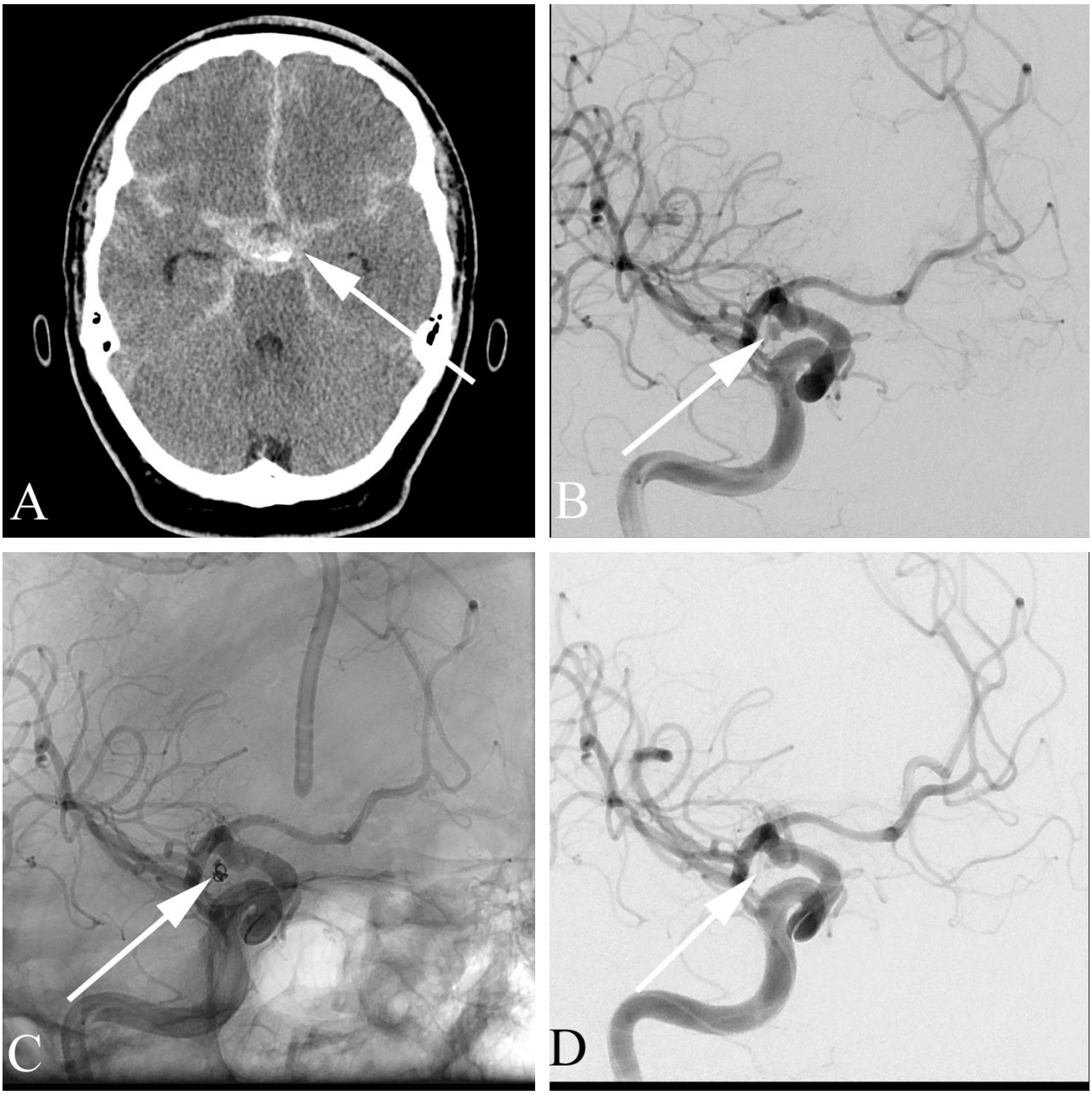

Figure 6. Subarachnoid hemorrhage from a bleeding aneurysm in a 60 year old man with thunderclap headache, nausea, and vomiting. A. Axial unenhanced CT study shows extensive subarachnoid hemorrhage in the suprasellar cistern (arrow) with extension between the hemispheres, around the brainstem, and along the sulci of the temporal lobes. B. Oblique image from catheter angiography demonstrates an aneurysm off of the distal internal carotid artery. C. Oblique image (nonsubtracted) from catheter angiography following coil deployment shows the coil at the former location of the aneurysm. D. Oblique image (digitally subtracted) from catheter angiography following coil placement demonstrates minimal flow into the aneurysm following successful coil placement (compare with "B").

Patients with a negative head CT suspected to have a subarachnoid hemorrhage on the basis of the clinical presentation (e.g. a thunderclap headache) need to undergo a lumbar puncture, because the lumbar puncture is more sensitive for the detection of small amounts of subarachnoid hemorrhage[11]. It is critical to identify these patients, because up to half of patients with major SAH will have a minor SAH or "warning leak" 6 to 20 days before the major leak[14], and identification of the aneurysm will allow treatment prior to the possible lethal rupture of the aneurysm.

Headache caused by intraparenchymal intracranial hemorrhage

CT in headache patients may demonstrate an intraparenchymal hematoma[15]. These hematomas may accompany a large variety of diseases including primary and metastatic brain tumors (Figure 7), hypertension with presumed vascular rupture (Figure 8), sympathomimetic drug abuse (e.g., methamphetamines or cocaine), as a complication of AIDS, amyloid angiopathy, bleeding diasthesis/anticoagulation (Figure 9) and parenchymal vascular malformations (Figure 10)[16]. These lesions will typically be referred to interventional neuroradiologists, neurosurgeons and neurologists. These specialists will order (in addition to the initial unenhanced CT study showing the intraparenchymal hemorrhage), further imaging studies, such as contrast-enhanced CTA, MRI done without and with contrast, and/or an angiography in order to establish the diagnosis (Figure 10).

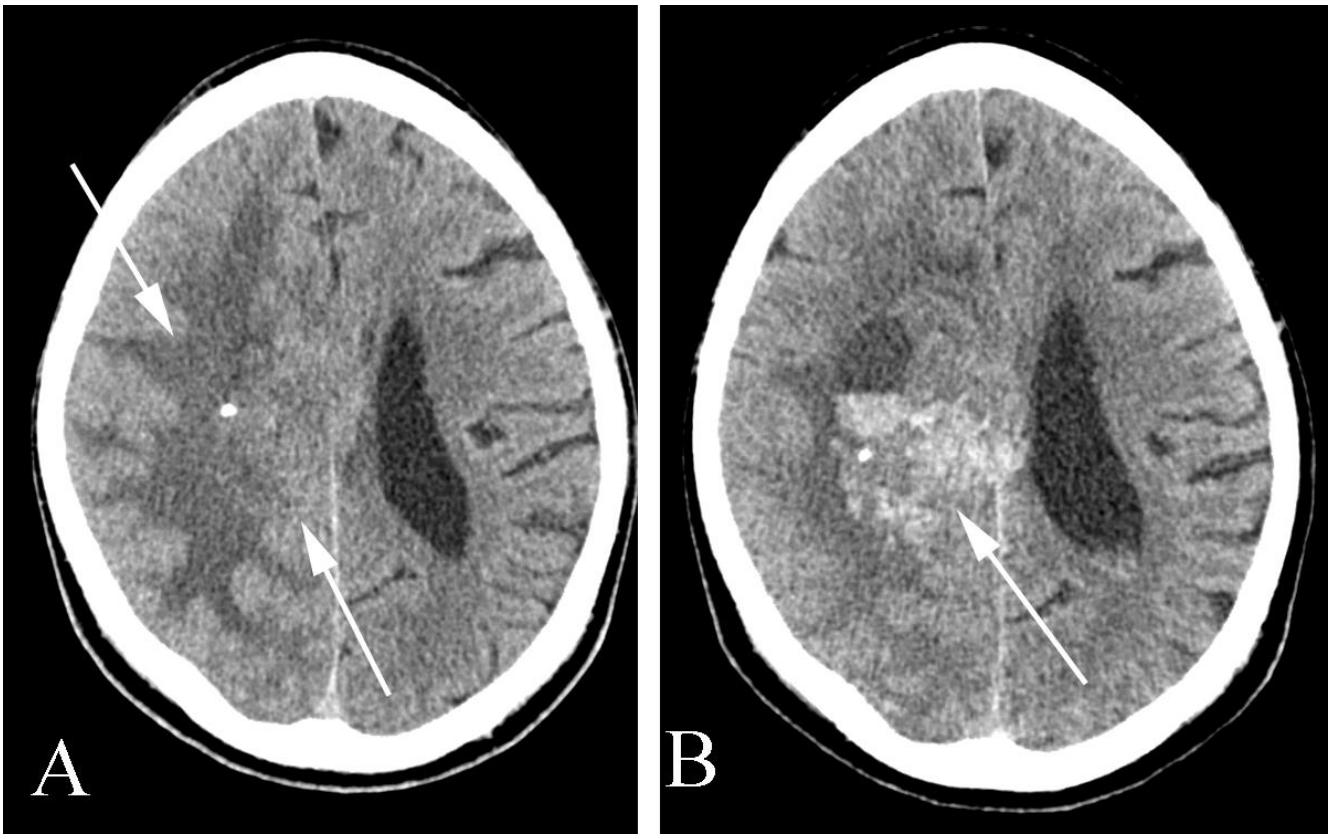

Figure 7. Intraparenchymal hemorrhage in a 40 year old man with a headache and a known right hemisphere oligodendroglioma. A. Axial unenhanced CT done prior to the headache as part of tumor monitoring shows a mass effacing the right lateral ventricle (arrows). B. Axial unenhanced CT done after the onset of a new headache demonstrates hemorrhage into the tumor (arrow).

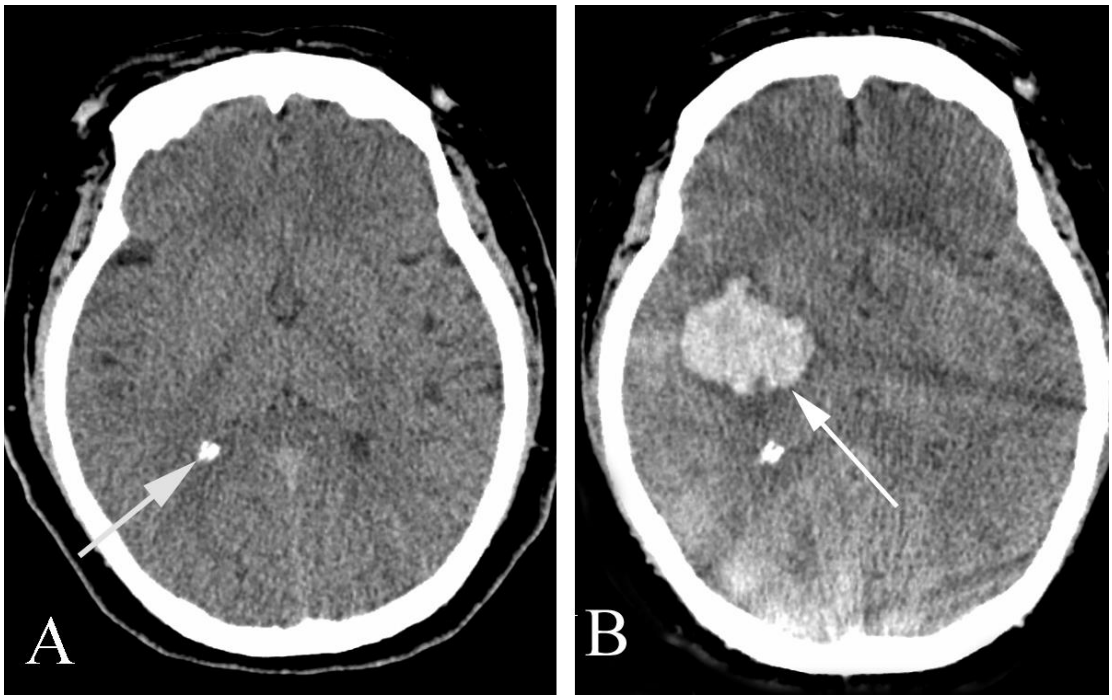

Figure 8. Intraparenchymal hematoma in a 51 year old with headache and slurred speech along with left-sided weakness. A. Axial unenhanced CT study done six months previous to the development of headache and neurologic symptoms demonstrates normal brain parenchyma. Incidentally noted are (normal) calcifications of the choroid plexus in the right occipital horn of the lateral ventricle (arrow). B. Axial unenhanced CT done at the time of the headache and neurologic symptoms demonstrates an acute hemorrhage into the right basal ganglia (arrow).

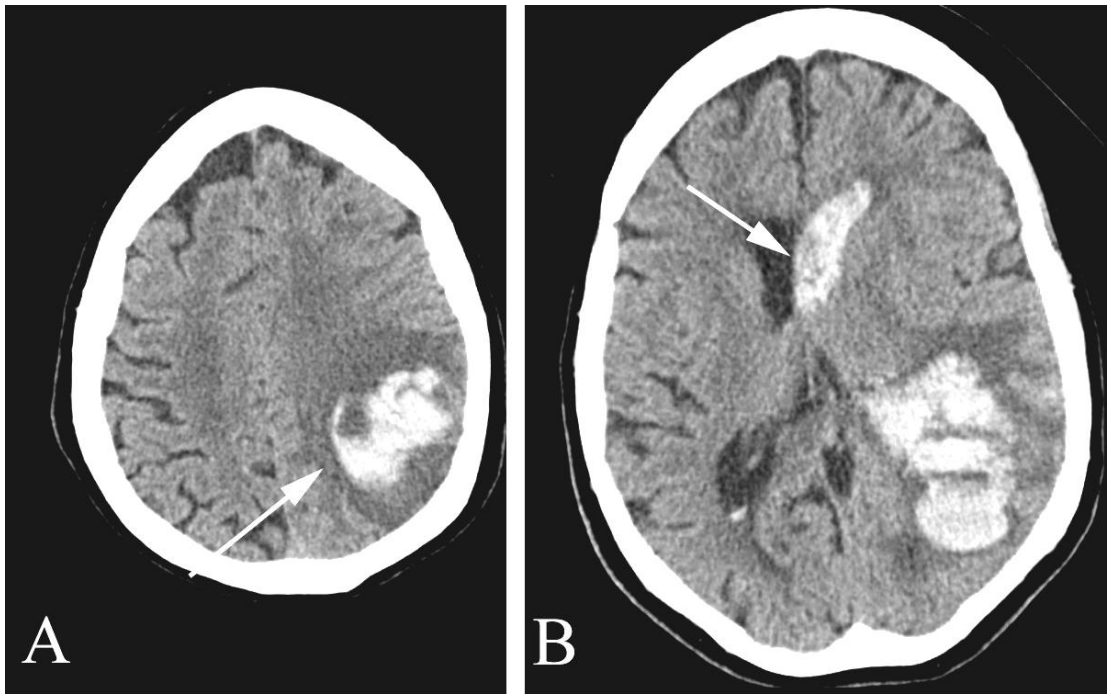

Figure 9. Intraparenchymal and intraventricular hematoma in an anticoagulated 79 year old with headache and acute mental status changes. A. Axial unenhanced CT shows extensive hemorrhage into the posterior left frontal lobe (arrow). B. Axial unenhanced CT demonstrates hemorrhage extending into the parietal lobe and into the lateral ventricle (arrow).

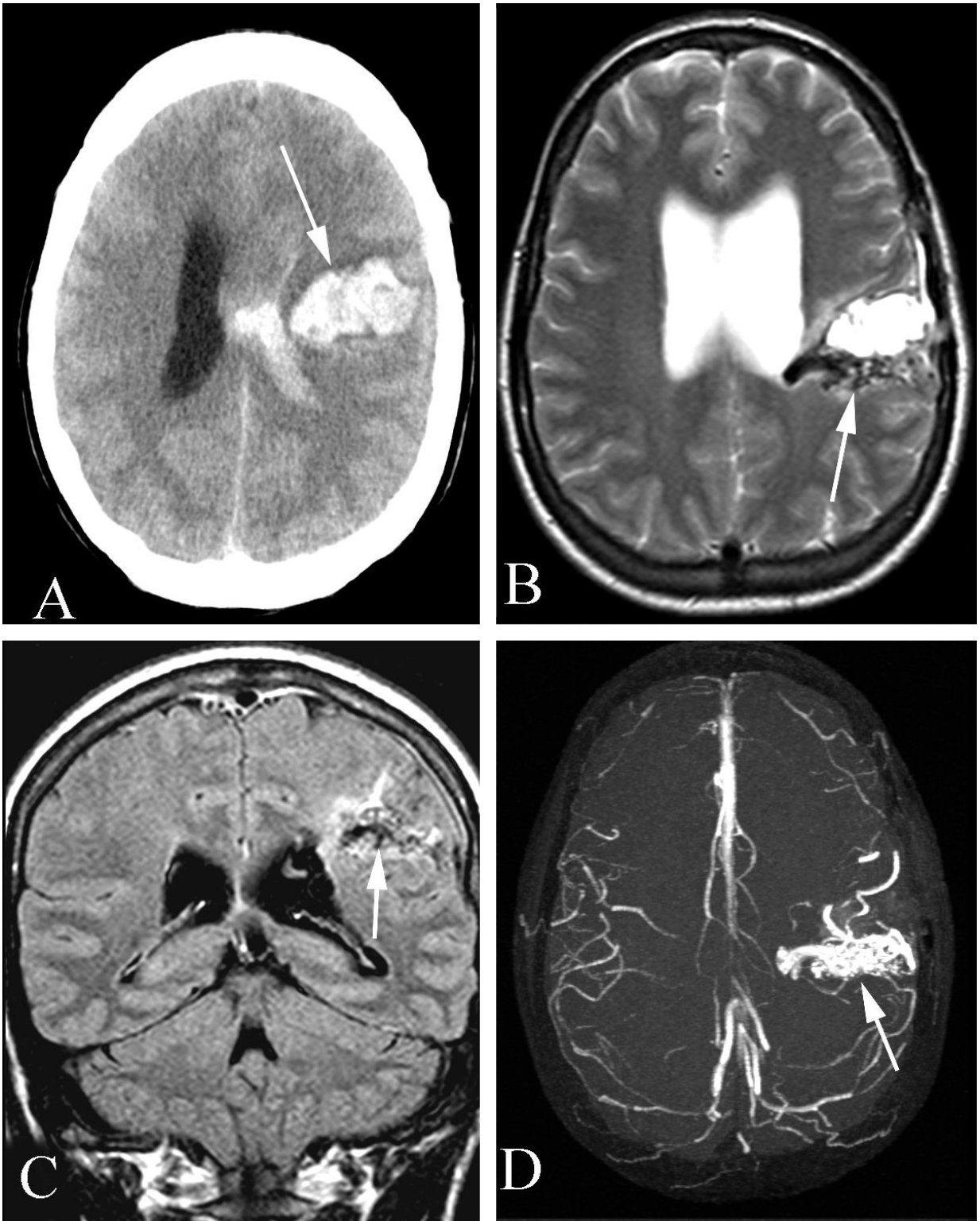

Figure 10. Intraparenchymal and intraventricular hematoma secondary to an intracranial arteriovenous malformation in a 45 year old woman with headache and new onset of left facial droop. A. Axial unenhanced CT demonstrates a left frontal lobe hematoma (arrow) as well as intraventricular hemorrhage. B. An axial T2 weighted MR study demonstrates the hematoma with abnormal vessels along the posterior margin (arrow). C. Coronal T1 weighted postcontrast MR study shows a complex “salt-and-pepper” appearance of the lesion. D. Axial MR angiogram demonstrates multiple abnormal vessels (arrow).

Headache caused by subdural hematoma

CT in headache patients may also demonstrate a subdural hematoma, seen more frequently in the elderly, particularly when anticoagulated or following trauma (Figure 11). The CT features are highly characteristic and diagnostic, and the clinical issue in these patients is whether it is worthwhile to surgically drain the hematoma (typically done through a burr-hole drilled in the calvarium) or to allow the body to resorb the hematoma without intervention.

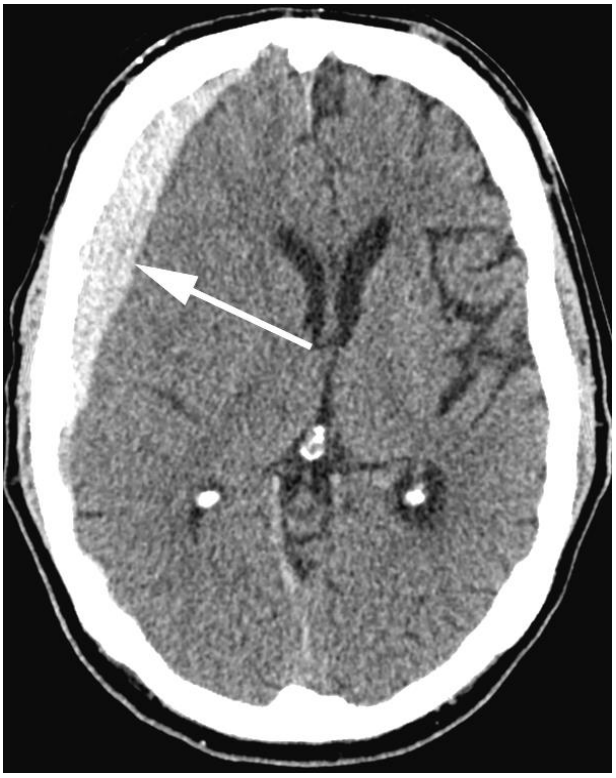

Figure 11. Subdural hematoma in a 66 year old man with headache and slurred speech along with decreased left-sided strength. Unenhanced axial CT study shows an acute subdural hematoma (arrow) compressing the right frontal lobe, and shifting the midline structures.

SECONDARY HEADACHES MAY RARELY BE INSIDIOUS AND MIMIC PRIMARY HEADACHES

While most patients with subarachnoid hemorrhage will present with a thunderclap headache or a similar dramatic event, and most patients with subdural hematomas, brain tumors, and strokes will have some feature in their history or on their physical examination to alert the clinician that they have something other than a primary headache, there are exceptions to this rule (Figure 12). As noted in the first section of this chapter, it is reasonable to not image patients with typical features of primary headache (except for cluster headache). As noted in the second section of this chapter, it is also reasonable to not image those patients with headache with no new or concerning features. Having said this, however, one should also note that headache is a common manifestation of, for example, brain tumor, and that one study of 111 patients with brain tumors found headache in about half. Tension type headache accounted for 77%, migraine type 9%, and other headache types 14% of headaches accompanying brain tumors[17]. Of course, as brain tumors grow, they will eventually produce neurological symptoms. Because of the possibility of a presumed primary headache actually representing a secondary headache, with the headache being secondary to a treatable cause, the decision as to which headache patients to image must ultimately rest with the clinician and the patient, and obtaining an imaging study may be a reasonable course of action for a patient where there is a significant suspicion of a causative lesion. When imaging is done in these cases, the preferred method is magnetic resonance imaging, done without and with contrast material. MR involves no ionizing radiation and is considerably more sensitive than CT to some causes of headache, which is relatively insensitive to certain parenchymal tumors (Figure 12).

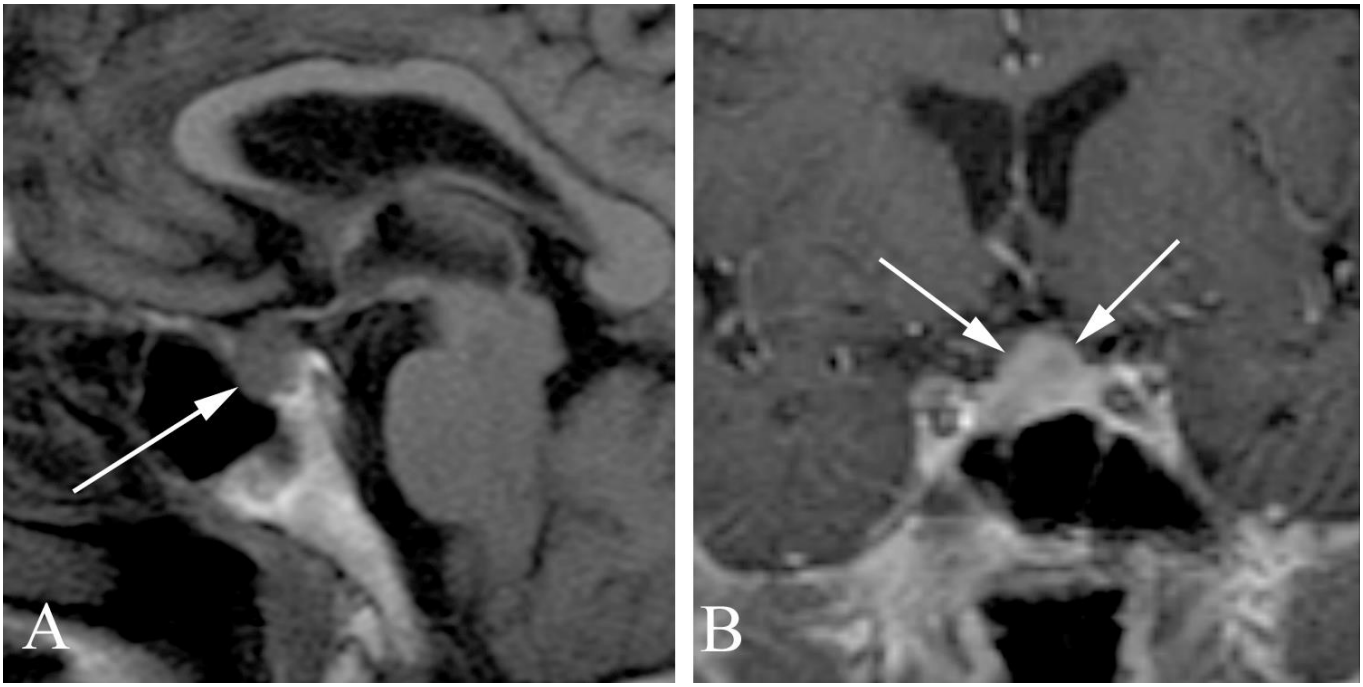

Figure 12. Pituitary adenoma in a 79 year old woman with headache, nausea, and vomiting. A. Sagittal unenhanced T1 weighted MR study shows a 10 mm pituitary adenoma (arrow). B. Coronal T1 weighted postcontrast MR study shows intense enhancement of the pituitary adenoma (arrows).

SUMMARY

Primary care providers see many patients with headaches, and most of these patients do not require imaging, particularly if they have straightforward features of tension or migraine type headaches. Cluster headaches should be evaluated with MR. Patients with headaches secondary to intracranial hemorrhage, tumors, or other processes may demonstrate "danger" signs, the most conspicuous of which is a "thunderclap" headache described as the first or worst headache of the patient's life. Such headaches should undergo immediate imaging, typically with CT, followed by lumbar puncture if the CT shows no hemorrhage or mass effect.

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16 For a unique, first person account of a patient who was herself a neuroanatomist (!) and who suffered an intracranial bleed from an arteriovenous malformation, see My Stroke of Inspiration by Jill Bolte Taylor, Viking Publishers, New York, 2008.

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Stroke, Seizures, Multiple Sclerosis, and Dementia

Donald L. Renfrew, MD

This chapter discusses the diagnosis and imaging of TIA/Stroke, seizures, and dementia. The five main points of this chapter are:

1. Neurologic symptoms need to be placed into one of several broad categories to plan imaging.
2. Both transient ischemic attacks and stroke require aggressive, timely management and work-up.
3. Patients with suspected epilepsy should be sent to a specialist for work-up, and MR should be performed.
4. Patients with possible multiple sclerosis should undergo MRI.
5. Patients with dementia should undergo MRI.

NEUROLOGIC SYMPTOMS NEED TO BE PLACED INTO ONE OF SEVERAL BROAD CATEGORIES TO PLAN IMAGING

Primary care practitioners see many patients who have a neurologic abnormality. Symptoms from these abnormalities may indicate an obvious specific diagnosis, such as the acutely hemiparetic patient from a stroke or the patient who has just suffered a loss of consciousness and tonic-clonic movements from epilepsy. Such patients belong in the emergency room or in the hospital, with imaging as outlined below. Other clear-cut symptoms include patients with likely abnormalities of the cranial nerves or the cranial nerves' associated central nervous system structures (e.g. anosmia, double vision, hearing loss/tinnitus/dizziness), discussed in Chapter 5.

Syncope, an abrupt transient loss of consciousness followed by complete recovery, most frequently represents a vasovagal attack. Other causes include cardiac disease (especially bradyarrhythmia or tachyarrhythmia). Often, syncope remains unexplained and neurologic disease accounts for very few cases[1]. Evaluation of patients with syncope uses primarily (non-imaging) cardiologic tests with neurologic testing of low yield unless there are suspicious neurologic findings (Figure 1)[2]. Patients with syncope following exertion or with accompanying angina should be seen urgently in the emergency room and/or by a cardiologist to exclude a cardiac cause, whereas patients with syncope and dyspnea should undergo urgent computed tomographic angiography of the chest to exclude pulmonary embolism[3] (see page 149).

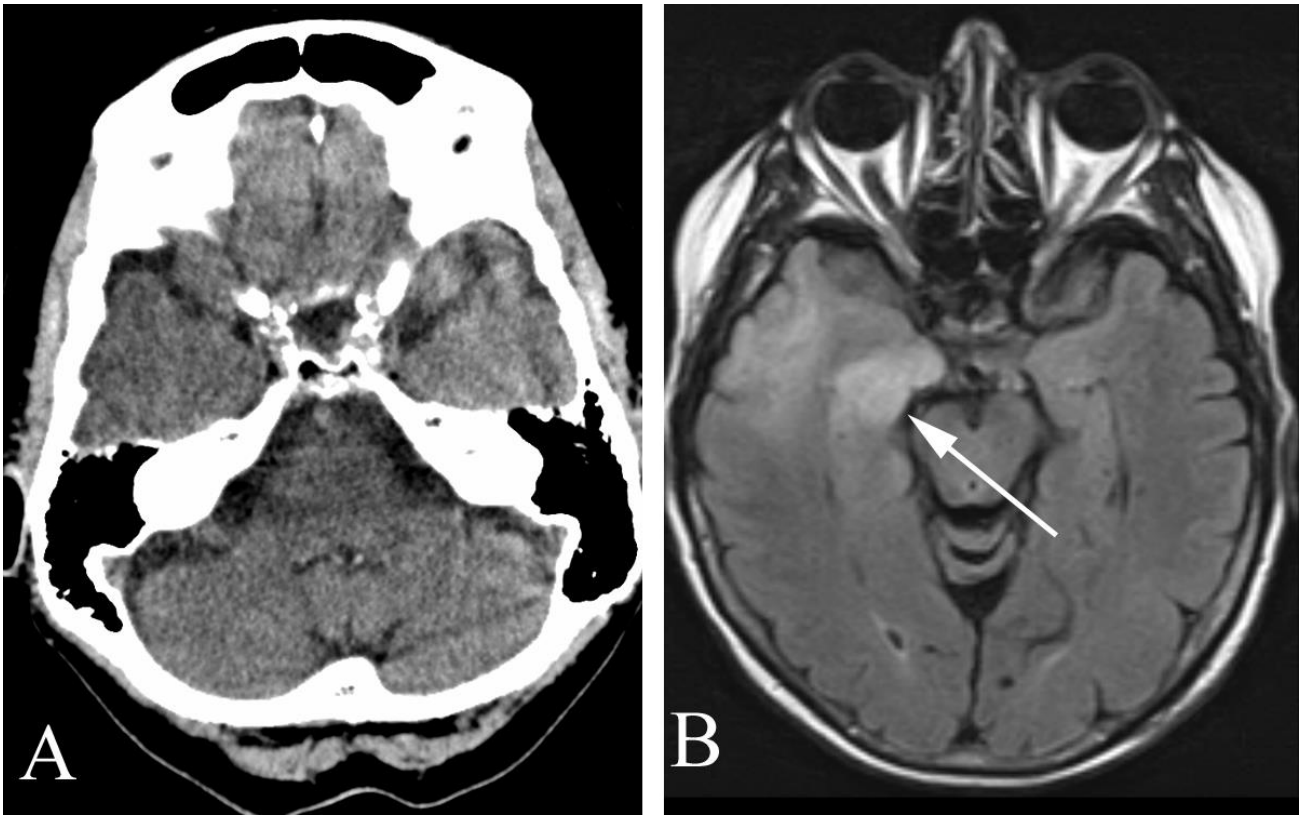

Figure 1. Temporal lobe astrocytoma in a 77 year old woman who experienced syncope. A. Nonenhanced axial CT study shows no abnormality. B. Axial FLAIR MR image demonstrates increased signal intensity along the left temporal lobe (arrow). Astrocytoma was found on biopsy.

Categorizing neurologic symptoms with more subtle or fleeting neurologic findings presents a challenge. Such symptoms often arise from one of a limited number of diseases including transient ischemic attack (TIA)/stroke, seizures, migraine auras (see pages 27-28), and multiple sclerosis. Historical features which help indicate a specific disease include whether the apparent neurologic symptoms are *positive or negative*, the *progression and course* of the symptoms, and the *duration* of the symptoms[4].

Positive or negative symptoms

Examples of positive neurologic symptoms include seeing bright lines or shapes, hearing noises, having a burning sensation or paresthesias, or experiencing jerking or repetitive rhythmic movements. Such symptoms indicate active discharge of central nervous system neurons[4], as will be encountered with seizures or migraine auras.

Negative neurologic symptoms include loss of vision, hearing, cutaneous sensation, or the ability to move a body part, and indicate a loss of neurologic function. Negative symptoms favor a TIA (if transient) or stroke (if fixed), and transient sensory deficits are the most common presentation of patients with multiple sclerosis (MS)[5]. Migraine auras, which often start with positive symptoms, may progress to negative symptoms in the same modality: for example, paresthesias may precede cutaneous numbness[4].

Progression and course of symptoms

The positive neurologic symptoms in seizure typically progress rapidly within a single modality, as do the negative neurologic symptoms of TIA/stroke and MS. The initially positive, then negative symptoms of migraine aura typically slowly progress and may switch from one modality (seeing bright lights) to another (paresthesias). Symptoms of MS characteristically come and go (the

catch-phrase describing the disease is "multiple lesions in time and space").

Duration

Duration of neurologic symptoms ranges from mere seconds to permanent. Seizures represent the shortest duration process with neurologic symptoms lasting from a few seconds up to a few minutes; many TIAs last shorter than five minutes; migraine auras often last 20 to 30 minutes[4]; multiple sclerosis attacks by definition last more than 24 hours[5], and strokes produce long-term and often permanent neurologic symptoms.

BOTH TRANSIENT ISCHEMIC ATTACKS AND STROKE REQUIRE AGGRESSIVE, TIMELY MANAGEMENT AND WORK-UP

The term "transient ischemic attack" (TIA) was originally defined as symptoms or signs of brain ischemia lasting less than 24 hours. The definition has been modified[6], recognizing that the original supposition that neurologic symptoms lasting less than 24 hours were not associated with brain infarction is false. In fact, ischemic symptoms lasting between one and twenty-four hours are often associated with brain infarction[7]. TIAs are currently defined as a transient episode of neurologic dysfunction caused by ischemia without acute infarction[6]. Even though they are defined as not being associated with infarction, TIAs may still represent the harbinger of a subsequent stroke[8], much like a sentinel headache may precede a severe or even fatal aneurysmal subarachnoid hemorrhage (see pages 32-34). For this reason, TIAs require urgent work-up and management, either within the hospital or on a very closely monitored outpatient basis[8].

Clinical risk assessment for impending stroke after TIA

Johnston et al[9] used a scoring system based on: age (>60 years = 1 point); blood pressure elevation when first assessed after TIA (systolic > 140 mmHg or diastolic > 90 mmHg = 1 point); clinical features (unilateral weakness = 2 points; isolated speech disturbance = 1 point); duration of TIA (> 60 minutes = 2 points, 10-59 minutes = 1 point; < 10 minutes = 0 points); and diabetes (present = 1 point) to stratify risk, with the estimated 2-day risk of stroke at 8.1% for scores of 6 or 7, 4.1% for scores of 4 or 5; and 1.0% for scores of 3 or less.

Imaging of TIA

The goals of imaging in a patient with an apparent TIA include: to exclude intracranial hemorrhage (more typically accompanied by a fixed neurological defect, headache, or both – see page 34 for further discussion of intracranial hemorrhage associated with headache); to evaluate for a possible alternative explanation of the neurologic symptoms such as brain tumor (Figure 2); to document any actual infarct accompanying the apparent TIA (which would, by definition, indicate that the transient symptoms do not, in fact, represent a TIA) (Figure 3); and to evaluate a (usually vascular) source of the TIA, including disease of the carotid bifurcations, intracranial vasculature, and heart (Figure 4). While CT may be used in the emergent setting to exclude hemorrhage, TIA patients by definition have no ongoing symptoms and should therefore be capable of undergoing MRI examination, which should be performed both without and with contrast, and which should include diffusion weighted imaging (DWI) sequences (Figure 3). DWI sequences will typically demonstrate cerebral infarction within minutes of onset, and are typically positive hours before T1 and T2 weighted sequences[10]. MRI examination performed with gradient echo sequences is also capable of detecting intracranial hemorrhage.

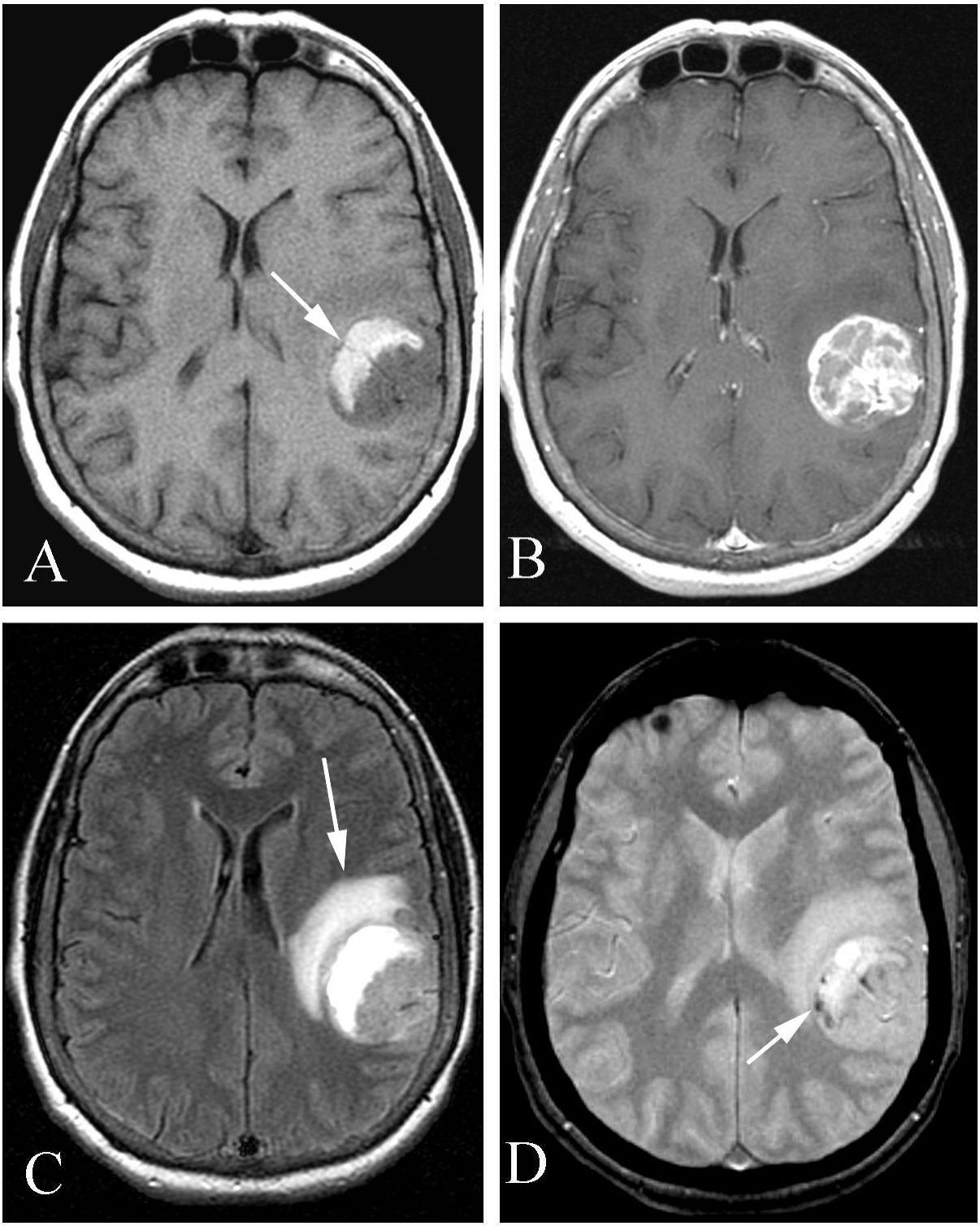

Figure 2. Glioblastoma multiforme in a 57 year old woman with transient verbal difficulty initially thought to be ischemic. A. Axial unenhanced T1 weighted image demonstrates a mass in the left temporal lobe with increased signal intensity along the anteromedial margin compatible with recent hemorrhage (arrow). B. Axial T1 postcontrast image demonstrates intense contrast enhancement of the lesion. C. Axial FLAIR image demonstrates the lesion and marked adjacent white matter vasogenic edema (arrow). D. Axial T2 image demonstrates focal areas of decreased signal intensity (arrow) compatible with intratumoral hemorrhage.

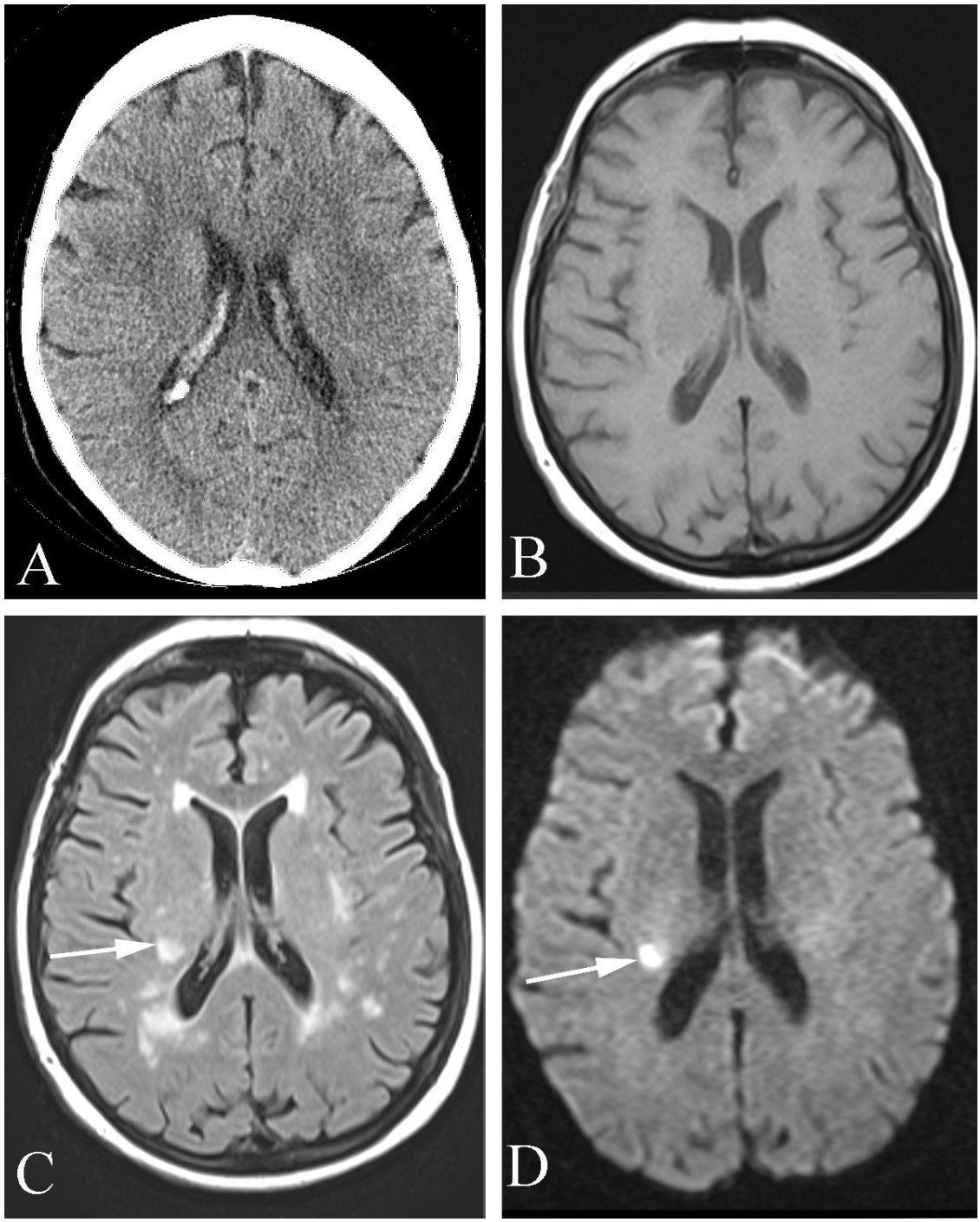

Figure 3. Stroke in an 85 year old woman with transient weakness six days prior to MR study. A. Axial unenhanced CT study shows no abnormality. B. Axial T1 MR weighted image also shows no abnormality. C. Axial FLAIR MR image demonstrates several focal areas of increased signal intensity, including one in the right cerebral hemisphere white matter (arrow). D. Axial diffusion weighted MR image demonstrates increased signal intensity indicating diffusion restriction (arrow) at the location of the lesion noted on image C., whereas the other white matter lesions seen on image C. show no diffusion restriction. These imaging findings indicate an acute infarction, and even through the patient's symptoms completely resolved she did not, by definition, have a TIA. The other areas of increased signal in C. likely represent chronic microvascular ischemic changes.

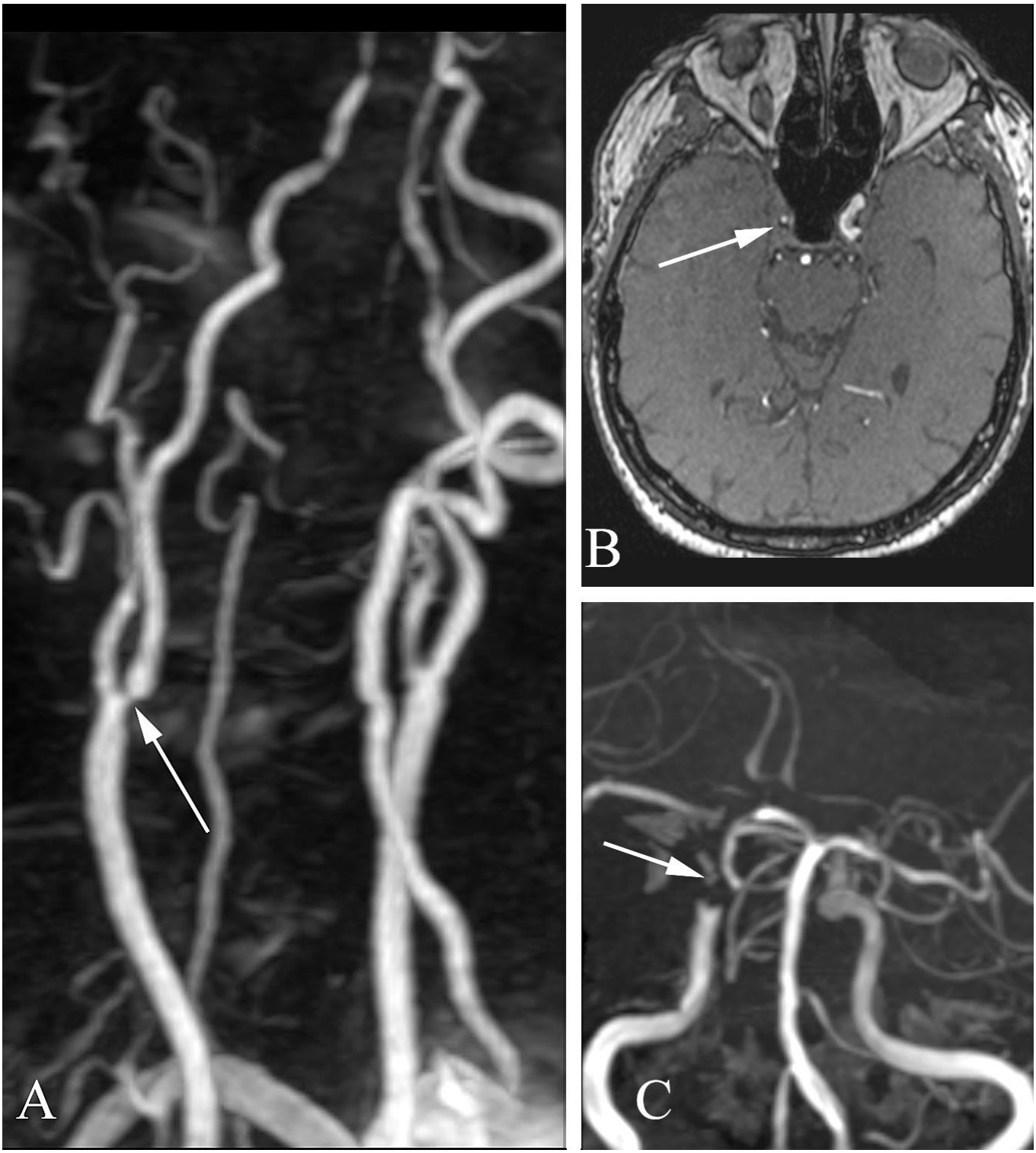

Figure 4. Extensive vascular disease in a 74 year old man with hypertension and a 2 minute episode of dizziness and sweating. Brain MR study (not shown) was normal. A. Arch and carotid MR angiogram shows multiple irregularities in the vascular tree including an approximately 75% stenosis at the origin of the right internal carotid artery (arrow). B. Axial magnetic resonance angiogram source images from the circle of Willis study demonstrates nearly complete occlusion of the right intracranial internal carotid artery (arrow). C. MR angiogram maximum intensity projection of the circle of Willis also demonstrates severe stenosis of the intracranial internal carotid artery (arrow).

Magnetic resonance imaging following TIA may include a magnetic resonance angiogram (MRA) for evaluation of the arch and carotid arteries (typically performed with contrast material, and using the same bolus of contrast as the contrast-enhanced brain MRI), and imaging of the circle of Willis(also known as MRA-COW which may be performed either without or with contrast material) (Figure 4). For patients who cannot undergo MRI (because of aneurysm clips, pacers, retained metallic foreign bodies within the orbit, etc.), CT of the brain without and with contrast, and CT angiography of the arch, carotids, and circle of Willis may be performed. Ultrasound examination of the carotid arteries may also be used to screen for carotid stenosis, ulceration, dissection, hematoma, and aneurysms. For further discussion of vascular evaluation of the arch, carotid arteries, and intracranial vasculature, see page 170. Transthoracic or transesophageal echocardiography (with greater sensitivity) evaluates for cardiac sources of emboli causing TIAs.

Imaging of Stroke

Patients with obvious stroke belong in an emergency room or in the hospital. The critical decision regarding treatment of these patients is whether to administer fibrinolytic therapy as soon as possible[11], at present limited to recombinant tissue-type plasminogen activator or tPA (alteplase). ER physicians and neurologists in stroke centers usually make this decision on the basis of multiple criteria including: duration of symptoms of less than 4.5 hours; historical exclusion criteria (stroke or head trauma in the previous 3 months, previous intracranial hemorrhage, major surgery in the prior 14 days, etc.); clinical exclusion criteria (spontaneously clearing stroke symptoms, minor or isolated neurologic signs, persistent blood pressure elevation, etc.); laboratory exclusion criteria (platelets < 100,000 cc^3, serum glucose < 50 mg/dl, INR > 1.7, etc.); and CT exclusion criteria (evidence of hemorrhage or evidence of multilobar infarction with hypodensity involving greater than 33% of the cerebral hemisphere (Figure 5)). Given the multiple exclusion criteria, few patients are truly eligible for alteplase. For those who are eligible, the increased chances of a complete recovery (38% versus 21% with placebo) must be weighed against the approximately 10-fold increase in symptomatic intracerebral hemorrhage[12].

Even more controversial than the intravenous administration of alteplase within 4.5 hours is the administration of IV alteplase after this time-limited therapeutic window (based on imaging studies) or intra-arterial administration of the same drug (also based on imaging studies). In explanation: both contrast-enhanced CT and contrast-enhanced MR are capable of creating perfusion maps of the brain following stroke. These maps differentiate *infarcted* brain (incapable of recovery) from *stunned but not infarcted* brain, which is at least theoretically capable of recovery. Preventing infarction of stunned brain is the purpose of administering alteplase and some centers use either CT or MR perfusion imaging to supplement the usual exclusion rules and time from the acute event to make decisions regarding intravenous alteplase[10]. In addition, both CTA and MRA may be performed acutely to delineate intra-arterial thrombus, which (particularly if accompanied by stunned but not yet infarcted brain) forms the target of intra-arterial alteplase.

Perfusion imaging studies to evaluate patients for possible alteplase therapy (either intravenous or intra-arterial) are typically done emergently within the setting of a stroke center. In patients who are not candidates for alteplase, CT still needs to be performed to evaluate for intracranial hemorrhage. In addition, the same MR imaging considerations listed for patients with TIAs above apply to patients with stroke: MR imaging of the brain without and with contrast should be performed to document the stroke (Figure 6) and exclude alternative diagnoses (Figure 7); vascular imaging should be performed, including *either* MRA of the arch and carotids and MRA-COW (Figure 6) *or* CTA of the arch and carotids and COW *or* ultrasound of the carotids; and echocardiography should be performed to exclude a cardiac source of embolism.

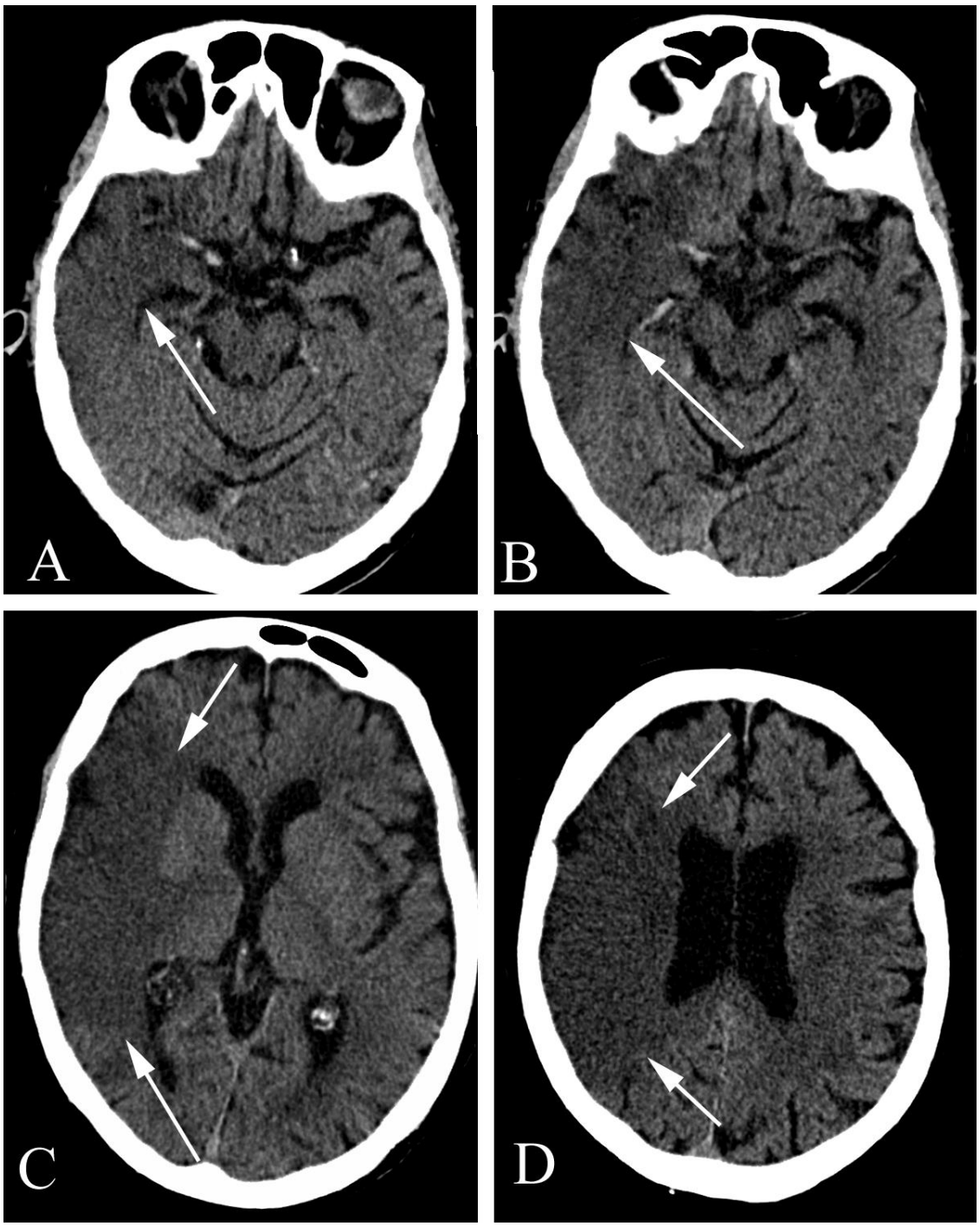

Figure 5. Huge acute cerebral stroke in an 88 year old woman who woke with left hemiparesis. A. Unenhanced axial CT at the level of the suprasellar cistern demonstrates hypodensity and effaced sulci in the right temporal lobe (arrow). Note the calcified vascular tree. B. Unenhanced axial CT superior to A. also demonstrates hypodensity and effaced sulci (arrow). C. Unenhanced axial CT at the level of the basal ganglia demonstrates a large area of the right cerebral hemisphere (basically the entire distribution of the right middle cerebral artery) with effaced sulci and hypodensity (arrows). D. Unenhanced axial CT through the mid lateral ventricles shows extension of the large, acute infarct through the cerebral convexity. Such a large acute stroke, involving more than 33% of the hemisphere, contraindicates fibrinolytic therapy for stroke.

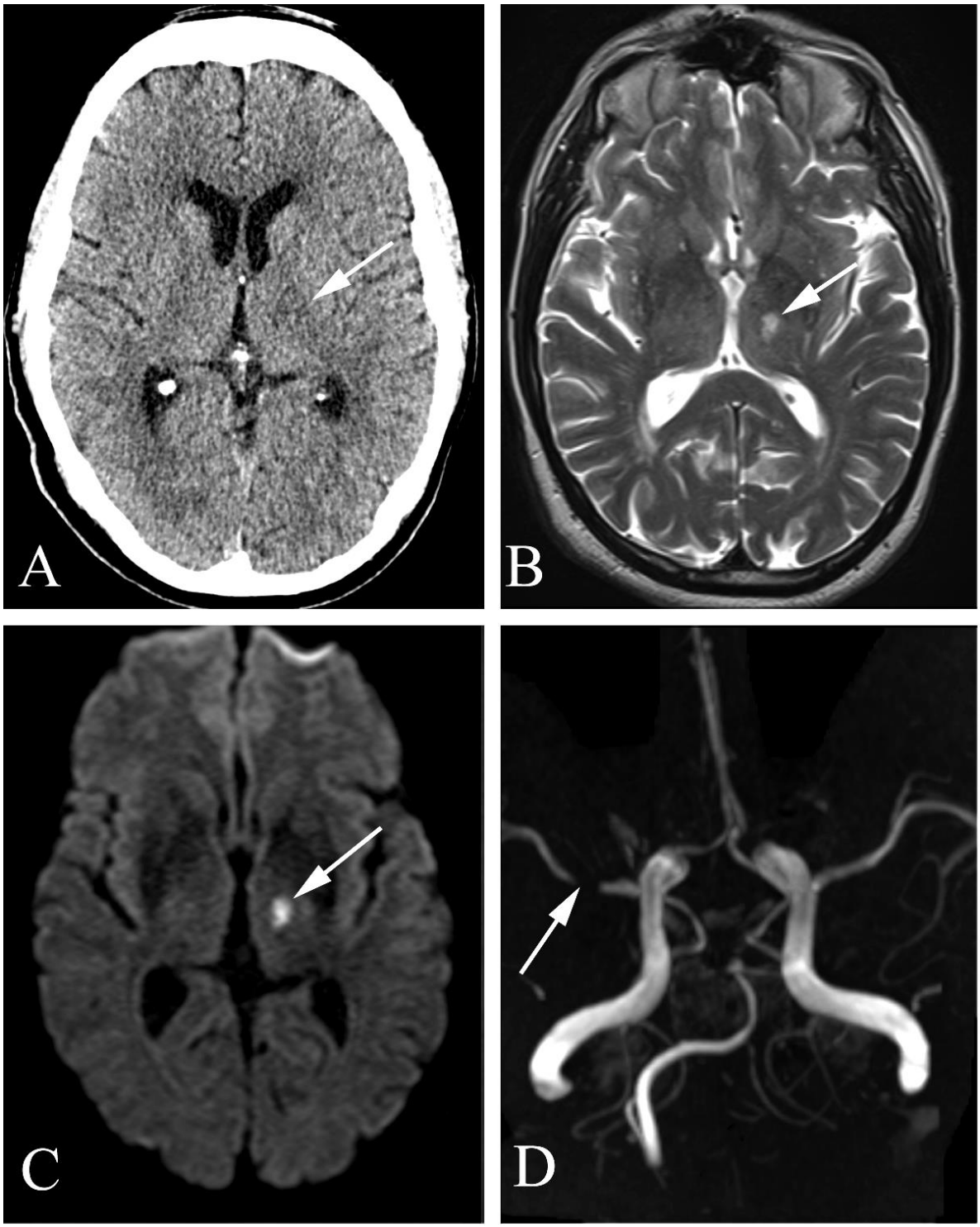

Figure 6. Acute left thalamic stroke in a 76 year old man with acute dizziness. A Unenhanced axial CT demonstrates a subtle hypodensity in the left thalamus (arrow). B. Axial T2 weighted MR image showing focal increased signal intensity in the left thalamus (arrow) C. Axial diffusion weighted MR image demonstrates increased signal indicating restricted diffusion (arrow). D. Circle of Willis magnetic resonance angiogram maximum intensity projection shows severe stenosis of the contralateral *right* middle cerebral artery, documenting severe vascular disease at a location other than that causing the patient's acute stroke.

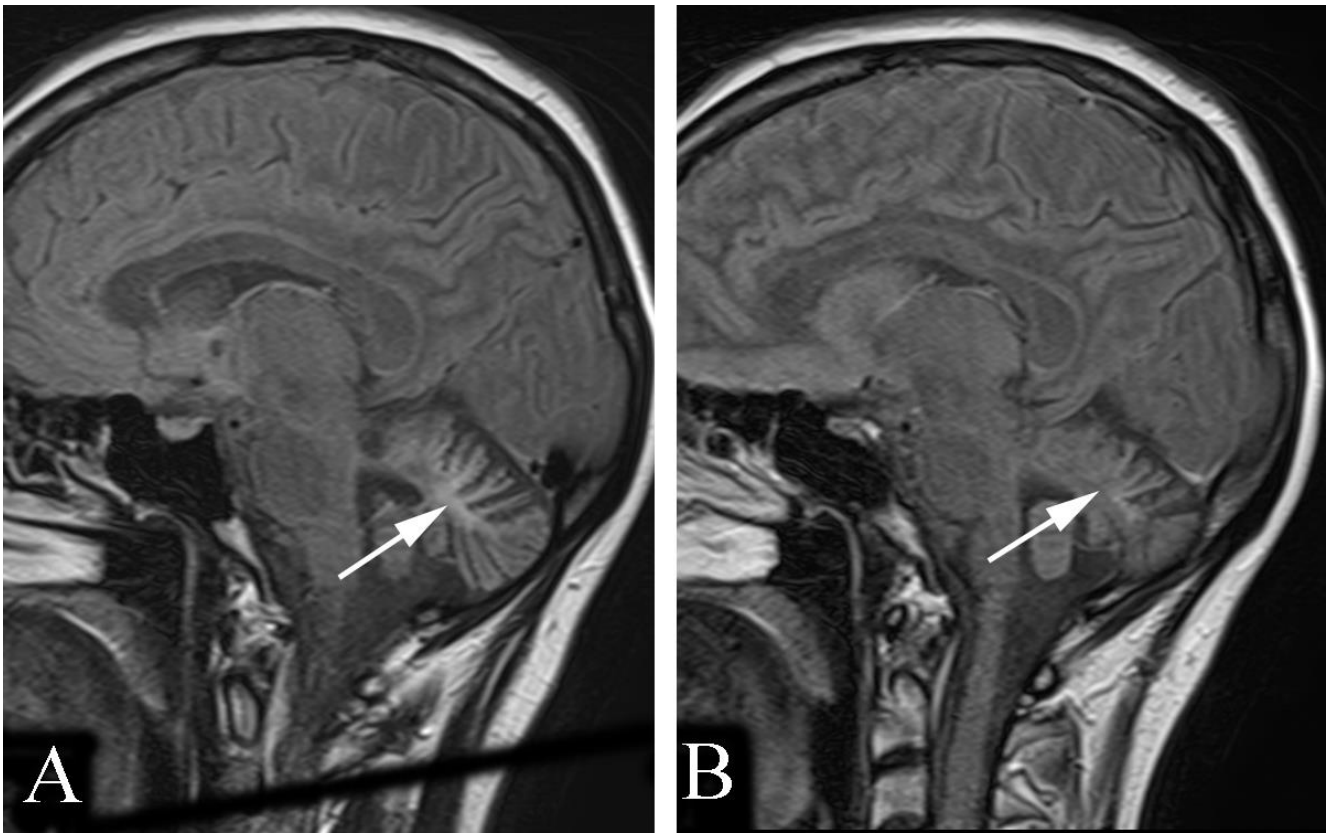

Figure 7. Viral cerebrellitis in a 48 year old woman with unsteady gait, dizziness, and blurred vision. A Sagittal magnetic resonance FLAIR image through the right cerebellar hemisphere shows abnormal signal (arrow). B. Sagittal magnetic resonance FLAIR image through the contralateral cerebellar hemisphere shows much less abnormal signal (arrow). The patient had viral meningitis six years before the onset of symptoms.

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PATIENTS WITH SUSPECTED EPILEPSY SHOULD BE SENT TO A SPECIALIST FOR WORK-UP, AND MR SHOULD BE PERFORMED

Seizures may be the result of a reversible medical disorder or epilepsy. Multiple medical disorders may provoke seizure, including: hypoglycemia; nonketotic hyperglycemia; rapid falls in serum sodium concentration; hypocalcemia; renal failure/uremia; hyperthyroidism; acute intermittent porphyria; cerebral anoxia (e.g. carbon monoxide poisoning, drowning), and drug toxicity or withdrawal[13]. Initial testing should be directed toward excluding such medical disorders. In the acute setting following the first seizure, a head CT will usually be obtained mainly to exclude intracranial hemorrhage, brain abscess, and tumor (Figure 8)[14].

Assuming that medical disorders have been largely or completely excluded and epilepsy remains the likely diagnosis in a patient with seizure, most primary care practitioners refer the patient to specialists or subspecialists for further evaluation[13], since epilepsy is relatively rare, the diagnosis has such a significant impact, and treatment is often life-long. With respect to imaging epilepsy, the goal is to find a structural cause of the epilepsy, particularly in those cases where surgery is contemplated secondary to failed medical therapy. Structural causes include hippocampal sclerosis, brain tumor, dysplasia, and vascular malformations[15]. Knake et al[16] showed that imaging performed on a high field strength magnet (3.0T rather than 1.5T) depicted causative lesions with much greater sensitivity and

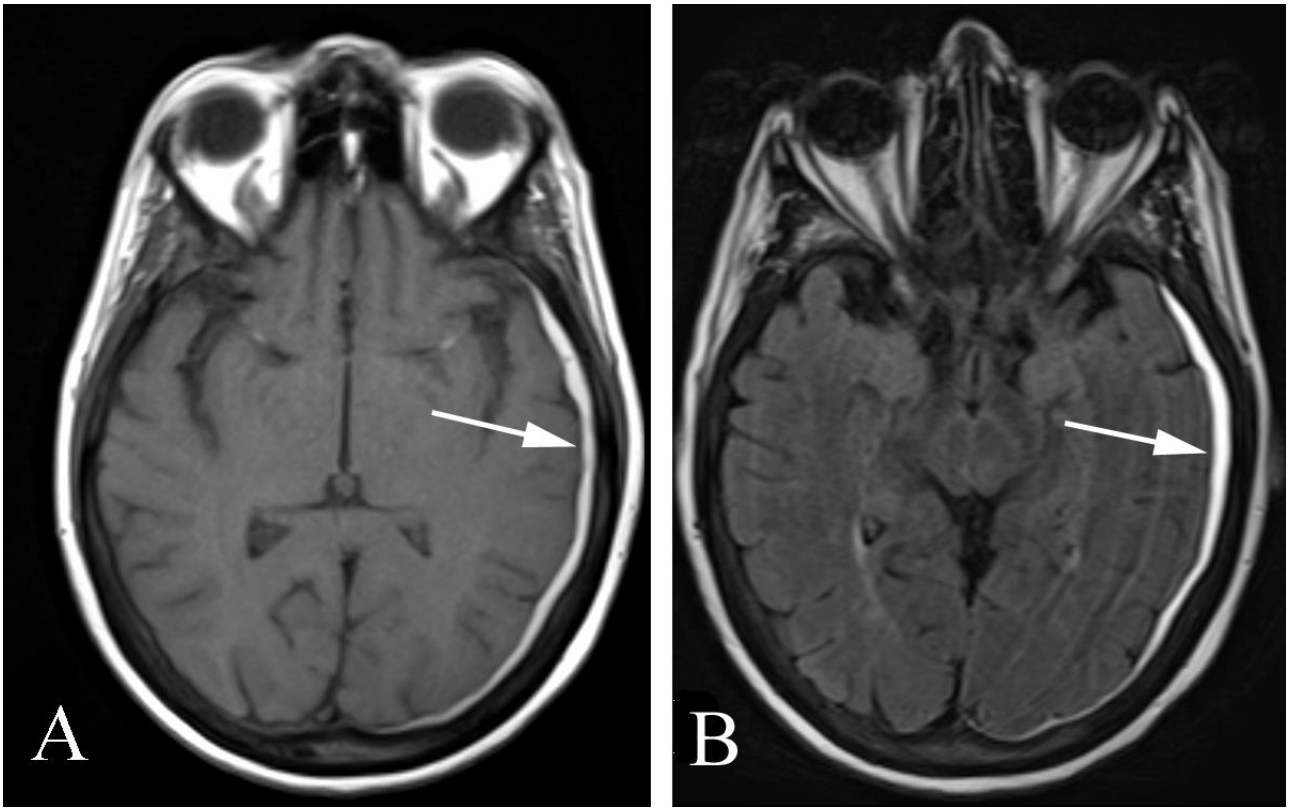

Figure 8. Subdural hematoma in a 60 year old anticoagulated woman with new seizures. A. Axial unenhanced T1 weighted MR image shows a crescent of increased signal along the left cerebral hemisphere (arrow), typical of a subdural hematoma. B. Axial FLAIR image also demonstrates a crescent of increased signal along the left cerebral hemisphere (arrow).

accuracy: in a subgroup of 23 patients with a normal interpretation at 1.5T, new lesions were detected on a 3.0T study in 15 (63%). Phal et al[17] found that epilepsy imaging performed on a 3.0T MRI showed increased imaging quality, detected more structural lesions, and improved characterization of lesions compared to 1.5T. While imaging at 1.5T may be diagnostic in some cases, it is not possible to predict which cases will be falsely negative or equivocal. Given the necessity to re-image with a 3.0T MR in negative or equivocal cases, direct referral for initial imaging at 3.0T performed at an epilepsy center may be preferred when feasible.

PATIENTS WITH POSSIBLE MULTIPLE SCLEROSIS SHOULD UNDERGO MR

While the hallmark of multiple sclerosis (MS) is multiple lesions in time and space (neurologic location), most patients (approximately 85%) will *initially* present with a clinically isolated syndrome[18], usually one of the following:

1. Transient sensory or motor deficits, affecting 40-50% of patients[5].
2. Monocular visual loss or visual field loss from optic neuritis, affecting 15-20% of patients[19].
3. Diplopia, affecting about 7% of patients.
4. Balance problems and/or vertigo, affecting about 5% of patients.

Patients who present with a clinically isolated syndrome should undergo contrast-enhanced MR imaging[20]. While diagnosis of MS was at one time confirmed using the Poser criteria, which require at least two clinical episodes, waiting for a second

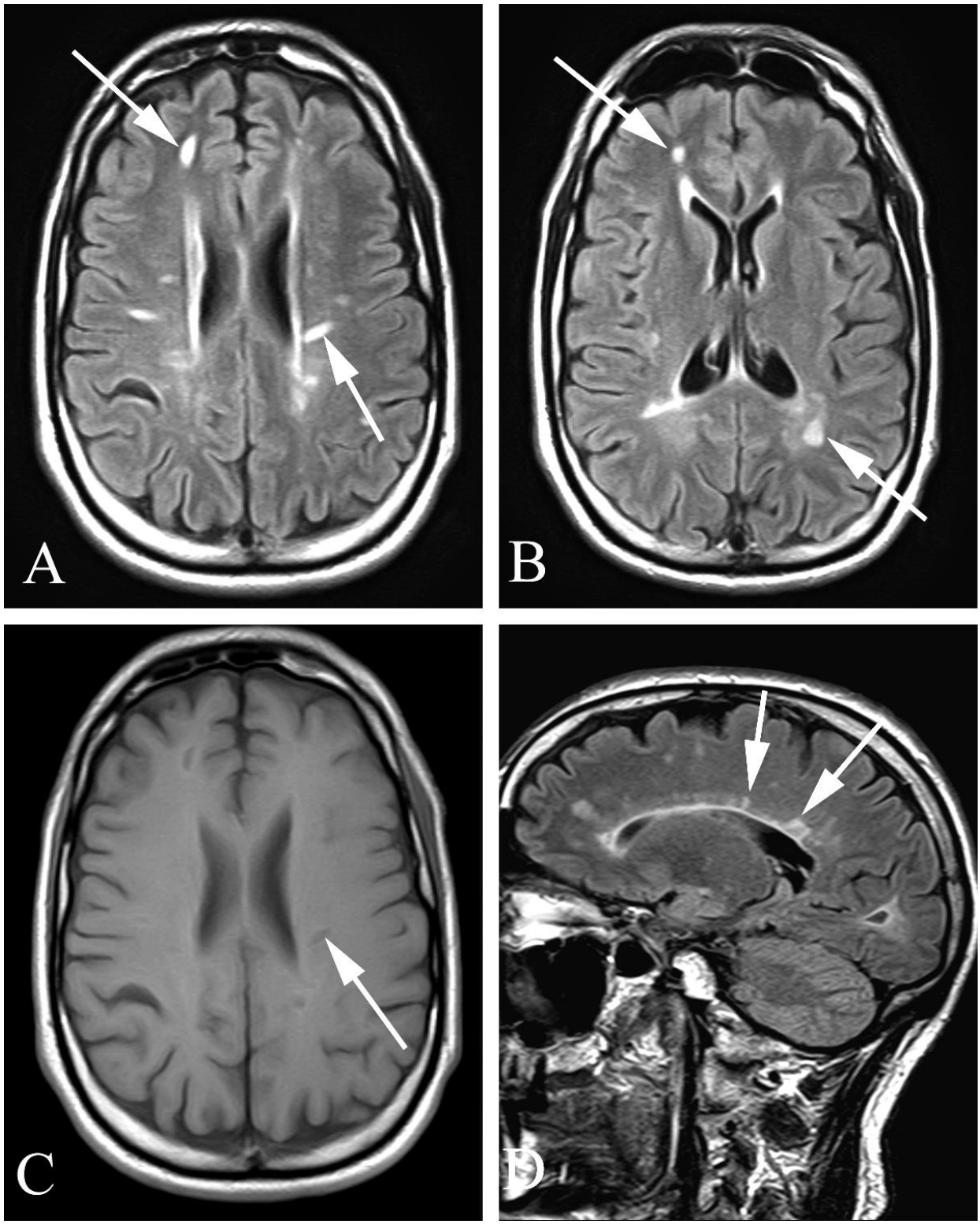

Figure 9. Multiple sclerosis in a 52 year old man with new onset poor coordination of the lower extremities. A Axial FLAIR brain MR shows multiple foci increased signal intensity (arrows). B. Axial FLAIR brain MR at a slightly lower location again shows multiple foci of increased signal intensity (arrows). C. Axial T1 weighted image shows decreased signal intensity at the location of the increased signal on B. D. Sagittal FLAIR brain MR shows multiple lesions of the corpus callosum (arrows), typical of multiple sclerosis.

episode is no longer acceptable since there is disease-modifying therapy[5]. Therefore, the McDonald criteria are used instead of the Poser criteria. The McDonald criteria allow a diagnosis of multiple sclerosis with one clinical attack and *either* abnormal cerebrospinal fluid protein with oligoclonal bands *or* an abnormal MRI, with three out of the following four MRI abnormalities: one gadolinium enhancing or nine T2-hyperintense lesions (if no gadolinium enhancing lesion is seen); one or more infratentorial lesions; one or more juxtacortical lesions; and three or more periventricular lesions[21]. As indicated in the McDonald criteria, MR abnormalities associated with multiple sclerosis include areas of increased signal on T2 weighted images (Figure 9), which sometimes show matched decreased signal on T1 weighted images and contrast enhancement. These are, of course, not specific findings and similar imaging features may be seen in acute disseminated encephalomyelitis, vasculitis, Lyme disease, and migraine headache. Systemic lupus erythematosis may not only demonstrate similar lesions at MR, but occasionally presents with recurrent neurologic symptoms prior to the systemic manifestations of the disease, complicating diagnosis[20].

Patients with multiple sclerosis may be followed with serial MR examinations to document progression or regression of lesions in response to therapy. Enhancing lesions indicate breakdown of the blood-brain barrier, which is generally taken as a proxy for "inflammation" and disease activity[22]. Several maneuvers may document additional contrast-enhancing lesions compared to standard technique (including magnetization transfer pulse sequences, triple-dose contrast, and delayed imaging), but such techniques are not routinely required and are not incorporated into MS diagnostic criteria[22].

PATIENTS WITH DEMENTIA SHOULD UNDERGO MR

The fourth edition of the *American Psychiatric Association Diagnostic and Statistical Manual* (also known as "DSM-IV") defines dementia as a disorder characterized by impairment of memory and at least one other cognitive domain (aphasia, apraxia, agnosia, or executive function) which represents a decline from a prior level of function severe enough to interfere with daily function and independence[23]. Self-reported memory loss does not appear to correlate with the development of dementia, while spouse (or other informant) reported memory loss is a much better indicator of the presence (or future development) of dementia[24]. The diagnosis typically rests on a clinical history, supplemented by cognitive tests such as the Mini-Mental State Examination[25], Clinical Dementia Rating[26], the "mini-cog" test[27] or formal neuropsychologic testing[24].

Major dementia syndromes include Alzheimer's disease (accounting for 60 to 80% of the total), vascular dementia, dementia with Lewy bodies, dementia with Parkinson's disease, and frontotemporal dementia[28]. Normal pressure hydrocephalus (NPH) may also be associated with dementia (along with gait disturbance and urinary incontinence), and is characterized by pathologically enlarged ventricles with normal opening pressure on lumbar puncture[29]. This diagnosis may be confirmed by the patient's clinical response to removal of 30 – 50 mL of cerebrospinal fluid, although there is little consensus regarding the diagnosis of NPH or the selection of patients with possible NPH for therapeutic shunt placement[29].

The American Association of Neurology recommends imaging with either CT or MRI in the routine initial evaluation of all patients with dementia[30]. MR imaging (preferred to CT) can accomplish the following in patients with newly diagnosed dementia:

1. Exclude subdural hematoma.
2. Exclude cerebral neoplasm.
3. Evaluate for disproportionate distention of the lateral ventricles relative to the sulci, suggestive of normal pressure hydrocephalus (NPH) (Figure 10).
4. Evaluate for disproportionate frontal lobe atrophy, suggesting frontotemporal dementia.
5. Evaluate for multiple prior strokes suggesting vascular dementia.

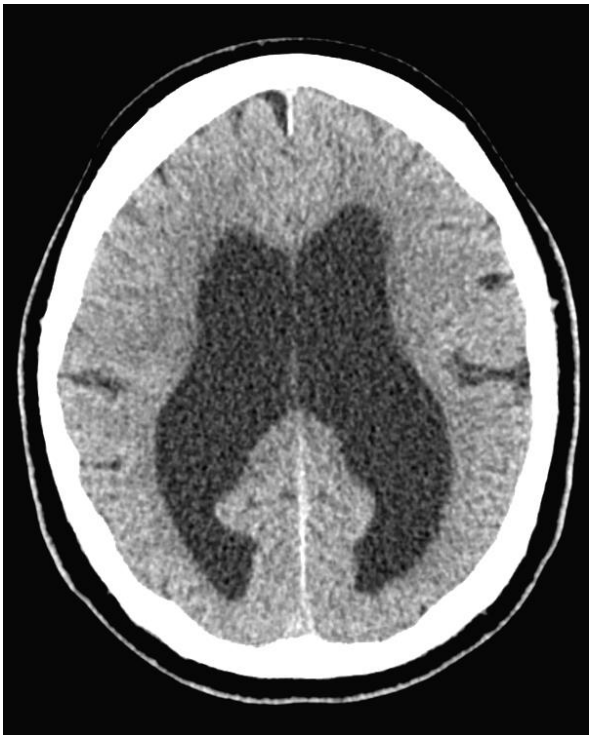

Figure 10. Normal pressure hydrocephalus in a 78 year old with dementia, incontinence, and gait abnormality. Axial unenhanced CT study shows bilaterally enlarged ventricles with disproportionate distension compared to the sulci.

Most MR studies will demonstrate nonspecific generalized atrophy, since this is the most common finding in Alzheimer's disease, and Alzheimer's disease accounts for the majority of dementias. While MR may also allow hippocampal volume measurement in Alzheimer's disease, it is not clear that this finding adds to the clinical diagnosis[31]. FDG-PET may be useful in distinguishing Alzheimer's disease from frontotemporal dementia, but typically there is little therapeutic imperative to make this distinction[31].

SUMMARY

Neurologic symptoms may indicate migraine aura, TIA/stroke, seizure, or multiple sclerosis. TIA and stroke require urgent work-up, seizures require specialist assessment and MR imaging (preferably at 3.0T if possible), and multiple sclerosis requires MR scanning. Dementia patients should undergo MR imaging upon initial diagnosis.

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Cranial Nerves, Sinuses, and Neck Masses

Donald L. Renfrew, MD

This chapter reviews imaging of symptoms related to the cranial nerves (or the central nervous system structures associated with the cranial nerves) and paranasal sinuses. It also reviews imaging of neck masses. The three main points of this chapter are:

1. Symptoms of cranial nerve abnormality may require MRI of the brain.
2. Patients with symptoms of sinusitis (purulent nasal discharge and/or facial pain/pressure) usually do not require imaging. CT is the study of choice when imaging is necessary.
3. Palpable thyroid lesions can undergo FNAB without imaging, whereas nonthyroid neck masses typically require CT scanning prior to biopsy.

SYMPTOMS OF CRANIAL NERVE ABNORMALITY MAY REQUIRE MRI OF THE BRAIN

Evaluation of patients with symptoms which may be related to the cranial nerves starts with a history and physical examination directed toward deciphering whether the symptoms are actually arising from the cranial nerve itself or because of an abnormality of the brainstem (or elsewhere in the brain). As a generalization, isolated involvement of a single cranial nerve is likely to be caused by either intrinsic dysfunction of the nerve (typically demonstrating either no imaging findings or manifesting as contrast enhancement along the nerve on an MRI study) or occasionally by a mass compressing the nerve. Symptoms from multiple cranial nerves or other additional symptoms (headache, pain, non-cranial nerve neurologic abnormalities) that suggest a brain abnormality require a brain MRI done without and with contrast. The routine brain sequences may be supplemented with thin cuts through the pituitary gland (for cranial nerves II - VI) or posterior fossa (for cranial nerves VII – XII) after intravenous contrast. The following paragraphs further discuss symptoms arising from each of the cranial nerves.

Cranial Nerve I symptom: Anosmia

Gradual onset of loss of smell may accompany sinus disease, allergic rhinitis, or dementia, while sudden onset of loss of smell may be secondary to head injury or viral infection[1]. Patients with acute onset of anosmia should probably be referred to an otolaryngologist for examination of the nasal cavity and paranasal sinuses. Imaging studies may include CT of the paranasal sinuses if there are symptoms of sinusitis (see below) and/or brain MR if there are symptoms of central nervous system disease.

Cranial Nerve	Major Function(s)	Associated Symptoms
I – olfactory	Smell	Anosmia
II – optic	Vision	Decreased or absent vision
III – oculomotor	Extraocular muscles; levator palpebrae; pupillary muscles	Double vision and abnormal eye motion; ptosis; abnormal pupil size
IV – trochlear	Superior oblique muscle	Double vision and abnormal eye motion
V – trigeminal	Facial sensation; muscles of mastication	Facial pain
VI – abducens	Lateral rectus muscle	Double vision and abnormal eye motion
VII – facial	Facial muscles	Bell's palsy
VIII – vestibulocochlear	Equilibrium	Dizziness, vertigo
	Hearing	Tinnitus, hearing loss
IX – glossopharyngeal	Taste, pharynx sensation	Loss of gag reflex and taste (bitter, sweet, sour, salty)
X – vagus	Muscles of larynx and pharynx, taste, heart rate, digestion	Impaired swallowing
XI – spinal accessory	Pharynx, trapezius and sternocleidomastoid muscles	Impaired swallowing
XII – hypoglossal	Tongue movements	Impaired swallowing

Table. Cranial nerves, function, and symptoms when functioning incorrectly.

Cranial Nerve II symptom: decreased vision and loss of vision

Chronic vision loss is typically the concern of the optometrist or ophthalmologist rather than the primary care provider. However, the primary care provider may see patients with acute vision loss. Acute transient monocular vision loss (amaurosis fugax) may be caused by embolic or hemodynamic vascular abnormalities[2]. This symptom represents a TIA/stroke equivalent, and should be managed as such, with aggressive timely work-up either on an inpatient or closely monitored outpatient basis. Imaging should include MRI of the brain and either magnetic resonance angiography (MRA) or computed tomographic angiography (CTA) of the arch and carotid arteries, or at least ultrasound of the neck vessels, to evaluate for vascular occlusion, stenosis, dissection, or other causative vascular abnormality (see pages 47-49).

When acute monocular vision loss occurs in a younger patient, particularly if there is associated movement disorder or an afferent papillary defect, MRI of the brain without and with contrast should be obtained to exclude multiple sclerosis[3] (Figure 1) (see pages 51-53).

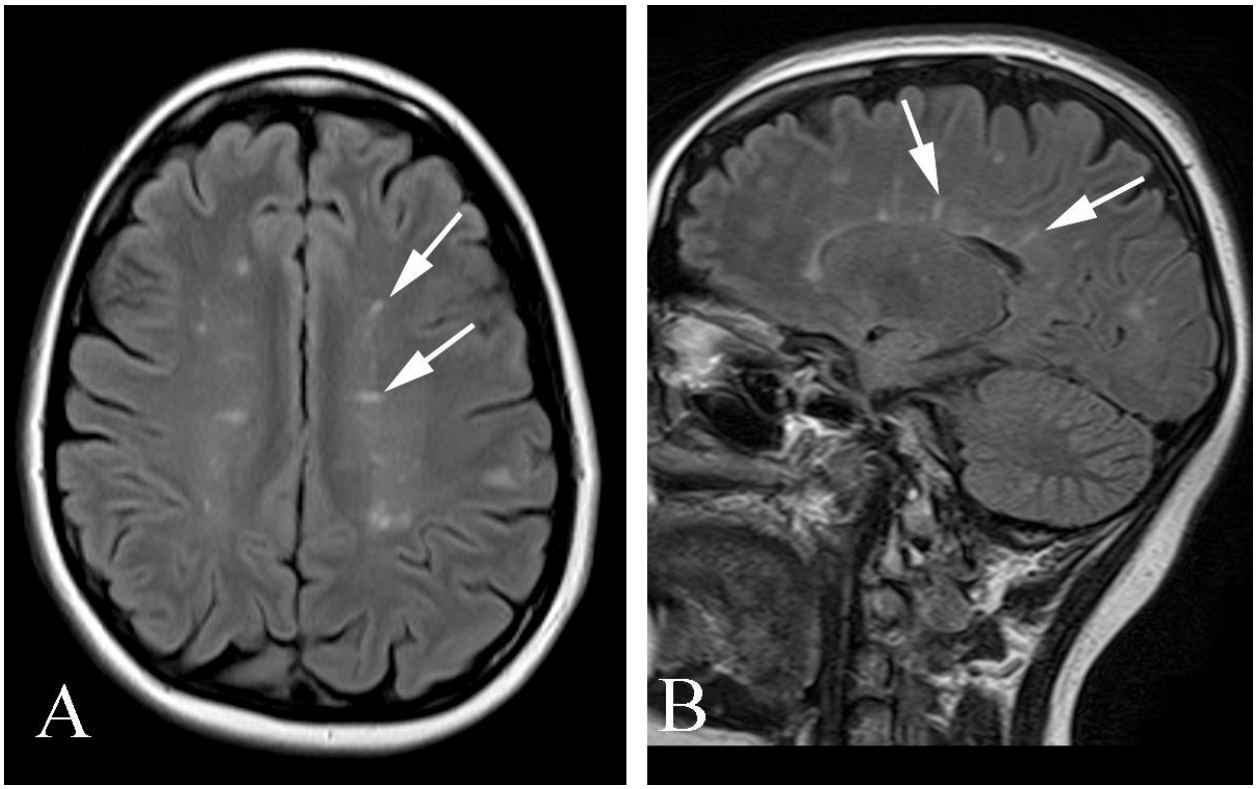

Figure 1. Multiple sclerosis in a 35 year old woman with blurred vision in one eye along with numbness in both hands. A. Axial FLAIR MR shows multiple foci of increased signal intensity (arrows). B. Sagittal FLAIR MR also shows multiple foci of increased signal intensity, including several lesions of the corpus callosum (arrows), typical for multiple sclerosis.

For patients with diminished temporal visual fields (bitemporal hemianopsia), routine brain MR sequences should be supplemented with examination of the pituitary fossa for adenoma; typically, contrast-enhanced thin cuts through the pituitary are obtained following IV contrast enhancement[4].

Cranial Nerve III, IV, and VI symptom: diplopia

Proper care of patients with diplopia depends upon differentiating the patients with double vision secondary to isolated cranial nerve abnormality from those with multiple abnormal cranial nerves. Isolated cranial nerve abnormality generally implies a benign cause and likely resolution (or at least improvement) of the process with time, whereas diplopia associated with multiple cranial nerve abnormalities requires an expedited work-up to evaluate for possible intracranial abnormalities[5]. For younger patients (where multiple sclerosis is a concern), MR done without and with contrast should be performed (Figure 2) (see pages 51-53). For older patients and others where a vascular cause is suspected, MRI without and with contrast and vascular imaging should be performed (Figure 3) (see pages 47-49).

Cranial Nerve V symptoms: trigeminal neuralgia

The fifth cranial nerve (also known as the trigeminal nerve because of its supraorbital, maxillary, and mandibular divisions) is primarily a sensory nerve, and the chief symptom arising from dysfunction is severe, unilateral lancinating facial pain (also known as tic douloureaux). This pain is highly characteristic, but because the same pain may be caused by multiple sclerosis (see pages 51-53) or a cerebellopontine angle tumor, MRI of the brain without and with contrast should usually be performed[6]. Note that unilateral facial pain may also be caused by dental disease, temporomandibular joint dysfunction, temporal arteritis, sphenoid sinusitis (see below), and cluster headache (see page 29)[6].

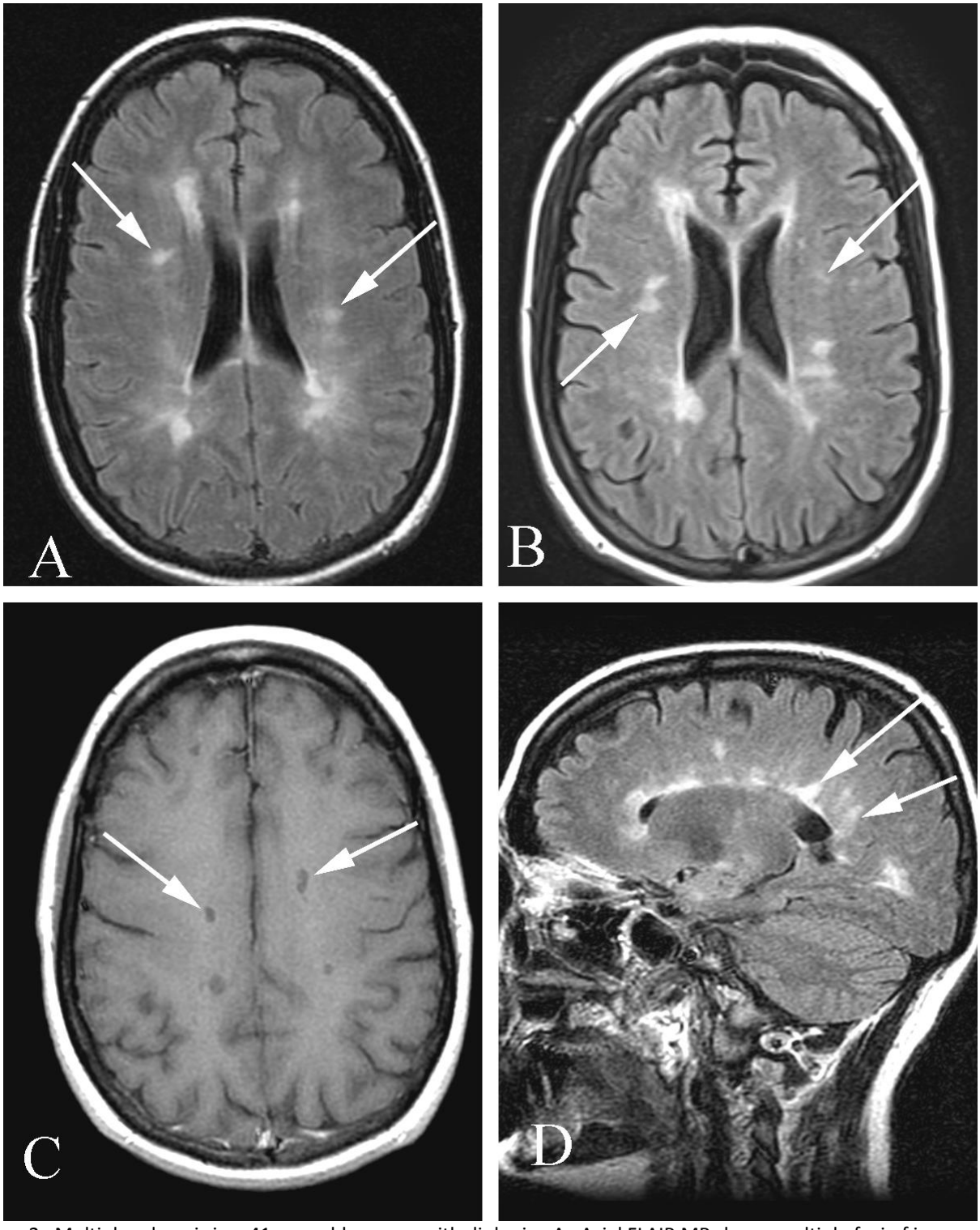

Figure 2. Multiple sclerosis in a 41 year old woman with diplopia. A. Axial FLAIR MR shows multiple foci of increased signal intensity (arrows). B. Axial FLAIR MR at a slightly different level shows multiple additional oblong foci of increased signal intensity (arrows) with the long axis of the abnormalities perpendicular to the lateral ventricles. C. Axial T1 weighted post-contrast examination shows multiple foci of decreased signal intensity (arrows) in the white matter of the cerebral hemispheres. D. Sagittal FLAIR MR shows multiple foci of increased signal intensity, including several lesions of the corpus callosum (arrows), typical for multiple sclerosis.

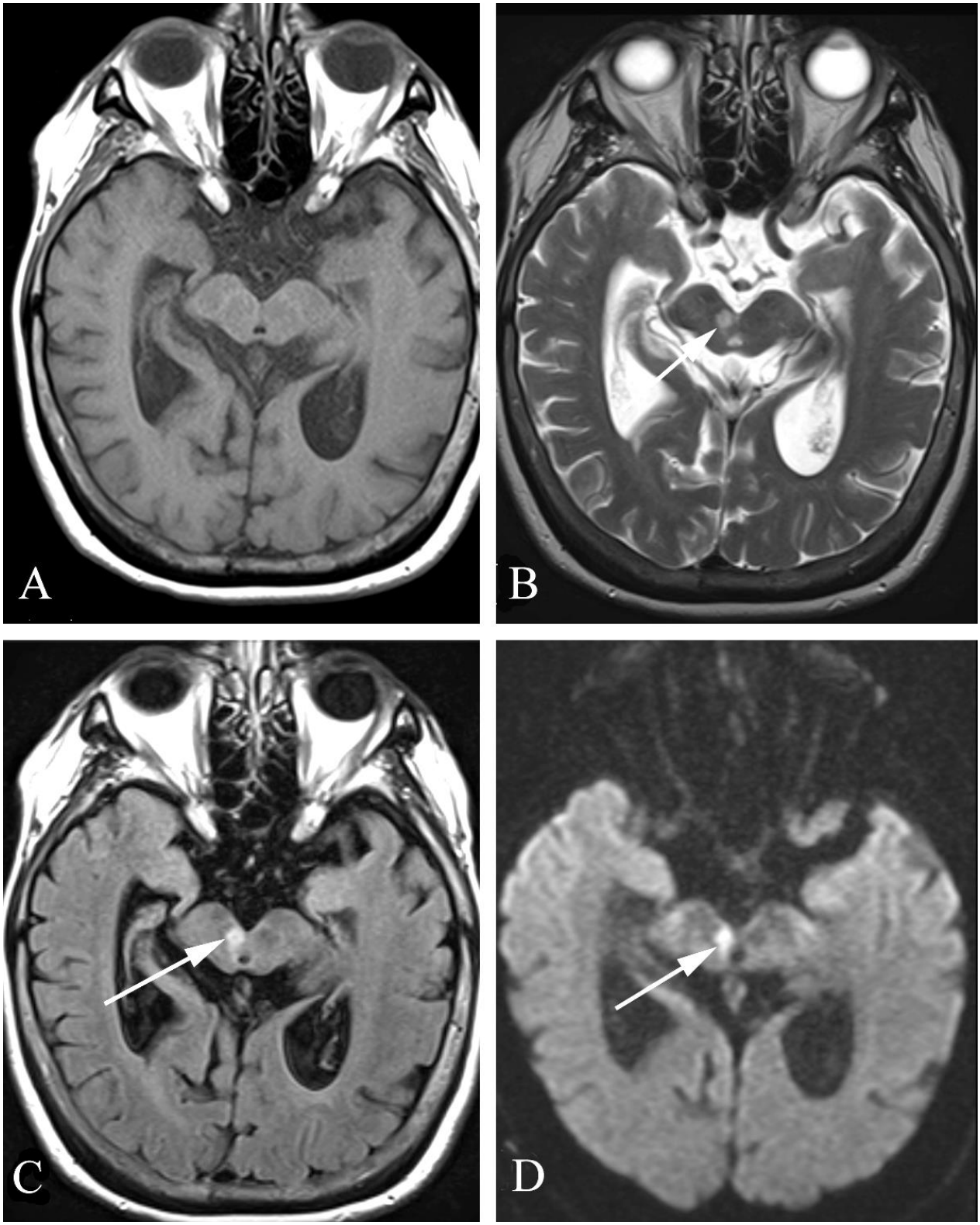

Figure 3. Stroke in a 70 year old woman with diplopia (and dizziness) beginning 4 days prior to the MR study. A. Axial T1 weighted MR is normal. B. Axial FLAIR MR shows a focus of increased signal intensity in the right brainstem (arrow). C. Axial FLAIR image also shows abnormal signal in the right brainstem (arrow). D. Axial diffusion weighted image demonstrates increased signal indicating restricted diffusion, characteristic of a stroke.

Cranial Nerve VII abnormality: Bell's palsy or idiopathic facial neuropathy

Abrupt paralysis of the facial nerve, or Bell's palsy, is typically highly characteristic, self-limited, and should be recognized by the primary care provider. It generally does not require imaging if isolated[7]. As with other cranial nerve symptoms, when multiple cranial nerve symptoms or central nervous system abnormalities are in question, MRI without and with contrast with additional thin cuts through the posterior fossa is in order.

Cranial Nerve VIII symptoms: tinnitus, hearing loss, dizziness, and vertigo

Eighth cranial nerve dysfunction may result in hearing loss or tinnitus if the cochlear division is involved, or dizziness/vertigo if the vestibular division is involved. Any patient with any of these four symptoms needs to first undergo an in office hearing examination including the Weber and Rinne test[8], along with a Dix Hallpike maneuver to evaluate for vertigo[9], after which it should be possible to categorize the patient into one of the following categories to decide whether imaging is necessary and, if so, which imaging study to order:

Isolated subjective tinnitus

Subjective tinnitus is an abnormal sound heard by the patient that the examiner does not hear, and is a frequently encountered symptom[10]. If the symptom is truly isolated, no imaging is probably necessary unless the patient is young and there is a suspicion of multiple sclerosis, in which case MR is advised.

Isolated objective tinnitus

Objective tinnitus, in which both the patient and the examiner detect the abnormal noise, is considerably less common than subjective tinnitus[11] and is most frequently caused by vascular lesions associated with turbulent or high velocity flow, for example around atherosclerotic plaque or through a vascular malformation. Brain MRI should be performed in patients with objective tinnitus, along with MRA or CTA of the skull base and neck vessels.

Isolated conductive hearing loss

Conductive hearing loss is caused by abnormality of the external auditory canal, tympanic membrane, ossicles, or cochlea (those structures which conduct sound to the 8th cranial nerve endings within the cochlea). Common causes include cholesteatomas, otosclerosis, and congenital abnormalities of the cochlea. Isolated conductive hearing loss does not necessarily require imaging, particularly if there is an obvious cause. When imaging is necessary, temporal bone CT is the study of choice, since it delineates the bony structures of the temporal bone much better than MR.

Isolated sensorineural hearing loss

In general, neural (8th cranial nerve or central nervous system) hearing loss is more likely to be caused by a life threatening abnormality (e.g. brain tumor) than is sensory (caused by inner ear abnormality) hearing loss[12], but the two may be difficult to distinguish. In cases of apparent neural abnormality or where there are ambiguous findings, MRI done without and with contrast with thin cuts is recommended to exclude vestibular schwannoma (formerly known as acoustic neuroma), brainstem stroke, and multiple sclerosis.

Dizziness and vertigo

Dizziness must first be distinguished from vertigo, which may be quite difficult given patients' tendencies to describe the symptoms similarly[13]. If the clinical history and Dix Hallpike maneuver implicate vertigo and the patient has any other symptom indicating a central process such as diplopia, dysarthria, dysphagia, weakness, or numbness[9] then MRI without and with contrast material with thin cuts through the posterior fossa is recommended. Even without these features, if the vertigo does not show rapid clinical improvement, imaging is probably indicated. Patients with balance and/or gait difficulties (whether dizzy or not), particularly when accompanied by other neurologic symptoms or findings, should undergo MR to search for a causative lesion (Figure 4).

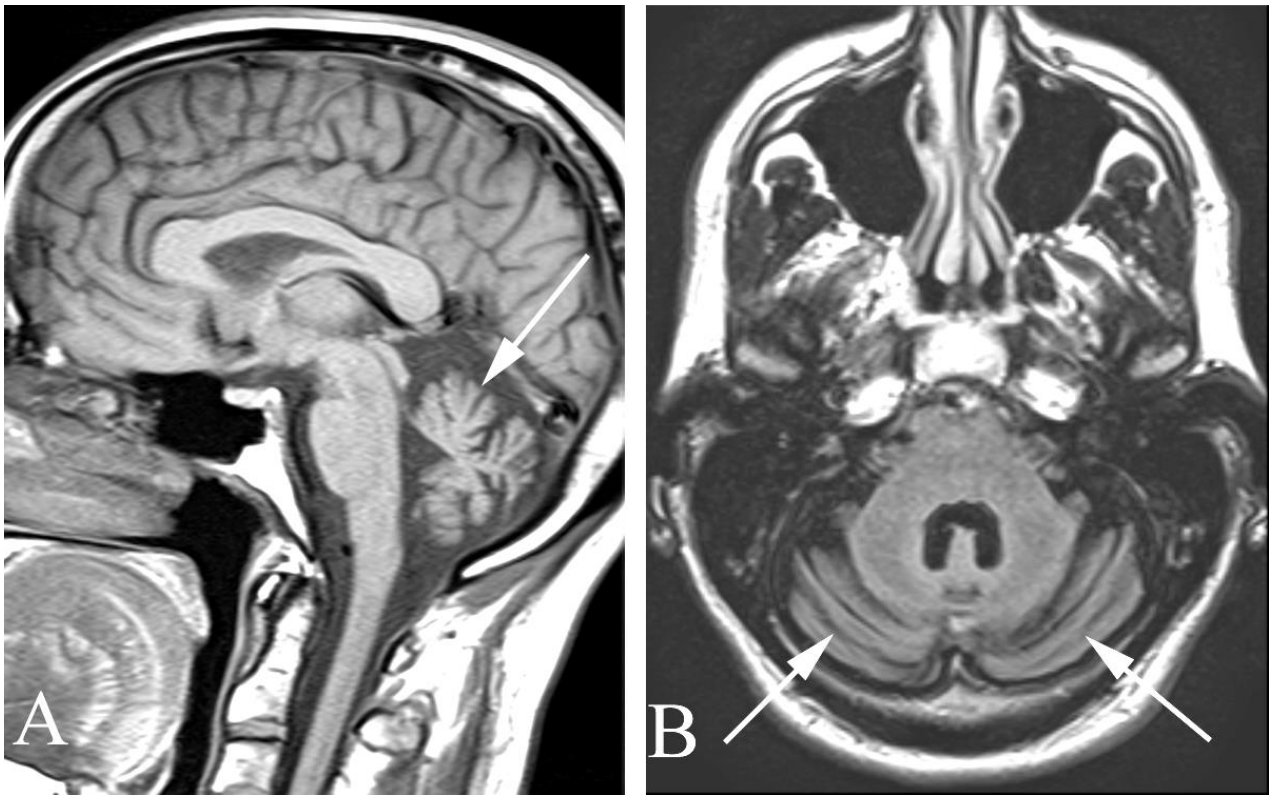

Figure 4. Cerebellar atrophy in a 36 year old man with balance difficulties and slurred speech. A. Sagittal T1 weighted MR shows loss of volume of the cerebellum (arrow). B. Axial T1 weighted image demonstrates bilaterally enlarged cerebellar sulci, compatible with cerebellar atrophy (arrows). The patient was a former alcoholic.

Mixed symptoms

If tinnitus, hearing loss, or vertigo is accompanied by any additional cranial nerve symptoms, headache, gait or coordination abnormality or other symptom suggesting a posterior fossa abnormality, MRI should be obtained to evaluate for vestibular schwannoma (Figure 5), stroke (see Chapter 4, Figure 6, page 49), brainstem tumor (Figure 6) or cerebellar tumor (Figure 7).

Cranial Nerves IX – XII symptoms: swallowing difficulty

Swallowing difficulty secondary to abnormalities of cranial nerves IX – XII may represent a stroke, and appropriate imaging needs to be performed when this is suspected (see pages 47-49). For a discussion of oropharyngeal dysphagia, see pages 98-99.

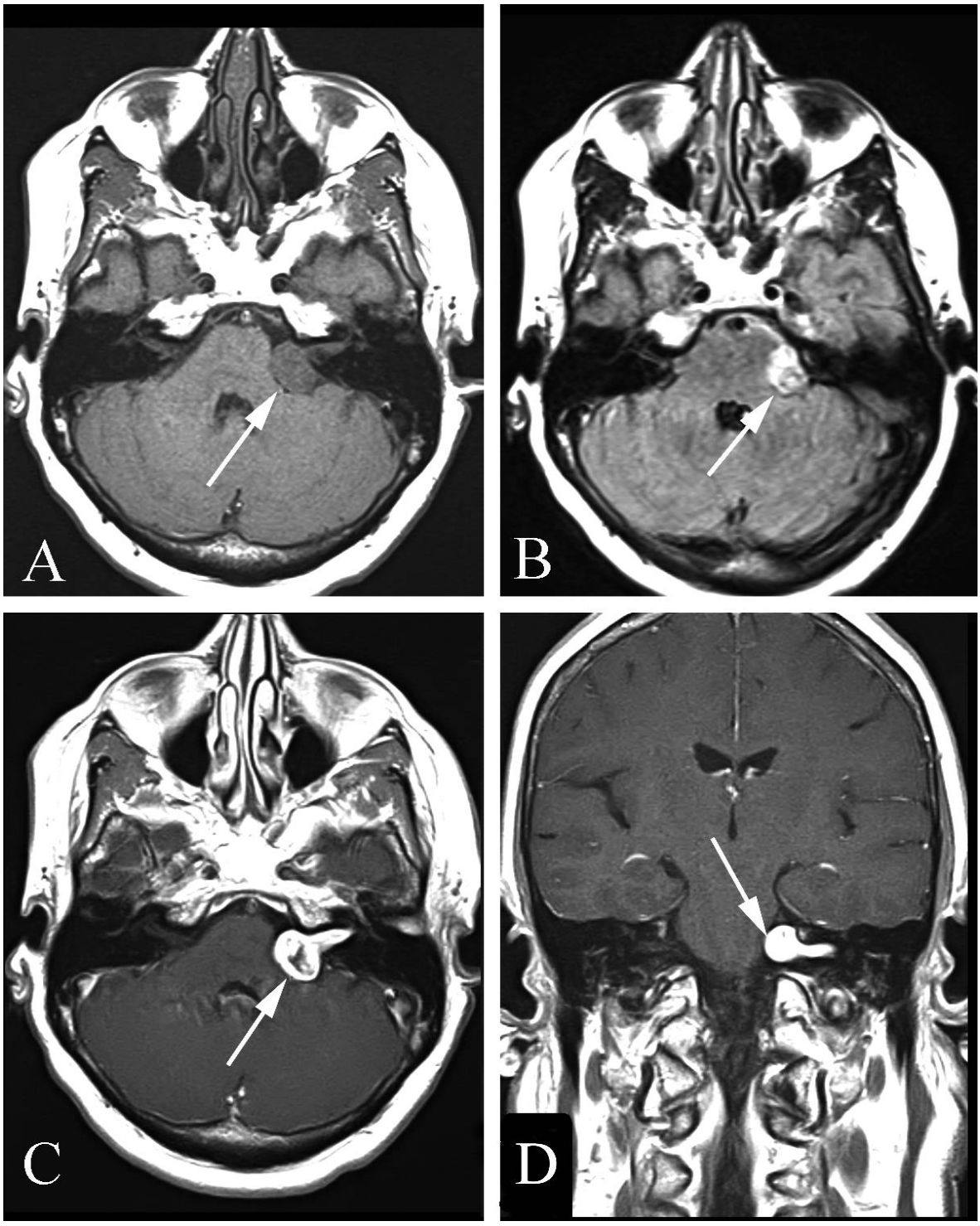

Figure 5. Vestibular schwannoma in a 55 year old woman with sensorineural hearing loss, tinnitus, and headache. A. Axial T1 weighted MR shows a cerebellopontine angle extra-axial lesion (arrow) which shows slightly decreased signal intensity relative to the adjacent brain. B. Axial FLAIR MR image demonstrates increased signal in the lesion (arrow) compared to the adjacent brain. C. Axial postcontrast T1 weighted image demonstrates the typical "ice-cream cone" appearance of the left vestibular schwannoma, with intense contrast enhancement particularly around the peripheral aspect of the tumor (arrow). D. Coronal postcontrast T1 weighted image also shows the "ice-cream cone" appearance and extension of abnormal contrast enhancement along the course of the vestibular-cochlear nerve in the internal auditory canal.

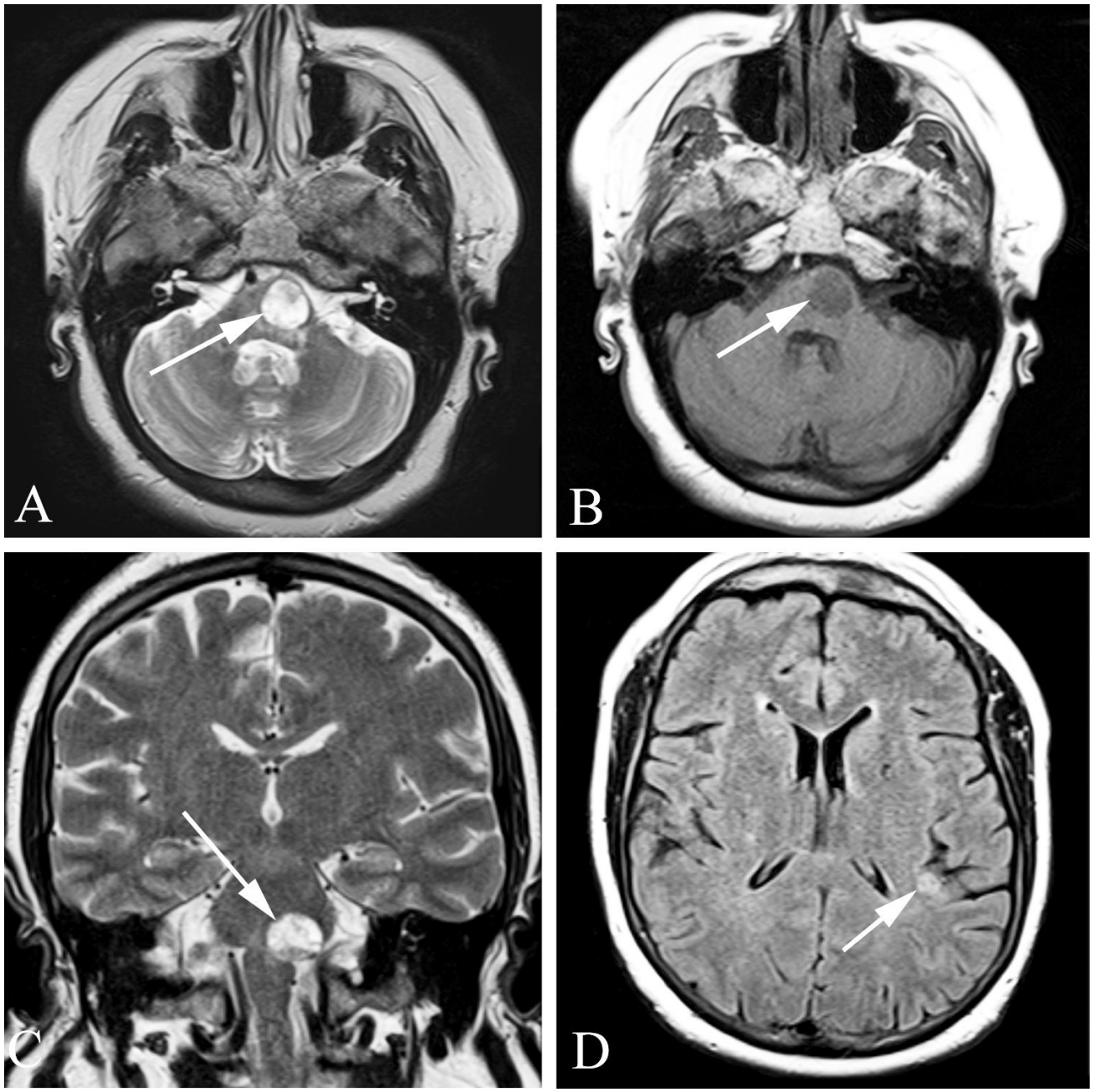

Figure 6. Metastatic disease in a 45 year old woman with tinnitus and headache who had a renal transplant. A. Axial T2 weighted MR image of the brain shows a mass with mixed (mostly increased) signal in the left brainstem (arrow). B. Axial T1 weighted image also shows the mass, with decreased signal. C. Coronal T2 weighted MR study also shows the brainstem mass. D. Axial FLAIR MR image demonstrates an additional lesion of the left cerebral hemisphere (arrow). Initially, the main consideration was for multiple brain abscesses in this immunocompromised renal transplant patient. Further study shows no extensive systemic findings of infection, however, and the patient was found to have a small cell carcinoma of the lung.

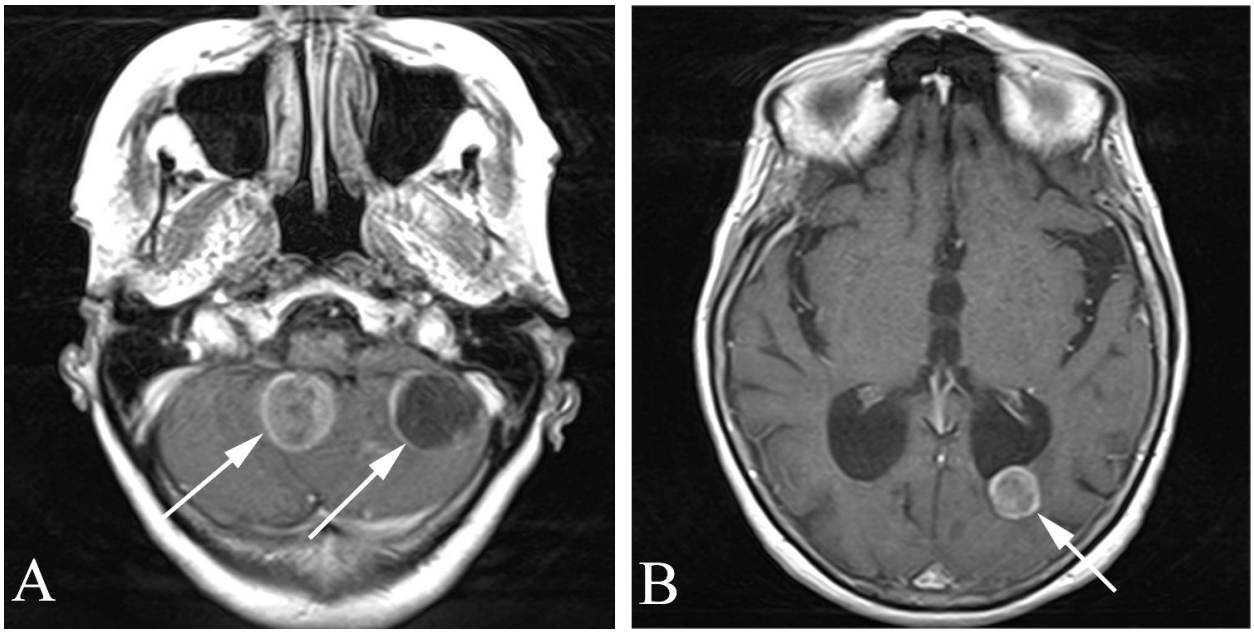

Figure 7. Metastatic disease in an 82 year old woman with balance difficulty, mental status changes, and vomiting. A. Axial contrast-enhanced T1 weighted MR exam demonstrates two separate lesions of the cerebellum (arrows), with the more midline lesion demonstrating relatively uniform contrast enhancement and the lesion of the left cerebellar hemisphere showing peripheral contrast enhancement. B. Axial contrast-enhanced T1 weighted MR exam shows an additional metastatic deposit in the left occipital lobe adjacent to the occipital trigone (arrow). The patient had known lung cancer.

PATIENTS WITH SYMPTOMS OF SINUSITIS USUALLY DO NOT REQUIRE IMAGING - CT IS THE STUDY OF CHOICE WHEN IMAGING IS NECESSARY

A diagnosis of sinusitis is based on purulent discharge and nasal congestion and/or facial pain/pressure[14]. It is not possible to tell from patient symptoms whether the patient has acute viral rhinosinusitis (AVRS) or acute bacterial rhinosinusitis (ABRS), although current recommendations favor supportive care rather than antibiotic treatment even for ABRS in the absence of severe pain or a temperature of over 101 degrees F (38.3 degrees C)[10].

Imaging of patients with a clinical diagnosis of acute sinusitis is not typically required or helpful, as many asymptomatic, healthy individuals have sinus abnormalities also seen in sinusitis[15], imaging cannot distinguish AVRS from ABRS[10], and imaging (even CT) does not, in general, correlate with the severity of the disease[16]. Plain films are both insensitive and nonspecific compared to CT studies in evaluation of sinusitis[10].

CT of the sinuses benefits patients with sinusitis symptoms in the following special circumstances:

1. When it may be helpful to *refute* sinusitis as the cause of pain. A normal CT supports a diagnosis such as allergy, non-allergic rhinitis, and atypical facial pain (as opposed to AVRS or ABRS) in a patient with equivocal symptoms[10].
2. When superimposed symptoms suggest complicated sinusitis. These symptoms include acutely diminished visual acuity, diplopia, periorbital edema, severe headache, and altered mental status[17]. The purpose of CT in these cases is to exclude extension of infection outside the sinuses (Figure 8) or alternative diagnoses presenting as sinusitis.
3. For mapping in cases where functional endoscopic sinus surgery (FESS) is contemplated[18]. In explanation: FESS was developed as a less invasive alternative to standard operations. In the classic standard surgical treatment for sinusitis, the Caldwell-

Luc procedure, the surgeon strips the mucosal lining from the sinus and opens the medial wall of the maxillary sinus to the anterior aspect of the inferior meatus in the nasal vault. Unlike the Caldwell-Luc procedure, FESS restores normal mucociliary transport by clearing obstructions to mucus flow, particularly along the maxillary infundibulum, nasofrontal duct, and ostiomeatal unit. Performance of this directed surgery depends upon[14,19]:

A. Obtaining a map of the exact location and cause of any obstructive process. This entails a sinus CT either performed in, or reformatted in, the coronal plane to best visualize the maxillary infundibulum, nasofrontal duct, and ostiomeatal unit (Figure 9).
B. Knowledge of any anatomic variants that may represent a hazard if not known. Given the endoscopic nature of the surgery, it is necessary to know about such anatomic variants as dehiscence in the lamina papricea (Figure 10) and sphenoid sinus walls (Figure 11) to avoid inadvertent puncture of the orbit or intracranial internal carotid artery. CT also provides this.

In cases where CT is performed, Harnsbarger[14] has noted that most patients can be classified into one of the following categories:

1. 40% have a normal study. As noted above, in the presence of sinusitis symptoms, a normal study supports the diagnosis of allergic sinusitis, non-allergic rhinitis, or atypical facial pain.
2. 30% had abnormalities that fit no specific pattern (sporadic, nonobstructive disease).
3. 30% had abnormalities that fit into a specific (nonsporadic) inflammatory pattern, where FESS should provide benefit. Subsets of this group include those with a maxillary infundibular pattern, a nasofrontal pattern, and an ostiomeatal unit pattern (Figure 9).

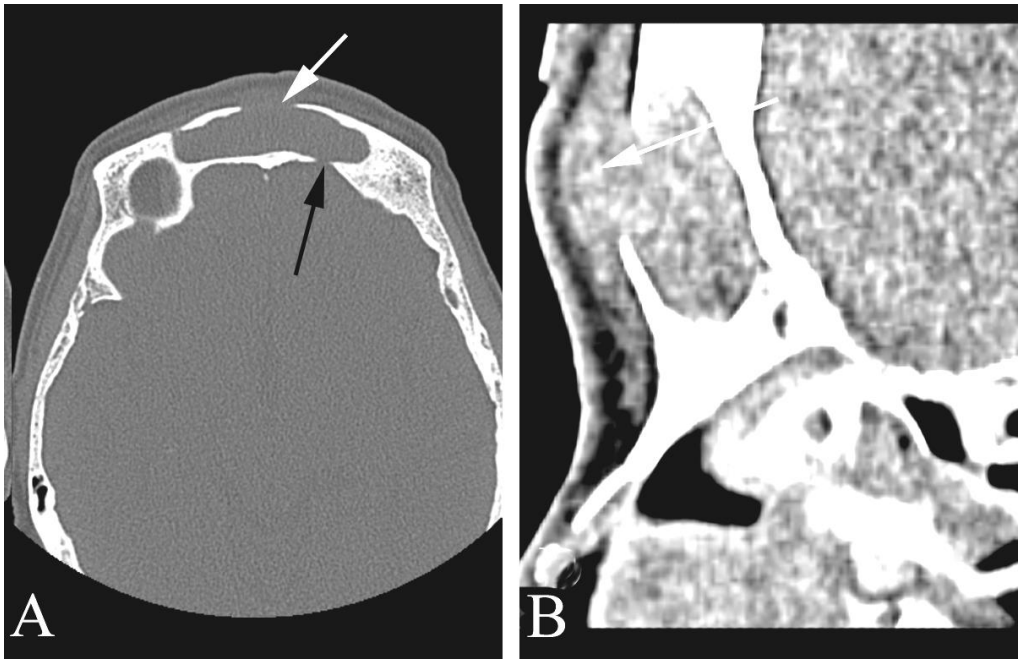

Figure 8. Sinusitis with extension of disease outside of the sinuses in a 77 year old with nasal drainage, headache, and a swollen forehead. A. Axial unenhanced head CT (bone windows) shows complete opacification of the frontal sinus as well as destruction of the anterior wall (white arrow) and posterior wall (black arrow) of the sinus. B. Sagittal reformatted CT image (soft tissue window) shows destruction of the anterior wall of the sinus with soft tissue extending anterior to the frontal bone (arrow) accounting for the patient's swollen forehead. This lesion is also known as "Pott's puffy tumor".

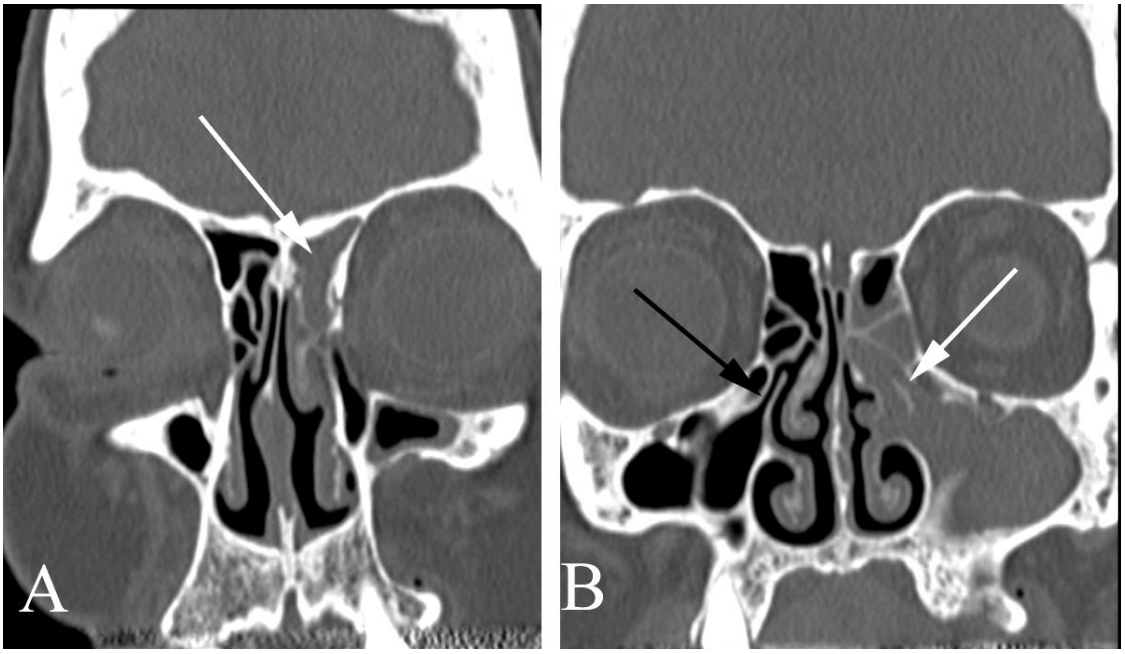

Figure 9. Sinusitis demonstrating an "ostiomeatal unit" pattern of infection in a 76 year old with chronic headache and nasal drainage. A. Coronal sinus CT (bone windows) shows opacification of left ethmoidal air cells (arrow). B. Coronal sinus CT (bone windows) at a more posterior location shows opacification of the left maxillary sinus including complete occlusion of the ostiomeatal unit (white arrow). Note the normal, open appearance of the contralateral ostiomeatal unit (black arrow).

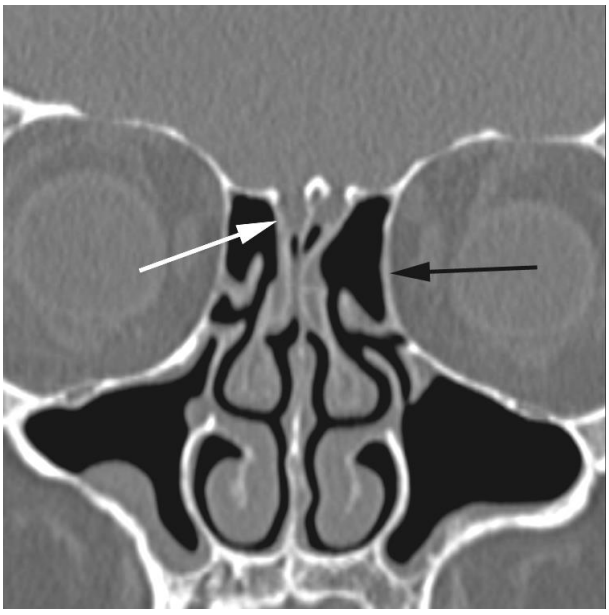

Figure 10. Possible hazards to functional endoscopic sinus surgery in a 64 year old woman with recurrent sinusitis symptoms. Coronal CT study (bone windows) shows a low-lying cribriform plate with a thin bony covering (white arrow) as well as a thin bony lamina papricea (black arrow).

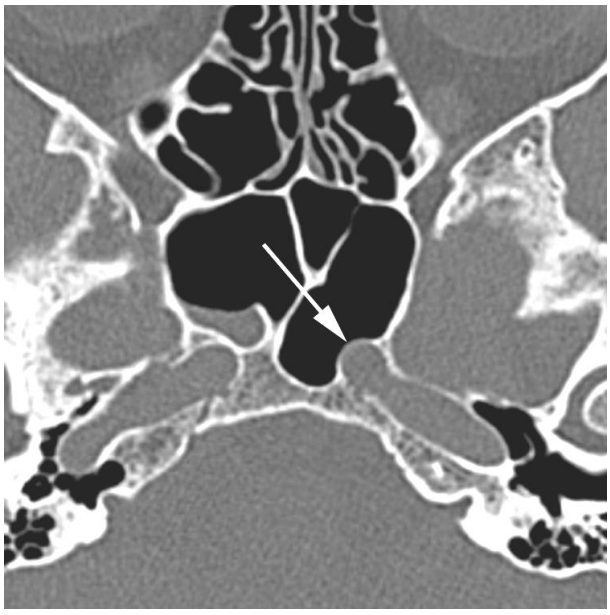

Figure 11. Possible hazard to functional endoscopic sinus surgery in a 57 year old woman with recurrent sinusitis. Axial CT study (bone windows) shows a thin bony covering over the carotid canal (arrow), which protrudes into the sphenoid sinus.

PALPABLE THYROID LESIONS CAN UNDERGO FNAB WITHOUT IMAGING WHEREAS NON THYROID NECK MASSES IN ADULTS TYPICALLY REQUIRE CT SCANNING

The first step in evaluation of a neck mass in adults requires distinction between thyroid lesions and lesions outside the thyroid gland. If it is not possible to make this distinction on clinical grounds, ultrasound may be performed.

Thyroid lesions

Many patients have thyroid lesions, and while most of these lesions are benign, it is not possible to say with acceptable accuracy which lesions are benign and which are malignant on the basis of clinical examination alone. While both nuclear medicine and ultrasound have been (and can be) used for evaluation of such lesions, imaging features are not sufficiently accurate to make the distinction between benign and malignant lesions, either: solid, mixed solid and cystic, and even apparently purely cystic lesions on ultrasound examination may be malignant[20]. It is more cost effective to obtain a fine needle aspiration biopsy (FNAB) of thyroid lesions than it is to perform imaging[16]. Biopsy results generally fall into one of four categories: nondiagnostic, requiring either repeat biopsy or excision of the nodule; benign, requiring at most follow-up; suspicious or indeterminate, typically requiring surgical excision; and malignant, requiring excision.

For thyroid lesions that are evaluated at ultrasound (which may or may not be palpable), current recommendations include biopsy of lesions greater than 15 mm unless microcalcifications are identified, in which case biopsy of lesions greater than 10 mm is recommended[21].

Nonthyroid neck masses

Adult neck masses outside the thyroid gland, particularly those in patients over the age of forty and with known risk factors such as smoking, must be considered malignant until proven otherwise. These patients typically need referral to an otolaryngologist for treatment[22]. While such clinical features as a soft, rubbery consistency and mobility favor a benign cause (compared to hard, fixed lesions), these clinical features are not, in themselves, diagnostic. Note that pediatric neck masses have a different set of considerations, and often represent benign cystic lesions or hemangiomas.

Almost all adult neck masses of unknown origin require a CT scan performed with contrast and extending from the arch of the aorta through the skull base (often called a "neck" or "soft tissue neck" or "head and neck" CT) (Figure 12). A marker should be placed on the lesion by the technologist performing the scan; if the patient cannot locate the lesion for the technologist, it is helpful for the primary care practitioner to mark the lesion with indelible ink prior to sending the patient for imaging so that a marker can be placed at the appropriate location. A CT of the chest may be performed at the same time, particularly if there are risk factors such as smoking, given the fact that chest primary tumors may either co-exist with neck primary tumors or be a source of metastatic deposit to the neck[18]. The CT may rarely suggest a cyst as may be seen with congenital/developmental anomalies such as branchial cleft cysts (Figure 13) or even provide a specific histologic diagnosis (Figure 14). However, the main purpose of the CT study is to demonstrate the exact location of the lesion, demonstrate any other nonpalpable lesions, and to search for a primary tumor, since many palpable lesions of the neck represent metastatic deposit from oropharyngeal mucosa primary tumors (Figure 12). The location of the palpable abnormality may provide a clue to the location of the primary tumor, as tumor cell drainage follows a typical pattern: for example, the lower lip, floor of the mouth, and apex of the tongue drain into the submental lymph nodes[23]. Knowledge of this pattern helps the radiologist and the otolaryngologist search the associated mucosal surface for primary tumors. Otolaryngologists will typically perform laryngoscopy and both esophagoscopy and bronchoscopy may also be necessary to identify the primary lesion[18]. Unfortunately, even with the location of the primary lesion and knowledge of the typical spreading pattern, the primary lesion may

not be found on CT or endoscopy, in which case an FNAB of the palpable lesion is performed. If the lesion is malignant when no primary tumor has been found, random biopsy of the nasopharynx, palatine tonsils, and base of the tongue may be performed[18].

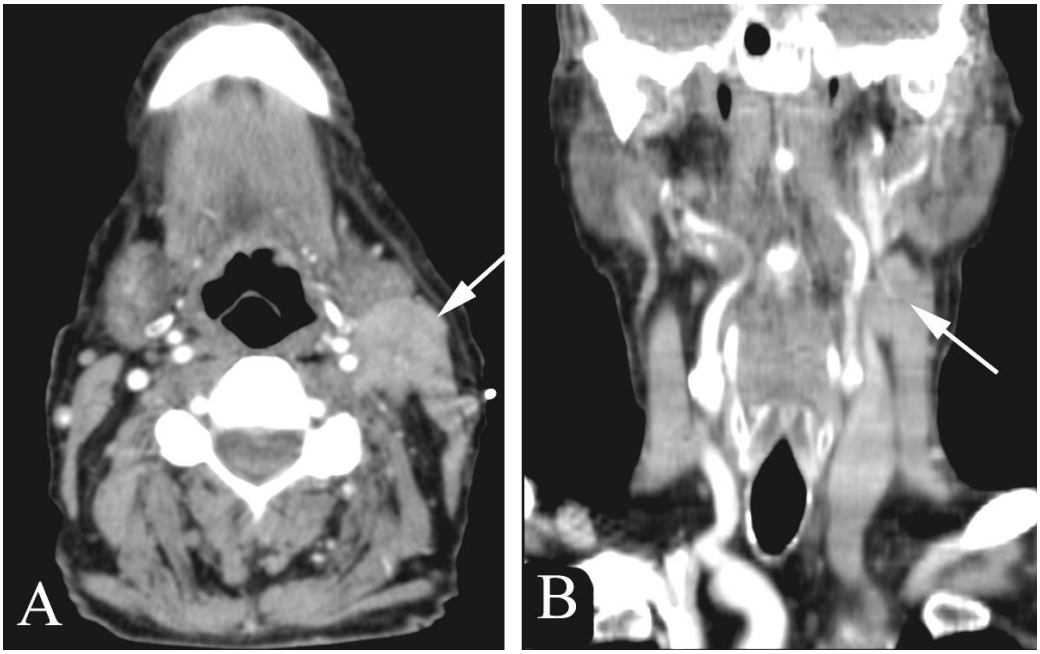

Figure 12. Metastatic squamous cell cancer of the head and neck in a 74 year old man with a palpable neck mass. A. Axial contrast-enhanced neck CT shows a mass (arrow) superficial to the carotid vessels in the left neck, representing metastatic deposit to lymph nodes. Note the marker (white dot) placed along the margin of the palpable lesion by the technologist. B. Coronal reformatted contrast-enhanced CT shows the mass (arrow) superficial to the carotid vessels.

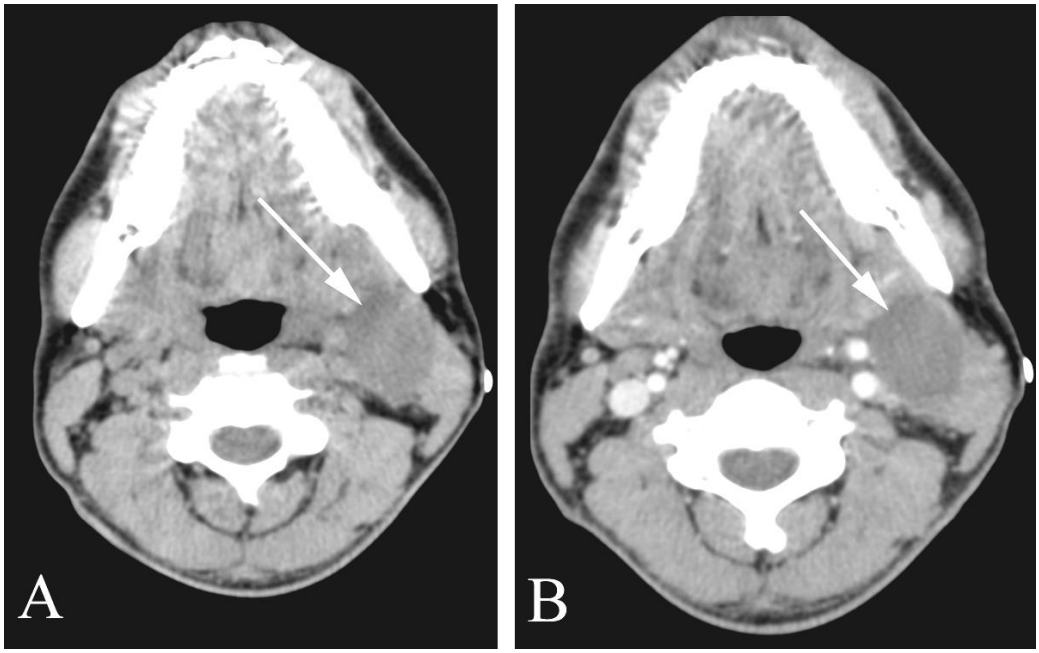

Figure 13. Branchial cleft cyst in a 32 year old man with a palpable neck mass. A. Axial noncontrast enhanced neck CT shows a mass superficial to the carotid artery and posterior to the mandible (arrow). B. Axial contrast-enhanced neck CT shows a lack of contrast enhancement of the lesion, which proved to be a branchial cleft upon removal.

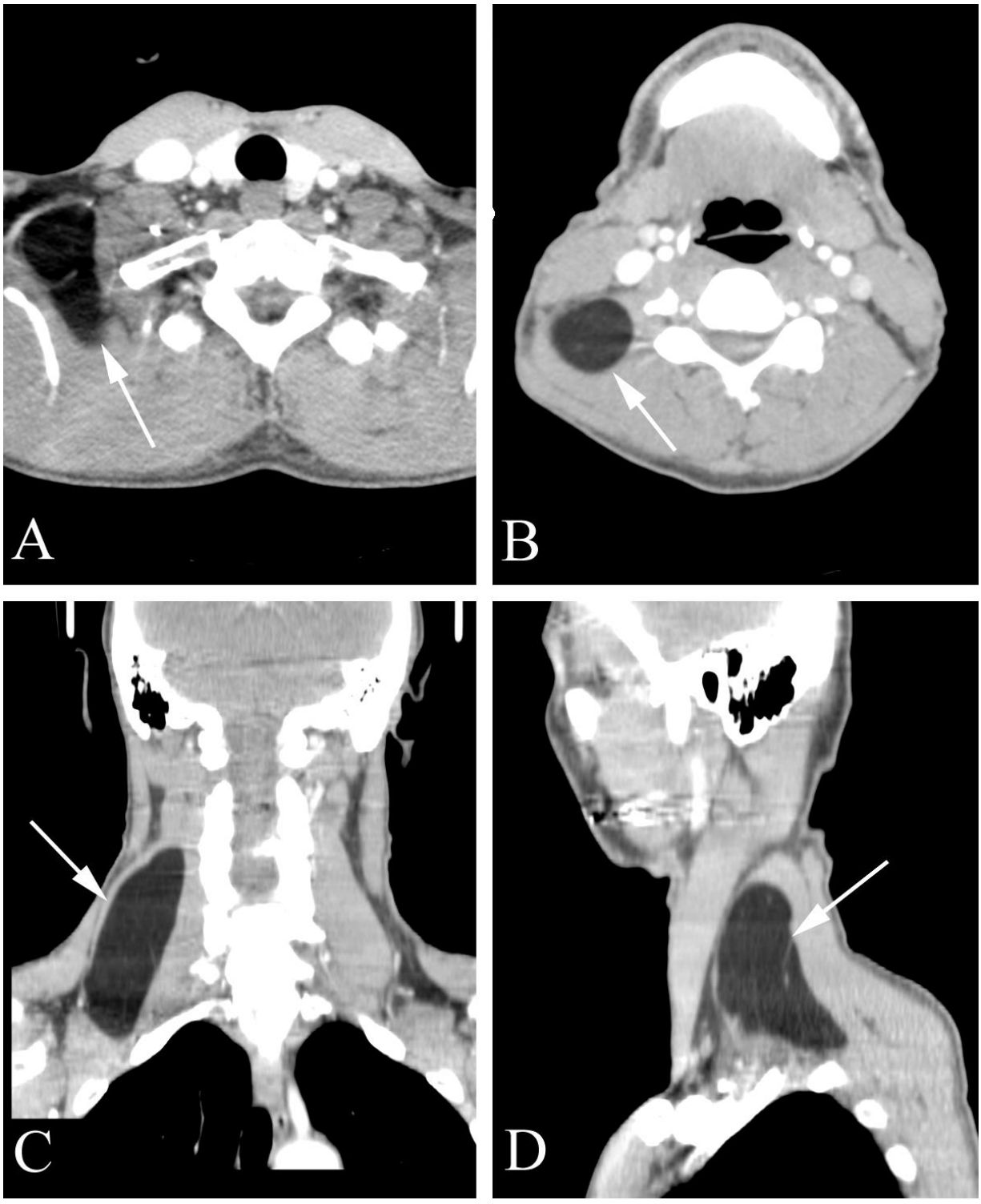

Figure 14. Benign lipoma in a 62 year old man with a palpable neck mass. A. Axial contrast-enhanced CT at the level of the thyroid gland shows a triangular soft tissue mass of fat density (arrow) in the right neck. B. Axial contrast-enhanced CT at the level of the aryepiglottic folds shows extension of the mass (arrows) superiorly, where it lies behind the sternocleidomastoid muscle (which is rotated slightly compared to the contralateral, normal side). C. Coronal reformatted contrast-enhanced CT study shows the lipoma (arrow) along the right neck, lateral to the spine. D. Sagittal reformatted contrast-enhanced CT shows the lipoma (arrow) posterior to the sternocleidomastoid muscle.

SUMMARY

Most patients with isolated cranial nerve symptoms do not require imaging, but when symptoms suggest multiple cranial nerves or a central process, brain MR should be performed. Uncomplicated sinusitis usually does not require imaging. CT of the sinuses may be performed to document sinusitis or prior to functional endoscopic sinus surgery (FESS). In patients with symptoms of complications from sinusitis orbit or head CT or MRI may be necessary. Palpable thyroid lesions can usually safely undergo percutaneous fine needle aspiration biopsy without imaging. Neck masses which do not arise from the thyroid usually require CT for evaluation.

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Spine Pain

Donald L. Renfrew, MD

According to Wikipedia[1]: "Back pain is one of humanity's most frequent complaints. In the U.S., acute low back pain (also called lumbago) is the fifth most common reason for all physician visits. About nine out of ten adults experience back pain at some point in their life, and five out of ten working adults have back pain every year." In caring for patients with spine pain (pain from the neck or low back and/or radicular symptoms), there are multiple diagnostic tests and therapeutic options available. Diagnostic imaging offers plain films, computed tomography, myelography, combined myelography/CT, nuclear medicine bone scans, magnetic resonance imaging, and fluoroscopically guided injections. Therapy includes doing nothing, oral medications, physical rehabilitation, spine injections, surgery, and then sometimes surgery again. This chapter covers three key concepts to guide the choice in diagnosis and treatment of spine pain. These concepts are:

1. "Red flags" in the patient's presentation call for priority imaging.
2. MRI has supplanted other modalities for the imaging work-up of spine pain.
3. Injections often provide diagnostic or therapeutic benefit for patients with spine pain.

"RED FLAGS" IN THE PATIENT'S PRESENTATION CALL FOR PRIORITY IMAGING

Gordon Waddell, a Glasgow spine surgeon, uses the term "red flag" to denote those clinical findings that indicate the potential of a medically serious diagnosis, and which should prompt priority imaging. Waddell's book *The Back Pain Revolution* (Churchill Livingston, 2004) is an excellent book for anyone who treats those with back pain.

Spine pain is such a common disorder, and so often runs a benign course, that common advice (although not necessarily often followed) is to wait 4-6 weeks before pursuing costly diagnostic measures. However, in the presence of a "red flag", it is prudent to expedite imaging. This does not necessarily mean that the examination has to be performed in the next five minutes, but it would probably be better to get imaging done this week rather than waiting a month.

Red flags include a personal history of malignancy, unremitting pain, pediatric patients with back pain, and constitutional symptoms (for example, weight loss or fever).

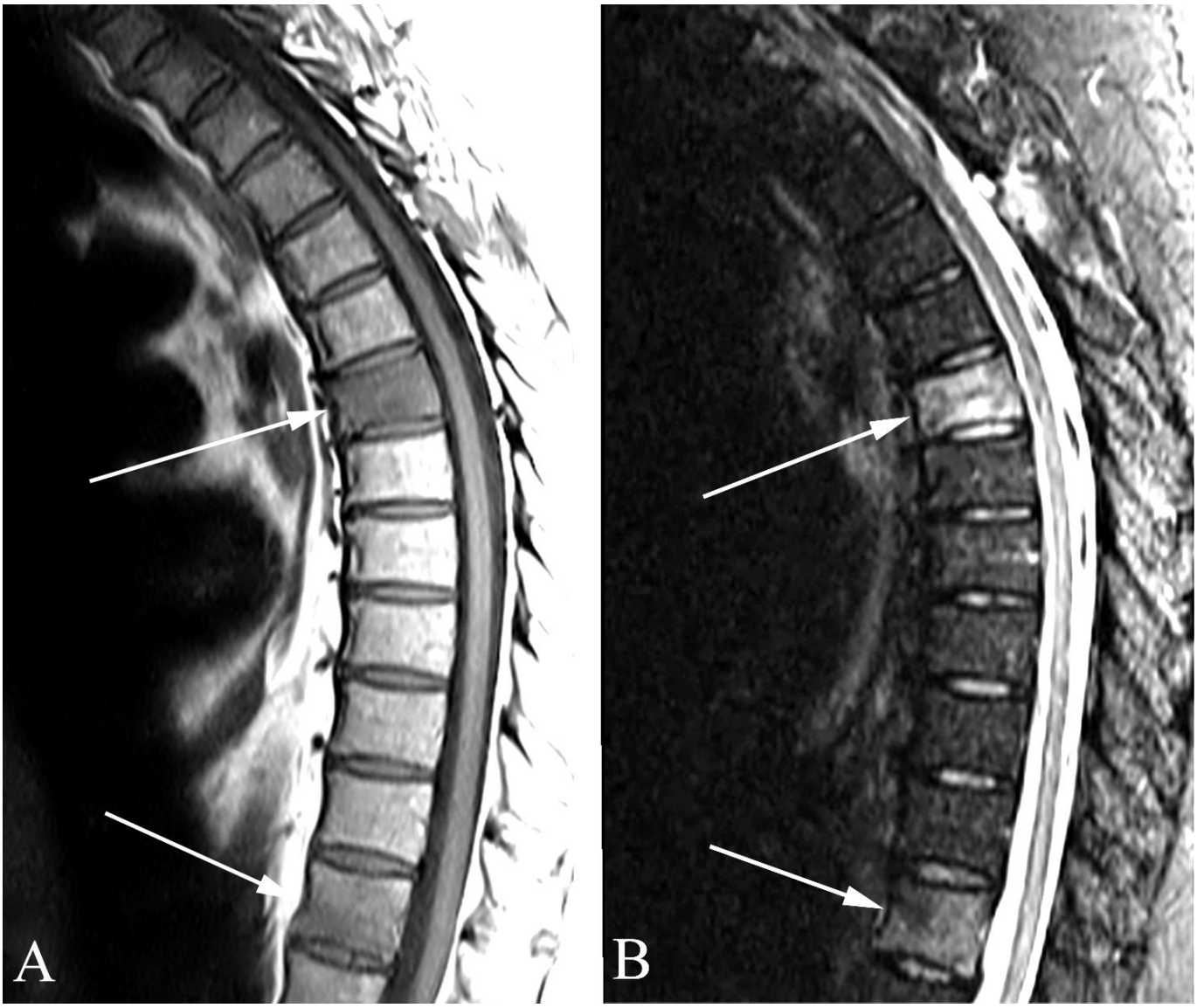

Figure 1. Metastatic lung cancer in a 65 year old man with back pain and known primary malignancy. A. Sagittal thoracic spine T1 weighted MR image shows decreased signal at T6 and T12 (arrows). B. Sagittal thoracic spine fat saturated T2 weighted MR image shows increased signal intensity (arrows), also at T6 and T12.

A personal history of cancer

This particular scenario, with patients presenting to primary care physicians with spine pain after successful cancer therapy, will likely increase in frequency as oncologists become better at curing, or at least putting into remission, various tumors. In the case of a patient with the new onset of spine pain and cancer, it makes most sense to first review any existing imaging studies, to see if they indicate (even in retrospect) a malignant cause of the pain. Studies done for tumor imaging such as CT of the abdomen and pelvis may show bone destructive changes which are easy to overlook. Nuclear medicine studies such as bone scans and PET-CT studies usually show more conspicuous and easily appreciated abnormalities which are less likely to be missed. If these studies do not show an explanatory abnormality, plain films of the painful region may be ordered but will likely not be the final study performed regardless of the outcome: if they are negative MR will need to be performed (because

plain films are insensitive), and if positive, MR will likely still need to be performed (to evaluate the extent of tumor including neural compression) (Figure 1).

Unremitting pain

This red flag emphasizes that, typically, benign spine pain is "mechanical" in the sense that it is brought on by mechanical factors (assuming a certain position, bearing a certain load), whereas spine pain secondary to such factors as tumor (Figure 2), osteomyelitis, or fracture is "non-mechanical". The patient cannot find comfort standing, sitting, or lying down, and finds little relief with medications which would normally offer benefit. Note that while a young, healthy adult would not normally sustain a spine fracture without significant trauma, the amount of trauma necessary to fracture an elderly, osteoporotic spine can be so trivial that it escapes notice, and therefore the patient may present with an osteoporotic fracture but not recall a specific incident that initiated the pain.

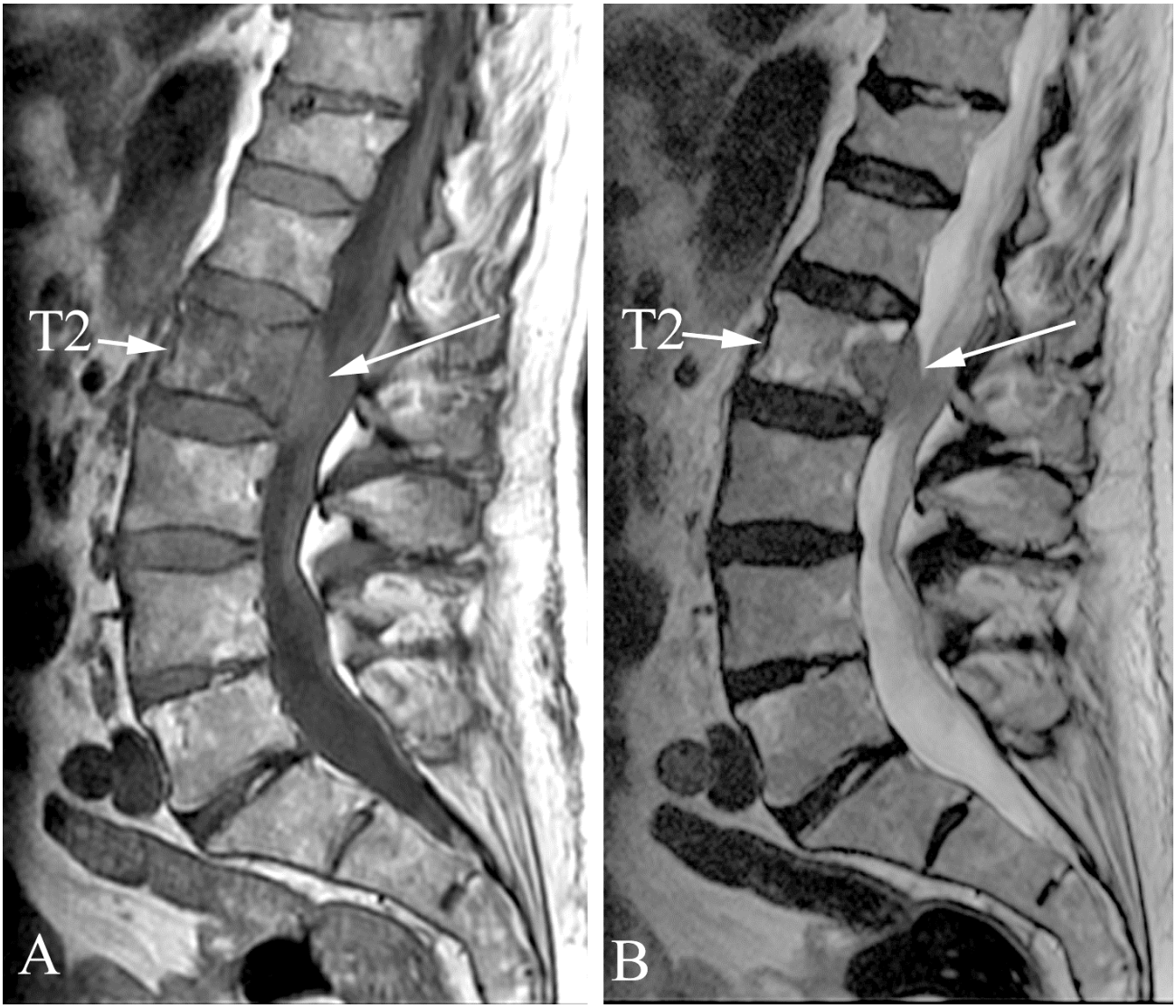

Figure 2. Lymphoma in a 65 year old man with new onset of unremitting back pain. A. Sagittal lumbar spine T1 weighted spine MR shows decreased signal in the L2 vertebral body and a mass extending into the spinal canal (arrow). B. Sagittal T2 weighted spine MR shows increased signal within the vertebral body, and also demonstrates the soft tissue mass.

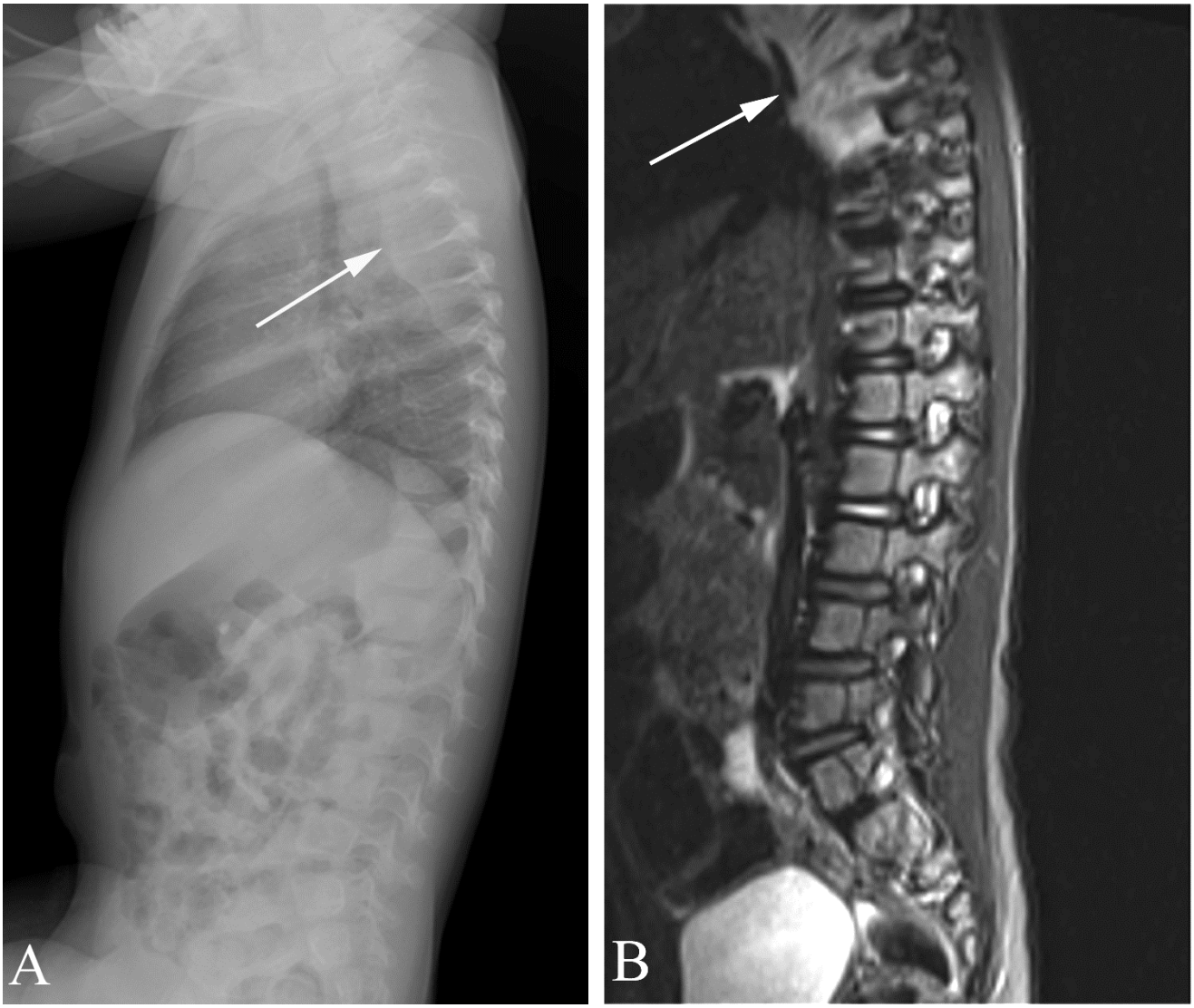

Figure 3. Neuroblastoma in a 2 year old with back pain. fatigue, and fussiness. A. Lateral plain film of the lumbar and thoracic spine shows a mass (arrow) along the posterior upper chest. B. Sagittal T2 MR shows a paraspinal mass (arrow).

The pediatric patient

Any child with spine pain should be evaluated carefully. Children rarely if ever have "degenerative" causes of backache. Occasionally, teenagers may present with central low back pain from spondylolysis. However, any younger child with spine pain should be suspected of having a possible serious medical condition such as tumor (Figure 3), infection, or unreported trauma.

MRI HAS SUPPLANTED OTHER METHODS FOR IMAGING BACK PAIN

In the short space of approximately 25 years, magnetic resonance imaging has revolutionized medicine. MRI has changed the way neurologists, neurosurgeons, and orthopedic surgeons evaluate and care for patients by providing detailed images of both pre- and post-operative anatomy that were the stuff of science fiction a few short decades ago. Paul C. Lauterber from the University of Illinois and Peter Mansfield from the University of Nottingham

won the Nobel Prize in Medicine for their key role in the development of magnetic resonance imaging in 2003, and rightly so, for this technology has allowed not only academic research on many of the most devastating diseases, but also found widespread use in community practice. It would be difficult to find a family in the United States that has not had a member to undergo MRI, and we are all familiar with the sports announcers' refrain "The team doctor is waiting for the MR results to make a decision on the player's return to action". MR not only demonstrates the causes of the "red flags" just mentioned, but also shows soft tissue and bony causes of back and leg pain.

Plain films may be obtained prior to performing an MR, but (as noted in the case of evaluating tumor patients, above) almost always need to be supplemented by MR. One possible exception: in cases of trauma where the plain film documents a simple compression fracture, MR is typically not necessary (although often even in this scenario, the MR will provide significant additional information; see below).

MR shows the causes of "red flags"

As noted above, patients with a history of cancer, unremitting pain, and pediatric patients may have serious medical diseases. These patients require priority imaging and prompt diagnosis and management. MR is the method of choice for evaluation of these patients. Please note that while many patients with tumor, fracture, or infection *do* demonstrate these red flags, probably as many do *not*, making MR all the more valuable.

MR shows symptom producing, benign soft tissue abnormalities

MR's superiority comes predominantly from its ability to visualize soft tissues. Prior to MR, imaging relied on the use of x-rays, either to produce plain films or myelograms, or CT scans. While a wonderful invention and tremendously useful, x-ray based techniques have limitations, the main one of which is that the x-ray attenuation of different tissues such as the intervertebral disc, muscle, synovium, and even tumor is virtually identical, and the x-ray attenuation of fluid within the cerebrospinal space is not much different. Neuroradiologists relied, for decades, on secondary phenomenon to diagnose spine disease: the lost intervertebral disc space on plain films as a sign of disc herniation, or the filling defect on myelography or myelo-CT. MR easily shows each type of tissue separately, MR evaluates different physical properties of protons within the patient to create pictures showing anatomic detail and unparalleled demonstration of disease processes.

Disc Herniation

Since the original description by Mixter and Barr in 1934[2], the herniated disc has gotten much press. The North American Spine Society (NASS) originally proposed, and various other medical societies have adopted, a specific nomenclature that distinguishes subtypes of herniation[3]. If viewed axially, the normal intervertebral discs are like a tree trunk with concentrically arranged layers of oblique fibers constituting the annulus fibrosus. In the middle of the tree trunk, having a consistency of toothpaste, is the nucleus pulposus. When the annular fibers degenerate and/or tear, the nuclear material may extend or herniate beyond the fibers of the annulus, and if this happens posteriorly the effect may be compression and/or inflammation of adjacent nerves (Figure 4). The NASS terminology calls small disc herniations "protrusions" and these are much less likely to be symptomatic. The NASS terminology calls large disc herniations "extrusions" and these are much more likely to be symptomatic. Note that large, acutely symptomatic disc herniations may show significant regression when sequentially imaged, even without surgical intervention.

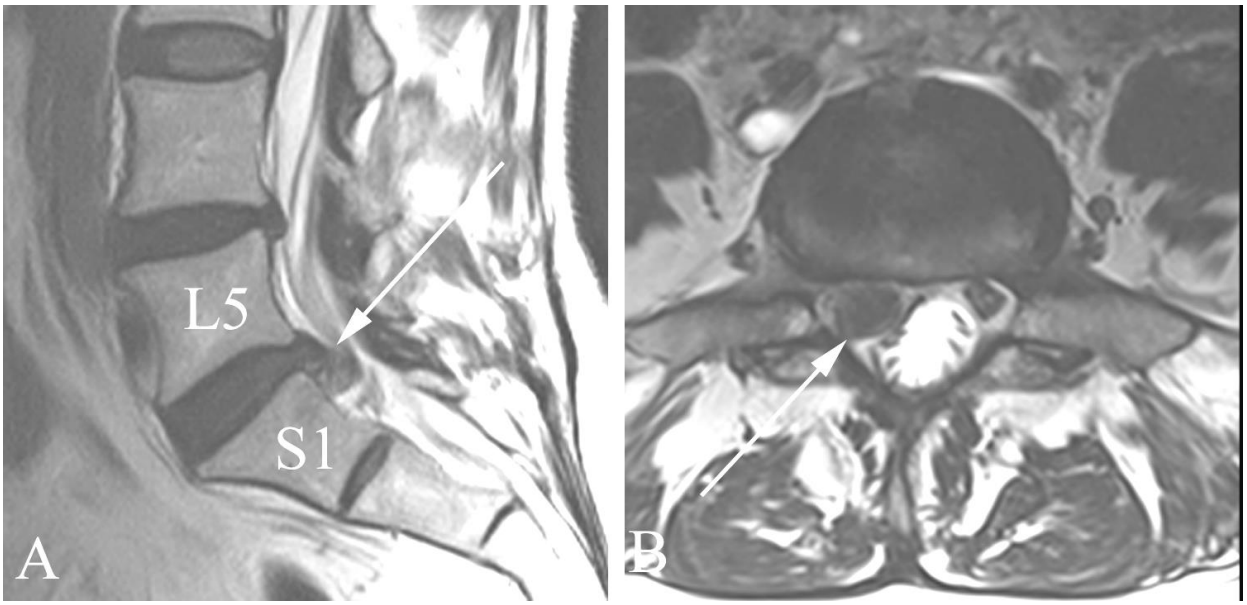

Figure 4. Disc herniation in a 59 year old woman with sudden onset of left back pain radiating down the posterior left lower extremity to the heel. A. Sagittal T2 spine MR image shows a caudally dissecting disc herniation (arrow). B. Axial T2 spine MR shows the disc herniation (arrow) indenting the thecal sac. The sagittal image has been cropped and originally included up to the T11 level.

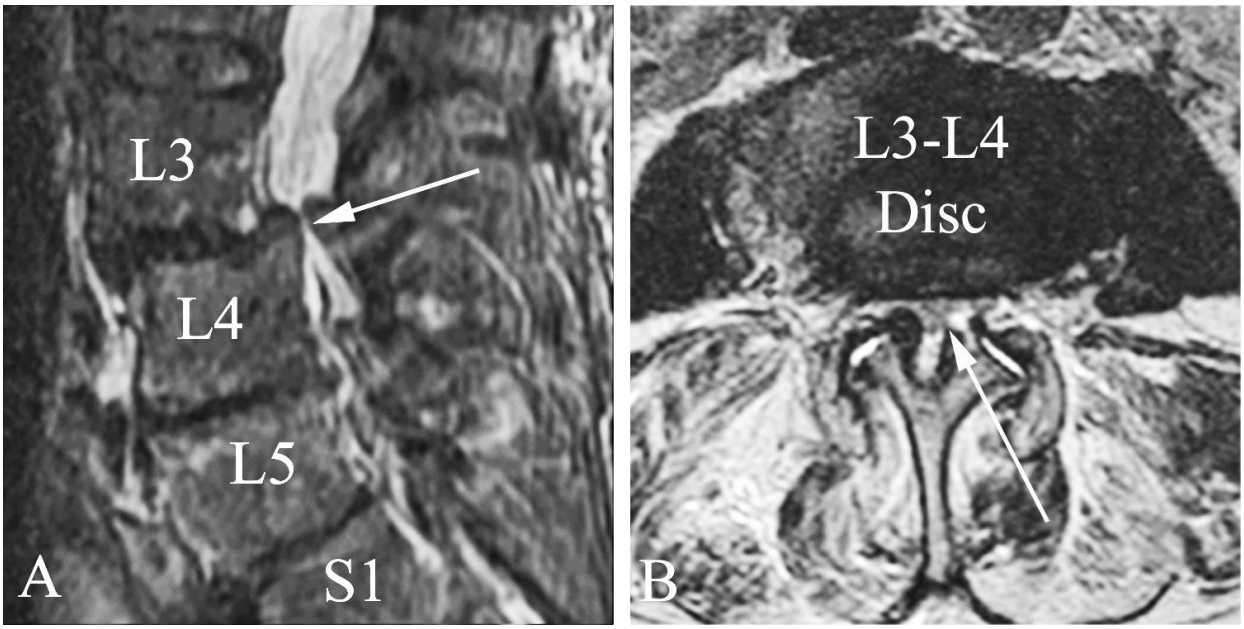

Figure 5. Spinal stenosis in an 82 year old woman with back pain and left leg pain. A. Sagittal T2 lumbar spine MR shows L3-L4 degenerative spondylolisthesis with stenosis (arrow). B. Axial T2 spine MR shows spinal stenosis (arrow). The sagittal image has been cropped and originally included from T11 through the lower sacrum.

Spinal Stenosis

Spinal stenosis refers to narrowing of those passageways through which the nerve roots and spinal segmental nerves travel, including the spinal canal, sub-articular recess, and neural foramen (Figure 5). Fibrocartilaginous tissue ("soft" tissue) causes this narrowing as or more frequently than bone ("hard" tissue), and therefore spinal stenosis is included in this section on soft tissue abnormalities.

While plain films usually show degenerative changes in patients with spinal stenosis, and CT often better shows the degree of narrowing, MR is capable of showing not only the narrowing but also demonstrating the neural structures, and any associated compression, directly. Compression of the nerves may result in pain or radiculopathy, but may also result in less specific generalized leg weakness and disability, a finding that may be exacerbated during extension and relieved during flexion. Indeed, these patients often find relief of their symptoms at the grocery store, for they use the grocery cart as an ambulation assistant which allows them to walk in a forward-flexed position which opens the spinal canal.

Synovial Cyst

A three-joint complex comprises each level of the lumbar spine. In addition to the intervertebral disc, a cartilaginous joint, in the front of the spine, there are two facet joints, which are synovial joints, at the back of the spine. These joints are prone to the same sorts of degenerative processes as other synovial joints, including cartilage loss, synovial proliferation, and secondary osteophytic spur formation. The synovial proliferation may occur posteriorly, in which case it is rarely symptomatic. Unfortunately, the proliferation and cyst formation may also occur anteriorly, where it may compress the nerve roots in the spinal canal (Figure 6) or the dorsal root ganglion in the neural foramen. These cysts may account for up to 10% of those patients that present with radicular pain[4]; clinically, these patients are indistinguishable from those with disc herniation.

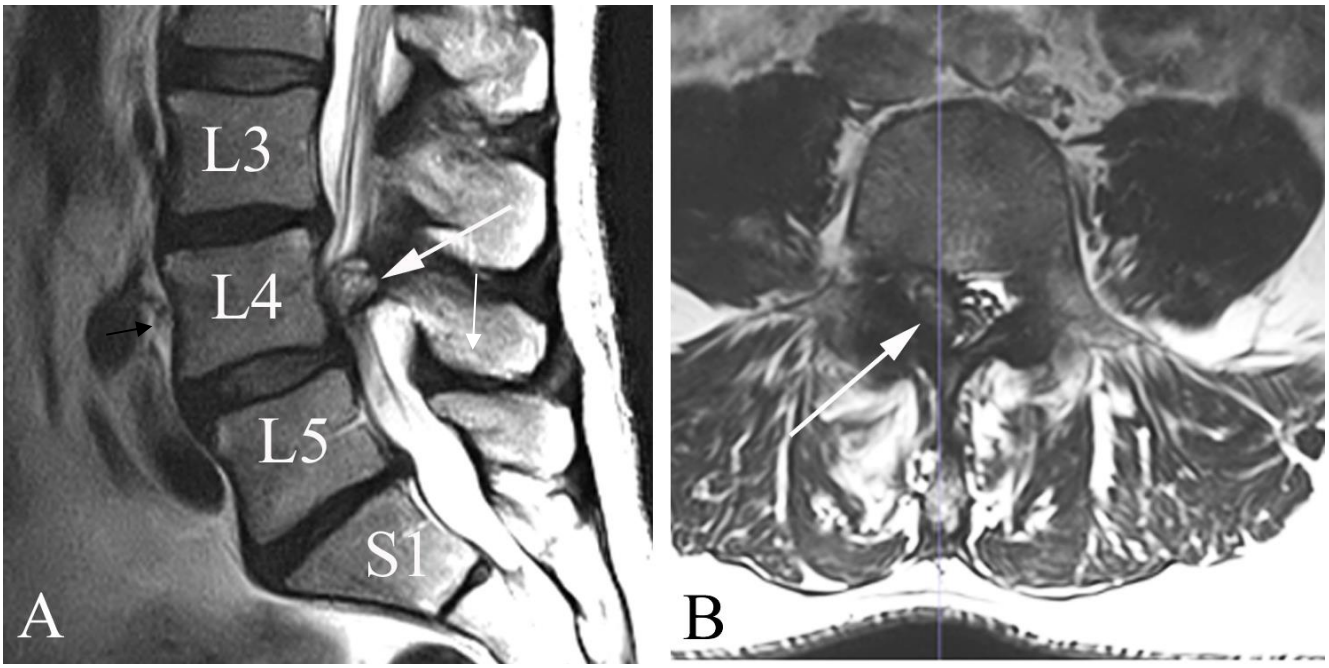

Figure 6. Synovial cyst in a 65 year old man with chronic back pain and new onset of right leg pain, right leg numbness, and right foot drop. A. Sagittal T2 lumbar spine MR shows a synovial cyst posterior to the L4 vertebra (arrow). B. Axial T2 lumbar spine MR shows a synovial cyst filling much of the right side of the spinal canal, compressing both the exiting L4 and traversing L5 nerve roots. The sagittal image has been cropped and originally included from T11 through the lower sacrum.

MR shows symptom producing, benign bone abnormalities

MR imaging is outstanding in the diagnosis of soft tissue abnormalities. It may be somewhat surprising to hear that MR is also outstanding in evaluation of most of the bone abnormalities afflicting the spine as well. This follows from the fact that while plain films can show cortical bone discontinuity and displaced bone fragments in the case of a fracture, plain films are relatively insensitive to marrow abnormalities. In fact, most of "bone" consists of bone marrow and/or trabecular bone. While MR does not show the trabeculae as well as, for example, CT, it reveals abnormal marrow tissue, whether from post-traumatic fibrovascular changes or hemorrhage, tumor, or infection. Direct visualization of the marrow allows MR to make diagnoses that are difficult or impossible with other imaging methods (Figure 7).

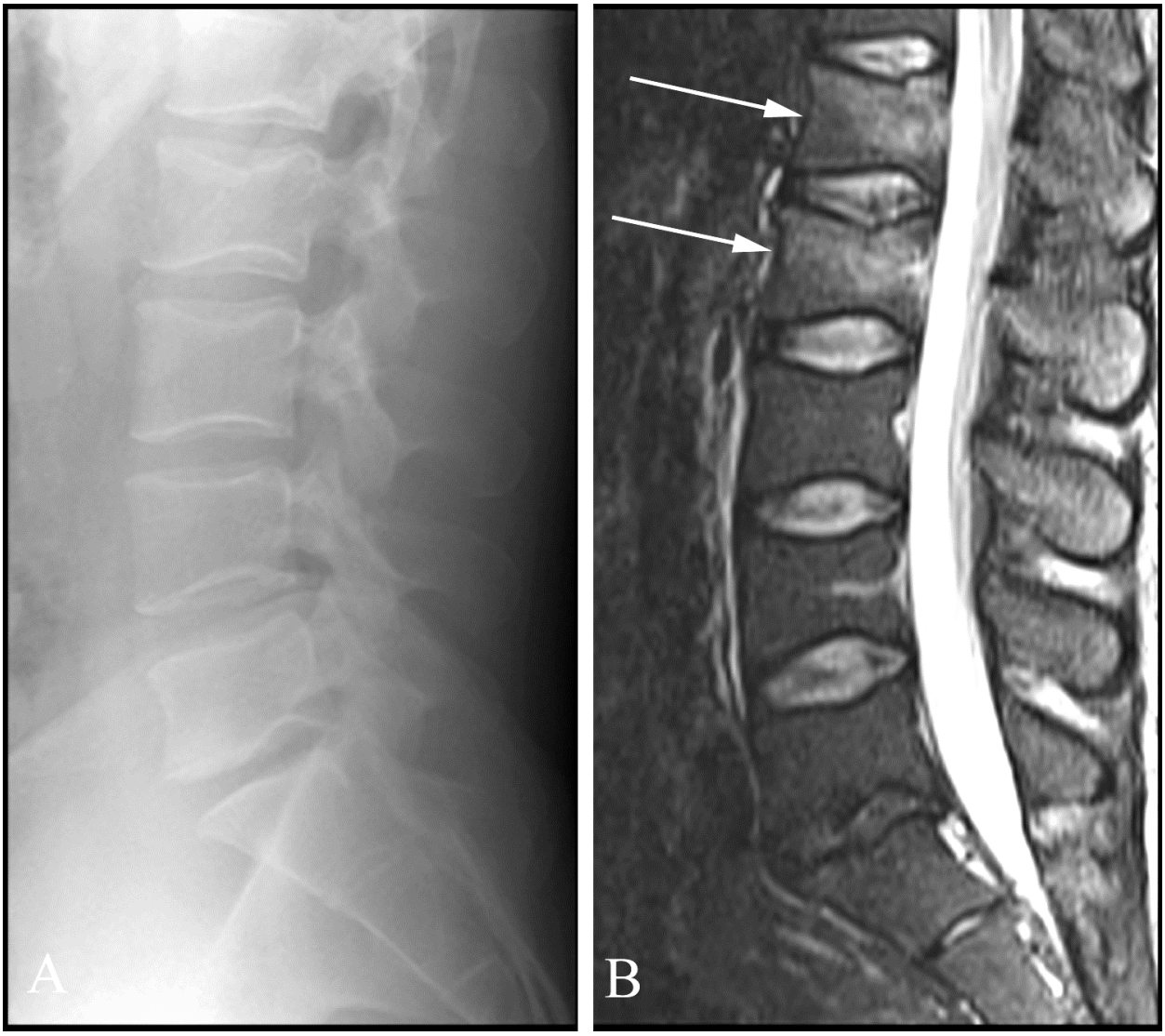

Figure 7. Radiographically occult post-traumatic fractures in a 39 year old man with back pain after falling off scaffolding. A. Lateral plain film of the lumbar spine taken in the emergency room is normal. The patient had persistent pain. B. Sagittal T2 spine MR (performed three days later, when pain persisted) demonstrates extensive abnormal marrow at the L1 and L2 levels (arrows) from contusion and trabecular fracture.

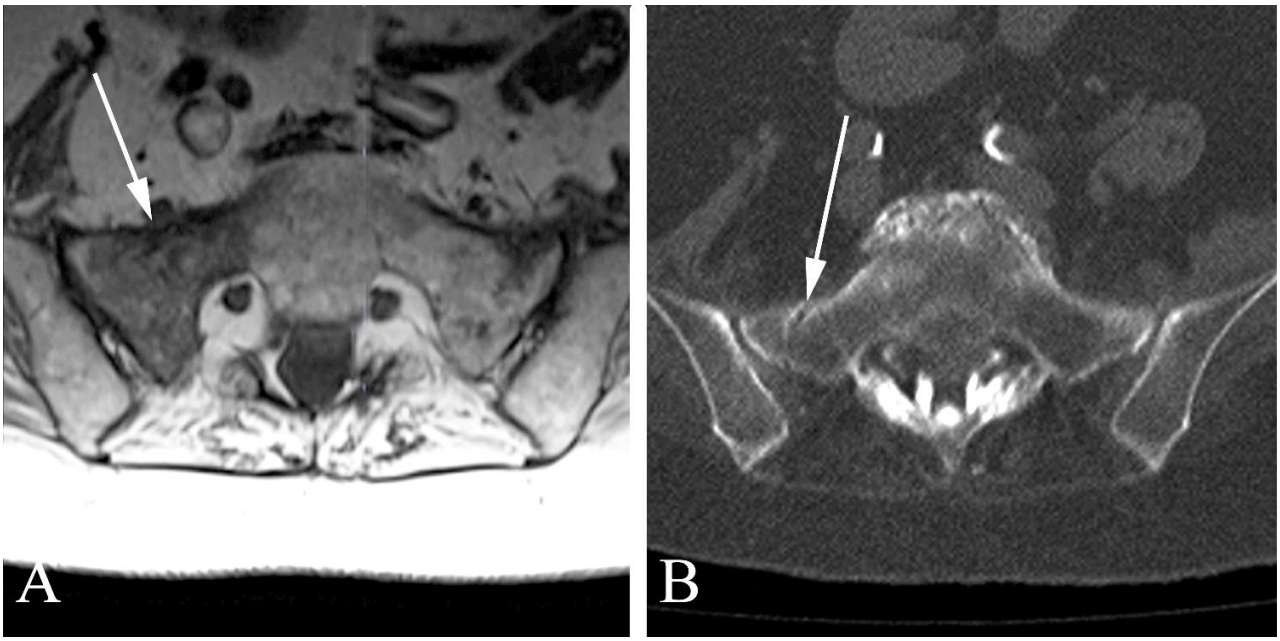

Figure 8. Radiographically occult stress fracture in a 73 year old woman with new pain following a change in exercise routine. A. Axial T1 sacral MR shows abnormal signal within the right sacral ala (arrow). B. Axial CT of the sacrum confirms a fracture lucency (arrow) corresponding to a healing stress fracture of the sacrum.

Radiographically occult post-traumatic fractures

Fractures through the marrow invariably result in fibrovascular tissue and hemorrhage. Nondisplaced but painful fractures may be impossible to see on plain films, even when you know exactly where the fracture is. At the same time, MR, through its superior soft tissue visualization, offers a specific diagnosis of bone contusion or fracture, evaluates any associated spinal canal compromise, and also excludes associated post-traumatic disc herniations (Figure 7).

Radiographically occult stress fractures

Sacral stress fractures (Figure 8) are difficult to diagnose since they may present with low back, sacro-iliac, gluteal, or hip pain, and plain films are notoriously unreliable in their diagnosis. Stress fractures of lumbar levels are much less frequent than sacral fractures.

Establishing fracture age

Many patients, particularly the elderly, will have sustained fractures earlier in life but not remember that they had prior trauma. The plain film, of course, will continue to show deformity of the vertebral body. Without a prior film, it is often not possible to distinguish between old and new fractures. MR is the study of choice for evaluation of fracture age (Figure 9). MR turns positive almost immediately and goes through a known temporal evolution of healing that allows more precise dating of the fracture. A nuclear medicine bone scan may be performed if MR cannot be done. The nuclear medicine study turns positive 1 – 3 days following injury, but may persist for months or in some cases years after the event, making precise fracture dating difficult

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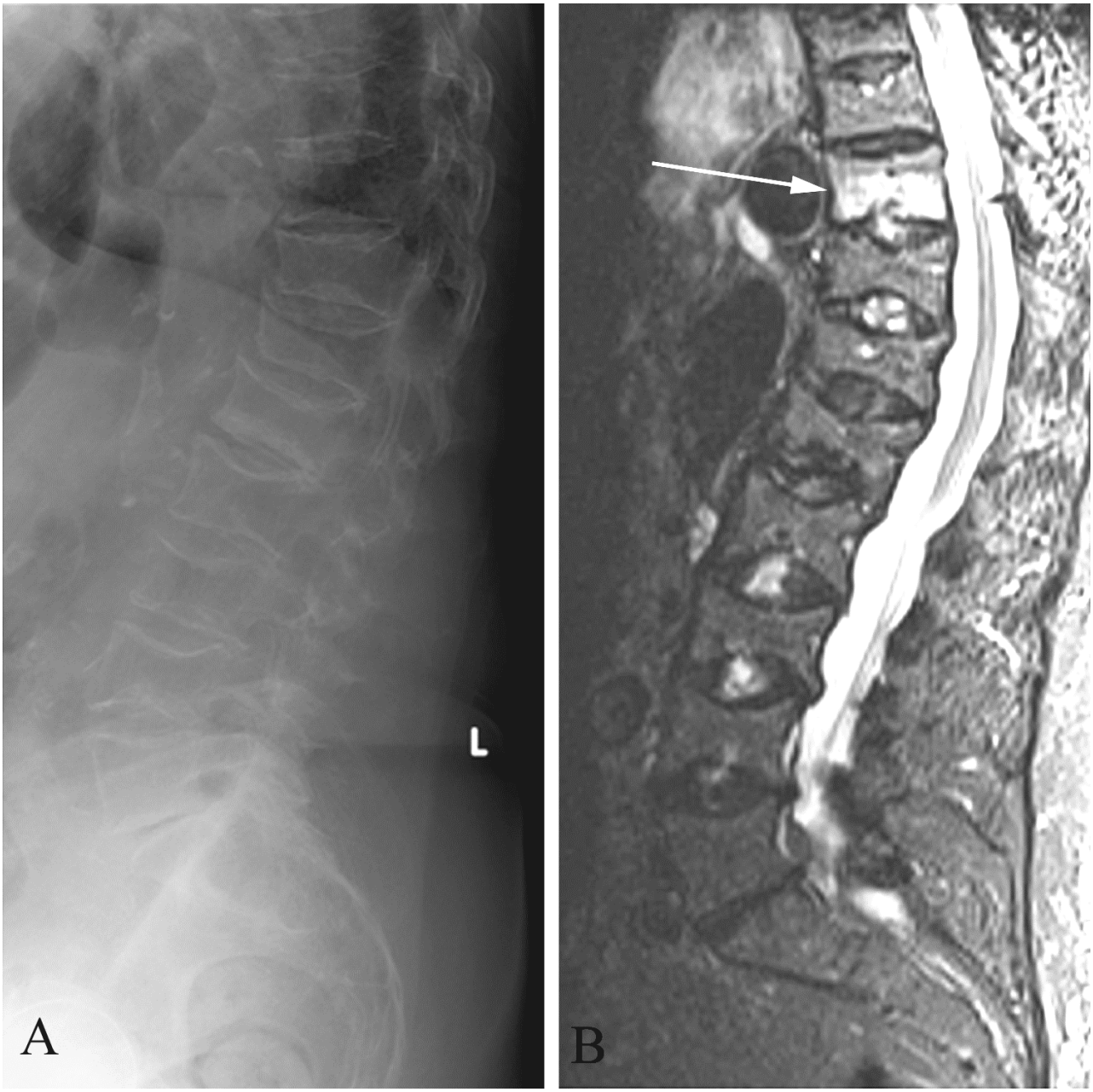

Figure 9. T10 fracture in an 85 year old white female with back pain for four days. A. Plain films showed multiple compression deformities, but no prior studies were available to determine how many, if any, of these were acute fractures. B. Sagittal STIR spine MR shows increased signal at T10 (arrow), establishing that this is the acute fracture.

SPINAL INJECTIONS MAY PROVIDE DIAGNOSTIC OR THERAPEUTIC BENEFIT

While diagnostic and therapeutic injections have been around for decades, they continue to evolve and to be more widely used. Diagnostic injections include nerve blocks, discography, facet injections including intra-articular injections and median branch blocks, and sacro-iliac joint injections. Therapeutic injections typically use the same techniques as diagnostic injections but add a long acting form of steroid to the injected material. Therapeutic epidural injections may treat multiple levels at one time. Injection of the hip and shoulder may also further delineate pain and differentiate pain emanating from these joints and the spine.

Injections may localize or treat a "pain generator"

The three fundamental assumptions of diagnostic and therapeutic injections include[5]:

1. Needle placement and injection close to or at the site of a symptomatic structure will stimulate nociceptors and thus reproduce the patient's typical pain.
2. Anesthetic placed through the needle will (at least temporarily) decrease activity within nociceptors and thus relieve the patient's typical pain.
3. Pain may be secondary to inflammation contributing to nociceptor stimulation and may respond to steroid injection.

Note that because of the placebo effect, regression to the mean, and the intermittent natural history of back pain, it is difficult to be certain that relief of pain upon injection of a structure is genuine "proof" that the structure is the cause of that pain.

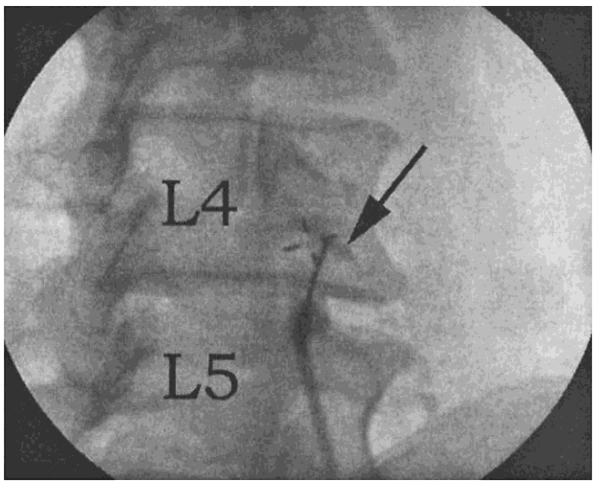

Figure 10. L4 nerve block. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located beneath the L4 pedicle and contrast material flowing along the L4 circumneural sheath (arrow).

Nerve blocks can localize and treat radicular pain

At each level of the spine, the two nerve roots come together at the level of the dorsal root ganglion, which then forms the spinal segmental nerve. The spinal segmental nerve carries with it a short sleeve connecting to the epidural space called the "circumneural sheath". Depending upon the amount of contrast material and/or medicine injected, material may seep back into the epidural space and cover other levels. For this reason, if one is performing a nerve root block for diagnostic purposes, it is necessary to limit the volume of injected material. Typically, this will consist of only 0.1 to 0.2 mL of nonionic contrast to establish that the needle is appropriately positioned, followed by injection of 0.3 to 0.5 mL of 2.0% lidocaine (Figure 10). If the patient has typical pain upon placement of the needle and during injection, and then excellent pain relief shortly thereafter, this constitutes a positive test. It is of note that placement of a needle tip in the vicinity of an irritated spinal segmental nerve is much more painful than is placement by a "normal" segmental nerve, even without injection.

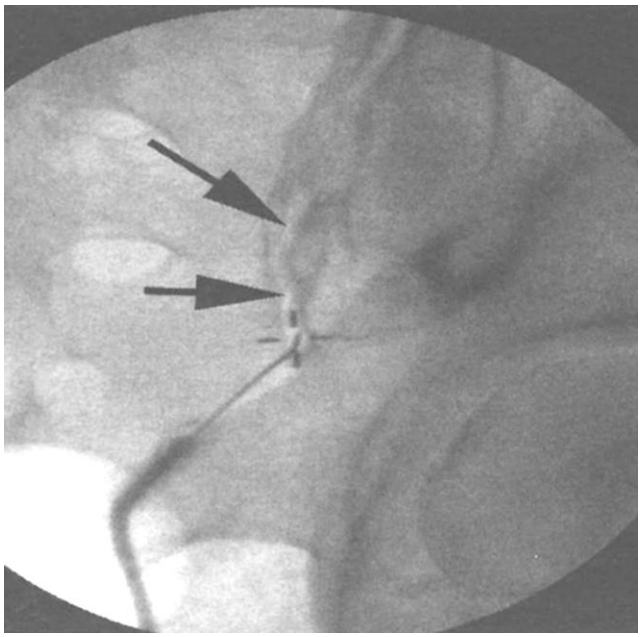

Figure 11. Sacro-iliac joint injection. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located at the inferior margin of the sacro-iliac joint, and contrast flowing superiorly into the joint (arrow).

Sacro-iliac joint injections can localize and treat SI joint pain

The sacro-iliac joint has been in and out of favor as a cause of low back and hip pain for the past 100 years or so. Since very few surgeons advocate intervention regardless of the results of injection,

there seems to be little diagnostic role for injections (Figure 11). The injections may provide pain relief.

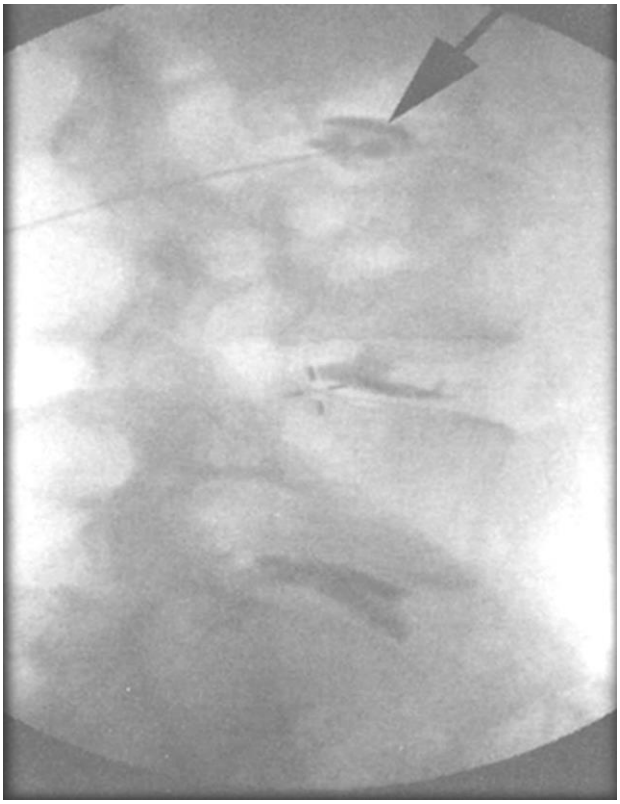

Figure 12. Discography. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located at the L3-L4 disc space with contrast within the disc (arrow). Contrast has already been introduced into the L4-L5 and L5-S1 levels.

Discography diagnoses "internal disc derangement"

Discography (Figure 12) remains the most controversial diagnostic injection done, and, for that matter, one of the most controversial diagnostic maneuvers done in medicine[6]. There are a number of reasons for this, not the least of which is the fact that the "disease" that discography is supposed to diagnose, "internal disc derangement", is very controversial itself, and has no widely accepted reference standard for diagnosis. Add to this the cost, risk, and pain of discography, and the fact that researchers continue to debate the role of false-positive diagnoses and even whether the injections may cause permanent exacerbation of backache, and you can understand why discography is so controversial. Advocates maintain that injection into a normal nucleus will not cause pain, whereas injection into a symptomatic nucleus will reproduce the patient's typical back pain. Surgeons who make use of the results of discography assume that fusing a painful level will eliminate or at least lessen the pain caused by the abnormal disc.

Epidural injections may be used to treat back and leg pain

Epidural injections, on the other hand, are much less controversial. They have been around for fifty years, and multiple controlled, randomized, blinded studies have shown efficacy of steroid over placebo[7]. While many patients tend to be frightened of the injections -indeed, the words "spine" and "needle" just don't seem to fit well in the same sentence for many patients– in fact the injections when properly performed take about five minutes and are about as painful as having blood drawn or an IV started.

It should be noted that it is fairly standard procedure to perform these procedures with fluoroscopic guidance and with the benefit of nonionic positive contrast material to document needle tip position and contrast flow. Studies have demonstrated that up to 30% of injections done without fluoroscopic guidance and contrast injection are incorrectly placed[8]. This number improves somewhat with increased experience on the part of the injector and favorable body habitus on the part of the injected patient, but is hard to get much below 10-15%. Given this fact, it is difficult to know what to make of a patient who has had a "blind" injection who doesn't improve. Was the material injected at the target location?

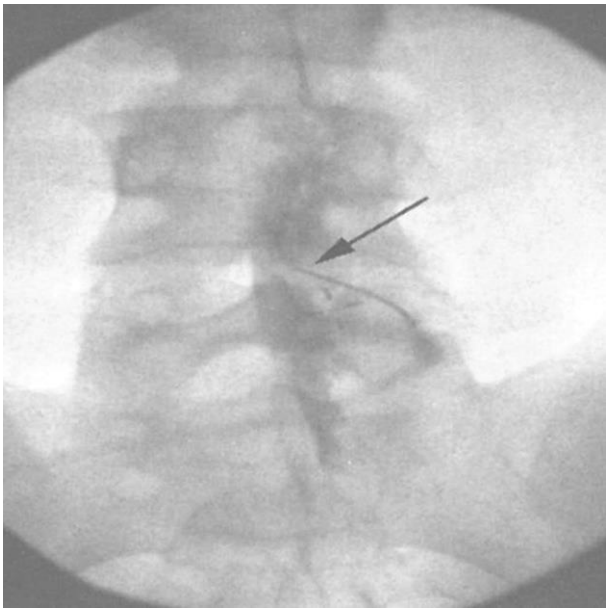

Figure 13. Lumbar interlaminar epidural injection. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located at the L4-L5 level through the interlaminar space (arrow) with contrast within the epidural space.

Lumbar interlaminar injections cover several levels

The simplest and typically least painful epidural injection to perform is the interlaminar lumbar injection (Figure 13). In this procedure the needle is advanced between the lamina of adjacent vertebrae and the injected contrast material (and drugs) will typically flow both superiorly and inferiorly for several levels. Typically, the material will also pass on both sides of the midline, although it often favors the side of the needle. Rarely, patients will have a dividing plica mediana dorsalis which keeps left sided injection from reaching the right side and vice versa. In general, the older the patient, the less likely the material will spread widely or well: scarring and limited flow within the epidural space seem to accompany gray hair.

Frequency of injection varies widely: some authorities advocate three injections done at one week intervals, while others use a single injection. One reasonable method is to plan to see the patient back a week after the injection if pain continues and if the patient is willing to undergo an additional injection. If the first injection provides partial relief, an additional injection usually provides additional benefit. If the first injection provides no relief, changing the method of injection (for example, from interlaminar to transforaminal, or from an epidural steroid injection to a facet injection) may be helpful.

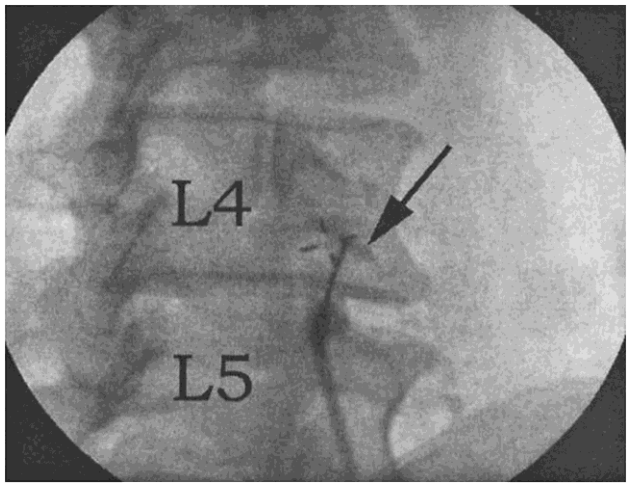

Figure 14. Transforaminal epidural steroid injection. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located beneath the L4 pedicle and contrast material flowing along the L4 circumneural sheath (arrow). Needle location (and the image) is identical to a selective nerve block (Figure 10).

Lumbar transforaminal injections target specific nerves

If this injection reminds you of the nerve block discussed above, there's a good reason: they are virtually the same procedure (Figure 14). The only real difference is the volume of material placed in the circumneural sheath. Since epidural injections are therapeutic maneuvers, it does not matter that the specificity of the nerve block is lost, and these injections bring medicine to an area in the epidural space (and therefore to certain neural structures) that may not be reached by interlaminar injections. As noted above, as people age, the flow of contrast in the epidural space diminishes, and posteriorly injected material may not reach the segmental nerves or the dorsal disc margin. In addition, material injected on the inside of the spinal canal may not pass readily through the foramen to the spinal segmental nerve if there is foraminal stenosis from osteophytic spurring along the disc margin and facet joint.

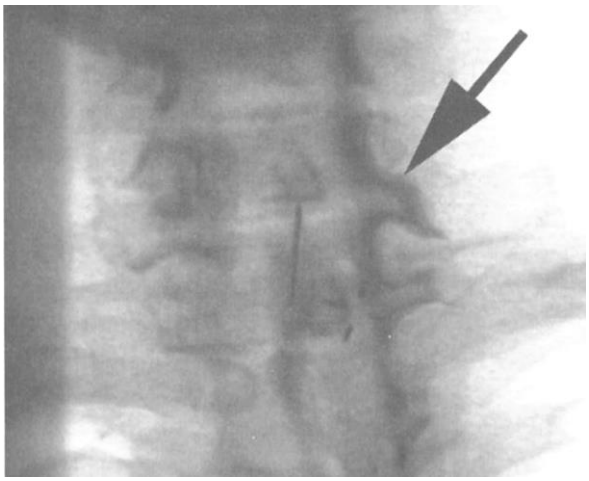

Figure 15. Cervical epidural injection. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located at the C7-T1 level with contrast within the cervical epidural space including along the circumneural sheath (arrow).

Cervical injections are possible

As noted above, most of the procedures and points stated with regard to back and leg pain, also apply to neck and arm pain coming from the cervical spine (Figure 15). Two words of caution[7]:

1. Disasters have occurred when injecting sedated patients in the cervical spine, secondary to placing the needle into the spinal cord and injecting steroid. This can cause permanent neurologic damage. While some patients are adamant that they be "knocked out" for any procedure more significant than clipping toenails, they should be warned that the ability to tell the practitioner about pain is an important part of the safety profile in injection therapy.
2. Disasters have occurred from transforaminal injections in the cervical spine because the needle tip may come to lie in a small, unnamed feeding artery on its way to the spinal cord. While these arteries may be extremely difficult to visualize during fluoroscopy even with contrast injection, multiple incidents of permanent neurologic damage have been reported from injection of steroids into these arteries.

Facet joint procedures diagnose and treat posterior element abnormalities

As noted previously, a three-part joint comprises each level in the spine, with the intervertebral disc positioned anteriorly and the two, paired facet joints posteriorly. While the intervertebral disc has occupied most of the attention of those caring for back pain for the last seventy years or so, some estimate that facet joint abnormalities cause the pain in at least 15% of patients with back pain[9]. 15% may sound like a small number until you multiply it by the number of people that have backache, with the result of several million patients in the United States.

As with other injections, the reasons to inject the facet joints are to diagnose whether they are the source of pain, and to treat this pain, if possible.

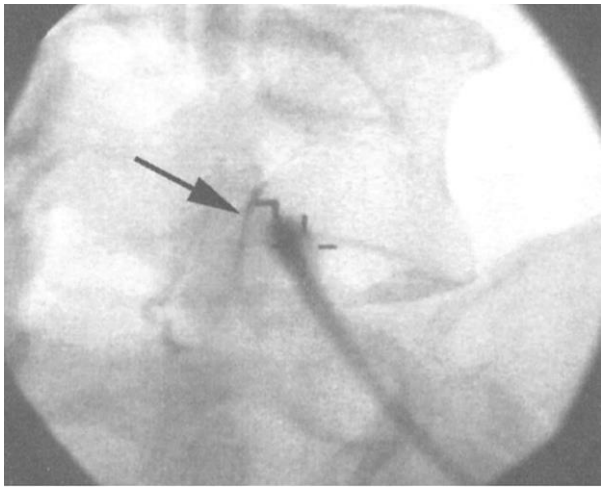

Figure 16. Facet joint injection. Fluoroscopically directed spot film obtained during the procedure shows the needle tip located in the L4-L5 facet joint. Contrast material fills the joint (arrow).

Facet joint injection can localize or treat facet joint pain

Experts have developed techniques to enter the facet joint (Figure 16). Facet joints are small structures, so the injection volume needs to be limited in order to make the injection specific. Just as with a diagnostic spinal nerve block, a total of only a few tenths of a cc of anesthetic is injected into the joint. If more than this is injected, the facet joint will rupture, and typically the material will flow into the epidural space and have the same effect as

an epidural steroid injection by covering several adjacent segments, and both sides, of the spine.

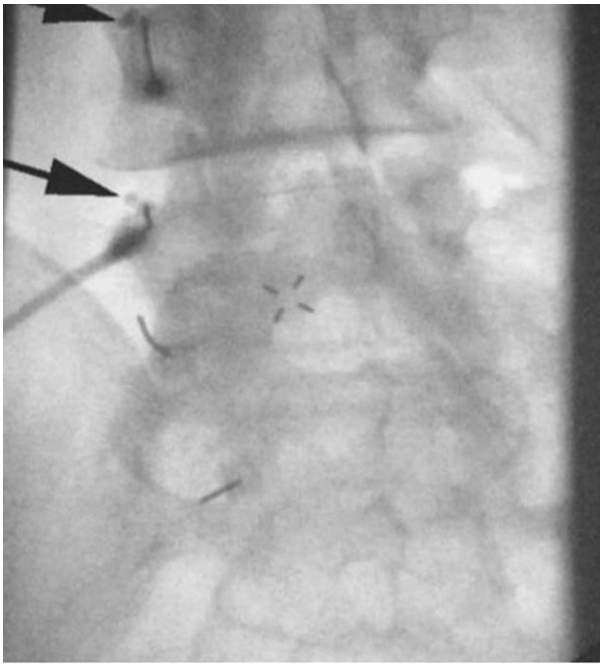

Figure 17. Facet blocks. Fluoroscopically directed spot film obtained during the procedure shows the needle tips located at the expected positions of the dorsal root branches which enervate the L4-L5 and L5-S1 facet joints. A small amount of contrast shows an appropriate, nonvascular appearance of the contrast material (arrows).

Rhizotomy can provide long-term relief from facet joint pain

In addition to injecting within the joint, the anatomy of the facet joints allows an alternative approach to diagnosis and treatment. This is the so-called median branch block (Figure 17). Facet joint ennervation comes from the median branches of the dorsal rami of the spinal segmental nerves above and below the level of the joint. What this means is that small amounts of anesthetic injected at the known location of the median branches will anesthetize the joint just as effectively as intra-articular injection. Figure 17 demonstrates needles in place at the location of the medial branches of the lower lumbar facet joints. If the patient achieves pain relief from these injections, the patient may be a good candidate for a rhizotomy, which is percutaneous lesioning of the nerves supplying the facet joints. These nerves typically perform no other function, and rhizotomies have been shown in randomized, controlled studies, to provide benefit beyond placebo needle placement[10].

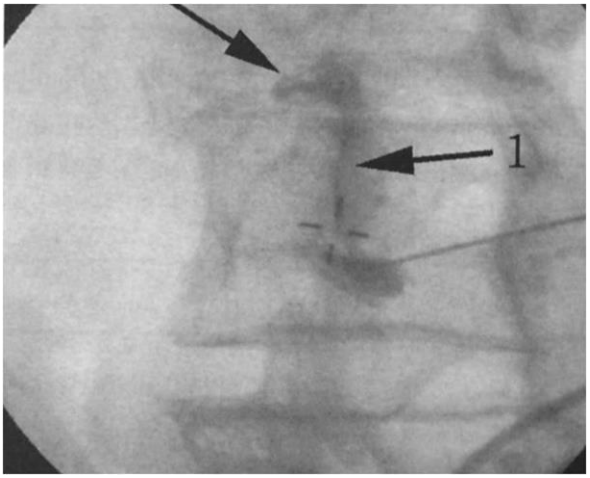

Figure 18. Facet injection performed for synovial cyst rupture. Fluoroscopically directed spot film obtained during the procedure shows the needle tip along the dorsal recess of the L4-L5 facet joint, with contrast material extending through the joint and into the synovial cyst (arrow).

Synovial cyst rupture can cure radiculopathy

As illustrated previously (Figure 6), synovial cysts may arise from the facet joints and extend into the spinal canal or neural foramen. When small, these cysts are typically considered an asymptomatic accompaniment of facet arthropathy. However, when large, they may compress nerve roots or spinal segmental nerves and thus cause radicular pain. When this happens, the patient has the same symptoms as if he or she had a disc herniation. With this diagnosis, the three major options are:

1. Treat with oral anti-inflammatory medication. The cysts are said to eventually go away, particularly if the joint inflammation subsides, and this is one way of approaching the problem, although for patients in radicular pain the wait may be agonizing.
2. Resect the joint and fuse the spine. This seems a particularly aggressive approach, but unlike disc herniations which are amenable to microscopic discectomy, fixing a bad facet joint generally entails removing

the entire thing, and stabilization is necessary following this maneuver.

3. Percutaneous rupture (Figure 18). By placing a needle in the joint and vigorously injecting it, it is often possible to rupture the joint, as mentioned above. In this case, that's a good thing, because it can cure the radicular pain in minutes.

These injections may help those patients with multilevel disease or with both disc and facet abnormalities by providing a more specific diagnosis. They may provide patients with back and leg pain, even without a specific diagnosis, relief from their discomfort.

One final pearl about these injections is that while the steroids injections would seem to have a time-limited effect, the relief of pain often extends for weeks or even months following injection. There are a couple of potential explanations for this prolonged benefit:

1. Back pain in general is a symptom given to exacerbation and remission. If the patient comes for injection when most painful, he or she would improve whether the injection was done or not, a phenomenon called "regression to the mean". Nonetheless, if their pain is relieved when it is most severe, this is of benefit.
2. Nerve nutrition requires appropriate blood flow, both on the arterial and venous side. Studies have shown that impaired flow prompts nerve swelling and a minimal superimposed insult may start a vicious cycle where the swelling actually contributes to the impaired flow. If the anti-inflammatory effect of the steroid makes the nerve smaller, blood flow may improve and the patients may therefore experience long-term relief of symptoms.

SUMMARY

1) "Red flags" in the patient's presentation call for priority imaging.
2) MRI has supplanted other modalities for the imaging work-up of back pain.
3) Injections often provide diagnostic or therapeutic benefit for patients with back and leg pain.

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"First Step" Imaging of Gastrointestinal Symptoms

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This chapter covers four main points designed to help you order the correct first test when evaluating patients with gastrointestinal (GI) complaints:

1. There are a limited number of "first step" studies for gastrointestinal symptoms: ultrasound (US), computed tomography (CT), and videofluoroscopy.
2. Ultrasound is the imaging study of choice for evaluating right upper quadrant pain and suspected acute pancreatitis. Ultrasound is the best way to figure out if someone has a diseased gallbladder and/or a dilated biliary tree. In pancreatitis, the study is done not so much to see the pancreas itself (although this may be useful) as it is to exclude a reversible cause of obstruction of the pancreatic duct (mainly, gallstones).
3. CT is the imaging study of choice for evaluation of "Abdominal Pain Plus". Of course, not all patients with abdominal pain need CT imaging, but there are many instances when they do. This chapter reviews the specific clinical scenarios (and the associated disease processes) when CT is in order.
4. Videofluoroscopy (a "swallowing study") is often the best first step in evaluation of oropharyngeal dysphagia, whereas endoscopy has supplanted the barium swallow for evaluation of esophageal dysphagia.

THERE ARE A LIMITED NUMBER OF "FIRST STEP" STUDIES FOR EVALUATION OF GASTROINTESTINAL SYMPTOMS

While there are multiple ways to image patients with GI complaints, "first step" studies are few in number, namely, ultrasound, CT, and videofluoroscopy. This section reviews the mechanics of ultrasound, CT, and swallowing studies from the technological point of view, while later sections look at each modality from the patient's and ordering clinician's point of view.

Ultrasound

Ultrasound is widely available, does not use ionizing radiation, and is typically considerably lower in cost than CT and MR. These factors have led, particularly in Europe (where there is much more concern about radiation exposure than in the United States), to a nearly universal use of these machines in physician's offices and emergency rooms.

It is easy to forget that ultrasound, like all imaging, is subject to continual improvements of technology. Machines are getting smaller and cheaper, although the bigger machines are still better at providing the best images. The software and hardware are undergoing continual refinements including such features as "harmonic imaging" and higher frequency transducers that allow prettier (and more accurate) pictures. A new type of transducer now allows routine acquisition of volumes of data with "isotropic voxels", enabling routine three-dimensional reformatted images in ultrasound that are widespread in CT. When machines equipped with this technology become widely available, this will greatly reduce the "operator dependence" that has been one of the main drawbacks with ultrasound. Instead of technologists laboring to find the ideal imaging plane and machine settings to visualize a particular part of, for example, fetal anatomy, the technologists will merely have to run the transducer over the patient once or twice, and the rest will be done with imaging processing at a workstation. The time to perform ultrasound studies will go from thirty to sixty minutes to five minutes, and the wait time to get an exam time should approach zero, just as it has for CT with the advent of multi-slice scanners.

When patients come to most radiology departments for ultrasound done for GI symptoms now, however, the technologists still do things the old fashioned way, which is to say, they scan the patient looking for specific items of anatomy. For a right upper quadrant US, the technologist will obtain images of the liver from a number of directions, and try to get the liver in the same scan as the kidney on at least a few pictures, because this allows an evaluation of the relative echogenicity of the liver and kidney. The denser (whiter) the liver is, the more likely it is to be a fatty liver. The tech will look for masses in the liver and assess the portal vein and hepatic vein, checking for patency and direction of flow in these vessels. Of course, one of the main areas of attention will be the gallbladder. For this organ, the lumen should be free of echoes and the wall thickness should be uniform and measure less than 2 mm, and the margins should be against either the liver or the bowel without intervening fluid. The technologist should note whether the patient is tender when the probe is placed directly over the gallbladder for scanning (which typically requires at least some light pressure), and record this as a "sonographic Murphy's sign". The intrahepatic ducts will be included as part of the evaluation of the liver. The common duct is typically measured where it is next to the portal vein. This may be either above the junction of the common hepatic duct (formed by the confluence of the right and left hepatic ducts) and cystic duct, or below this junction. Therefore, it is often not possible to say whether the measurement pertains to the common hepatic duct or the common bile duct, so the correct wording is probably "common duct". Regardless, the measurement should be 7 mm or less in patients less than 60 years of age, and 10 mm or less in older patients[1], at least in patients who still have their gallbladder. The pancreas is often not well seen on abdominal ultrasound, particularly the tail. The body may be relatively well evaluated depending on bowel contents, and longer periods of fasting are generally associated with better visualization of the pancreas. Even seeing a portion of the body and head allows evaluation of the pancreatic duct, which is important to see in patients with possible pancreatitis, to make sure it is not dilated. The right kidney is routinely included in this evaluation, because right kidney hydronephrosis, a stone, or tumor could cause right upper quadrant pain.

Radiology departments usually offer both "right upper quadrant" ultrasound studies for evaluation of the liver, gallbladder, biliary tree, pancreas, and right kidney, and a "complete abdomen ultrasound" which evaluates the same structures plus the left kidney, aorta, and inferior vena cava. The "complete" study is typically done to survey the entire abdomen, and in this regard the study comes

in a poor second to the abdominal CT for most disease processes, even a CT done without IV contrast (see below). However, it can be done as a portable exam. Even so, "complete abdominal ultrasound" is generally not a "first step" study for gastrointestinal complaints.

Computed Tomography

There has been a revolution over the past decade or so in CT scanning as multi-slice scanners changed the industry. Isotropic voxels (imaging volumes with the same resolution in all three planes) allow multiplanar reformatting and other methods of data interrogation that leads to improved diagnosis. In addition, the time required to perform a CT scan dropped from an hour or more to a few seconds, and most of the time "scanning" a patient is now spent either waiting for orally administered contrast material to travel through the gastrointestinal system, intravenously administered contrast material to travel through the circulatory system, or to get the patient on and off the table.

Note that, for the most part, a radiologist will be responsible for setting the protocol for performance of the CT study, taking into account the clinician's request and the patient's condition. It is certainly possible to perform a CT study with neither IV nor oral contrast material, a so-called "CT-KUB". As noted in Chapter 1, the main use of the CT-KUB is for suspected renal stone disease. Typically, it is best to perform more than the CT-KUB for evaluation of GI issues. Obviously, the bowel is much better seen with contrast material in the lumen, and it is only possible to fully assess many solid organ tumors, vascular abnormalities, and bowel wall lesions with IV contrast. Therefore, most studies will be performed with some combination of oral and IV contrast.

While the radiologist sets the protocol for CT studies based on patient symptoms and laboratory data, the necessity to improve quality by maintaining a uniform technique results in many studies being done with what may be called a "routine" technique for abdomen and pelvis examination involving oral and IV contrast. Positive oral contrast may be provided by dilute oral barium (more often used) and dilute oral water-soluble non-ionic contrast (preferred in those cases where there is any question of contrast extravasation from perforation). IV contrast has, of course, evolved through time. Modern nonionic contrast is designed to be less nephrotoxic than the ionic contrast material of yesteryear, and some recent papers have challenged the entire concept of whether modern nonionic water soluble contrast materials are nephrotoxic at all[2]. Nonetheless, most departments routinely assess renal function and use a combination of features including serum creatinine, glomerular filtration rate, and patient circumstances to determine whether to give intravenous contrast or not. In some cases, either hydration (for borderline renal insufficiency issues) or premedication with steroids (in cases of patient allergies) may be used. See Chapter 16 for a longer discussion of IV contrast material.

With regard to enteric contrast, in some cases, particularly in patients with known inflammatory bowel disease undergoing work-up for small bowel inflammatory changes, the routine dense, positive contrast within the bowel impedes visualization of inflammatory changes in the bowel wall which may be seen following IV injection of contrast material. Manufacturers have developed a low density contrast material for studying these patients. Woo et al[3] found that whole milk is just as good at bowel distension and visualization, cheaper, and preferred by patients.

In addition to, or instead of, enteric *oral* contrast material, enteric *rectal* contrast material allows excellent distention and study of the large bowel, which is typically poorly distended with only oral contrast. Rectal contrast may be particularly useful in patients suspected of diverticulitis or appendicitis.

With regard to the timing of scans following intravenous injection of contrast material, the ability to acquire a scan in a few seconds with multi-slice CT scanners permits evaluation at any of several phases of vascular opacification. Routine studies are typically performed as "two phase" examinations. The first phase is the so-called "portal venous phase" (which in most normal patients occurs 60 – 90 seconds following beginning of the IV injection) which shows the portal venous system, but also the arterial tree. A later, delayed, scan allows evaluation of the urinary tract collecting system including the renal pelves, ureters, and bladder, as

well as the dynamics of any contrast enhancing lesions such as hemangiomas of the liver.

The radiologist may alter this sequence, typically in one of a few specific ways:

1) Obtaining a precontrast study to evaluate renal stones which may be obscured following contrast material, solid tumors such as adrenal adenomas where the pre-contrast images may allow density measurements to allow histologic specificity, and aortic wall density to check for hematoma formation. Of course, it may not be clear that you need a non-contrast study until after the fact, and therefore the patient may need to return to the department at a later time. Performing all studies without IV contrast as a routine would be possible but low yield, and, it is thought, does not justify the additional expense and radiation exposure.
2) Obtaining an arterial phase study, to get an even better look at the arterial tree than is allowed by the portal venous phase and to check for early contrast enhancement of some tumors.
3) Obtaining additional, more delayed studies, usually to confirm liver hemangiomas or to evaluate the urinary tract when there is pathologically delayed filling.

Barium Studies and Plain Films

There was a time twenty to thirty years ago when barium studies formed a large proportion of the work done in a radiology department. At that time, barium studies using oral contrast were routinely employed to evaluate mucosal disease, namely esophagitis, gastritis, gastric ulcers, duodenitis, duodenal ulcers, malignancies of the esophagus, stomach, and proximal small bowel, whereas barium administered rectally was used to evaluate polyps, villous adenomas, and malignancies of the large bowel. Water soluble upper GI studies were used in the post-operative situation, and water-soluble enemas for both post-operative situations and for evaluation of suspected diverticulitis. Almost all of these indications now result in the ordering of different studies, usually either optical endoscopy or cross-sectional imaging. There is basically a single exception where a barium study is still considered a "first step" examination for evaluation of gastrointestinal symptoms and that is videofluoroscopy for oropharyngeal dysphagia.

Dysphagia, or difficulty swallowing, may be divided into oropharyngeal dysphasia, accompanied by difficulty initiating a swallow, choking, and an abnormal feeling in the throat or cervical region, and esophageal dysphagia, or difficulty swallowing with the abnormal sensation in the lower chest and typically occurring several seconds following swallowing[4]. Videofluoroscopy studies, done for oropharyngeal dysphagia, are done in conjunction with a speech pathologist. These studies are typically recorded on videotape. The study allows evaluation of swallowing function using a variety of substances, typically starting with thin (water consistency) barium, and proceeding to nectar thickness, honey thickness, applesauce, thin liquid barium with fruit cocktail, barium smeared over pieces of meat, and barium on a graham cracker. Usually, the test is finished off by observing the passage of a 13 mm barium tablet through the esophagus. The idea is to test the function of the oropharynx, hypopharynx, epiglottis, and proximal esophagus in the usually smooth, even flow of material from above to below, avoiding the larynx and trachea. With the swallowing study, the main report on the examination, as well as the recommendations for treatment, will typically come from the speech pathologist performing the procedure, with a radiology report usually providing a relatively limited summary of the main findings. Endoscopy has largely supplanted the barium swallow in the evaluation of esophageal dysphagia.

Plain films of the abdomen have largely been replaced by ultrasound (for right upper quadrant pain suggesting biliary disease) and CT (for other indications). The role of plain films is limited because the plain films are rarely definitive: typically, they add little and another imaging examination (most frequently CT) is done anyway[5].

ULTRASOUND IS THE IMAGING STUDY OF CHOICE FOR EVALUATING RIGHT UPPER QUADRANT PAIN AND SUSPECTED PANCREATITIS

Ultrasound is the examination of choice for suspected biliary disease[5]. The first finding to look for in the case of someone with right upper quadrant pain is a gallstone (or gallstones) in the gallbladder (Figure 1). Such stones are likely the cause of the patient's abdominal pain, particularly if accompanied by: a classic history; a positive physical examination finding of tenderness over the gallbladder; a positive "sonographic Murphy's sign"; or additional imaging findings such as the stone being lodged in the gallbladder neck or accompanied by gallbladder wall thickening or pericholecystic fluid. However, it is good to keep in mind that many people harbor asymptomatic gallstones, and right upper quadrant pain and gallstones may co-exist even when the stones are not the cause of the pain. When the pain is atypical or not accompanied by other imaging features, be suspicious of alternative diagnoses such as gastritis, duodenitis, or ulcer disease. On the other hand, elderly patients may have cholecystitis *without* classic right upper quadrant pain, epigastric pain, or enzyme abnormalities: they may simply have nausea and vomiting[6].

In addition to gallstones, a finding which may accompany gallstone disease is a dilated biliary duct. The upper limit of a normal duct is typically taken to be about 7 mm, although slightly larger measurements may be encountered in the elderly. A dilated biliary duct in a patient with cholelithiasis may occur because of a stone lodged in the duct downstream from the dilatation (choledocholithiasis) or residual dilatation from prior passage of stones. Since the common duct is tricky to examine through its length by ultrasound, additional tests including nuclear medicine studies, magnetic resonance cholangiopancreatography (MRCP), or endoscopic retrograde cholangiopancreatography (ERCP) may be necessary to evaluate patients when there is a strong suspicion of choledocholithiasis (See page 106). The determination of whether to perform these tests is usually made by a gastroenterologist or GI surgeon.

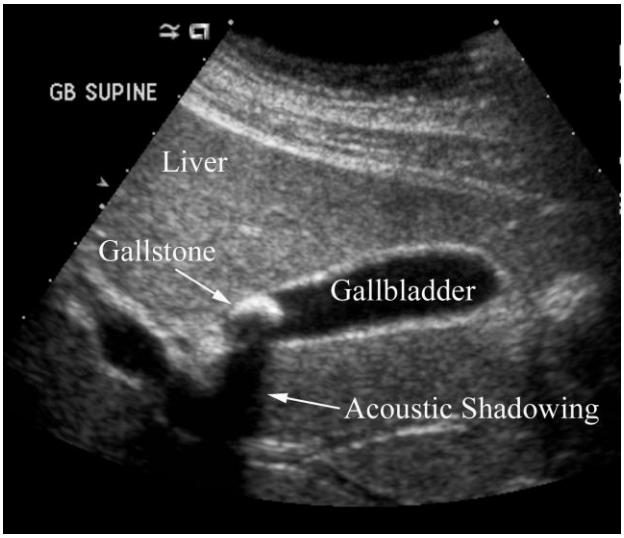

Figure 1. Gallstone disease in a 31 year old woman with right upper quadrant pain. Ultrasound demonstrates a hyperechoic, shadow casting structure within the gallbladder diagnostic of a gallstone.

Other additional features of gallstone disease include a thick walled gallbladder and pericholecystic fluid, which suggest inflammation from acute cholecystitis. Again, clinical features are the key: if accompanied by recurrent right upper quadrant pain and gallbladder tenderness, or a sonographic Murphy's sign, the diagnosis is obvious. The issue is more complicated in patients with congestive heart failure, because accompanying hepatic congestion may cause right upper quadrant pain and ascites, and ascites may collect around the gallbladder as pericholecystic fluid, mimicking the inflammatory fluid of cholecystitis.

Regarding suspected pancreatitis, it is important to exclude gallstone pancreatitis, caused by choledocholithiasis downstream from the insertion of the pancreatic duct into the common bile duct, as a reversible cause of acute pancreatitis, and ultrasound is the study of choice to accomplish this goal. CT will typically better demonstrate the inflammatory changes around the pancreas than ultrasound. In severely ill patients, CT allows evaluation of significant peripancreatic fluid collections which may require drainage, and also allows evaluation of pancreas perfusion (which

provides important prognostic information about the likely course of pancreatitis for a given patient). It may be necessary to perform both US and CT in a given individual if the diagnosis is in doubt. If the ultrasound documents a normal appearance of the gallbladder and pancreas and the patient has typical clinical and laboratory features of mild pancreatitis, particularly if there is an identifiable offending agent such as alcohol involved, it is probably reasonable to treat the patient as having pancreatitis without CT or other additional imaging.

CT IS THE IMAGING STUDY OF CHOICE FOR EVALUATION OF "ABDOMINAL PAIN PLUS"

The simple way of thinking about pain is that it arises from stimulation of nerve fibers. The autonomic nerve fibers of the viscera react to distension, whereas both these visceral nerve fibers and the somatic nerve fibers of the peritoneum react to inflammation. Abdominal nerve fibers tend not to react to cutting or tearing. Therefore, those things that cause distension and inflammation lead to pain. In general terms, endoscopy tends to work best for evaluation of those diseases which cause inflammation of the mucosal lining, whereas imaging offers a better evaluation for those processes causing obstruction, particularly if the obstruction has resulted in distension of the obstructed structure. Examples of the obstruction-distension-pain process include: gallstones in the gallbladder neck, cystic duct, or biliary tree; appendicoliths or lymphoid tissue obstructing the appendix, and fecaliths obstructing diverticulae. In many cases, the obstruction also produces inflammation secondary to the dilated structure's lining stretching to the point where it leaks: typically, the contents of these structures irritate the adjacent tissue or (in the case of some bowel contents) infects them, leading to inflammatory pain. From the clinical standpoint, it may be quite difficult to tell prior to the work-up which of the two categories (mucosal inflammation or obstructed structure) the patients fits into, and whether endoscopy or imaging may offer the best chance of providing a diagnosis. Additional features of the pain may help in the process of test selection. In the scenario of abdominal pain with features suggesting biliary colic or pancreatitis, as noted above, ultrasound is the first study of choice. In most other instances of "abdominal pain plus another feature" where imaging needs to be performed, CT is the first imaging study of choice. This section reviews those features.

Abdominal pain and inflammation

Patients may present with abdominal pain and features of inflammation, such as fever or an elevated white blood cell count, ESR, or C-reactive protein. Statistically, you are looking for one of a few disease processes: appendicitis, diverticulitis, and appendagitis epiploicae.

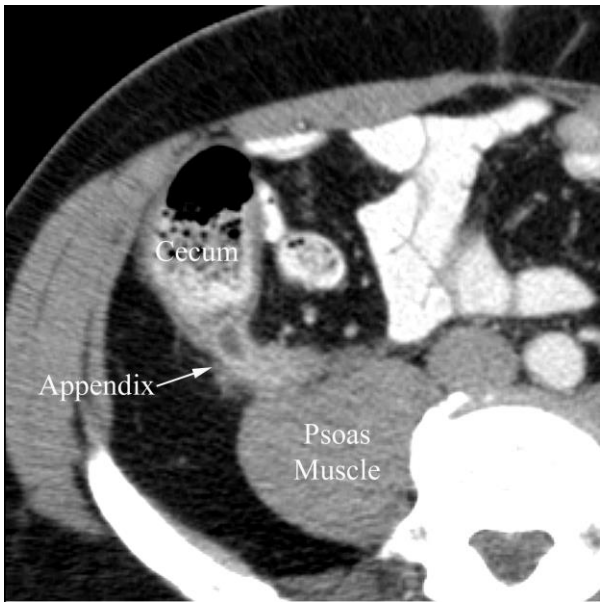

Figure 2. Appendicitis in a 52 year old man with right lower quadrant pain. CT study through the right lower quadrant performed with oral and IV contrast shows a swollen appendix with marked contrast-enhancement of the appendix wall along with periappendiceal fat stranding.

Appendicitis

Appendicitis may follow obstruction of the appendix, typically by an appendicolith but also by lymphoid tissue along the base of the appendix[7]. This obstruction results in distension of the appendix and subsequent leakage of material from the appendix, with inflammatory change of the periappendiceal fat and/or fluid in the peritoneal

cavity (Figure 2). The process may eventually result in rupture of the appendix with extraluminal fluid and/or air in the right lower quadrant. This process may take several hours, and patients typically initially have generalized pain which, through time, localizes to McBurney's point (halfway between the umbilicus and anterior superior iliac spine). These patients typically lose all appetite and often have nausea and vomiting. When the patient has a classic clinical presentation accompanied by an elevated WBC, emergent surgical consultation should follow, which will likely be followed by emergent appendectomy.

Not all presentations of appendicitis are classic: frequently, patients will not have an elevated white count, or will have hematuria or pyuria, complicating clinical diagnosis. When you suspect appendicitis but the diagnosis remains uncertain following clinical and laboratory examination, CT is usually the study of choice. Ideally, this CT will be performed with both oral and intravenous contrast material. The use of oral contrast in these cases may present a problem. The benefit of oral contrast is that it opacifies the bowel and makes it easier to diagnose bowel abnormalities, which are among the chief causes of abdominal pain. Detriments include that the nauseated patient may not tolerate oral contrast, that CT scanning should optimally be performed at a minimum two hours *after* beginning to ingest oral contrast to allow time for the contrast to reach the large bowel, and that if the patient goes to surgery immediately following the CT exam, the enteric contents increase the risk of GE reflux, vomiting, and aspiration pneumonia. Accordingly, it may be reasonable to perform a so-called "CT-KUB" or unenhanced CT of the abdomen and pelvis, as is done for suspected renal stone disease (see pages 6-7), since renal stones are in the differential diagnosis for many of these patients anyway. Such an unenhanced exam will often demonstrate appendicitis well, particularly in patients who sport a few extra pounds (where intraperitoneal fat surrounds bowel loops and the appendix). Of course, the CT-KUB may be equivocal or nondiagnostic, in which case the best next step is usually to give the patient oral contrast and scan the patient in two hours with both oral and IV contrast.

Ultrasound (US) examination of the appendix has long been advocated as a useful study in evaluation of patients with suspected appendicitis and remains popular in some radiology departments as the *initial* study of choice, particularly for pregnant women and pediatric patients. Ultrasound findings include demonstration of appendiceal swelling or an appendicolith. US is highly "operator dependent," however, and unless the ultrasonographer is skilled and experienced with ultrasound for appendicitis, the exam is prone to false-negatives. Since CT usually follows a negative US in a patient where appendicitis needs to be excluded, the addition of ultrasound to the work-up may simply add expense and delay diagnosis. A more recently developed alternative to performing CT in pregnant women suspected of appendicitis is MR, which may demonstrate appendiceal swelling and periappendiceal inflammation[8]. MR almost certainly has fewer adverse consequences for the fetus than CT, but because of the relative novelty of the use of MR for appendicitis, it makes sense to consult the radiologist prior to ordering the exam.

Diverticulitis

Similar to gallstone disease and renal colic, diverticulitis likely follows obstruction. In diverticulitis, the obstruction occurs at the lumen where the diverticulae communicate with the bowel. The diverticulum then swells and leaks (microperforation), causing pericolic inflammation and associated fat stranding, or frankly ruptures (macroperforation), leading to greater degrees of inflammation or free air in the peritoneal cavity[9] (Figure 3). Bowel wall thickening may be secondary to inflammation, or it may be secondary to circle muscle hypertrophy which accompanies chronic diverticulosis. Since most diverticulae are in the sigmoid colon, most patients have low pelvic pain, but diverticulae, and diverticulitis, may occur anywhere along the course of the large bowel. While patients with diverticulitis may have changes in bowel habits, they often do not, and simply present with lower abdomen or pelvic pain. While diverticulitis preferentially affects the elderly, more people are being diagnosed with diverticulitis at a younger age. This is probably secondary to dietary

changes leading to diverticulosis earlier in life, and the widespread use of CT to evaluate abdominal pain which would have, in earlier eras, been treated empirically without a specific diagnosis.

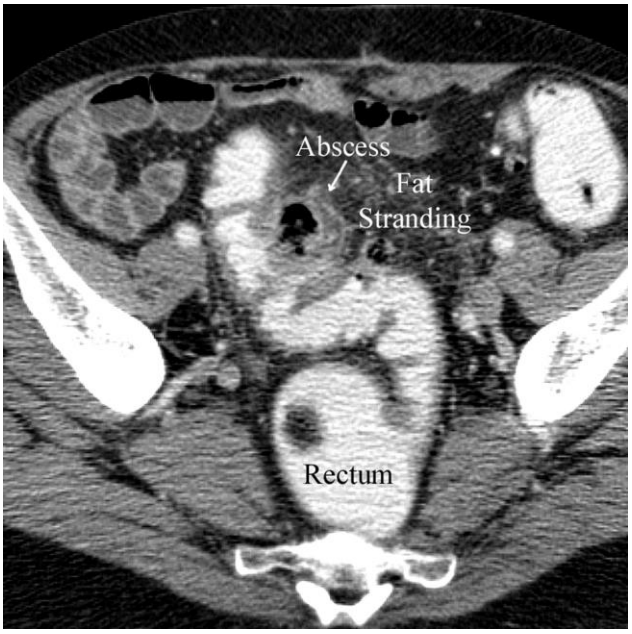

Figure 3. Diverticulitis in a 62 year old man with lower abdomen and pelvic pain. Axial CT performed with rectal contrast shows sigmoid colon wall thickening, pericolic fat stranding, and a pericolic abscess.

Patients with suspected diverticulitis typically undergo CT performed with oral and IV contrast. Oral contrast often does a poor job of filling the colon regardless of the time of the scan, however, and some radiology departments advocate routine use of rectal contrast in cases of suspected diverticulitis, a practice which is unpopular with both the patients and the technologists.

Inflammatory change in pericolic fat adjacent to a diverticulum in the setting of abdominal pain establishes the diagnosis of diverticulitis, and the patient will need appropriate treatment with antibiotics. These patients must subsequently undergo colonoscopy (typically done a few weeks after the acute bout of inflammation has regressed), not to document the diverticulae or any residual inflammation, but to exclude causative or coincident neoplasm in the colon. Large perforations of the sort associated with extraluminal air (or infection with gas producing organisms) and abscesses both merit emergent surgical consultation. The surgeon may opt for emergent surgery or elect to admit the patient for IV antibiotics and bowel rest prior to intervention. Percutaneous abscess drainage may be performed prior to surgery.

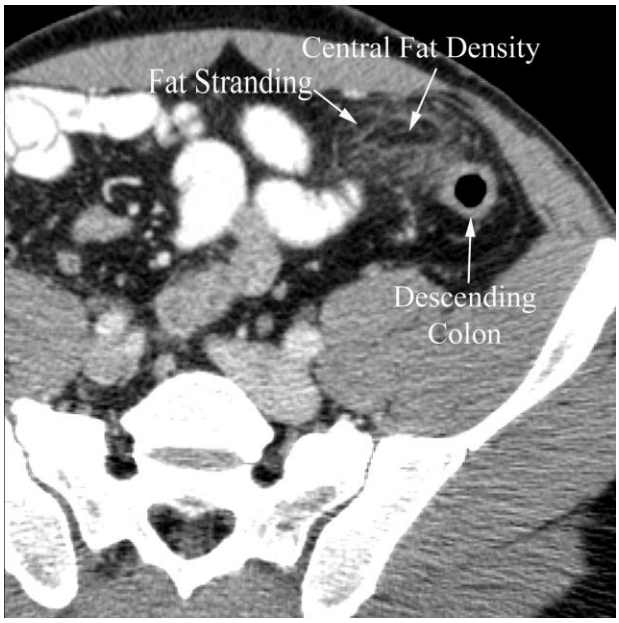

Figure 4. Appendagitis epiploicae in a 35 year old man with right lower quadrant pain. Axial CT performed with oral and intravenous contrast demonstrates soft tissue stranding in the pericolic fat along the descending colon with a central area of fatty density.

Appendagitis Epiploicae

Appendagitis epiploicae is another painful inflammatory condition of the bowel which may mimic diverticulitis[10]. This self-limited entity was likely under-diagnosed in the past because it usually resolves without the necessity of surgery (and, therefore, a lack of a correlating pathologic diagnosis). With the widespread use of CT in the evaluation of patients with acute abdominal pain and features of inflammation, appendagitis epiploicae is being diagnosed more frequently. Appendagitis epiploicae likely follows torsion of the epiploic appendages (fatty outpouchings along the bowel margin), which results in infarction of the fat and a highly characteristic CT picture (Figure 4). These patients nearly always respond to conservative measures (pain relievers), with prompt resolution of symptoms.

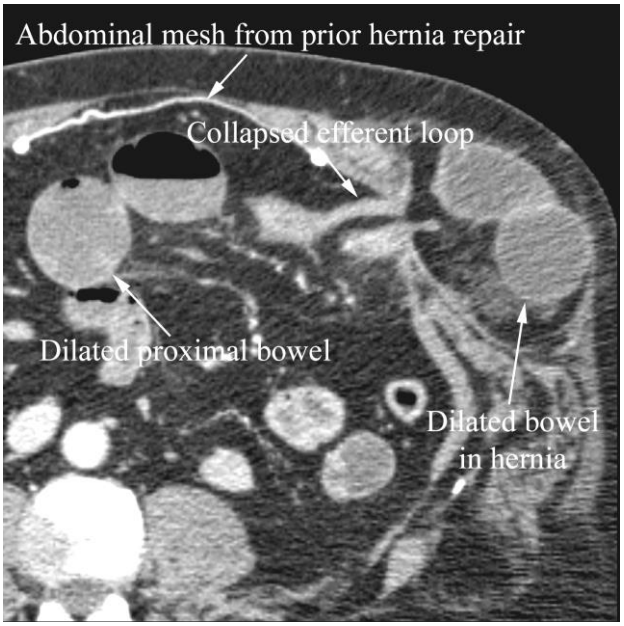

Figure 5. Small bowel obstruction from hernia in a 52 year old man with abdominal distension and a history of prior surgery. Axial CT shows dilated bowel loops proximal to and within the hernia, collapsed distal loops, and a transition point in the loop leading out of the hernia. Note the mesh in the anterior abdominal wall from prior hernia repair.

Abdominal pain and suspected obstruction

Patients who present with abdominal pain with distension may or may not have nausea and vomiting[5]. Those who have nausea and vomiting, of course, must be suspected of having bowel obstruction. The physical exam should focus on the presence and quality of bowel sounds and hernias. There is ongoing debate regarding the utility of plain films in the evaluation of such patients. Certainly, the plain film may show features of bowel obstruction such as distended small bowel loops, air fluid levels, and a paucity of colonic contents. However, the study may be falsely negative, and typically does not provide the location or cause of the obstruction. CT can often provide both (Figure 5). In cases of severe obstruction with vomiting, the study should be performed with only IV contrast (the patient will simply vomit the oral contrast anyway). The patient may tolerate oral contrast in cases of low grade or intermittent obstruction. Key findings in bowel obstruction include distended small bowel (normal small bowel should measure no more than about 25 mm) and the presence and location of a *transition point*. The transition point, or location where the small bowel abruptly changes caliber from dilated to collapsed, is the key to the diagnosis[11]. There may be a hernia, volvulus, or a mass at the transition point, or there may be inflammatory changes suggesting a stricture from inflammatory bowel disease. If there is a transition point and no specific cause is identified, the bowel obstruction is likely secondary to adhesions. If there is no transition point and the entire bowel is distended, ileus or pseudo-obstruction is more likely.

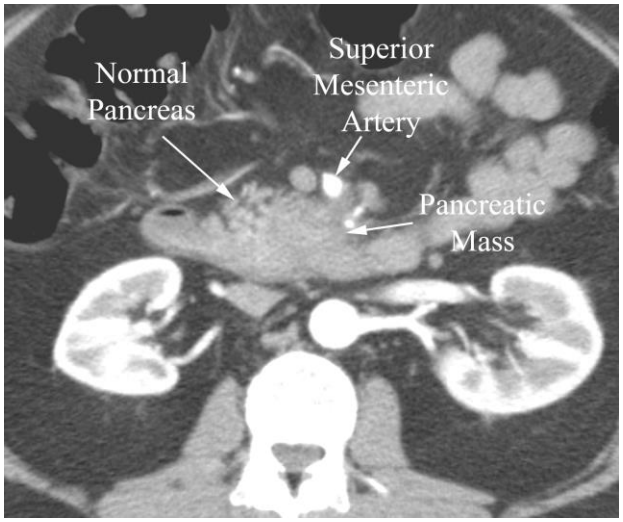

Figure 6. Pancreatic cancer in a 43 year old man with weight loss and abdominal pain. Axial CT performed during the arterial phase demonstrates tumor in the pancreas effacing the normal lobulated (with fat) appearance. The tumor surrounded the superior mesenteric artery more superiorly (not shown).

Abdominal pain and weight loss

Another scenario in which CT should be considered is when patients have abdominal pain and such constitutional symptoms as weight loss (Figure 6). Possible causes include intestinal ischemia (particularly for those with such risk factors as known vascular disease or diabetes) (see page 177) and occult malignancy (particularly of the pancreas).

Abdominal pain and diarrhea

Multiple diseases may cause colitis and result in both abdominal pain and diarrhea, chief among them pseudomembranous colitis[12] (Figure 7) and ischemic colitis[13]. Both may be associated with CT

findings of diffuse colon wall thickening and pericolic fat stranding. Patients with either pseudomembranous colitis or ischemic colitis will likely require hospitalization. CT is the imaging study of choice, but as with other diagnoses associated with abdominal pain and diarrhea, the patient may undergo endoscopy first with no CT scan performed.

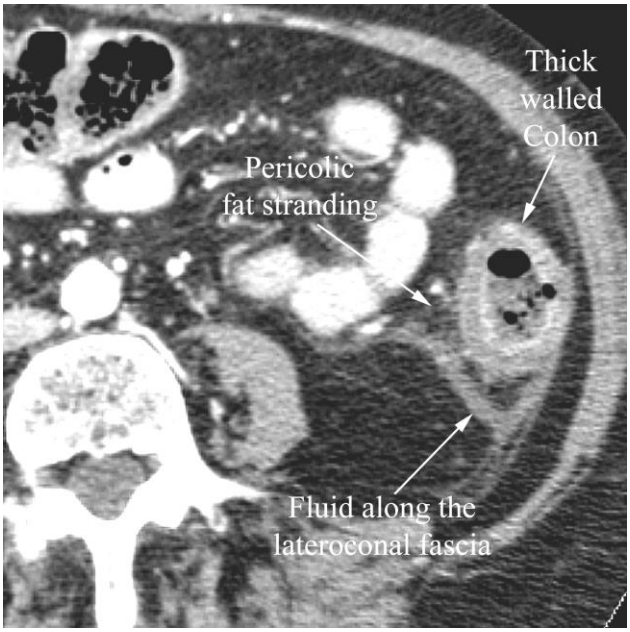

Figure 7. Pseudomembranous colitis in an 88 year old woman with diarrhea following antibiotic treatment. CT demonstrates a thick walled colon and pericolic fat stranding as well as fluid along the lateroconal fascia.

This should not leave you with the impression that all abdominal pain needs to be worked up with CT examination. As noted above, in patients with suspected biliary disease and in those with straightforward acute pancreatitis where no complication such as abscess or pancreatic necrosis is suspected, the first step of the work-up is with ultrasound rather than CT. Also as noted above, abdominal pain of mucosal origin is now largely evaluated with endoscopy, which has replaced barium studies. Cross-sectional imaging is performed as a complementary procedure in patients undergoing endoscopy. For example, in patients with colon cancer diagnosed with endoscopy, CT is typically performed to access not the colon mucosa or the primary tumor (although the primary tumor is often visible), but to evaluate lymphadenopathy and possible liver lesions. In addition, many patients have chronic abdominal pain which has been worked up extensively in the past, and reimaging such chronic pain patients is infrequently rewarding (see page 101).

VIDEOFLUOROSCOPY IS OFTEN THE BEST FIRST STEP IN EVALUATION OF OROPHARYNGEAL DYSPHAGIA

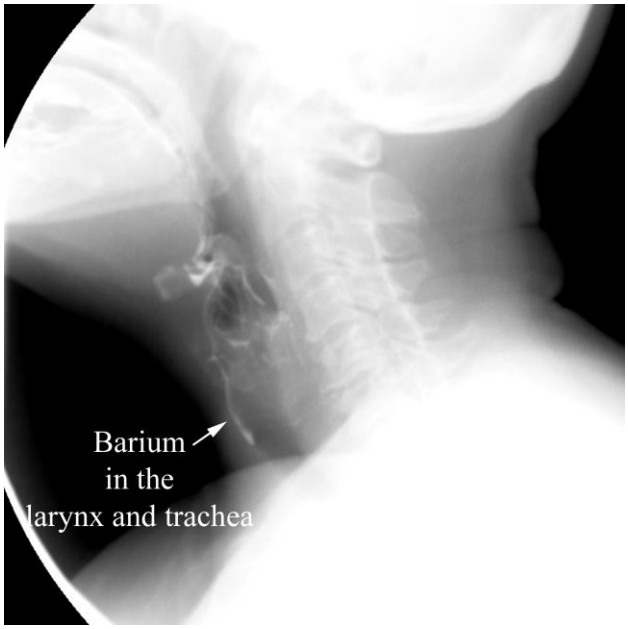

Figure 8. Aspiration in a 75 year old woman with oropharyngeal dysphagia. Lateral image obtained during videofluoroscopy shows aspirated barium in the larynx and trachea.

Videofluoroscopy swallowing studies are designed to test the function of the oropharynx, hypopharynx, epiglottis, and proximal esophagus in the usually smooth, even flow of material from above to below, avoiding the larynx and trachea[14]. While videofluoroscopy allows analysis of the swallowing mechanism in great detail, the main reproducible findings are *penetration*, or barium flowing abnormally past the epiglottis and into the supraglottic larynx, and *aspiration*, or barium flowing past the true vocal cords into the trachea [15](Figure 8). These are both associated with a significantly increased risk for pneumonia, and are responsible for the choking episodes characteristic of oropharyngeal dysphagia.

A lesion that may be diagnosed with either videofluoroscopy or with a barium esophagram is a Zenker's diverticulum (Figure 9). These more frequently lead to oropharyngeal dysphagia than esophageal dysphagia, since they are proximally located in the esophagus and give rise to such symptoms as difficulty shortly after initiating a swallow, gurgling, or a full sensation in the throat. Retention of food within these diverticulae can compress the esophagus, which leads to difficulty after the first or second swallow. The patient then has to wait for the diverticulum to empty, which may result in regurgitation of food, before being able to resume eating.

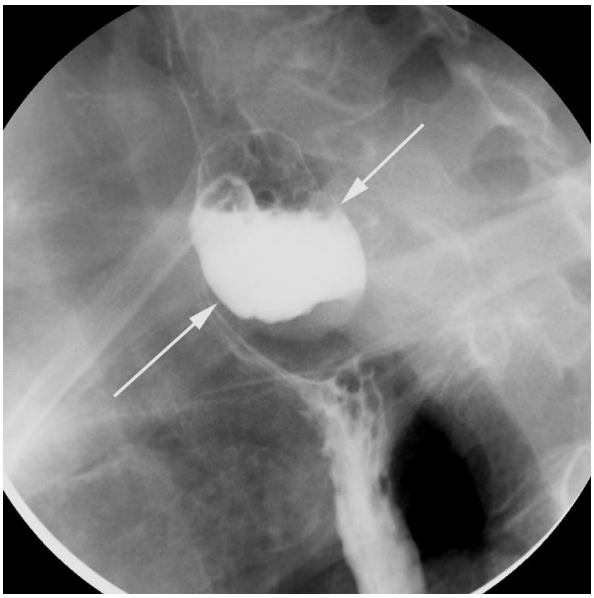

Figure 9. Zenker's diverticulum in a 75 year old with dysphagia. There is a large Zenker's diverticulum in the typical location along the posterior, proximal esophagus.

SUMMARY

While there are many different imaging studies which may be performed on patients with gastrointestinal complaints, first step studies include ultrasound for evaluation of biliary colic and pancreatitis, CT for "abdominal pain plus", and videofluoroscopy for oropharyngeal dysphagia.

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"Second Step" Imaging Of Gastrointestinal Symptoms

Donald L. Renfrew, MD

Chapter 7 covered the "first step" in the imaging evaluation of gastrointestinal symptoms, and noted that while there are many imaging options available to evaluate gastrointestinal symptoms, the "first step" imaging options are relatively few. This chapter picks up where the prior chapter leaves off, and addresses situations when the first step has been taken, but more imaging is warranted. The major points of this chapter are:

1. Women with low abdomen/pelvic pain may need both computed tomography (CT) and ultrasound (US) for evaluation.
2. More than US may be necessary for evaluation of RUQ pain and suspected biliary disease, particularly with abnormal enzyme studies.
3. Small bowel imaging may require additional evaluation after a standard CT study.
4. CT may demonstrate incidental lesions or be performed in asymptomatic patients.

WOMEN WITH LOW ABDOMEN/PELVIC PAIN MAY NEED BOTH CT AND US FOR DIAGNOSIS

Because of the proximity of the bowel, bladder, uterus, ovaries, and fallopian tubes, many disease processes may result in low abdomen/pelvic pain in women[1]. Often, women will have ambiguous or even frankly misleading signs and symptoms because of the proximity of the disease processes. Diagnosis may be elusive without performing *both* CT and US (and, occasionally, even *when* performing both).

CT for bowel abnormalities

If the primary suspicion is for a bowel related abnormality, CT is the study of choice, particularly if there are features of inflammation or obstruction (see page 97). CT is performed to evaluate for diverticulitis, appendicitis, appendagitis epiploicae, and colitis. CT abnormalities in the pelvis may be further characterized with ultrasound, which can often provide a more specific diagnosis (Figure 1).

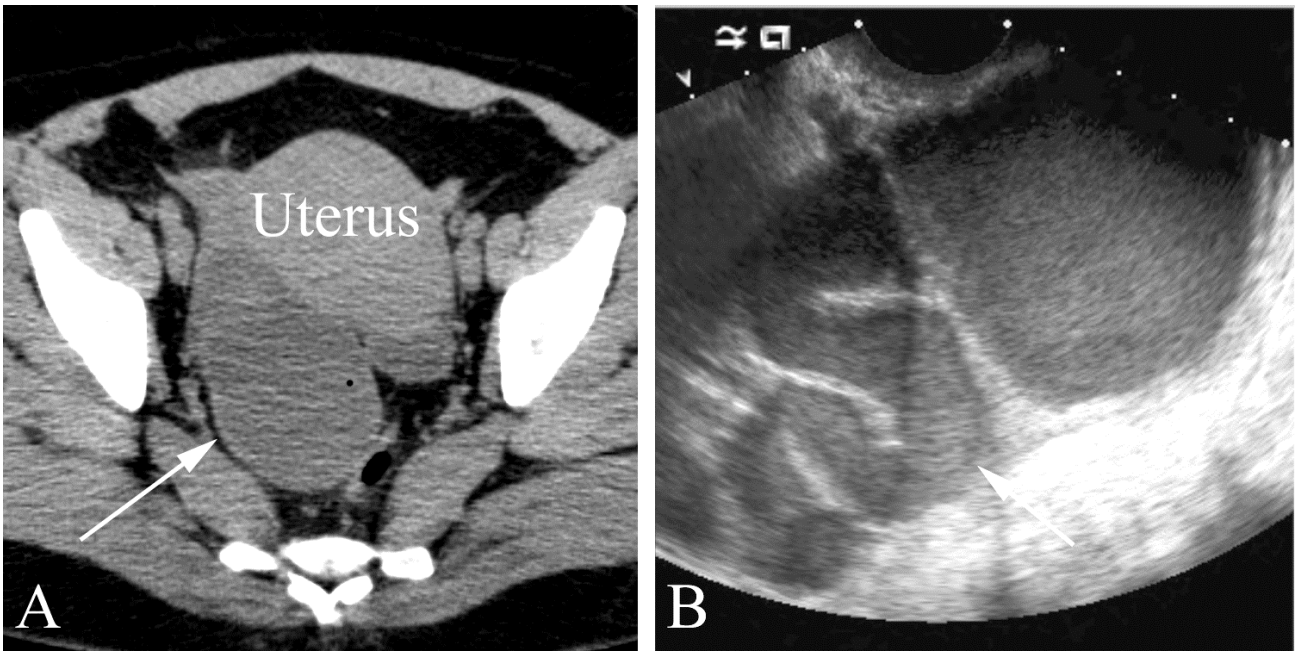

Figure 1. Pyosalpinx in a 42 year old woman with chronic right adnexal and lower abdominal pain. CT supplemented with US to arrive at a diagnosis. A. Axial CT shows an apparent large right adnexal cyst (arrow). B. Pelvic US demonstrates a "tadpole" appearance, characteristic of a hydrosalpinx or pyosalpinx. The patient had surgical resection of the right ovary and fallopian tube demonstrating pyosalpinx superimposed upon a chronic hydrosalpinx.

US for uterus/ovarian abnormalities

If the primary suspicion is for an abnormality of the uterus or ovary, ultrasound is the first study of choice (see pages 16-21). Ultrasound is performed to evaluate for ovarian torsion, adnexal masses, uterine fibroids, ectopic pregnancy, and other disorders that may present with either low abdominal and pelvic pain or a mass. Sometimes, CT (Figure 2; see also Figure 7, page 20) or MR (see Figures 6 and 8, pages 19-20) is obtained for further evaluation of a mass discovered on ultrasound. If the ultrasound is negative, a CT may provide an explanation for the symptoms.

CT, US, and even MR for "lining abnormalities"

In *cystitis* (inflammation of the bladder wall), imaging studies will typically be normal unless the cause of the symptoms is actually outside the bladder (Figure 3). In *endometritis* (inflammation of the uterine lining), the ultrasound may be normal or demonstrate (in severe cases) a thickened endometrial stripe with air bubbles if there is a gas-producing infection. In *colitis* (inflammation of the colon wall), the bowel wall may show thickening and associated pericolic fat stranding (Figure 3; see also Figure 7, page 98). *Endometriosis* (ectopic endometrial tissue outside of the uterus) may cause pain in the abdomen and be challenging to diagnose with either US or CT, which may simply show a nondescript soft tissue mass (or masses) of the peritoneal cavity or adnexa (see Figure 5, page 18). MR may assist in making this diagnosis because of the ability to detect blood breakdown products which are often seen in endometriomas.

MORE THAN ULTRASOUND MAY BE NECESSARY FOR EVALUATION OF RUQ PAIN

While ultrasound is the first step in evaluation of suspected biliary colic and pancreatitis (see pages 93-94), there are occasions when the evaluation will not stop with the ultrasound. Further evaluation may be directed according to the pattern of liver enzyme abnormalities, patient symptoms, or the abnormalities seen on the ultrasound study.

Liver enzyme abnormalities

Many times, RUQ ultrasound will be requested for "abnormal LFTs". Occasionally, abdomen CT will

be requested for the same reason. Purists will maintain that the terminology is often used incorrectly – that "LFT" is the wrong term to apply to, for example, the aminotransferases, which are more properly called "liver *damage* enzymes". These purists reserve the "liver function test" label to measures of synthetic function such as serum albumin and prothrombin time[2].

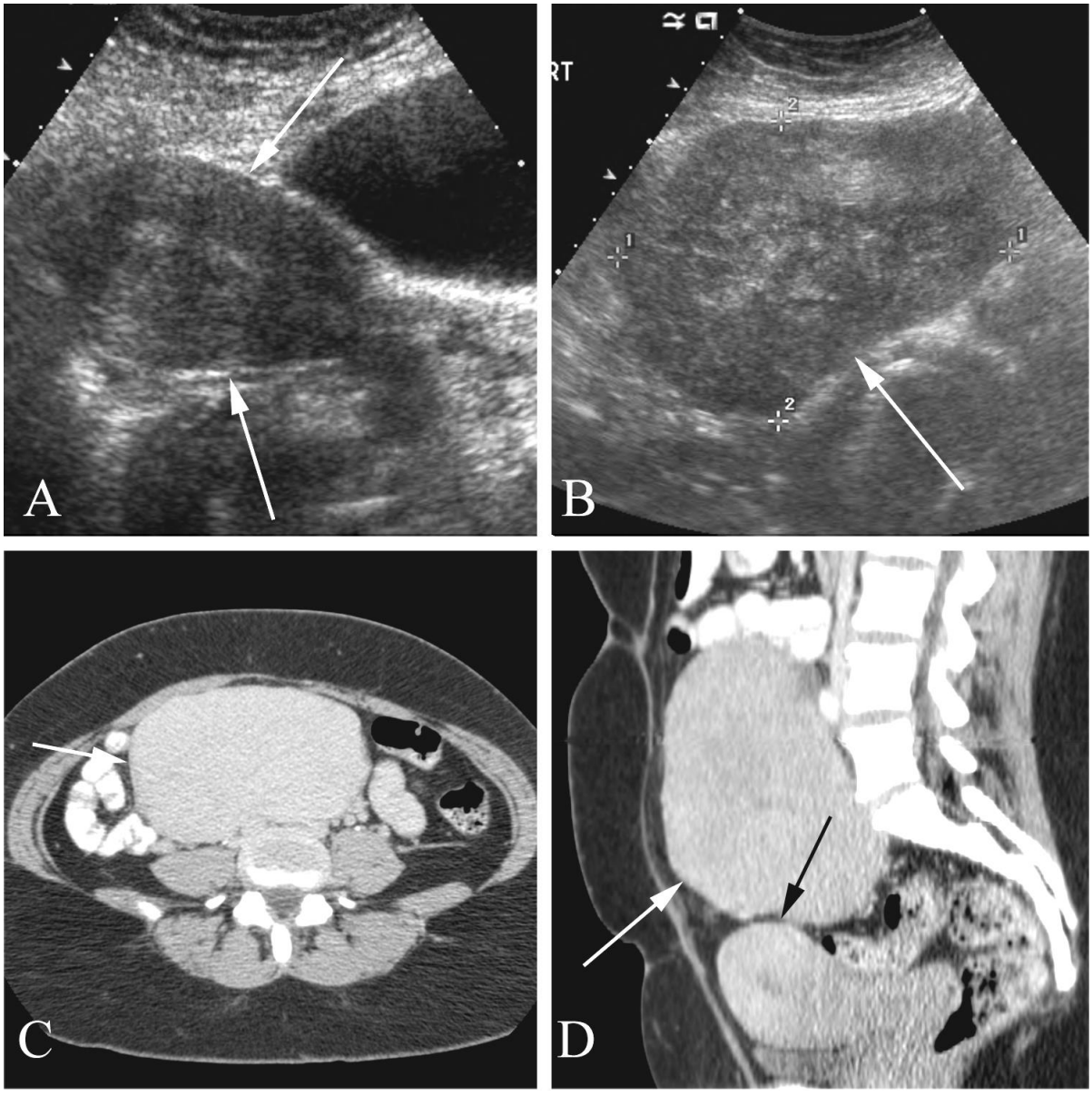

Figure 2. Extrauterine leiomyoma in a 40 year old woman with a lower abdominal and pelvic mass. US supplemented with CT to arrive at a diagnosis. A. Transabdominal US demonstrates a normal appearing uterus (arrows). B. Transabdominal US lateral and somewhat superior to the uterus demonstrates an isoechoic-to-hyperechoic mass (arrow) which had the texture of a fibroid. It was difficult to separate this lesion from the uterus. C. Axial CT study confirms a large, homogeneous mass of the lower abdomen (arrow). D. Sagittal reformatted image demonstrates the mass (white arrow) with a clear demarcation plane separating the mass from the patient's uterus (black arrow). CT-directed biopsy (not shown) was consistent with a leiomyoma. Subsequent surgical removal showed an extra uterine, intra-abdominal leiomyoma.

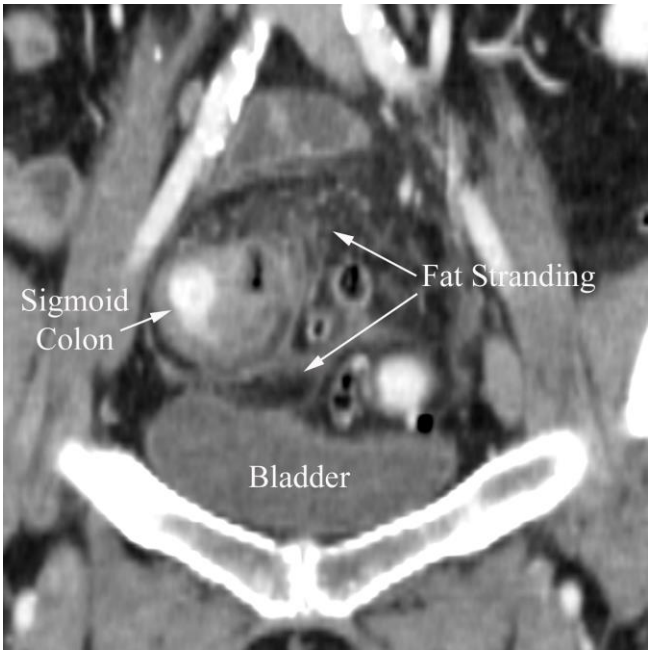

Figure 3. Diverticulitis in a 68 year old woman with urinary frequency and bacteriuria. Coronal reconstruction CT demonstrates the proximity of the urinary bladder to the inflamed loop of sigmoid colon, providing an explanation of the patient's symptoms.

From a radiologist's perspective, this distinction (between *function* and *damage*) is probably not nearly as important as the general *pattern* of enzyme abnormality, which provides at least a clue as to the diagnosis associated with the "abnormal LFTs". An important point to remember here is that the radiologist may not have access to the exact lab values when interpreting the study, and therefore it makes sense to provide not only "Abnl LFTs" on the requisition, but also the pattern of this abnormality so the radiologist is on alert for findings associated with the appropriate disease. So, what are the patterns?

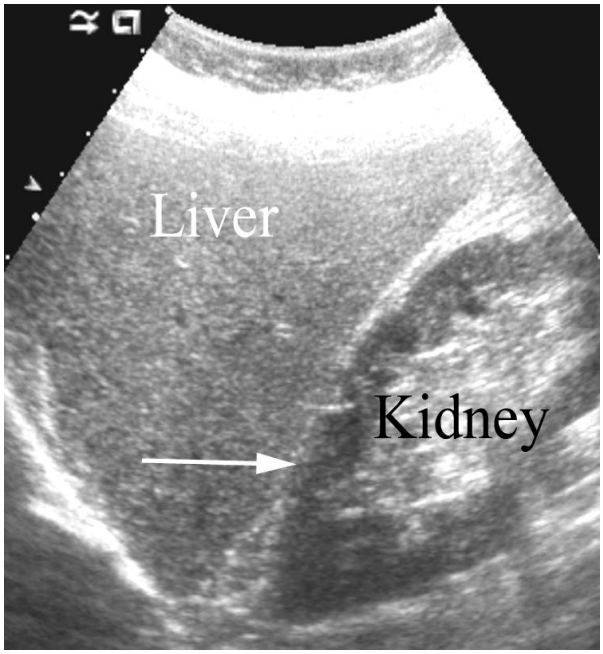

Figure 4. Fatty liver in a 55 year old woman with elevated transaminases and right upper quadrant pain, compatible with steatohepatitis. US shows a diffusely echogenic liver, with the liver parenchyma demonstrating increased echogenicity compared with the renal cortex (arrow).

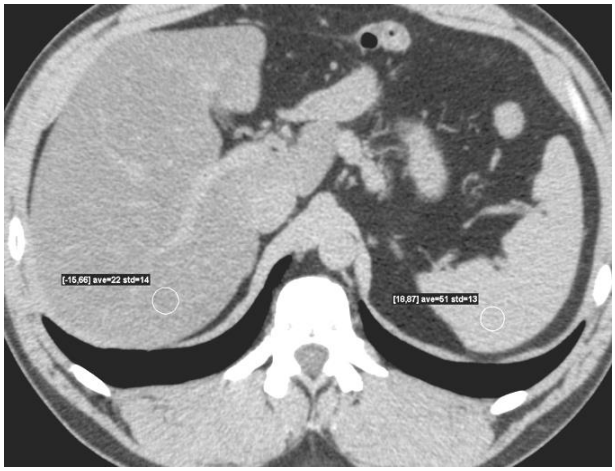

Figure 5. Fatty liver in a 40 year old man with dyslipidemia and elevated AST and ALT. CT shows diffusely decreased density compared to the spleen, with Houndsfield Units [HU] of 22 in the liver target area versus HU of 51 in the spleen target area.

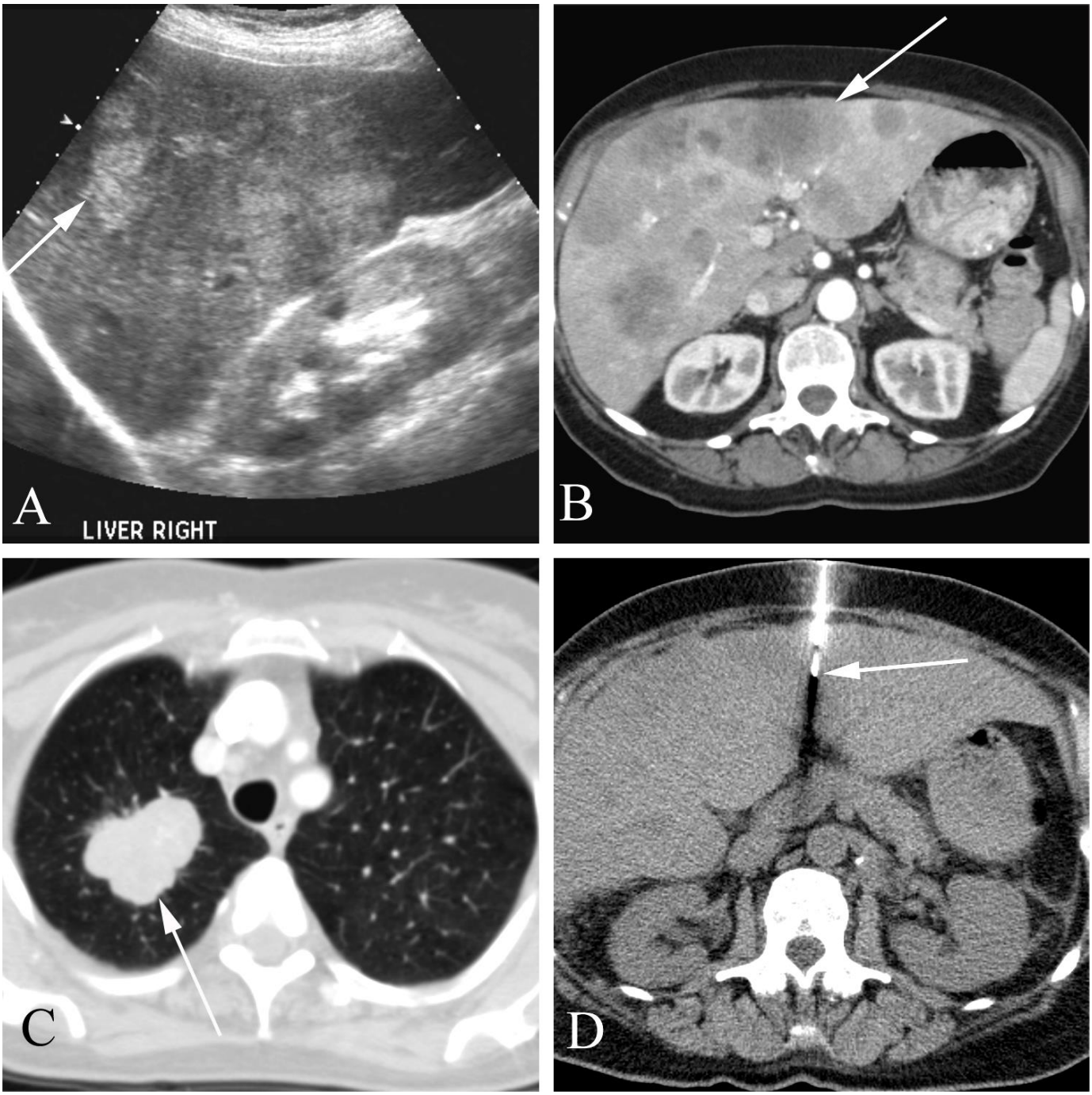

Figure 6. Metastatic liver disease in a 66 year old woman with abnormal liver function tests (elevation of AST, ALT, alkaline phosphatase, and GGT). A. The hepatobiliary ultrasound shows multiple, hyperechoic lesions scattered through the liver (arrow). B. The CT scan of the abdomen confirmed multiple liver lesions (arrow). C. The chest CT, done at the same time as the abdominal CT for tumor evaluation, demonstrates a tumor of the right upper lobe (arrow). D. CT-directed biopsy shows the needle tip in the left lobe of the liver, at the location of the lesion seen on the contrast-enhanced exam (arrow). As no contrast was given, the liver lesions are much less conspicuous on this study. Biopsy results indicated a metastatic lung tumor.

Hepatocellular damage pattern

Hepatocellular abnormalities typically manifest as disproportionate elevations of aminotransferases compared to alkaline phosphatase[2]. Multiple disease processes may cause hepatocellular injury, but many of these demonstrate either no imaging abnormality or a nonspecific imaging abnormality, at least until late in their course. Statistically, abnormal aminotransferases, especially if the elevation is mild, have either no cause of liver disease found, or have hepatitis, alcoholic liver disease, or nonalcoholic steatohepatitis (NASH). While an ultrasound study (Figure 4) or a CT study (Figure 5) may show a fatty liver, the imaging study cannot differentiate between asymptomatic steatosis and NASH. Note also that ultrasound, while less expensive, is not as accurate in evaluation of fatty liver as CT or MR[3]. The ratio of aspartate aminotransferase (AST) to alanine aminotransferase (ALT) may help in discriminating between NASH and alcoholic hepatitis: the AST/ALT ratio is typically *less* than one in NASH and *greater* than one in alcoholic hepatitis. In general, with the hepatocellular pattern of enzyme abnormalities, the expected imaging finding is a normal study or a fatty liver, and with either a normal or fatty liver, further evaluation for hepatitis (with serum screening), alcohol use, other drug use (including prescription drugs), and follow-up is probably necessary. In at least some cases, a liver biopsy will ultimately be necessary to establish a diagnosis and may be considered for those with persistent two-fold elevation of aminotransferases[2]. Of course, there are nonhepatic causes of elevated aminotransferases, including muscle disorders (e.g., injury and pyomyositis), thyroid disorders, celiac disease, adrenal insufficiency, and anorexia nervosa[2].

Cholestatic pattern

When alkaline phosphatase elevation exceeds that of the aminotransferases, the patient is said to have a cholestatic pattern of enzyme abnormalities, and a different set of diseases needs to be considered, namely partial bile duct obstruction, primary biliary cirrhosis (PBC), primary sclerosing cholangitis, drugs (particularly anabolic steroids), and unsuspected metastatic cancer[2]. In patients with this pattern of enzyme abnormality, as with all patients with suspected hepatobiliary issues, the first imaging study to order is ultrasound, which will evaluate for diffuse metastatic disease or biliary distension. If the ultrasound shows diffuse liver abnormality then liver biopsy will typically be performed (Figure 6). If the ultrasound demonstrates biliary distention, the patient is said to have extrahepatic cholestasis, and endoscopic retrograde cholangiopancreatography (ERCP) will usually follow to diagnose the cause (typically choledocholithiasis) and provide treatment (e.g., stone removal or stent placement). In some patients, magnetic resonance imaging cholangiopancreatography (MRCP) may represent an alternative to ERCP, for example when the likelihood of intervention is small[2]. Note that this is an evolving area and local availability and expertise in ERCP and MRCP vary widely, so discussion with local radiologists, surgeons, and gastroenterologists in these patients certainly makes sense.

Nuclear medicine study for physiology and blockage

In patients with suspected biliary colic, as noted in Chapter 7, the initial study is hepatobiliary ultrasound. In many cases, this study will be definitive. It may be definitively *positive*, showing gallstones and features of inflammation such as pericholecystic fluid and a positive sonographic Murphy's sign allowing the surgeon to proceed with cholecystectomy. Or, it may be definitively *negative*, where the suspicion of acute cholecystitis was relatively low and the negative ultrasound supports the clinical diagnosis of, for example, gastritis. In patients where the clinical suspicion for hepatobiliary disease is high and the ultrasound study is either normal or ambiguous, further imaging may be helpful. As noted above, the pattern of enzyme abnormality – if the patient has both enzyme abnormalities and biliary pain – may help direct the search: patients with a cholestatic pattern (elevated alkaline phosphatase relative to aminotransferases) more likely need ERCP or MRCP, whereas those with a hepatocellular pattern more likely benefit from biopsy, if a definite diagnosis is clinically required. In patients with biliary pain and normal or ambiguous enzyme abnormalities, a

nuclear medicine hepatobiliary scan may provide additional important information.

The nuclear medicine study for gallbladder disease has two phases, but only the first phase may be necessary in some patients. In the first phase of the study, the patient is injected with an intravenous dose of radioactively labeled iminodiacetic acid, which the liver conjugates and secretes in the bile. Sequential imaging should demonstrate the liver, biliary tree, gallbladder, and small bowel (Figure 7). In patients with acute biliary pain and no gallstones seen on right upper quadrant ultrasound, non-visualization of the gallbladder by one hour indicates acute cholecystitis. If the patient is direly ill, the test will likely be terminated at this point and the patient taken to surgery[4].

In the absence of a direly ill patient, the test will continue. If the gallbladder is not seen by one hour and sequential follow-up never reveals the gallbladder, then the diagnosis remains acute cholecystitis until proven otherwise (Figure 8). If the gallbladder visualizes between one and four hours, then the diagnosis is *chronic* cholecystitis. If the gallbladder visualizes within an hour, then the second phase of the nuclear medicine study is performed. Note that in almost all cases, these patients are *not* the acutely ill patients suspected of having acalculous cholecystitis, but, rather, are patients with chronic pain suspected to have sphincter of Oddi dysfunction. The clinical features of these patients include epigastric or right upper quadrant pain, elevated aminotransferases, elevated bilirubin, or elevated pancreatic enzymes or pancreatitis-type pain[5]. Sphincter of Oddi dysfunction has a number of synonyms, including papillary stenosis, sclerosing papillitis, biliary spasm, and biliary dyskinesia. As is frequently the case when so many terms apply to one thing, there is confusion about what constitutes the condition, what causes the abnormality, and how to diagnose and treat the disease. With respect to the nuclear medicine study, the second phase of the study is designed to provide some information regarding hepatobiliary function. The patient is injected with cholecystokinin (a naturally occurring endogenous peptide), and the patient is imaged for up to approximately one hour (exact protocols differ). A normal response consists of an ejection fraction of greater than 40% (Figure 7), whereas a lower ejection fraction is compatible with gallbladder dyskinesia and the patient is a candidate for cholecystectomy[5] (Figure 9).

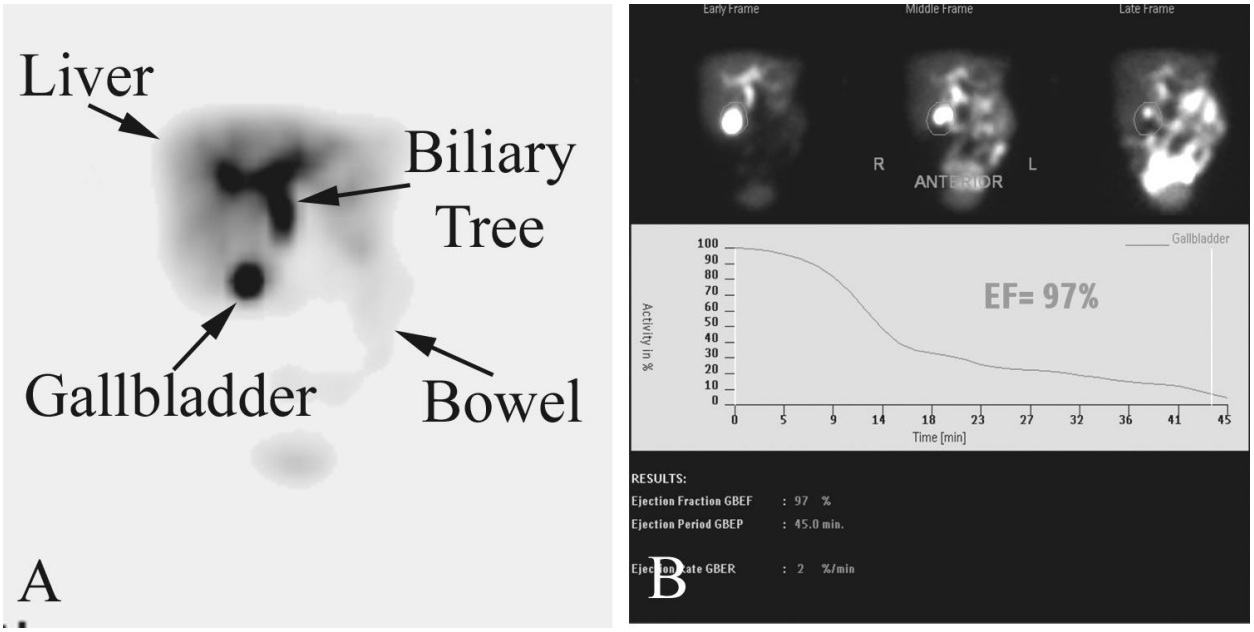

Figure 7. Normal nuclear medicine hepatobiliary scan in a 42 year old woman with chronic right upper quadrant pain. A. Images obtained during the first hour show normal visualization of the liver, biliary tree, bowel, and gallbladder. B. Following injection of cholecystokinin, there was a 97% ejection fracture, far above the normal cut-off value of 40%. Subsequent endoscopy showed gastritis.

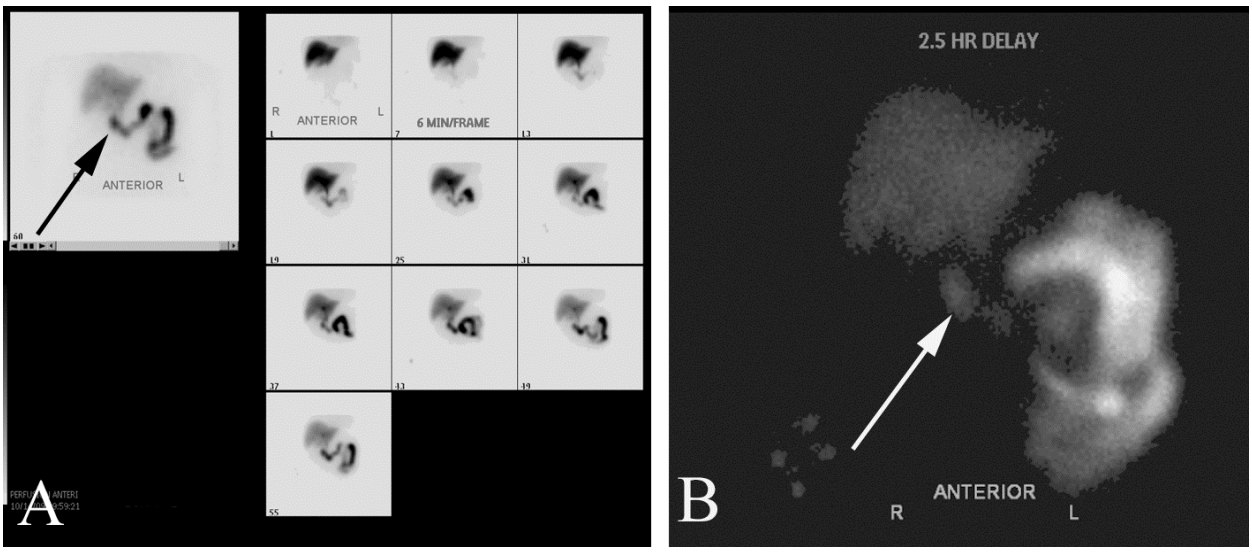

Figure 8. Cholecystitis on a nuclear medicine study in a 79 year old man with equivocal right upper quadrant pain and gallstones seen on an ultrasound study (not shown). A. HIDA scan through one hour shows no visualization of the gallbladder, although there is visualization of the liver, duodenum (arrow), and small bowel. B. Nuclear medicine scan done at 2.5 hours still shows only the duodenum (arrow) without gallbladder activity. The patient was taken to the operating room where acute cholecystitis was confirmed.

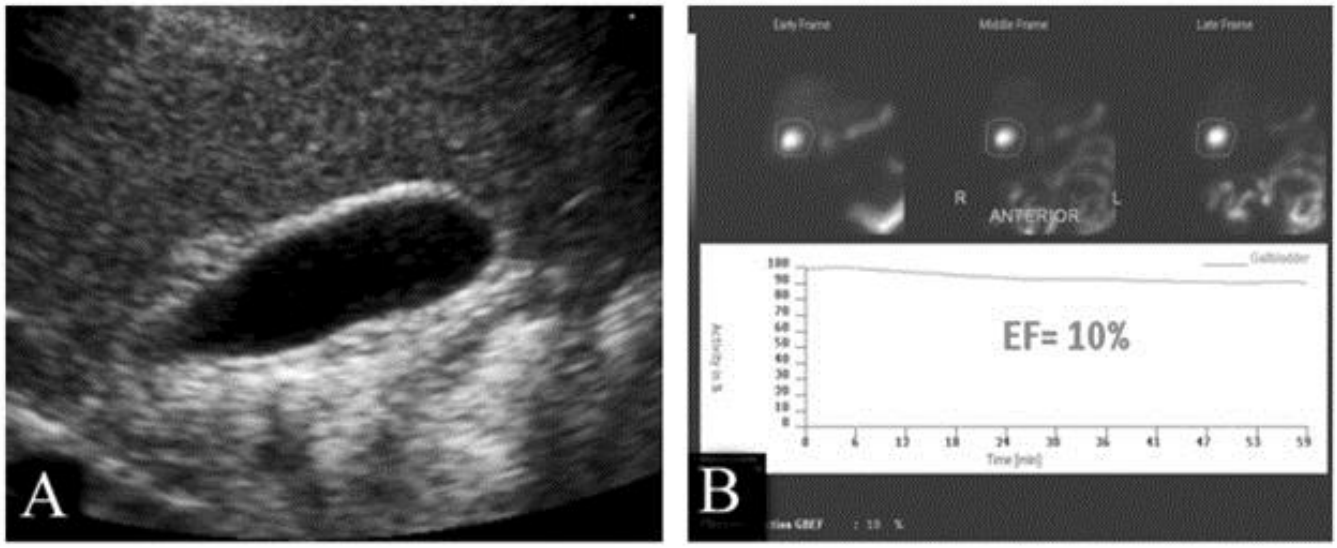

Figure 9. Sphincter of Oddi dysfunction in a 50 year old man with chronic right upper quadrant postprandial pain. A. Ultrasound study of the gallbladder is normal. B. Nuclear medicine hepatobiliary scan shows an abnormally diminished ejection fraction. The patient underwent elective cholecystectomy with relief of his symptoms.

Additional abnormalities seen on US

As noted above and in Chapter 7, the first step in evaluation of patients with right upper quadrant pain is biliary ultrasound. In some cases, this study will show abnormalities that prompt further evaluation with additional imaging studies. For example, extensive liver abnormality may be followed by CT and biopsy (Figure 6), whereas renal abnormalities may be followed by CT and renal excretion studies (Figure 10).

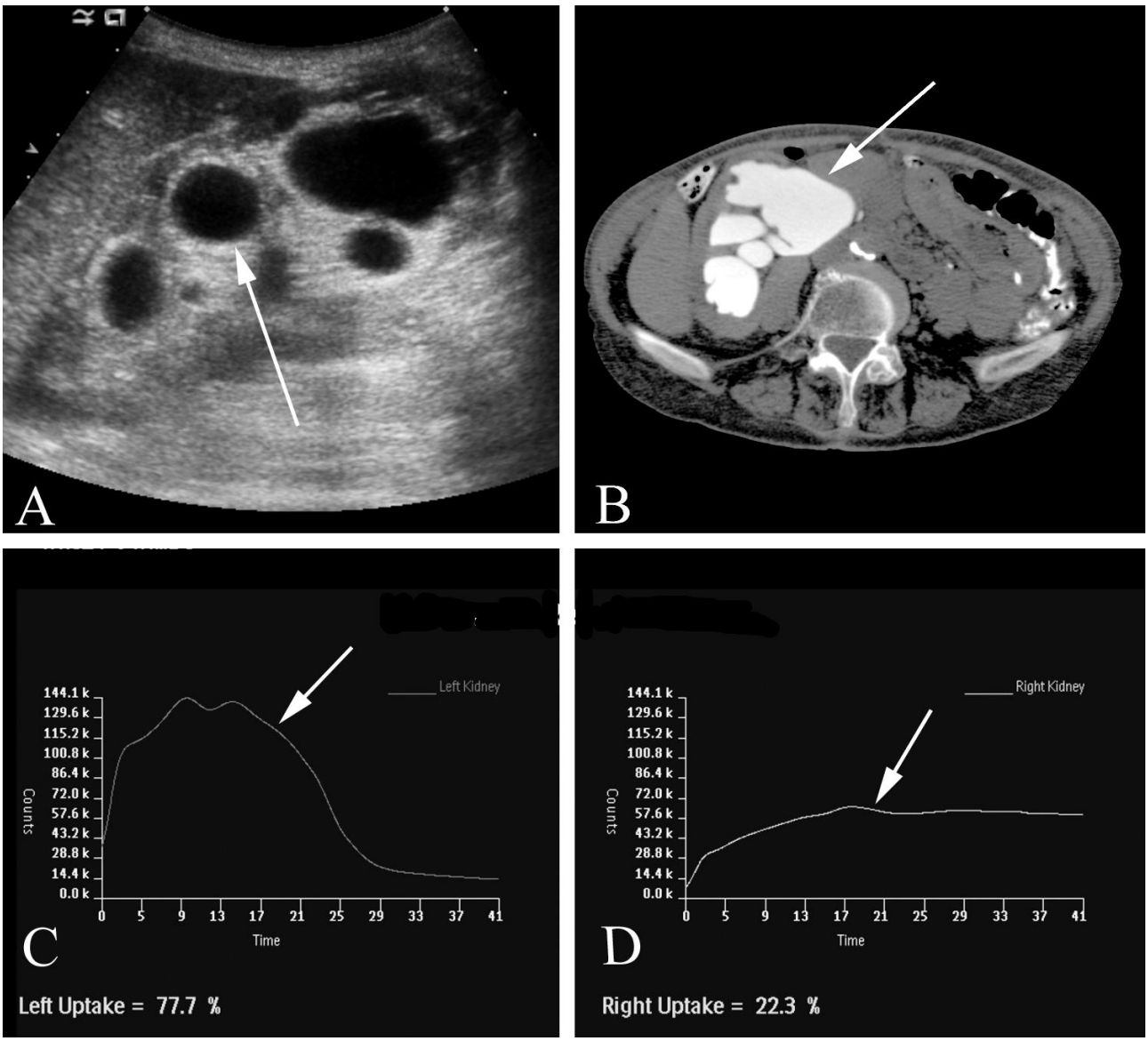

Figure 10. Ureteropelvic junction obstruction in a 57 year old woman with intermittent right upper quadrant pain. A. RUQ ultrasound examination showed a dilated right renal collecting system (arrow). B. Axial CT shows a dilated right collecting system (arrow). Both ureters (not shown) were of normal size. C. Nuclear medicine time activity curve for the left kidney (arrow) shows a normal appearance, with 77% of the measured excretion of injected radionuclide exiting the left side. D. Nuclear medicine time activity curve for the abnormal right kidney (arrow) shows a flattened appearance, with 23% of the measured excretion of injected radionuclide exiting the left side.

SMALL BOWEL IMAGING MAY REQUIRE ADDITIONAL EVALUATION AFTER A STANDARD CT STUDY

Most small bowel abnormalities that can be diagnosed with CT will be diagnosed with routine CT studies of the abdomen and pelvis. As noted in Chapter 7, these are performed with barium or iodine containing contrast material, both denser than most body tissues. The bowel will appear as a rim of grey tissue around the white contrast in the lumen of the bowel. Typically, the bowel wall should be quite thin – as if drawn with a pencil. If the bowel wall looks thicker – as if drawn with a brush – then the wall is probably too thick (Figure 11). Bowel wall thickening is not a specific finding and may be seen in any disease which causes inflammation (e.g. Crohn's disease), hemorrhage (e.g. anticoagulation) or infiltration of tumor cells (e.g. lymphoma) within the bowel wall. Focal tumors may appear as filling defects in the contrast column, rather than diffuse wall thickening.

As noted in Chapter 7, alternatives to positive (dense, white) contrast have been developed for evaluation of the small bowel. These alternatives are usually employed *after* the diagnosis is established in a patient with small bowel disease. The diagnosis is usually established by a combination of features including clinical history, endoscopy, and standard CT of the abdomen and pelvis, so alternatives to the standard CT are generally a "second step" and thus included in this chapter. Note, however, that such studies may be the exam of choice when a patient returns with an exacerbation of symptoms.

The alternatives to positive contrast in evaluation of the small bowel may be performed in one of two ways: an easy way and a hard way. The easy way consists of substituting a lower density oral material for the positive contrast usually given, and having the patient drink approximately 1300 cc of the chosen substance over a one hour period. As noted in Chapter 7, there is a commercially available product for this purpose (VoLumen), but Woo et al[6] found whole milk to work as well. The main advantages to the lower density contrast material is that it may distend the bowel slightly better, and that it allows evaluation of bowel wall enhancement with IV contrast. Recall from Chapter 7 that the portal venous phase study is typically performed about two minutes or so after IV injection of contrast material starts, and at this time inflamed loops of bowel will demonstrate abnormal enhancement along the bowel wall. However, the positive contrast usually given for CT will obscure this enhancement. The advantage of the lower density oral contrast is that the enhancement is much easier to see.

Crohn's disease is one of the most frequent indications for a dedicated small bowel study. In Crohn's disease, CT performed with low density contrast allows evaluation of enhancement of the bowel wall, bowel wall thickening, detection of fat stranding, abscess formation, and fistula formation. This permits the radiologist to accomplish the specific goals of imaging the small bowel in patients with Crohn's disease, which are to evaluate the presence, severity, and extent of disease, to evaluate disease activity, and to evaluate extraintestinal complications[7]. As noted above, administration of oral low density contrast material is the "easy way" of doing the study. The "hard way" is adapted from a fluoroscopic/plain film technique called "enteroclysis" which means "bowel washing." Enteroclysis requires placement of a tube within the small bowel, ideally far enough into the duodenum (or with a specially designed balloon near the tip) to prevent backflow into the stomach (then the esophagus and then the exam room floor). Such reflux will occur because the rate of infusion is fast enough to cause bowel distension. This high rate of infusion (accomplished with a special pump) allows superb small bowel distention and beautiful pictures of the small bowel. However, these exams are not widely available, and consultation with the local radiology department is in order before scheduling such an exam.

Wireless video capsule endoscopy, which involves swallowing a disposable 11 x 26 mm camera which transmits images to receivers outside the patient, may also be used to evaluate the small bowel[8]. Since high-grade bowel stenosis is a contraindication to this study, the patient will probably need to go through a CT scan performed with oral contrast *before* capsule endoscopy.

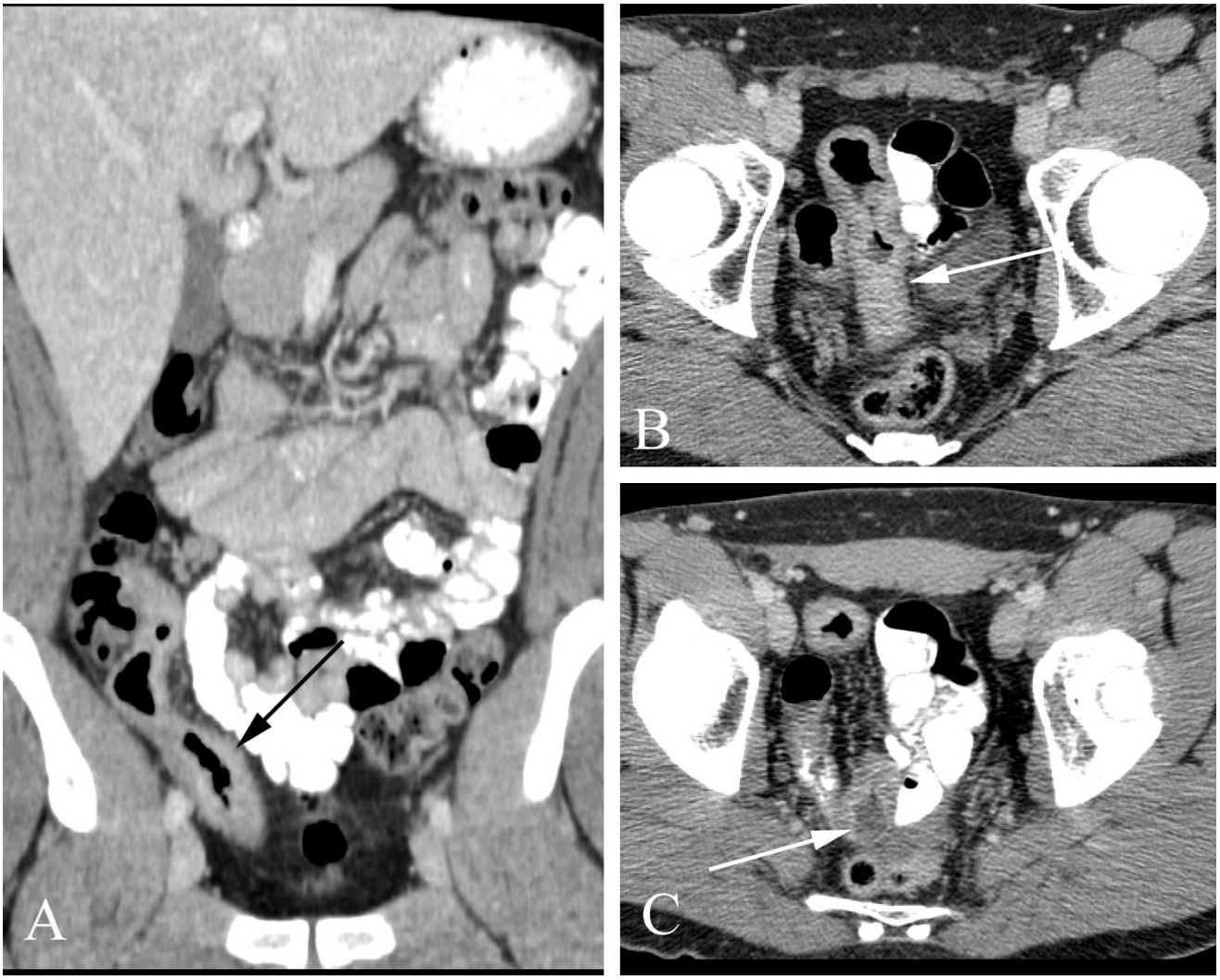

Figure 11. Crohn's disease in a 24 year old man with acute onset of abdominal pain and bloody diarrhea. A. CT coronal reformatted image performed with standard oral and intravenous contrast material demonstrates that the terminal ileum has a thick wall (arrow). B. Axial CT study also demonstrates the thick walled terminal ileum (arrow). Compare to the adjacent bowel loops, which have a normal, thin bowel wall. C. A lower CT cut shows free fluid in the pelvis, an abnormal finding in a man and an indication of intraperitoneal inflammation.

CT MAY BE PERFORMED IN SOME ASYMPTOMATIC PATIENTS

Incidentalomas and "Full Body CT"

The expanded use of imaging – particularly CT scanning – has created a new class of lesion. The term "incidentaloma" refers predominantly to lesions of the adrenal incidentally found when scanning the patient (and this section reviews these lesions first), but this term also applies to those other incidentally discovered lesions found when scanning the patient produces a finding not related to the reason for getting the scan. The most commonly encountered such lesions are in the adrenal, liver, and lung. This chapter covers the first two, whereas Chapter 10 deals with incidentally discovered lung lesions. One of the main things to keep in mind when dealing with incidentally discovered lesions is that the CT scan functions as a screening tool for whatever body part is scanned – indeed, many entrepreneurs have opened up centers

for "CT screening" to evaluate asymptomatic or largely asymptomatic patients for occult but significant disease[9]. Such practice has engendered much interest on the part of the press and lay public, but at this time is not approved by the FDA[10]; the FDA notes ". . . the FDA has never approved CT for screening of any part of the body for any specific disease, let alone for screening the whole body when there are no specific symptoms of disease at all. No manufacturer has submitted data to the FDA to support the safety and efficacy of screening claims for whole-body CT screening". While a full review of such issues is beyond the scope of this chapter, H. Gilbert Welch has written an excellent book on the topic of cancer screening called "*Should I Be Tested for Cancer? Maybe Not and Here's Why"* (University of California Press, 2006). As the book notes, it is difficult to show the efficacy of screening, including screening with imaging. Indeed, there is even controversy about such widely accepted screening studies as mammography and the use of prostate specific antigen. These issues are good to keep in mind when dealing with incidentally found lesions on imaging studies, because even if these lesions are malignant, they may represent "pseudodisease" or "cancers that will never matter to patients". With respect to "Full Body CT", as noted by Jackson et al[9], "Primary providers, who may not have ordered or discussed the study with the patient before it was done, are expected by the patient to provide management recommendations and education without clear supporting evidence".

Adrenal "incidentalomas"

Adrenal incidentalomas are lesions greater than 1 cm in diameter incidentally discovered on an abdomen CT performed for reasons other than investigation of possible adrenal abnormality. There are two sets of issues regarding these lesions: clinical and imaging. From the clinical standpoint, patients with incidentalomas (even small, definitely benign ones – which will generally be the majority of them) need to undergo endocrine evaluation because some of these incidentalomas will actually be functioning, even though the symptoms are mild enough to escape detection[11]. The common tumor types determine the symptoms and laboratory abnormalities, but since the tumor type is unknown prior to full evaluation, it is necessary to screen for all three possibilities: cortisol producing, adrenaline producing (pheochromocytomas), and aldosterone producing. The cortisol producing tumors may produce clinical or subclinical Cushing's disease and be associated with obesity, hypertension, glucose intolerance or diabetes, or hypercholesterolemia. The pheochromocytomas may produce paroxsysmal hypertension or flushing and anxiety. Aldosteronomas may produce hypertension and sodium retention. Recommendations for laboratory testing include a dexamethasone suppression test for Cushing's, a 24 hour catecholamine test (urine collection) for pheochromocytoma, and evaluation of serum sodium and (if there is hypertension) measurement of serum rennin and aldosterone for suspected aldosteronoma.

Regarding imaging, algorithms have been devised to guide work-up[12] (see Table). Radiologists may differ in their recommendations as to which additional test to perform, but in general the patient will need to return to the radiology department for either an unenhanced CT (Figure 12) (likely followed by an additional enhanced study, even if one has already been performed, because of timing differences between the "routine" CT and the optimal adrenal protocol work-up) or an MR. CT is cheaper, more available, and (with "washout" tests based on contrast clearance from the adrenals) very effective in differentiating benign from malignant adrenal incidentalomas[12]. These tests are done to evaluate the size and fat content, which are the imaging features which determine further work-up. In addition to this imaging evaluation, most of these patients will need to return again on a periodic basis to evaluate incidentaloma growth, because an increase in size is an indication for removal.

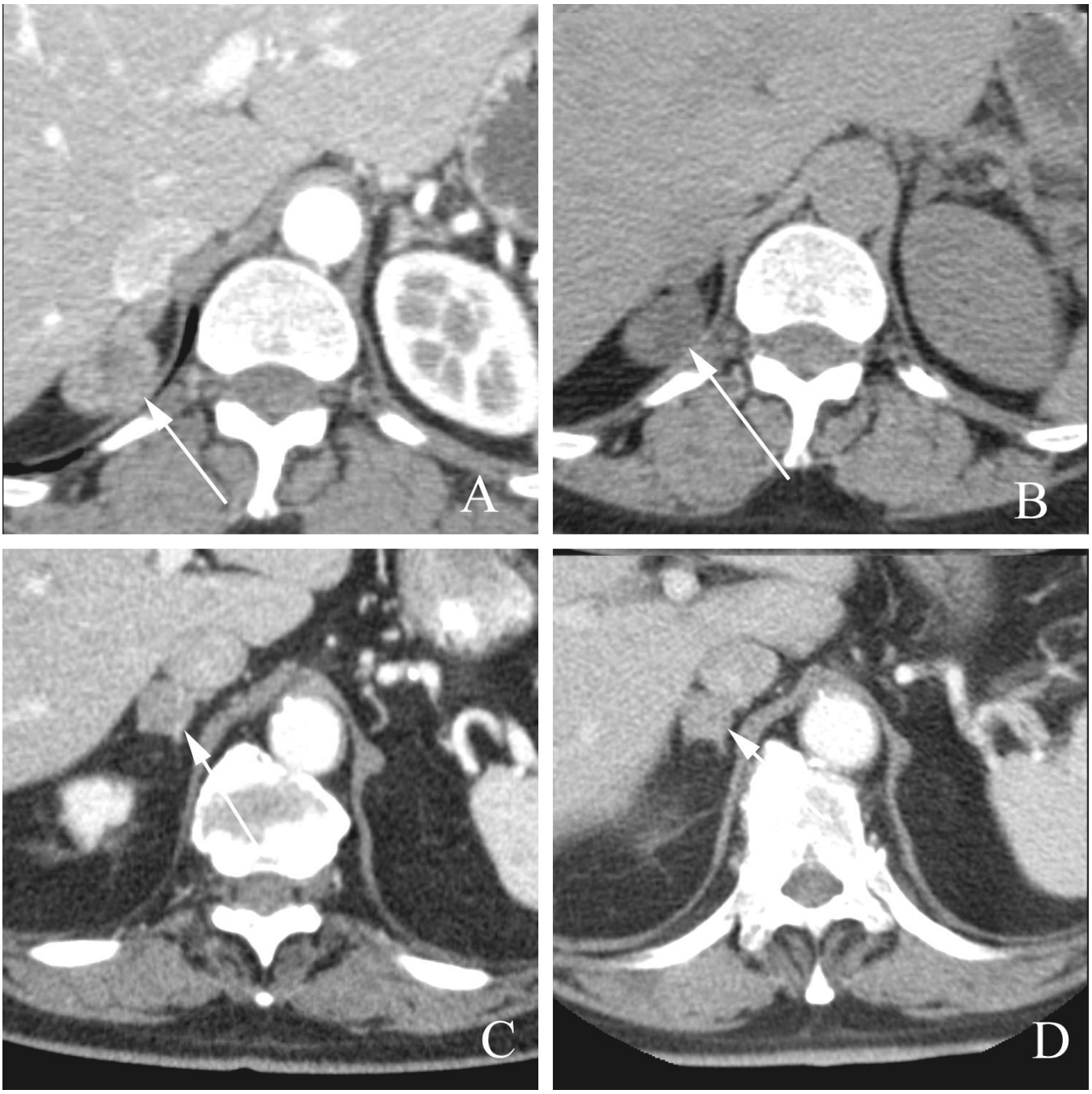

Figure 12. Adrenal incidentalomas where additional images demonstrated features of definitely benign adenoma. A. An axial CT done during the arterial phase of a CT performed for hematuria in a 60 year old woman shows a mass of the right adrenal (arrow). B. A noncontrast study performed at the same level shows the adrenal lesion to be hypodense (HU < 0), establishing the diagnosis as a benign adenoma. C. An axial CT done during work-up for esophageal carcinoma in a 78 year old man shows an adrenal lesion (arrow). D. Comparison with a study done eight years earlier shows that the lesion has undergone no interval change, diagnostic of a benign adrenal adenoma.

Size			Category
< 3 cm			Probably benign adenoma
3 – 5 cm	CT w/o contrast – density evaluation	< 0 HU	Benign adenoma
		0 - 10 HU	Probably benign adenoma
		10-43 HU	Indeterminate
		> 43 HU	Suspicious for cancer
	CT contrast dynamics (done with contrast)	% relative washout >40% or enhancement washout >60%	Probably benign adenoma
		% relative washout <40% or enhancement washout <60%	Indeterminate
	MRI	Lipid present	Probably benign adenoma
		Lipid not present	Indeterminate
> 5 cm			Suspicious for cancer

Recommendations	
All nodules	Endocrinologic evaluation
Benign adenoma	Done
Probably benign adenoma	f/u CT at 6 and 18 months
Indeterminate	f/u CT at 6 and 18 months or biopsy
Suspicious for cancer	Biopsy or surgery

Table. Evaluation of incidentalomas, courtesy of Dr. Timothy Seline, Radiology Associates of the Fox Valley. The table assumes that an adrenal lesion has been found on imaging. For most incidental lesions of less than 3 cm in patients without cancer, endocrinologic work-up and follow-up CT scanning at 6 and 18 months are usually all that is necessary (if negative). Otherwise, the patient will return earlier for specific additional imaging, with recommendations as noted above.

Liver "incidentalomas"

In addition to the incidentally discovered adrenal lesion, CT may disclose incidental lesions of the liver (or other body parts). In the absence of liver enzyme abnormalities or multiplicity, these lesions are usually benign abnormalities of some sort. The likelihood of primary tumor such as hepatocellular carcinoma or cholangiocarcinoma being found on CT (particularly in a non-cirrhotic patient) is quite small. Of course, multiple liver lesions, particularly with a history of malignancy or clinical features of cancer, need further evaluation, with biopsy often necessary (Figure 6). For isolated, incidentally discovered liver lesions, the radiologist may recommend further imaging. Similar to the situation with adrenal incidentalomas, imaging consists of additional CT exams done either without or with various phases of intravenous contrast enhancement, or MRI. MRI has the ability to detect smaller lesions and to "fully characterize" (i.e. offer a firm diagnosis) of certain small lesions such as cysts, which is difficult or impossible with CT. Of course, it may ultimately be necessary to sequentially image an abnormality over several months or years or to perform biopsy to establish the benign nature of the lesion. The utility of a diagnosis in such cases must

be weighed against the cost, anxiety, and morbidity of obtaining an answer – which is difficult to do when you don't know the histology of the lesion. As of this writing, in the U.S., the usual path (perhaps spurred as much by fears of a lawsuit as by the pursuit of good medicine) is to err on the side of over-evaluation rather than under-evaluation. It is a tough call as to how aggressive to be in the work-up of these incidentally found abnormalities, and it may be worth a discussion with the radiologist who finds the incidentaloma and recommends further evaluation to see what particular diagnosis (or what differential diagnosis) he or she has in mind before proceeding with further testing. It may be that upon weighing the options the patient may choose to forego the option of certainty, if the likelihood of potential benefit is small and the costs of certainty are high. Of course, estimation of probabilities in such a case is devilishly difficult and the most that can be said about such lesions is "It is not clear *what* the lesion is for certain, and it *may* be something quite bad".

CT colonoscopy

Another scenario where asymptomatic patients undergo imaging is the screening study of virtual colonoscopy. This technique involves a complete large bowel prep followed by a CT scan performed with rectally insufflated air. Dedicated software is used to process the resulting data, producing not only the standard axial, coronal, and sagittal reformatted images, but also a "fly through" version simulating the endoscopist's view. The resulting images and all the technology to produce them are stunning.

As impressive as all this technology is, it is difficult to make any sweeping statements about when to order or use the study. Advocates note a lower perforation rate than with optical colonoscopy, the fact that optical colonoscopy may miss some lesions hidden behind bowel folds, and the lack of a need for sedation (or a driver to take you home). They also note the ability to screen extraintestinal organs for disease, although for this particular "asset", see the above discussion about CT screening. Advocates of optical colonoscopy point to the facts that optical colonoscopy is the reference standard for polyp and carcinoma detection, that polyps may be removed and carcinoma biopsied during colonoscopy, and that most patients prefer optical colonoscopy as a procedure. Also of note is that, at least as of 2010, the Centers for Medicare and Medicaid Services (CMS) do not cover most screening virtual colonoscopy, and many insurance companies follow the CMS's lead in this regard.

Whether to recommend virtual colonoscopy over or as an alternative to optical colonoscopy is therefore a controversial and evolving question, and the best advice is to take into account patient preferences, local expertise, and insurance company stipulations when counseling (and ordering studies on) patients.

SUMMARY

This chapter covers the "second step" in gastrointestinal imaging. Women with low abdomen and pelvic pain may need both CT and US for evaluation, occasionally supplemented by MR. Small bowel imaging may require more than a standard CT study, with additional imaging performed with low density contrast material administered orally or via an intestinal catheter. CT may demonstrate incidental lesions that require additional work-up. Finally, CT colonoscopy is an evolving method of evaluation of the large bowel, often used for screening to find colon polyps prior to malignant transformation.

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Breast Imaging

Donald L. Renfrew, MD

Breast cancer is the most frequent non-skin cancer diagnosis in women, with an estimated 192,370 new cases in 2009[1]. Knowing what diagnostic imaging tests are available, which test to order when, and what to do with the results presents a challenge to the primary care practitioner. This chapter reviews three key concepts regarding breast imaging:

1. There are certain relatively widely accepted rules about how to screen asymptomatic women, and how to image symptomatic women.
2. Mammography is the mainstay of diagnosis, frequently supplemented by ultrasound, with MR playing a minor role.
3. Careful follow-up and handoff of the patient is critical for optimal patient care.

RULES TO GUIDE BREAST IMAGING

There are a few relatively widely accepted rules regarding breast imaging that are helpful to know when ordering imaging studies. Breast imaging studies may be divided into screening and diagnostic exams, and the rules differ for these two categories of exams. This chapter first covers screening studies, done on asymptomatic patients to detect possible breast cancer. It then discusses diagnostic studies.

Screening studies

Screening studies are usually chosen for a combination of factors including relatively low cost and high sensitivity: the screening test should pick up as much disease as possible, with the idea that subsequent studies will provide more specificity regarding the diagnosis.

Screening mammography

Mammography remains the king of breast imaging (Figure 1). It has been shown in multiple trials to reduce mortality in the screened population by about 30%[2]. It's the best screening test we have. That being said, it has problems as a screening test: it is relatively insensitive, it involves ionizing radiation, it is at least somewhat painful for most women, and it can be inconvenient. It also results in a fair number of false positives, causing a lot of needless worry on the part of patients and driving up the costs of medical care. If we had some alternate method of early diagnosis – for example, a serum test for tumor markers – this would be a great advantage. This may happen, but it hasn't yet, so we continue to do mammography.

General recommendations are that women have screening commencing at age 40, and continue as long as life expectancy is at least ten years[3]. For

patients who have had a mother, sister, grandmother, or aunt diagnosed at a young age (prior to 40) with breast cancer, it is generally accepted that screening should begin at an age earlier than 40. One commonly used rule is to start screening at 5 years prior to the age of diagnosis of cancer in the relative.

Note that a screening mammography report will usually contain one of two recommendations: 1) a recommendation to return for an annual screening mammography in one year, if the study is normal; or 2) a recommendation for additional imaging studies if the screening study is abnormal (see below). Usually, the additional imaging study is either additional mammography, with, for example, spot compression or magnification views, or ultrasound evaluation. It is uncommon to proceed directly to biopsy on the basis of a screening study.

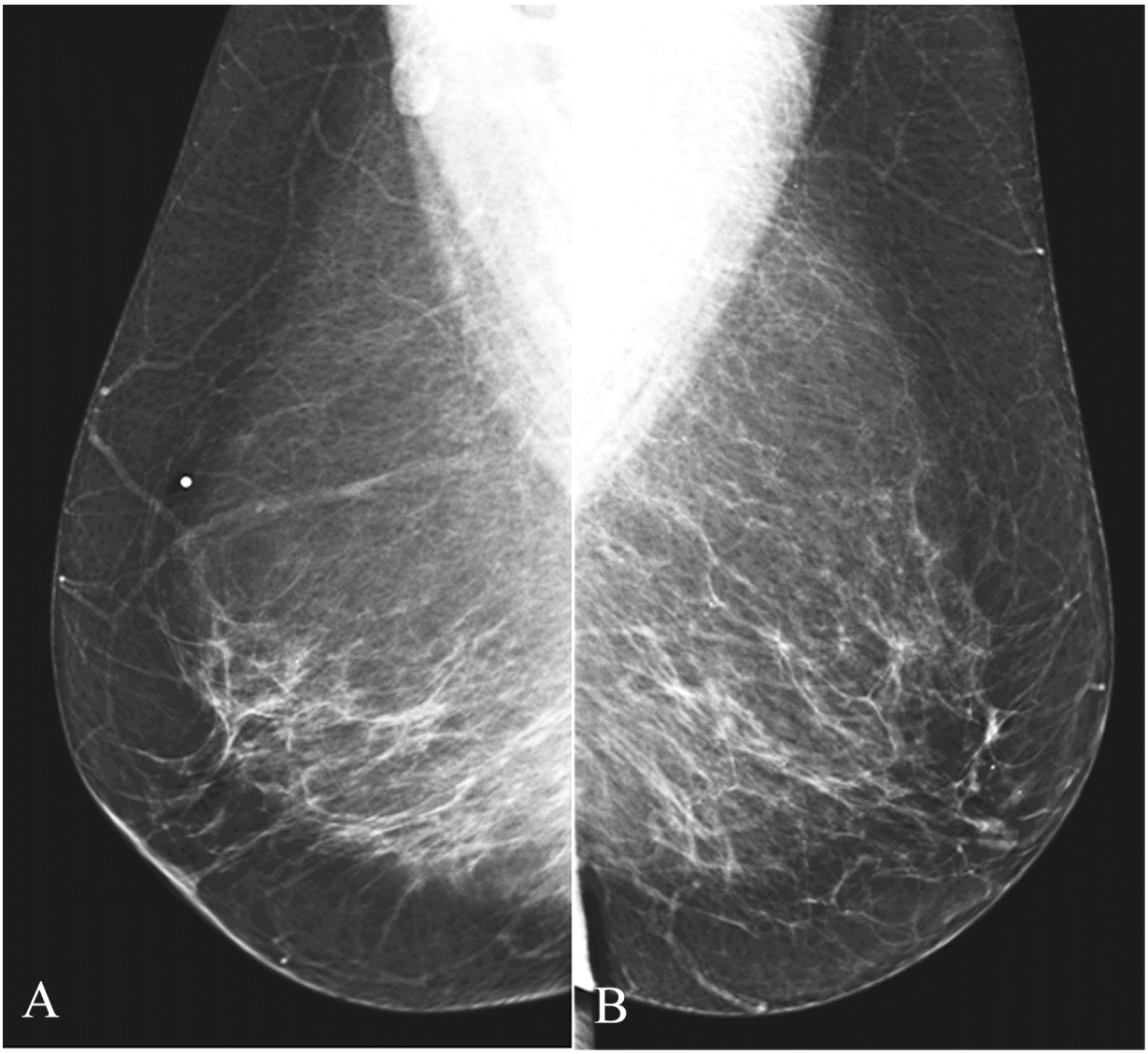

Figure 1. Normal digital screening mammogram, mediolateral oblique (MLO) views. Modern digital mammography technique shows exquisite detail of breast tissue allowing screening for malignancy. Note the inclusion of the pectoralis muscle along the posterior margin of the study. Screening mammography usually includes both bilateral mediolateral oblique views (shown) and craniocaudal views (not shown).

Screening MRI

MR is more sensitive than mammography in the detection of breast cancer. The generally accepted sensitivity for MRI is over 90%, but it will miss small cancers or areas of DCIS[4]. There are two major problems with breast MR, however: 1) specificity is only in the 50-70% range secondary to false positives from fibroadenomas and other benign lesions, and 2) cost. The false positives necessitate either biopsy or follow-up MR, both of which are also costly. However, because of the increased sensitivity of MR compared to mammography, there are multiple organizations, including the American Cancer Society, that advocate screening MRI for patients with a 20 – 25% lifetime risk of breast cancer[5]. Patients will generally fall into this high risk category if they have a breast cancer gene (BRCA1 or BRCA2), or if they have close relatives with breast cancer. There are several online calculators which will allow precise determination of cancer risk, for example at: http://www.cancer.gov/bcrisktool/.

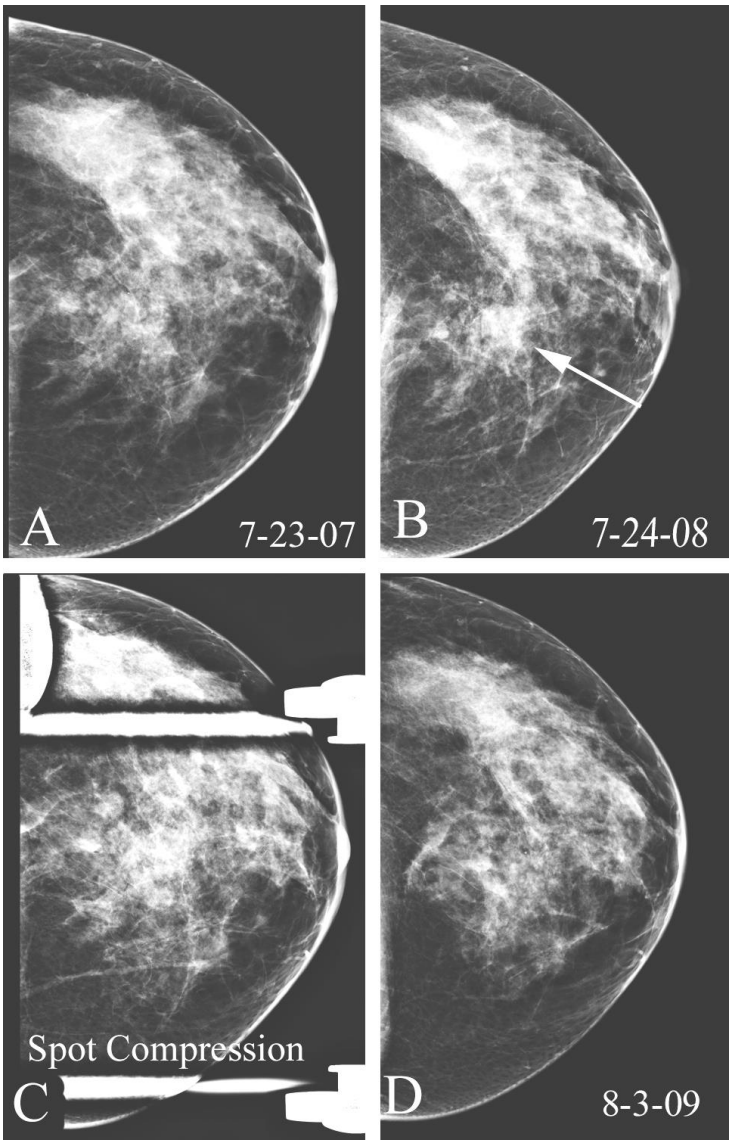

Figure 2. Abnormal screening mammogram, prompting recall of the patient for a diagnostic mammogram with additional views showing normal tissue. A. Screening mammogram from 7-23-07 is normal. B. The patient's left craniocaudal view from 7-24-08 shows an apparent developing mass in the inner aspect (arrow). C. Spot compression study shows no discrete mass but normal, although dense, breast tissue. D. Follow-up mammogram study of 8-3-09 is normal.

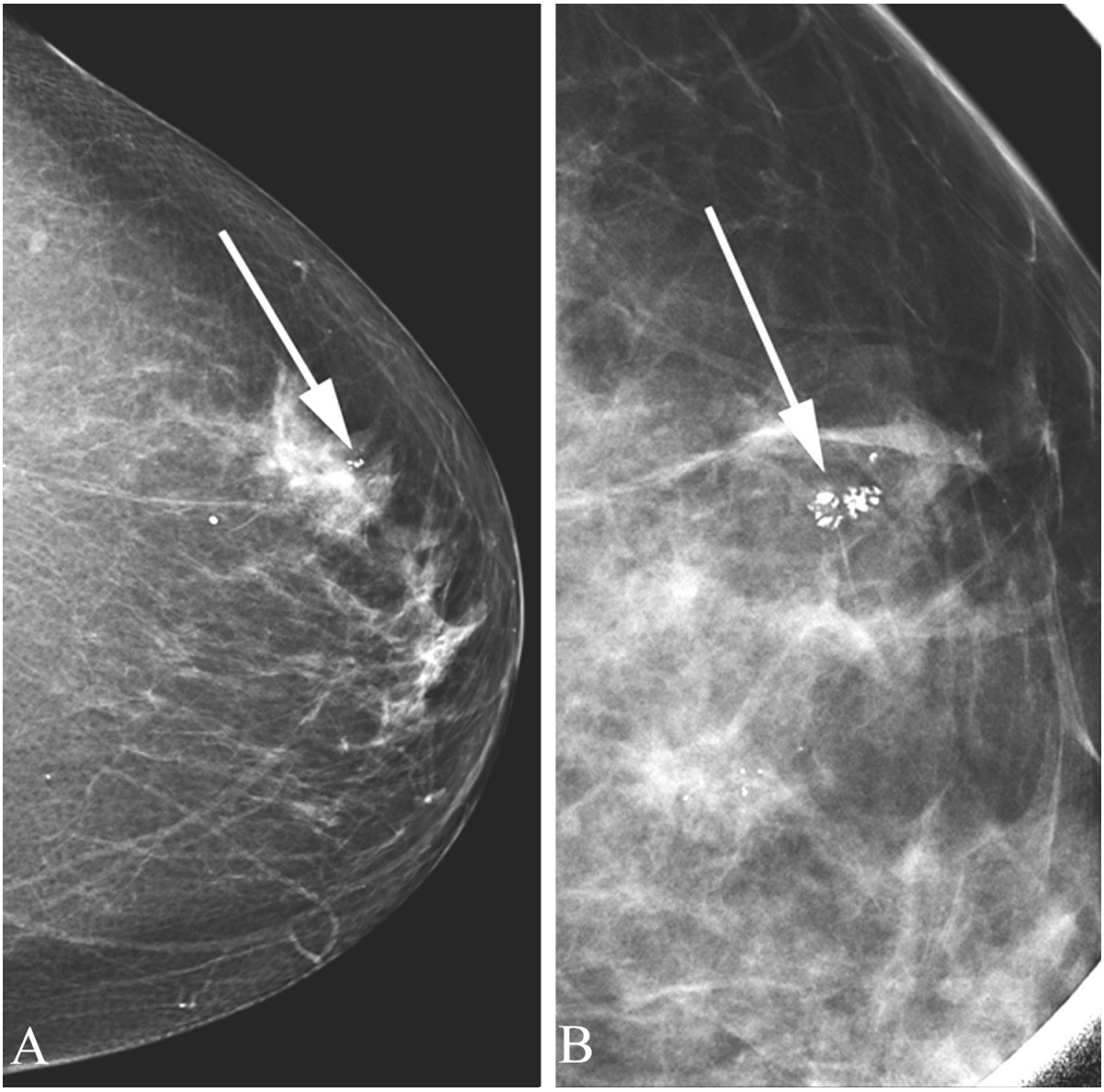

Figure 3. Abnormal screening mammogram, prompting recall of the patient for a diagnostic mammogram with additional views prompting biopsy. A. Screening cradiocaudal mammogram shows a small, dense cluster of calcifications (arrow). The patient was recalled for a diagnostic mammogram. B. Spot magnification craniocaudal mammogram better shows these calcifications (arrow), which demonstrate variable size. Stereotactic needle biopsy was performed, and the pathology interpretation was an involuted fibroadenoma and focal ductal hyperplasia without atypia.

Screening ultrasound

Ultrasound is presently not routinely used as a screening study, although the modality is undergoing evaluation as an adjunct (or possible replacement) to mammography, particularly in patients with dense breasts[6] [7].

Diagnostic Studies

Screening mammography is done on asymptomatic patients with no known imaging abnormality. *Diagnostic mammography* is performed when there is either an abnormality on a screening examination (also known as a callback) or the patient has symptoms. Ultrasound and MR may also be used as

diagnostic studies, and again this usually occurs either because of an abnormal screening examination or patient symptoms.

Abnormal screening studies resulting in diagnostic studies

Nowadays, most radiology departments handle callbacks internally, with the department notifying the patient that additional evaluation is necessary. If the results of that additional evaluation are clearly benign (Figure 2), then the patient returns to a yearly screening schedule. If the results of the additional evaluation are not clearly benign, it may be necessary to proceed with biopsy (Figure 3). Ordering of studies and the decision to proceed with biopsy should generally follow the radiologist's recommendations.

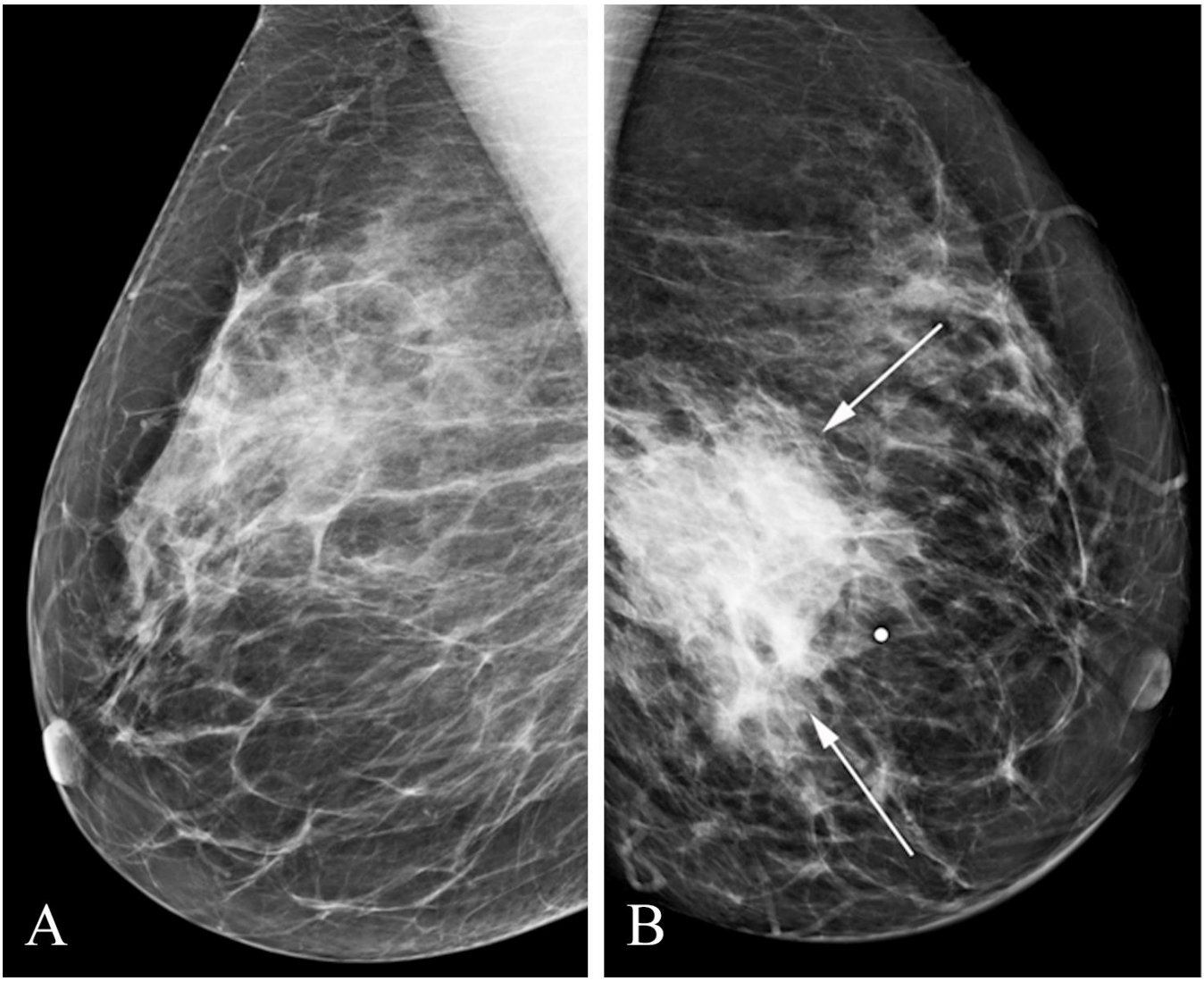

Figure 4. Infiltrating ductal carcinoma in a 39 year old woman with a breast mass found at breast self examination. A. Right mediolateral oblique (MLO) diagnostic mammogram is normal. B. Left MLO diagnostic mammogram demonstrates a large, dense mass (arrow).

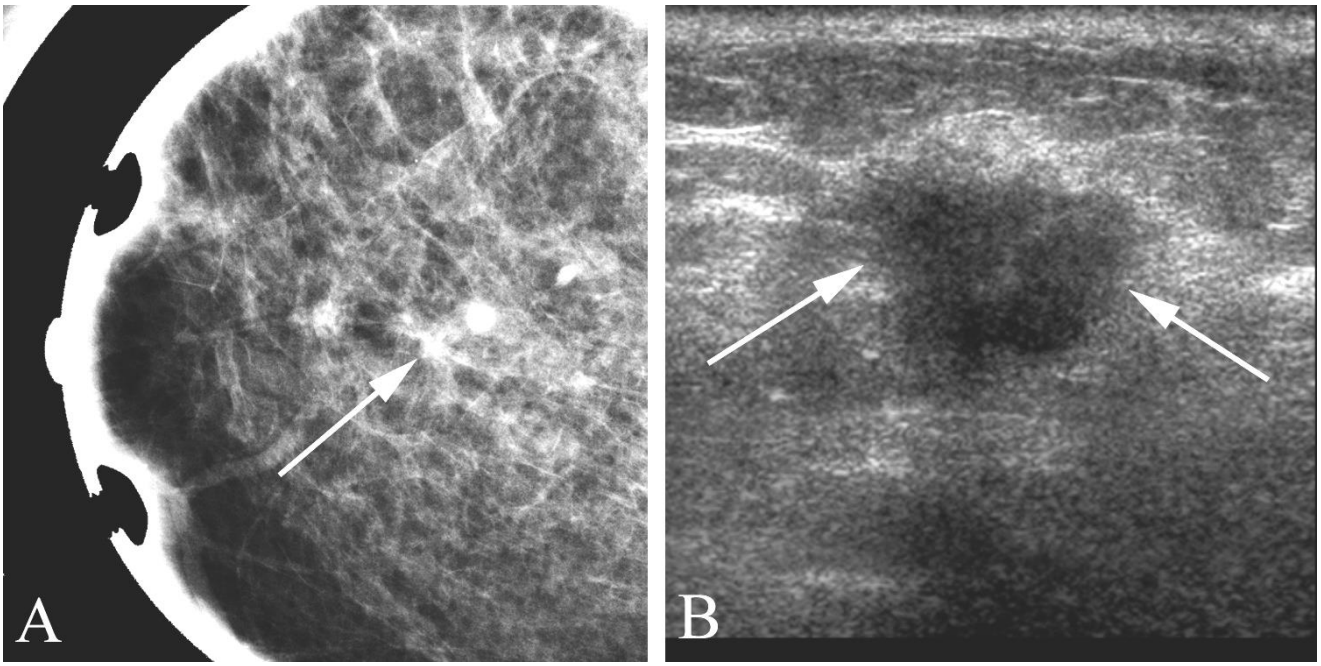

Figure 5. Infiltrating ductal carcinoma in a in a 75 year old woman with a palpable lesion found at clinical breast examination. A. Craniocaudal mammogram spot compression views (following initial full field exam) shows a subtle lesion of the right breast by the radio-opaque marker. B. Breast ultrasound demonstrates a hypodense, shadow-casting, irregular lesion (arrows) worrisome for malignancy. Biopsy revealed infiltrating ductal carcinoma.

Breast lump or focal pain, age > 35

Generally speaking, lumps and focal pain should be worked up in a similar fashion. Lumps found at clinical breast examination (CBE) or breast self examination (BSE) are both evaluated using the same algorithm, although lumps found at CBE are more likely to be malignant than those found at BSE[3].

For patients over the age of 35 with a lump or focal pain, mammography should be performed first (Figure 4), with ultrasound to follow if necessary (Figure 5)[8]. The mammogram should be scheduled as a "diagnostic" (not a "screening") study, and the technologist will typically put a radiographic marker at the location of the palpable lump or area of maximum pain. If the palpable abnormality is subtle on clinical exam, particularly if the patient cannot feel the abnormality herself, it is best to mark the patient's breast at the time of the physical examination, prior to sending the patient for imaging. This way, the technologist will know where to place the radiographic marker. The mammogram should include both breasts if the asymptomatic breast has not undergone mammography in the past year.

If the mammogram fails to show, or does not adequately characterize, an explanatory lesion at the location of the palpable abnormality or focal pain, the patient will typically proceed to ultrasound (Figure 5). The ultrasound study is done because ultrasound will demonstrate some malignant lesions that escape detection on mammography, and ultrasound may better demonstrate some lesions which are poorly demonstrated on mammograms.

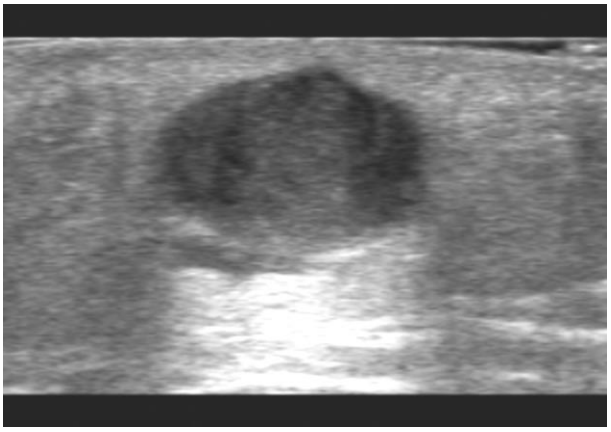

Figure 6. Ruptured epidermal inclusion cyst in a 23 year old woman with a palpable abnormality. The ultrasound exam demonstrates a hypoechoic lesion. Since the abnormality did not represent a simple cyst or a prominent but normal ridge of breast tissue, it was surgically excised, and the pathologic diagnosis was a ruptured epidermal inclusion cyst.

Breast lump or focal pain, age < 35

For patients under the age of 35, ultrasound should be performed first, followed by mammography if necessary[8] (Figure 6). These women have denser breasts and a lower pretest probability of having a malignancy with a higher likelihood that the palpable lesion is a cyst or benign but prominent ridge of breast tissue. Therefore, it makes sense to perform ultrasound first, followed by mammography if the ultrasound provides no explanation but there is still a strong suspicion of a lesion.

Breast discharge

Multipore, blood-negative, expressed-only discharge is best categorized as benign physiologic discharge, and is not worrisome for malignancy. Such discharge may require medical evaluation and medical work-up[9].

Unilateral, single pore discharge, particularly if bloody, is worrisome and needs further evaluation[10]. The first imaging step in evaluation is usually ultrasound, particularly in patients under 30, to detect dilated ducts and focal masses. This may be followed by mammography, and if these tests do not provide a definitive answer, then a ductogram (also known as a "galactogram") may provide a diagnosis (Figure 7). The ductogram is performed by cannulating the nipple pore that shows the discharge with a small, specially designed blunt catheter and injecting contrast material into the duct in a retrograde fashion.

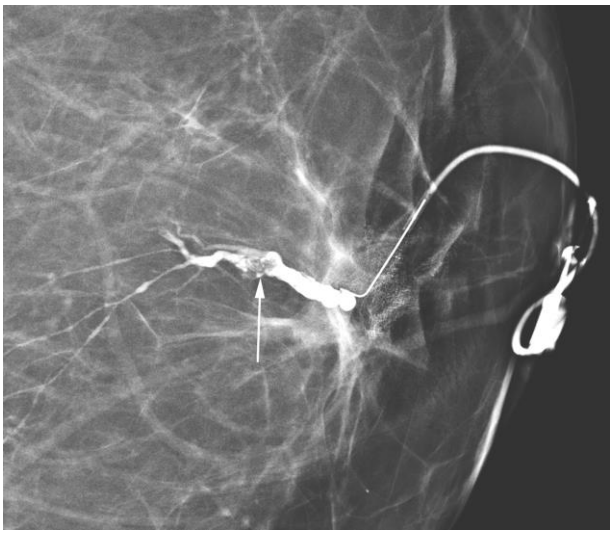

Figure 7. Intraductal papilloma in a 55 year old woman with bloody single pore nipple discharge. A standard mammogram (not shown) failed to demonstrate any cause of discharge. The catheter tip is at the nipple, and contrast material fills the dilated duct which has a filling defect (arrow), found at pathology to represent a benign intraductal papilloma.

What NOT to image

For patients with diffuse pain, or with bilateral, multipore discharge, no imaging beyond standard, age-appropriate screening mammography is useful.

MAMMOGRAPHY IS THE PRIMARY IMAGING MODALITY, SUPPLEMENTED BY ULTRASOUND, WITH A SMALL ROLE FOR MRI

As noted above, mammography is the screening modality of choice, and is the most frequently used diagnostic modality as well. Since mammography is the primary method of breast evaluation in both the screening and diagnostic roles, how do radiology departments know that they are doing a good job?

Mammography quality assurance

Mammography quality assurance has evolved through the years in part because of work done by

the American College of Radiology (ACR), and in part because of legislation known as the Mammography Quality Standards Act (MQSA).

ACR Lexicon and BI-RADS

In response to complaints about the variability of mammography reports, the American College of Radiology developed a lexicon of mammography terms[11]. As it turns out, this lexicon has not been universally adopted although the ACR publishes an excellent handbook illustrating these terms[12]. At the same time they developed the lexicon, the ACR also developed the Breast Imaging Reporting and Data System or BI-RADS (Table 1). BI-RADS is a quality assessment tool, but it is also directly clinically relevant, because it forces the radiologist to reduce the mammogram result to a single number which determines the next step in patient care. As noted by Sistrom and Langlotz writing on the topic of improving radiology reporting "One of the greatest benefits of the entire BI-RADS initiative arises from the mandated forced choice between clinically diagnostic categories".[13] Each BI-RADS category is linked to a specific next step, making management unambiguous (Table 1)

BI-RADS Category	Description	Next Step
0	Incomplete assessment	Return for additional imaging or obtain prior comparison studies
1	Negative	Return for routine screening
2	Benign findings	Return for routine screening
3	Probably benign findings	Return for initial short term follow-up (usually 6 months)
4	Suspicious abnormality	Biopsy should be considered
5	Highly suggestive of malignancy	Appropriate action should be taken
6	Known malignancy	Appropriate action should be taken

Table 1. BI-RADS categories with descriptions and resulting actions.

Screening mammography metrics

The BI-RADS categories allow relatively easy evaluation of large amounts of data. The United States Department of Health and Human Services has created benchmarks or metrics for community radiologists which may be calculated with the use of these categories[14]. Of these metrics, the most useful are probably recall rate, biopsy rate, biopsy yield, and cancer detection rate*. Note that these metrics can be calculated from the BI-RADS codes given to the screening studies and follow-up on those specific studies where biopsy was recommended (which should represent about 1% of the screening exam results). Also note that the cancer *prevalence* rate is different in those women undergoing screening mammography for the very first time than the cancer *incidence* in patients undergoing annual screening. While the general recall rate is set at 10%, the recall rate is also different between women undergoing their first study (where 10% is a reasonable figure) versus women undergoing repeated screening (where 3% or 4% is probably more reasonable[15]). However, the prevalence data (exams for first-time screening mammograms) and incidence data (exams with prior studies for comparison) are often pooled in evaluating mammography quality assurance. An example for data in one small community hospital is presented in Table 2. Note that in the "Analog" column, the data represents a two year period and demonstrates adequate performance with respect to the recall rate, biopsy yield, and cancer detection rate. The biopsy rate is higher than the benchmark (1.6% versus 1.0%), but given that the biopsy yield is still significantly above the benchmark, this is acceptable.

* Other benchmarks or metrics include: sensitivity of at least 85%, prevalent cancer detection rate of 0.6 – 1.0%, incident cancer rate of 0.2 – 0.4%, less than 25% with positive lymph node metastases at the time of diagnosis, mean tumor size of less than 1.5 cm, at least 30% DCIS or invasive cancer < 1 cm; at least 50% stage 0 or 1 cancer.

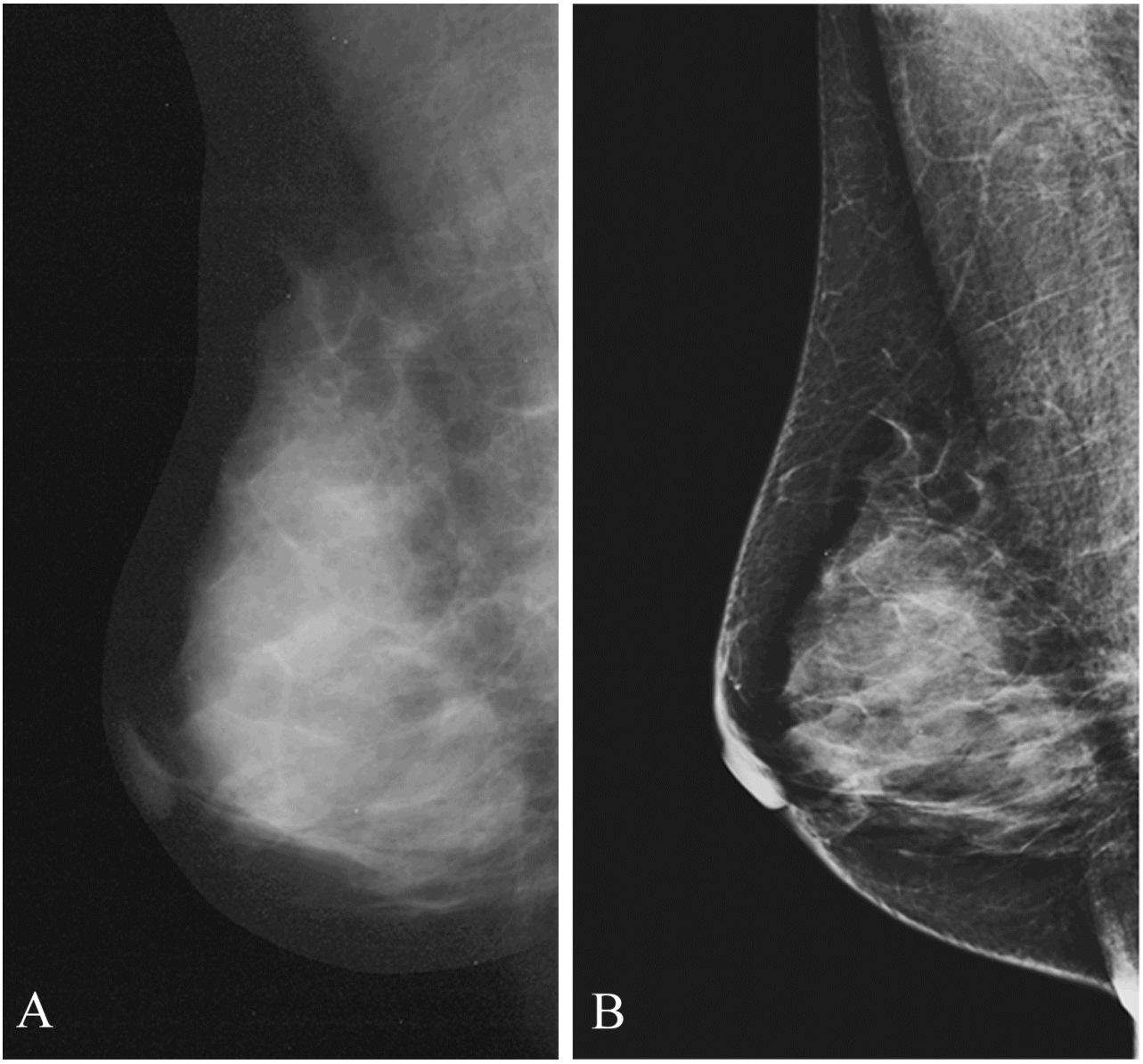

Figure 8. Superior detail with digital mammography. Film-screen (A.) versus digital (B.) normal screening mammogram. Note the superior visualization of both the central, dense parenchymal tissue, and also the peripheral, predominantly fatty breast tissue, with digital mammography.

Digital mammography

Digital mammography uses different technology than analog mammography, and provides greater detail, particularly in the superficial tissues and in dense breasts (Figure 8). Image data is collected, stored, and displayed electronically rather than with film. Digital mammography shows greater sensitivity for detection of cancer in women with dense breasts, as seen in women under the age of 50 or women who are premenopausal and perimenopausal[16]. In addition, Sala et al demonstrated a significant reduction of the call-back rate for digital mammography versus film mammography in return patients (2.4 % versus 3.6%)[15], without a decrease in the rate of malignancy detection. This reduction in call-back rate is important, since women being recalled for additional views may experience significant, ongoing anxiety[17]. When comparing the data at the same small community hospital (Table 2 again), note that following implementation of digital mammography, there was a decrease in the recall rate (in this table, both initial and return recall rates

are pooled), similar to the Sala et al study, while the biopsy *rate* (the percentage of screening patients eventually undergoing biopsy) fell, while the biopsy *yield* (the likelihood that a given biopsy demonstrated cancer) increased. The cancer detection rate showed a statistically insignificant, small decrease.

Wherever mammography is done, these metrics should be available. If, as is often the case, there is more than one available location for mammography service, these metrics provide a handy way to compare the locations.

Metric	Benchmark	Analog (5742)	Digital (6128)
Recall Rate	<10%	6.3%	4.6%
Biopsy Rate	<1%	1.6%	1.1%
Biopsy Yield	>25%	30.4%	40.6%
Cancer Detection Rate	0.2 – 0.5%	0.49%	0.46%

Table 2. Mammography data from Door County Memorial Hospital, Sturgeon Bay, WI. Rates are for screening mammograms performed in a community hospital, with historical comparison between Analog and Digital examinations.

Ultrasound is used frequently and MR is used occasionally for problem solving

Ultrasound is used to distinguish normal tissue and cysts from solid masses. Ultrasound can be used to evaluate palpable lesions, focal tender spots, or lesions seen on mammography or MRI requiring further work-up. Lesions seen on ultrasound may be placed into one of four basic categories, two of which typically require no further evaluation or work-up. If a normal ridge of breast tissue or a cyst explain the abnormality, then no further evaluation is necessary (Figure 9). If a solid lesion is identified, this typically requires biopsy, although some solid lesions are relatively typical of benign lesions such as fibroadenomas (Figure 10),

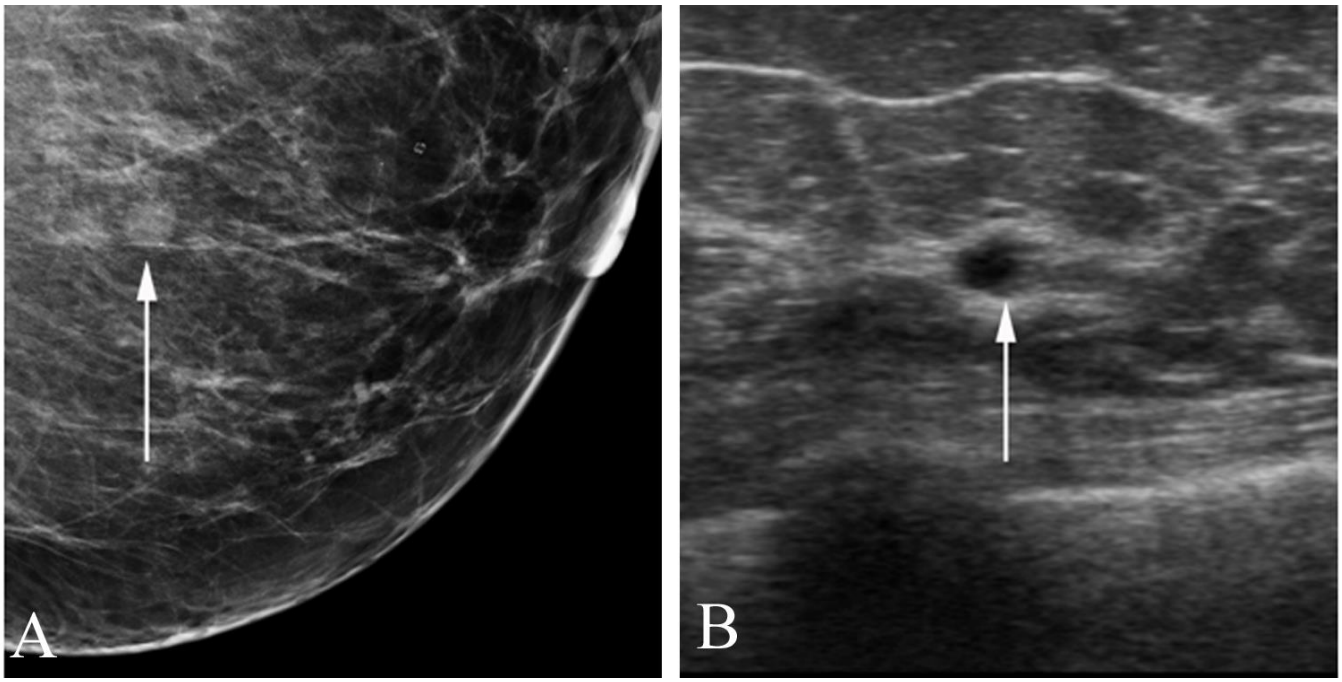

Figure 9. Cyst in a 47 year old woman with an abnormal screening mammogram, with ultrasound demonstrating a benign cyst at the location of the new lesion. A. Craniocaudal screening mammogram (cropped) shows a circumscribed hypodense lesion of the inferior right breast (arrow). B. Ultrasound (right) demonstrates a simple cyst at the location of the lesion (arrow), and no further work-up required.

while others are quite suspicious for malignancy (Figure 5). Some women would rather have even benign appearing solid lesions removed rather than followed, whereas other women would rather avoid biopsy. Malignant appearing solid lesions should certainly undergo biopsy.

MRI shows malignancy as a mass or enhancing tissue

In addition to its role as a screening tool in patients with a high risk of breast malignancy, MR may be used to evaluate the ipsilateral breast for mammographically occult disease, the contralateral breast in a patient with known malignancy (Figure 11), and, on occasion, to better characterize a lesion seen on mammography or ultrasound[18].

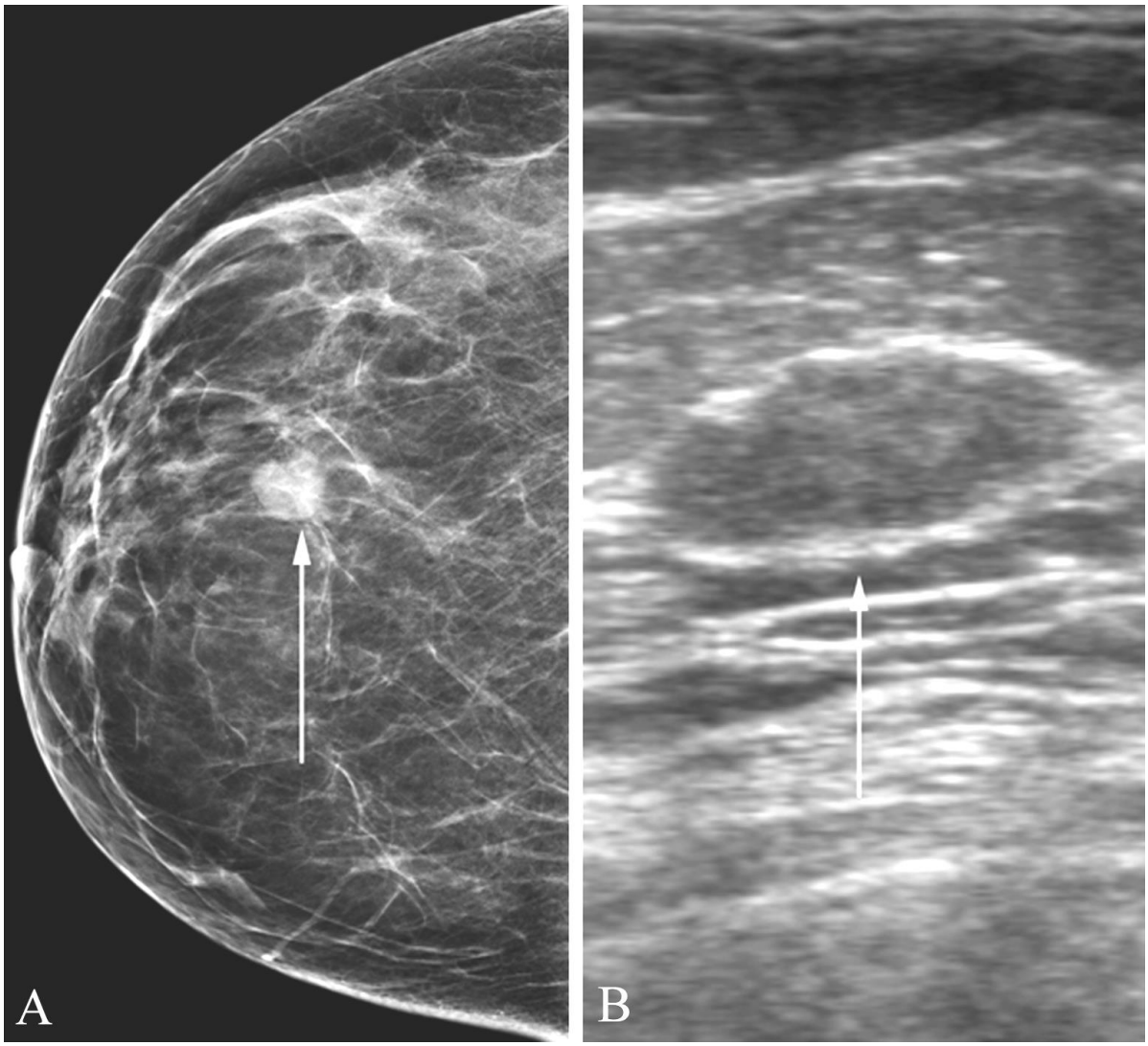

Figure 10. Fibroadenoma in a 48 year old woman with an abnormal screening mammogram, with ultrasound demonstrating a solid, benign appearing lesion at the location of the abnormality. A. Screening mammogram shows a circumscribed isodense mass (arrow) in the right breast. B. Breast ultrasound (with a different magnification) shows an oblong, sharply marginated, isodense solid mass without shadowing (arrow), characteristic of a fibroadenoma. The patient wanted the lesion removed despite its benign appearance, and pathology confirmed a fibroadenoma.

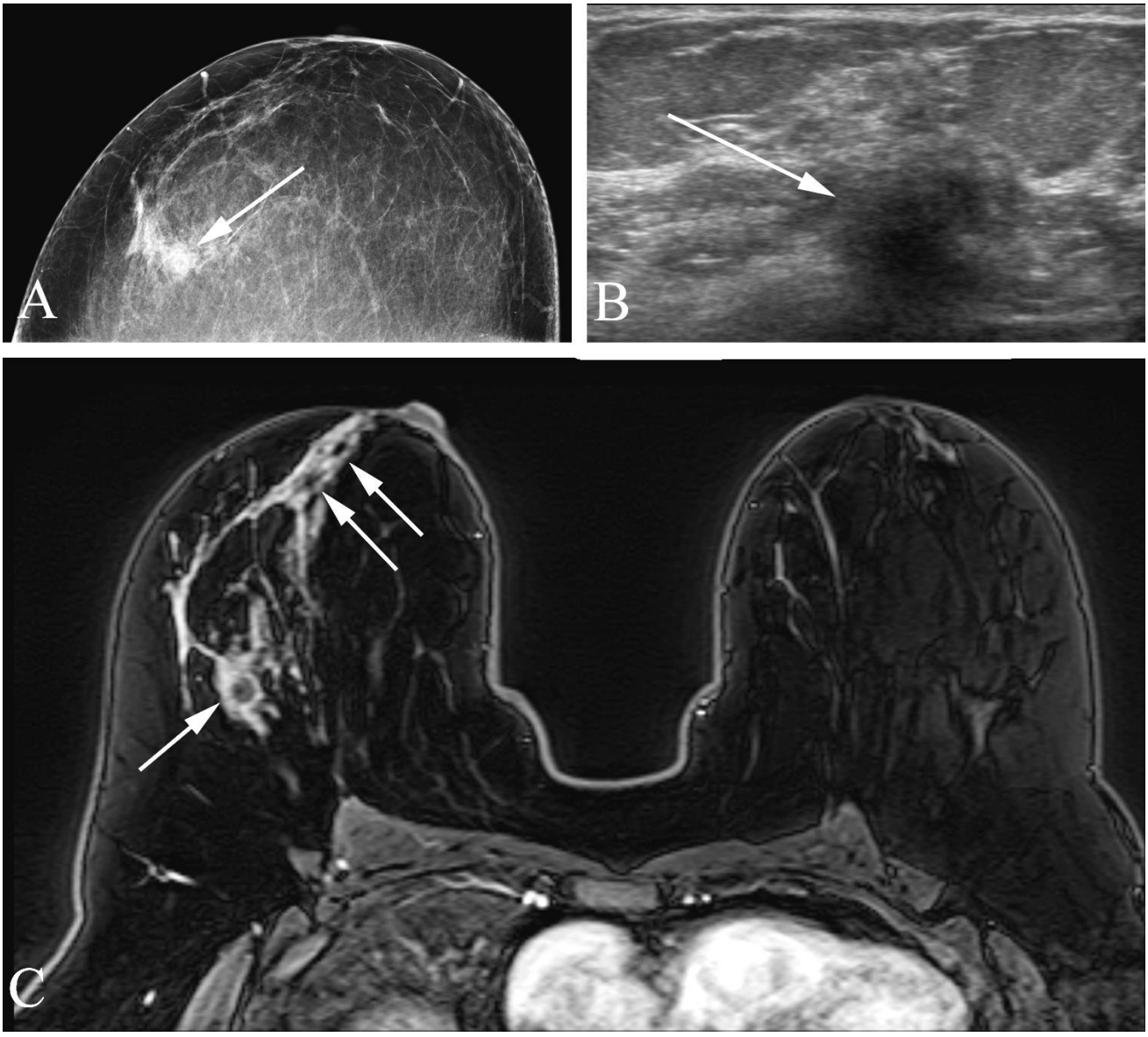

Figure 11. Infiltrating ductal carcinoma in a 41 year old woman with MRI demonstrating additional disease not detected at initial surgery. A. Right craniocaudal screening mammogram shows a mass in the lateral breast (arrow). B. US of the breast confirms a malignant appearing mass (arrow). C. Contrast enhanced MR examination of the breasts done following excision of an infiltrating ductal carcinoma demonstrates the operative site (arrow). Abnormal tissue extends from the biopsy site to the nipple (double arrow). Imaging directed biopsy of this region demonstrated multifocal high-grade DCIS beyond the margins of the initial surgery.

CAREFUL HANDOFFS ENSURE THE BEST PATIENT CARE

Careful handoffs from practitioner to practitioner prevent the tragic mistakes that can happen because of missed reports "falling through the cracks". With the development of BI-RADS, the responsibility to notify the patients to return for additional views or ultrasound examination largely shifted from the referring physician to the radiology department. Many of these same departments also schedule and

perform ultrasound-directed biopsy or stereotactic biopsy, whereas at other locations biopsies are performed by surgeons. Local referral patterns, as well as preference for ultrasound directed biopsy, core needle biopsy with mammographic guidance, and biopsy using needle localization techniques vary with locations as well as patient circumstances[18]. Regardless of the local distribution of duties, it is imperative that all involved physicians know the pathway the patient is taking. In the unfortunate event of a bad outcome, all parties will likely be held liable, so it is good to have redundancy built into the system in those instances when a patient is sent to biopsy. There are various mechanisms to achieve this, such as keeping a list of patients you know are going to biopsy *and* setting up automated forwarding of pathology results to you from the laboratory. Making sure the patient knows who to call, and that she *should* call someone, if she does not hear about her results, adds an additional layer of security. Do *not* assume the patient will call if she hears nothing: there are patients who, hoping for the best, will assume that "no news is good news".

SUMMARY

Breast imaging usually follows several widely accepted rules about when and how to screen patients, and when and how to image the breast symptoms of a palpable mass or focal breast pain. Mammography remains the mainstay of diagnosis, frequently supplemented by ultrasound with MR typically playing a minor role. Careful follow-up and handoff of the patient are critical for optimal patient care.

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Cough, Dyspnea, And Lung Nodules

Donald L. Renfrew, MD

This chapter covers four main points designed to help you order the correct first test when evaluating patients with cough and dyspnea and to understand imaging of pulmonary nodules:

1. The main decision about imaging of the pulmonary symptoms of cough and dyspnea is when to order a chest computed tomography (CT) study.
2. For patients with cough, a chest x-ray (CXR) is done first, usually followed by evaluation and treatment of common causes of cough, with a chest CT ordered only after eliminating common causes or if there are red flags in the clinical history.
3. For patients with dyspnea, a CXR is done first, followed by an urgent CT if there are red flags for a pulmonary embolism.
4. Pulmonary nodules typically will undergo CXR and CT, along with biopsy/excision, sequential CT, or positron emission tomography (PET) depending on the circumstances of the patient and size of the lesion.

CXR AND CT

The primary care provider has few options when ordering studies for evaluation of such cardiopulmonary complaints as cough and dyspnea. For the most part, the main decision will be whether to order a plain film or not, and when to proceed to CT. With regard to plain films of the chest, most texts emphasize that chest plain films are not always necessary to work up common cardiopulmonary complaints[1]. While this is certainly true in an academic sense, most patients with chest symptoms, in fact, seem to undergo plain film examination relatively early in the diagnostic work-up, and it is difficult to fault this practice since the downside is minimal, the patient expects that the study will be ordered, and the safety of having excluded a large chest malignancy is reassuring[2]. CT exams, on the other hand, are ordered much less frequently and usually later in the course of evaluation: they cost more, generally require the injection of IV contrast material, subject the patient to higher doses of radiation, and also reveal asymptomatic incidental pulmonary nodules requiring additional work-up.

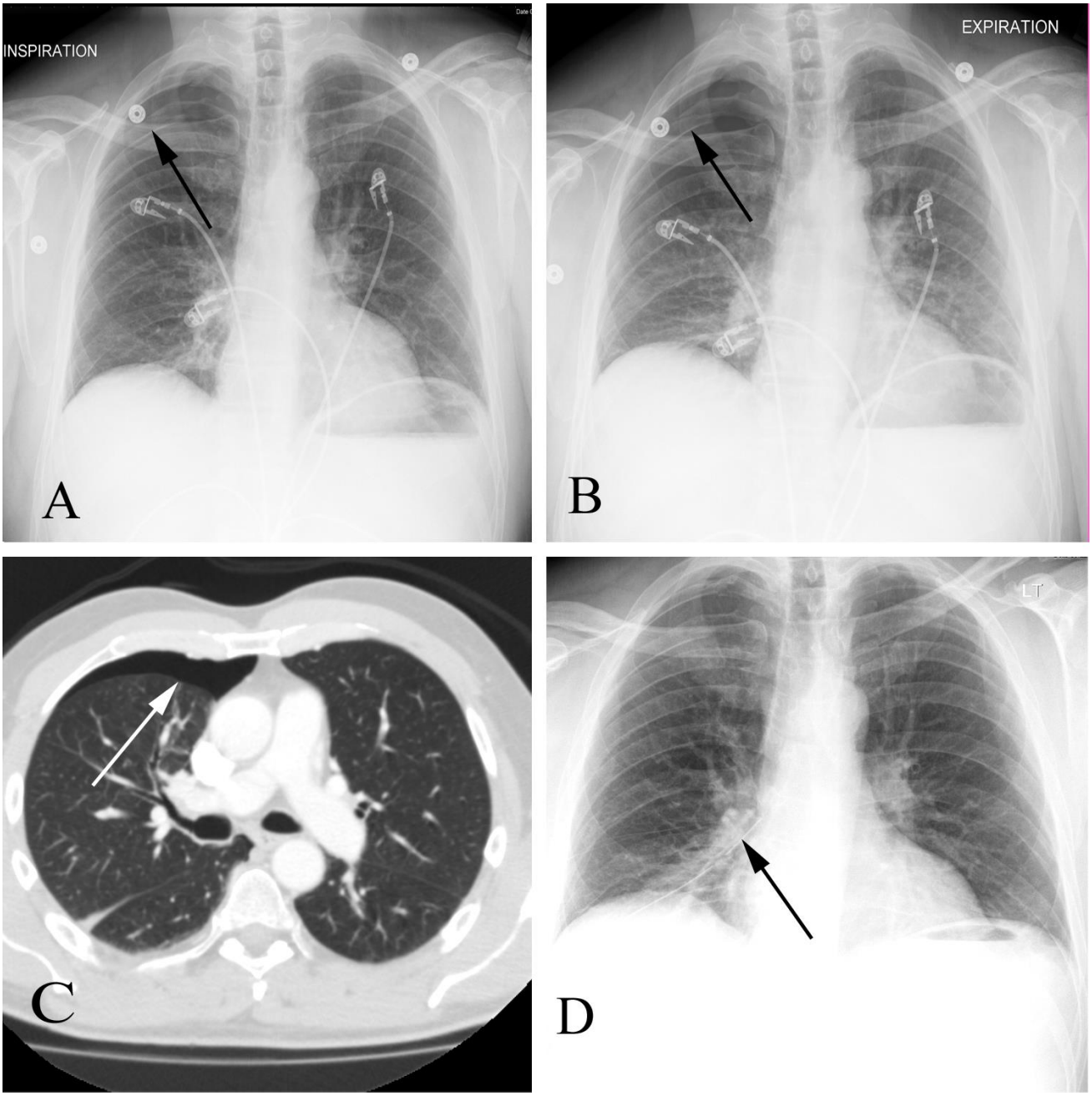

Figure 1. Pneumothorax in a 53 year old man with chest pain and dyspnea following trauma. A. PA inspiration chest x-ray shows a subtle pleural line (arrow) on the right side. B. PA expiration chest crowds the pulmonary vessels. C. On the CT study, the pneumothorax is much more conspicuous (arrow). D. Chest x-ray shows resolution of the pneumothorax following chest tube placement (arrow).

The standard two view chest plain film examination consists of a frontal exam taken with the anterior aspect of the chest closer to the film[1]

("PA" or posterior-to-anterior with respect to the path of the x-rays through the patient) and the x-ray source approximately 6 feet (72 inches) away from the film. The patient typically stands for the examination if possible, with the arms brought forward so the scapulae project to the side (or along the lateral margins) of the lungs. The exam is taken in full inspiration. The lateral study is done with the left side closest to the film/recording device. Note that variation in any of several technical aspects (distance, portion of the patient closest to the film/detector, degree of inspiration) may produce large changes in the appearance of the radiograph.

Alternatives to the "standard" upright PA and lateral view, like changes in the technical factors of the study, will alter the result. Typical variations include obtaining the study in a portable fashion, usually done in an AP manner so that the patient's back is to the film, which magnifies the heart. Non-portable supine exams also universally use the AP technique, again magnifying the heart. These studies are also performed at less than 72 inches from the x-ray tube to the film/recording device, another factor that magnifies the heart. Note also that when the patient is supine rather than upright, pleural fluid will flow to a dependent position along the dependent posterior chest wall so that, instead of demonstrating a dense (and easily recognized) band across the lower chest, the fluid will cause the entire associated lung to look slightly denser than it otherwise would. A similar phenomenon occurs with air in the pleural space: it moves to the anterior aspect of the chest and the entire lung looks slightly more lucent than it otherwise would. A crescent of air along the superior chest is typically easy to identify on an upright chest radiograph, but generalized lucency of the hemithorax much less so.

Decubitus films may solve the problem of identifying fluid or air in the pleural space. In these exams, the patient is placed with one side down (the side of the suspected effusion, or opposite the suspected pneumothorax) and a film is taken (usually AP). Layering of the fluid along the dependent aspect of the patient or air along the non-dependent aspect facilitates diagnosis.

Chest CT may be performed as a more accurate alternative to decubitus views (Figure 1), and is also almost always the study of choice as the next imaging study in the workup for any significant, worrisome, or confusing finding on a chest radiograph. Chest CT has undergone the same, relatively rapid, evolution with the advent of helical/multidetector scanners as has CT of other body parts. Twenty years ago, most deployed CT scanners were so slow that it was only practical to scan most patients at 8 or 10 mm slice thickness, and a contrast bolus would only result in vascular enhancement of a handful of slices. Nowadays most facilities have machines capable of (and use protocols calling for) 2 mm slices with excellent contrast of the vascular tree throughout the study. This has blurred or eliminated the distinction between "standard" and "high resolution" CT of the lungs, since routine CT scans are now performed with a technique that would once have been considered "high resolution". In addition, many radiologists routinely use workstations for image interpretation which allow image manipulation with, for example, construction of what are called "maximum intensity projection" maps or "MIPs" which allow easier and better detection of pulmonary nodules (Figure 2).

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1 I will use the term "film" throughout the chapter, but a recording device has replaced film in modern radiography equipment.

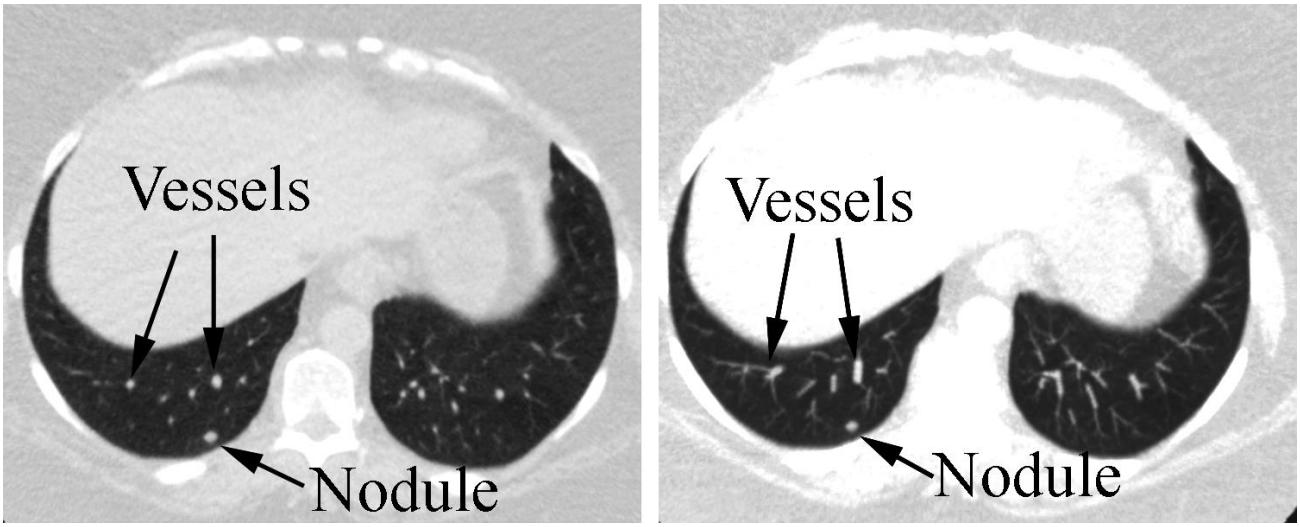

Figure 2. Chest CT exam showing the value of "Maximum Intensity Projections" or "MIPs". A. Routine chest CT filmed at lung windows shows a similar appearance for both the nodules and the vessels (imaged "on end"). B. MIP CT image demonstrates that these 10 mm thick slices (which record only the densest pixels from the slab) show the nodule as an oval density (as before), but the vessels as tubular, branching structures. These images make evaluation of the lung parenchyma for small nodules much easier.

The two main decisions facing the ordering physician when it comes to CT are: 1) "When do I order the CT?" and 2) "Do I order it without, or with, IV contrast material?" The first question is the subject of most of the rest of this chapter. The second question is best answered: "With contrast, if at all possible". Contrast allows visualization of the vascular tree, which is essential for diagnosis of pulmonary embolism and pulmonary arteriovenous malformations, and which is helpful to distinguish mediastinal vessels from lymph nodes and masses. The main reasons *not* to use contrast are if the patient is in renal failure (see pages 251-254 for further discussion of rules for contrast injection in renal failure), or if the scan is being done only to follow up a pulmonary nodule (or pulmonary nodules), (see later section in this chapter on "Pulmonary Nodules").

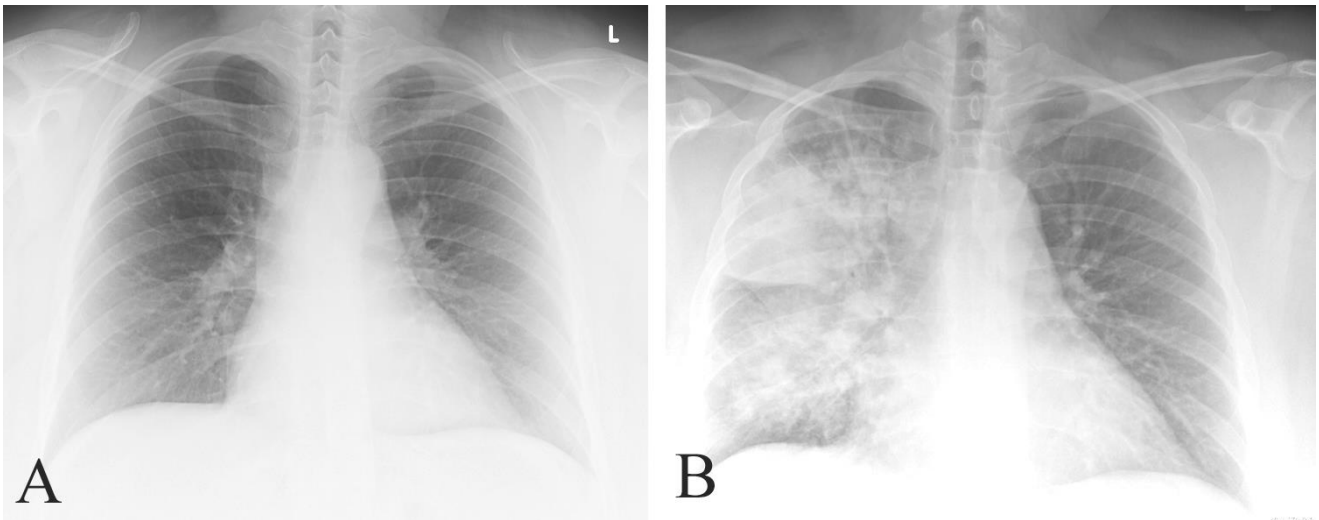

Figure 3. Pneumonia in a 34 year old woman with cough, fever, and shortness of breath. A. PA chest obtained prior to illness shows clear lungs. B. PA chest obtained during illness (right) shows extensive right lung opacity.

COUGH

One may divide patients with cough into those with acute cough (generally less than 3 weeks duration) and chronic cough (more than 3 weeks duration)[3]. Patients with acute cough, particularly when accompanied by a febrile illness or productive cough, will typically have a chest x-ray to identify consolidation indicating pneumonia (Figure 3). In rare cases, the radiograph may demonstrate an unpleasant surprise in the form of a pulmonary mass (Figure 4), in which case the patient may still have pneumonia which has developed secondary to an obstruction "upstream" from the mass. These patients typically require CT for further characterization of the mass and associated hilar and mediastinal nodes and the remainder of the pulmonary parenchyma for synchronous independent tumors and metastases.

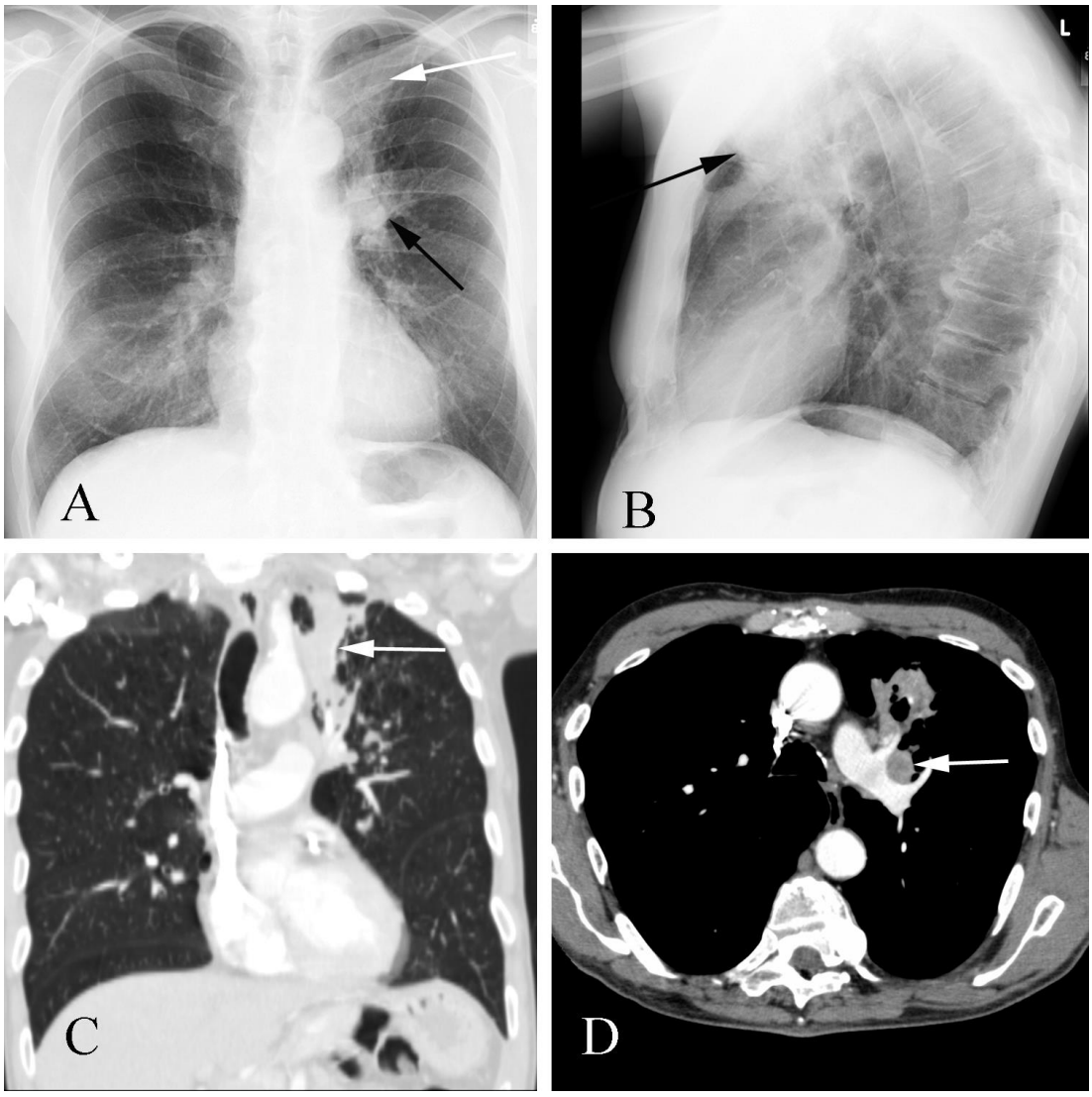

Figure 4. Lung cancer in a 68 year old man with hemoptysis. A. PA chest shows consolidation of the left upper lobe (white arrow) and left hilar fullness (black arrow). B. Lateral exam confirms abnormal opacity in the so-called "anterior clear space", anterior to the trachea. C. Coronal reformatted CT shows the area of consolidation in the left upper lobe corresponding to the plain film findings. D. Axial CT shows lymphadenopathy along the left pulmonary artery (white arrow) as well as lung consolidation. Lung cancer was subsequently diagnosed.

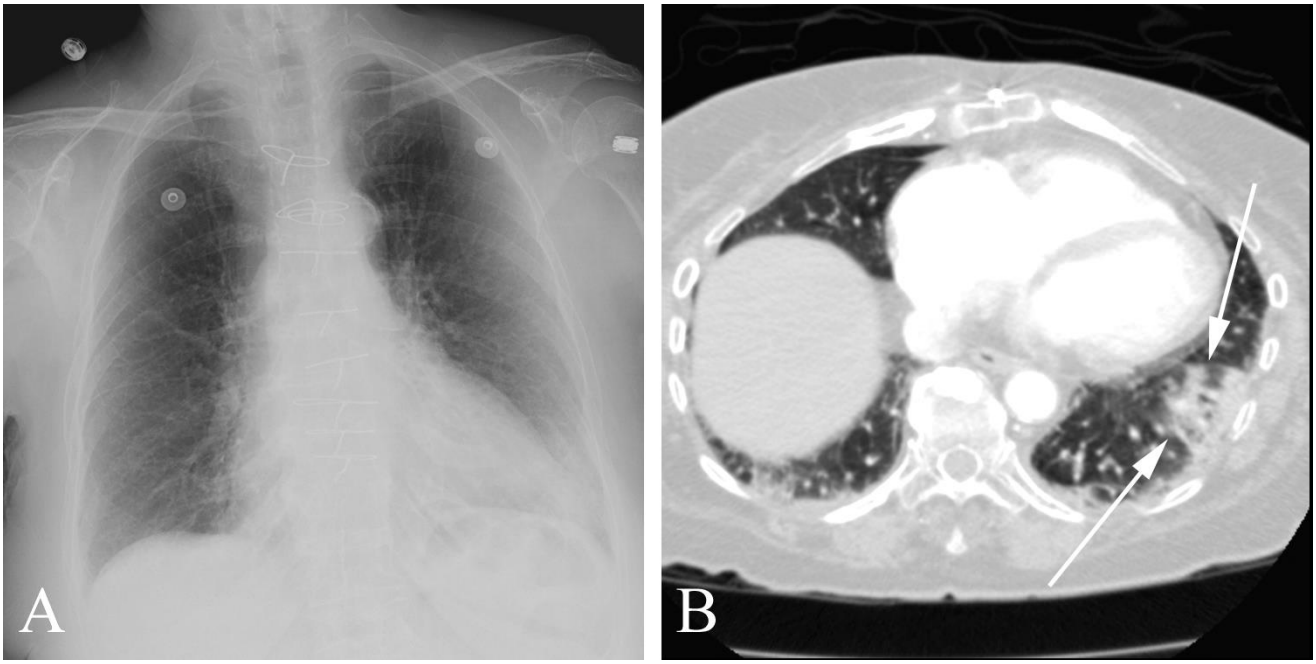

Figure 5. Radiographically occult pneumonia in a 78 year old woman with cough, syncope, and weakness. A. PA chest obtained at the time of the illness shows no obvious consolidation. B. Chest CT performed to exclude pulmonary embolism shows obvious opacity in the left lung base (arrows). There were no pulmonary emboli, and the patient's symptoms resolved and her elevated white blood cell count returned to normal with antibiotic treatment.

Patients with pneumonia occasionally have normal chest radiographs early in the course of the disease, or when dehydrated or immunocompromised[4] (Figure 5). CT will usually demonstrate abnormal opacity of the lung parenchyma in such cases and can be performed if there is a quandary about whether or not to treat the patient or if the diagnosis of pneumonia needs imaging documentation.

In patients with acute cough, several "red flags" should provoke earlier ordering of both the chest x-ray and the subsequent CT. Red flags include symptoms such as fever, sweats or chills, unintentional weight loss, hemoptysis, and dyspnea, which suggest underlying infection, tumor, and/or pulmonary embolism[5]. Note that while many smokers do not seek medical attention for their cough, if a smoker does come in for evaluation of cough the most important feature is whether there has been any change in the character of the cough: chronic, unchanged cough likely represents chronic bronchitis whereas a changed cough is worrisome for the development of a malignancy[3].

In patients with chronic cough, a chest x-ray will typically be obtained although it will usually be normal. This follows because most cases of chronic cough are secondary to post-nasal drip[2], gastroesophageal reflux disease, and asthma (especially variant asthma), and these diseases either produce no chest x-ray findings or subtle nonspecific findings[6]. Diagnostic algorithms typically call for a careful review of systems in patients with chronic cough to elicit any subtle history of these diseases, with further diagnostic testing in those cases where there are suggestive symptoms. Suspected post-nasal drip may be further evaluated with sinus films, suspected GERD with pH monitoring of the esophagus (*not* endoscopy or a barium esophagram), and suspected asthma with spirometry and metacholine challenge.

In those patients with no features to suggest one of these three diseases, the patients still probably has

[2] More recent texts often use "upper airway cough syndrome" rather the term "post-nasal drip".

one of the three diseases but simply are not clinically manifesting any symptoms other than cough. In this case, the options include either testing for the three diseases as noted above, or treating the patient as if they had one of the diseases and noting the response, with the assumption that eradication of the cough with treatment for the disease (nasal glucocorticoids and/or antihistamine-decongestant combination for post-nasal drip, H2 blockers for GERD, bronchodilators for asthma[7]) proves that the patient has the disease.

At some point in this evaluation process – usually after all else has failed, although perhaps earlier if there is an abnormal chest x-ray – a CT may be obtained. Unfortunately, such CT studies provide clinically meaningful information in a minority of cases[1]. Helpful findings which may be seen include bronchiectasis, unsuspected tumor, and interstitial lung disease. Regarding interstitial lung disease, volumes have been written regarding the radiographic appearances of the literally dozens of diseases that fit into this category, and from a radiologist's viewpoint the best summary I can offer is that the CT findings are almost always nonspecific. While combinations of imaging features and clinical findings allow formulation of a reasonable differential diagnosis, lung biopsy is nearly always required to secure a specific diagnosis.

In some 10 to 25 percent of patients, despite all diagnostic testing listed here, the cause of cough may remain unclear[3].

DYSPNEA

The term "dyspnea" indicates unpleasant or uncomfortable breathing and for the purposes of discussion here will be assumed to include patients with shortness of breath. Dyspnea, like cough, is a nonspecific symptom associated with many diseases, and the usual decisions to make in imaging are when to order a chest x-ray and when to proceed to a chest CT. "Red flags" prompt earlier ordering of chest CT exams. As with cough, red flags include symptoms such as fever, sweats or chills, unintentional weight loss, and hemoptysis, which suggest underlying infection, tumor, and pulmonary

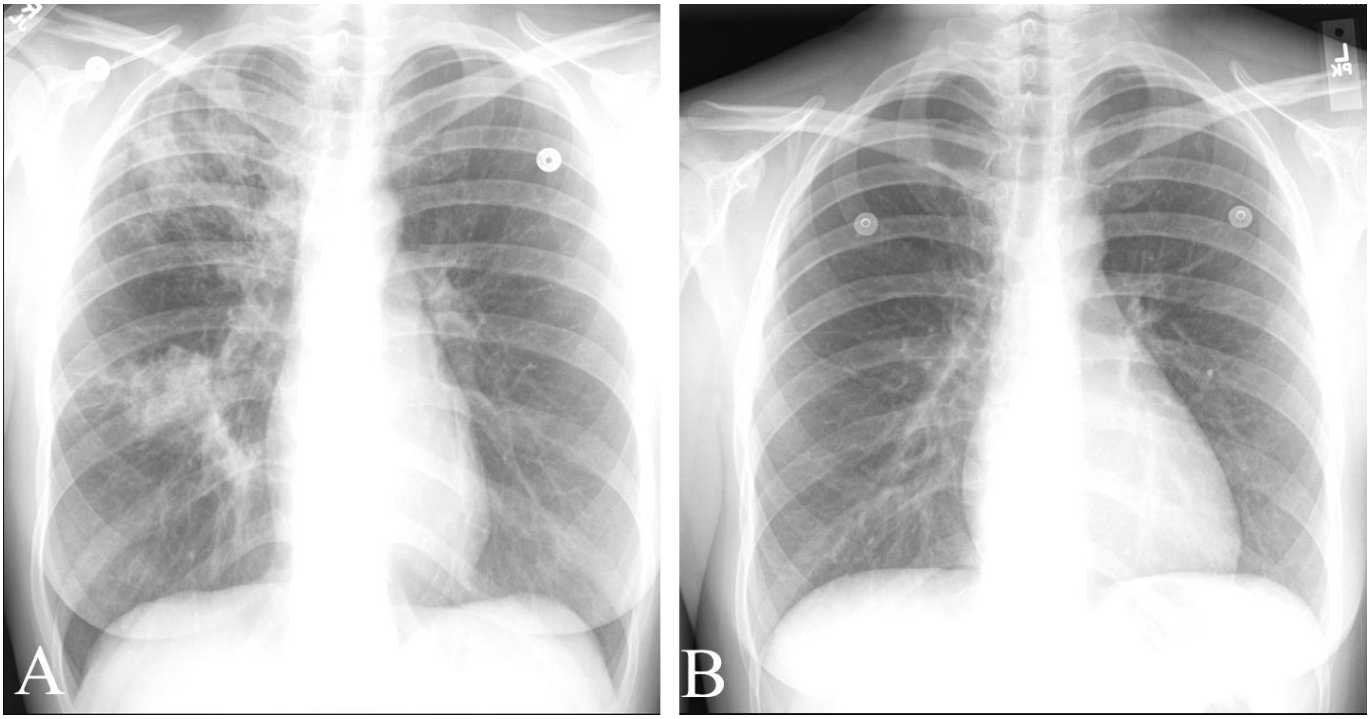

Figure 6. Pneumonia in a 33 year old woman with dyspnea. A. PA chest obtained at the time of the illness shows extensive consolidation of the right lung. B. PA chest obtained following the illness shows clearing of the pneumonia.

embolism[5]. Many patients with pneumonia present with dyspnea rather than cough (Figure 6).

Severe, acute onset dyspnea, particularly when associated with chest pain, may herald one of two critical diagnoses: pneumothorax and pulmonary embolism. Chest x-rays should be obtained immediately in any patient with severe acute onset dyspnea, and while a plain film will demonstrate pneumothorax (Figure 1), contrast-enhanced CT is necessary to diagnose pulmonary embolism (Figure 7). In cases of suspected pulmonary embolism, measurement of D-dimer may be helpful as an elevated level suggests pulmonary embolism and should prompt urgent CT examination.

Dyspnea caused by congestive heart failure (Figure 8) may be further evaluated with echocardiography. Typically, these patients do not need to undergo CT scanning.

Imaging results in patients with dyspnea are less likely to be normal than imaging results in patients with chronic cough. The imaging studies will often demonstrate at least some cause of the symptoms, even if the exact diagnosis is elusive and requires further testing. Nonetheless, many diseases that produce dyspnea demonstrate either few CT findings (asthma) or nonspecific findings (chronic obstructive pulmonary disease or COPD).

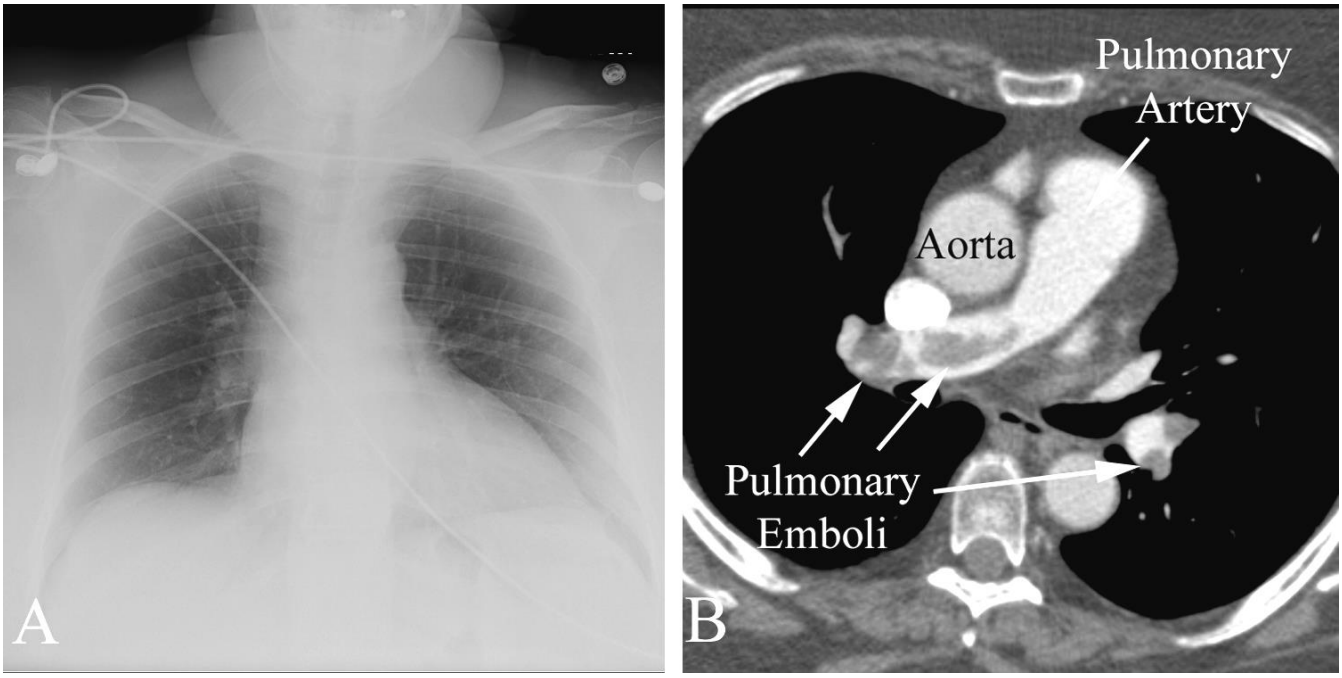

Figure 7. CT examination demonstrating pulmonary embolism in a 74 year old woman with acute onset of shortness of breath. A. Portable chest plain film examination was normal, as is often the case with pulmonary embolism. B. Chest CT examination demonstrates multiple filling defects within the pulmonary arteries, diagnostic of pulmonary embolism.

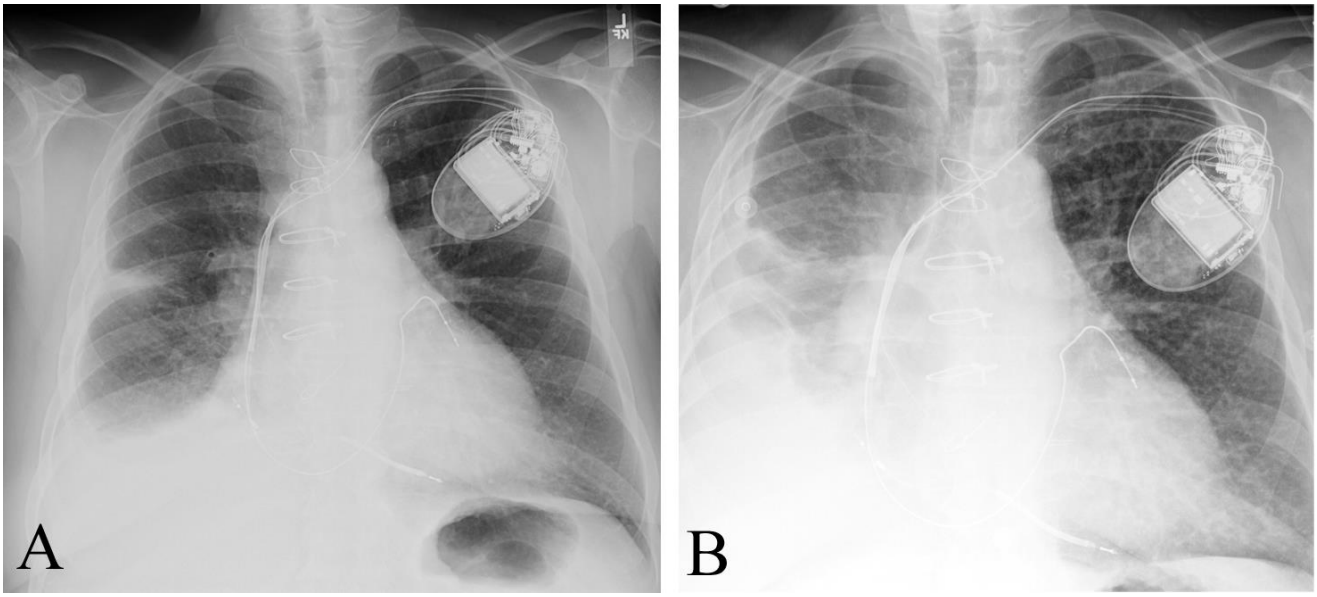

Figure 8. Worsening of congestive heart failure in a 66 year old woman with shortness of breath and a 15 pound weight gain over the past week. A. Baseline PA chest radiograph shows cardiomegaly, a pacer, and a right pleural effusion. B. PA chest at the time of weight gain shows a larger pleural effusion, increased size of pulmonary vessels from pulmonary venous hypertension, and increased lung density from pulmonary edema.

PULMONARY NODULES

Pulmonary nodules and masses may be symptomatic, discovered as part of a search for metastatic deposit, or discovered as an incidental finding. Symptomatic lesions, for example a chest mass that has involved the chest wall or which has undergone necrosis causing chest pain, requires prompt work-up (Figure 9). CT with possible CT-guided biopsy may be appropriate, with likely early referral to a chest surgeon, oncologist, or both for treatment.

In patients with known malignancy undergoing screening for metastatic deposit, new chest masses are usually malignant. Note that in this regard, oncologists frequently monitor cancer patients with CT (rather than simply plain films) because of CT's increased sensitivity (Figure 10). Lesions incidentally discovered on chest x-ray typically undergo CT as the next step in evaluation, unless there is an old chest x-ray establishing stability for at least two years (see below). The CT may demonstrate that an apparent pulmonary nodule on the chest x-ray represents a benign abnormality (as noted below).

With incidentally discovered lesions found at CT scanning, the work-up depends upon the size of the lesion and risk status of the patient (Table) in a way that is difficult to memorize. In general, the goal is to identify and remove malignancies as early as possible, while not routinely removing benign lesions. This is very difficult to accomplish. One way to think about the process is to attempt to establish whether the lesion has any features which unequivocally demonstrate that it is benign and therefore may be ignored. These include:

1. Typical benign pattern of calcification. While not all calcification within a pulmonary nodule indicates a benign lesion, most does, especially if the calcification is uniform (Figure 11) or "popcorn" like in appearance.
2. The lesion contains fat. Fat, though far less frequently encountered than calcification, indicates a benign lesion. A uniformly fatty lesion is a lipoma, whereas one with a small amount of fat distributed among other tissue is a hamartoma (Figure 12).
3. The lesion represents a typical arteriovenous malformation. These lesions demonstrate conspicuous feeding and draining vessels and obvious enhancement with contrast. If

large and symptomatic, these lesions may be embolized (Figure 9).

4. The lesion shows stability on sequential scans. The typical cut-off for assuming that stability equates with a benign lesion is two years, although this is somewhat arbitrary and controversial[8]. There are, of course, two ways to establish stability: to look backward at old exams, and to sequentially follow the patient forward. As for looking backward, questioning the patient for possible prior studies of any kind may be helpful – for example, nodules at the lung base may be visible on prior CT scans of the abdomen (Figure 13). If no old films are available, then one must decide whether to:

   A. Do nothing. This is an option if the lesion measures less than 5 mm and the patient has no risk factors. This follows from the fact that such small pulmonary nodules are common (seen in up to 50% of patients[7]) but almost always benign (less than 1% malignant[7]).
   B. Follow the lesion with sequential CT studies (Figure 14). This is usually done with lesions of intermediate size particularly absent patient risk factors (see Table).
   C. Proceed to PET imaging. This is a good choice for lesions at least 8 – 10 mm in size. Lesions that are hypermetabolic (Figure 15) are nearly always malignant and require removal, whereas those that are not hypermetabolic can be followed with sequential CT studies. Nonhypermetabolic lesions should *not* be ignored unless they have been proven stable for at least two years, as there are a few cancers (especially bronchioalveolar carcinoma) which are not hypermetabolic. So called "ground glass" lesions (named because they have a density which is greater than normal lung tissue, but are not so dense that they obscure vessels, on CT study) are more worrisome and biopsy or prolonged (3-5 year) follow-up of these lesions should be performed.
   D. Any increase in lesion size is a cause for concern and unless there is some overwhelming reason not to do so, lesions showing an increased size on sequential studies should probably be removed.

Size	Further Evaluation	
	No smoking, asbestos exposure, or known malignancy	Smoking, asbestos exposure, or known malignancy
< 4 mm	None	CT @ 12 months; if no change, stop
>4 to 6 mm	CT @ 12 months; if no change, stop	CT @ 6 months; if no change repeat at 24 months
> 6 to 8 mm	CT @ 6 months; if no change repeat at 24 months	CT @ 3 months; if no change @ 9 months; if no change @ 24 months
> 8 mm	CT @ 3, 9, and 24 months or PET or biopsy	CT @ 3, 9, and 24 months or PET or biopsy

Table: Recommended follow-up for incidentally discovered nodules discovered at non-screening CT for patients 35 years or older. Adapted from MacMahon H et al. Guidelines for management of small pulmonary nodules detected on CT scans: a statement from the Fleischner Society, Radiology 2005;237:395-400.

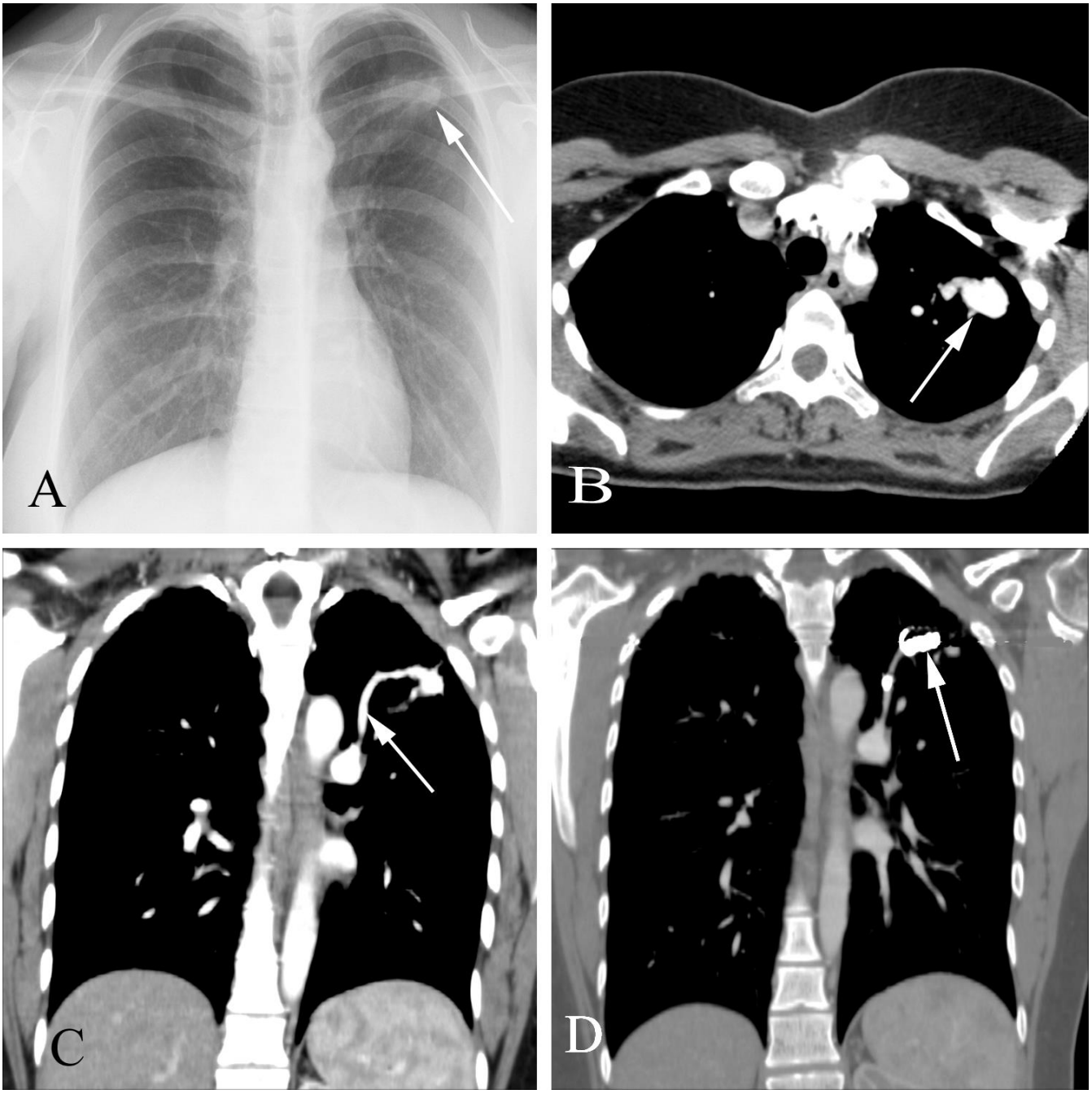

Figure 9. Arteriovenous malformation in a 34 year old woman with chest pain. A. Chest radiograph shows a mass in the left upper lobe. B. Axial contrast-enhanced CT shows an intensely enhancing lesion. C. Coronal reformatted contrast enhanced CT shows a feeding vessel characteristic of an arteriovenous malformation. The patient underwent embolization therapy. D. Follow-up coronal CT filmed at bone windows shows embolic material within the lesion. The patient's pain remitted following treatment.

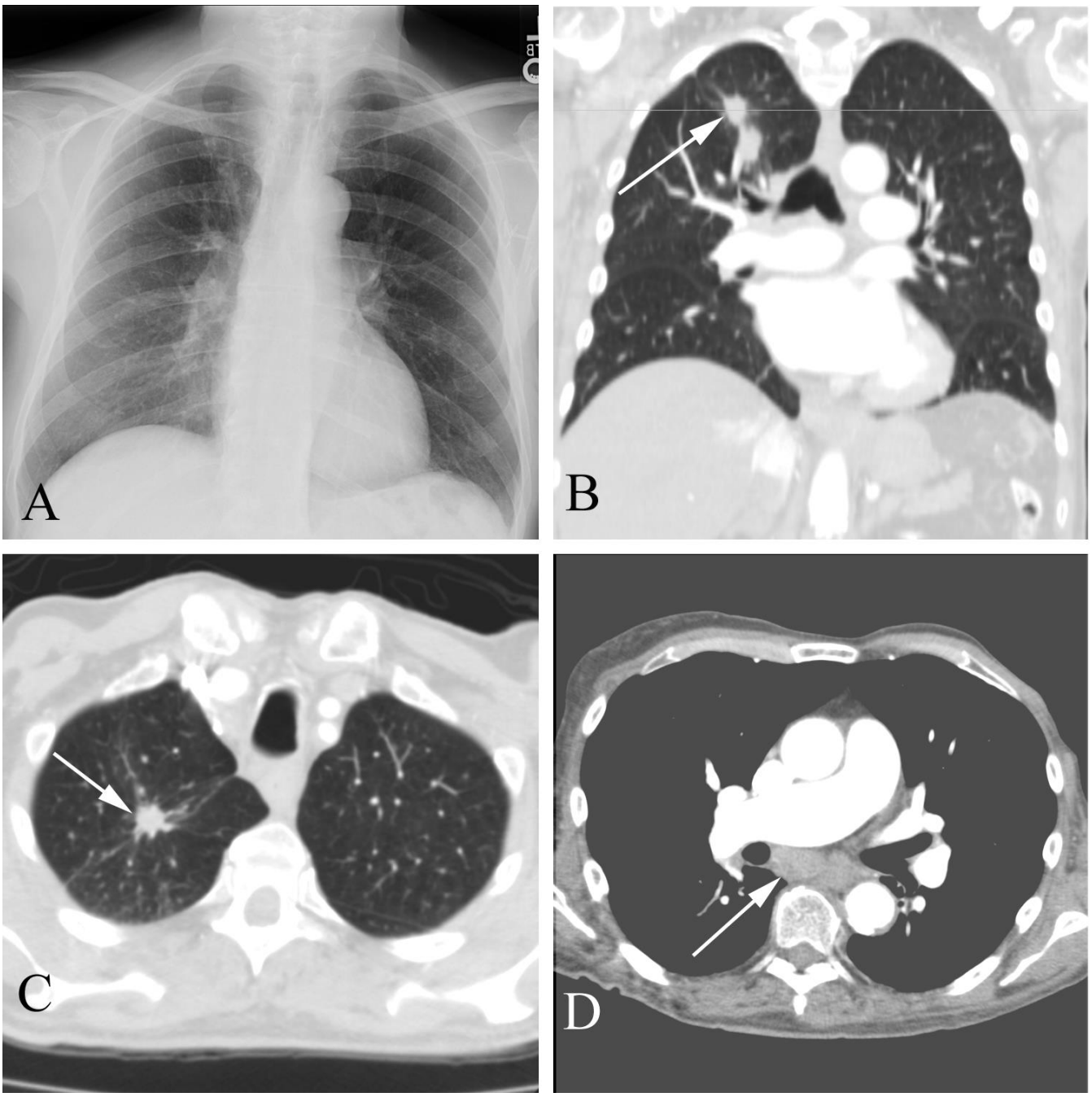

Figure 10. Metastatic disease in a 74 year old man with both breast and prostate cancer (!), with CT demonstrating lesions not seen on plain film examination. A. PA upright chest shows subtle, easily missed opacity in the medial right upper lung. B. Coronal reconstruction CT clearly shows a spiculated mass in the right upper lobe (arrow) along with an additional, more inferior lesion. Axial localizing line is at the plane of the axial slice in C. C. Axial CT also shows a spiculated mass. D. More inferior image shows bulky lymphadenopathy from metastatic deposit posterior to the right pulmonary artery (arrow).

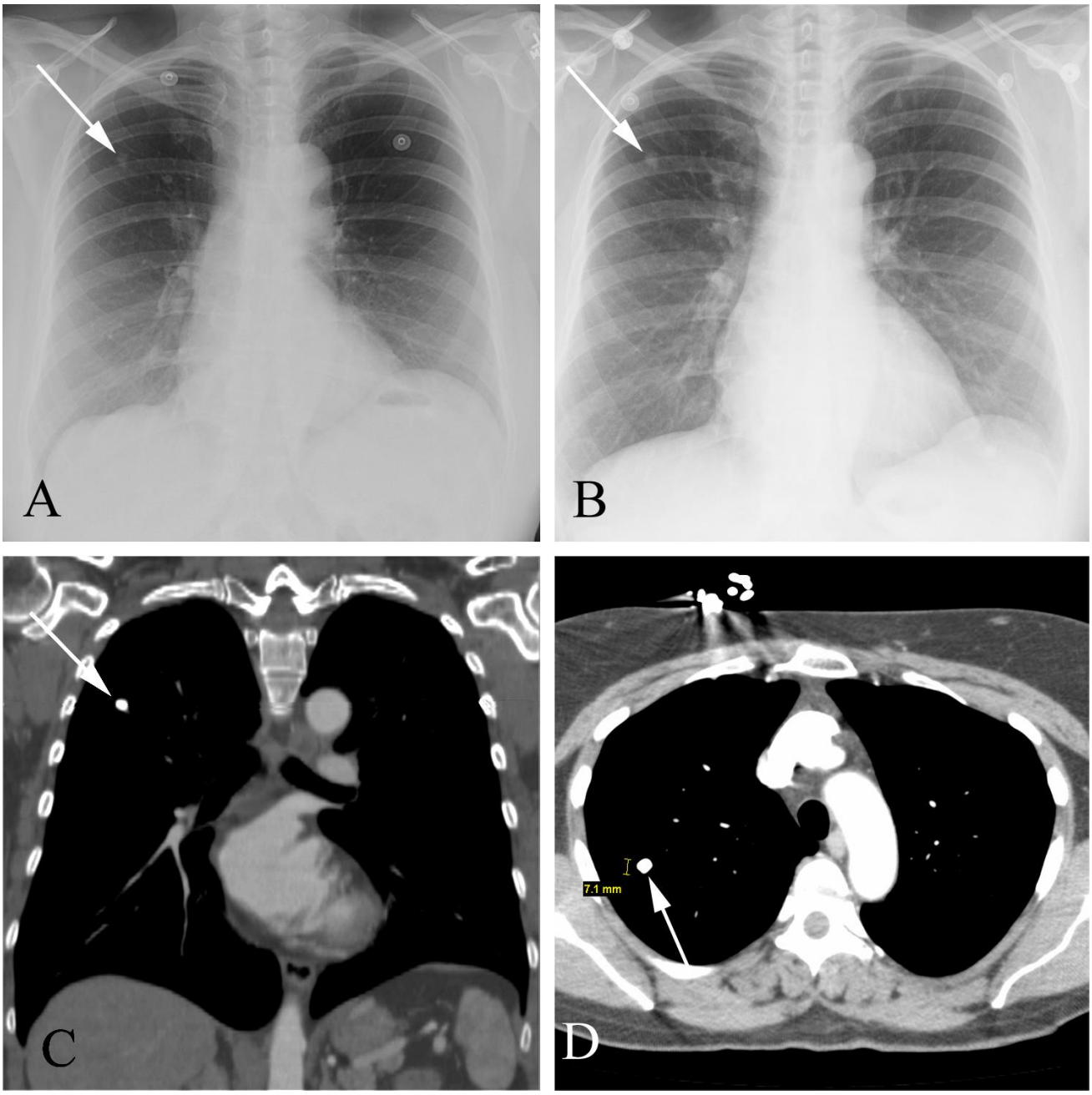

Figure 11. Benign granuloma in a 67 year old woman. A. Baseline PA plain film examination shows a right upper lobe pulmonary nodule. B. Later PA plain film obtained for pain shows an apparent increase in size of the lesion. C. Coronal reconstruction shows a densely calcified lesion (arrow) at the location of the nodule on the chest radiograph. The horizontal localizing line is at the plane of the axial slice. D. Axial CT also shows a densely calcified lesion. No further work-up was required, as the lesion has a completely benign appearance.

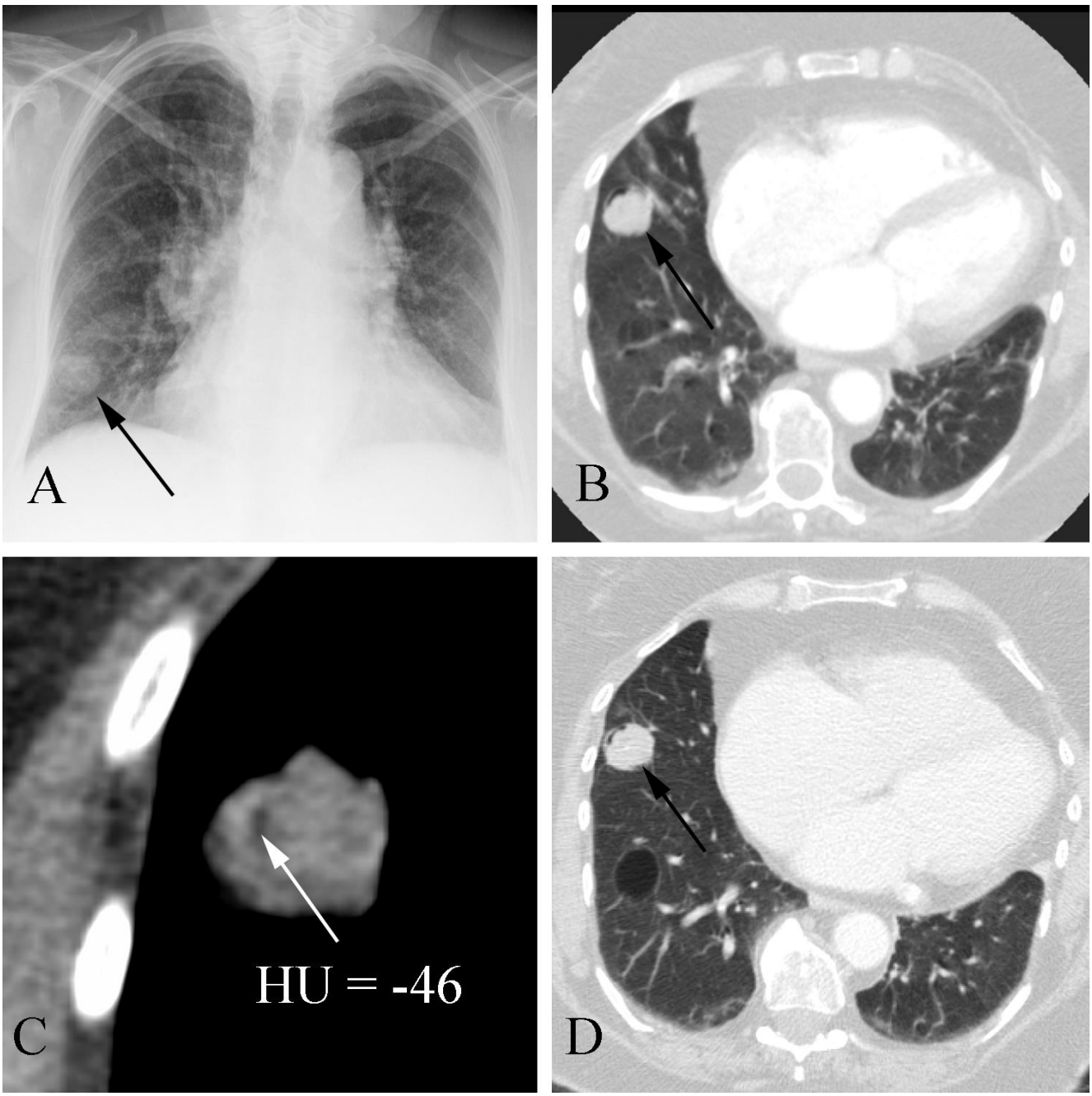

Figure 12. Hamartoma in a 74 year old woman with an incidentally discovered chest lesion. A. Chest radiograph obtained for shortness of breath demonstrates a lesion in the right middle lobe (arrow). B. Axial CT study filmed on pulmonary windows shows a mass (arrow). C. Magnified CT shows fat within the lesion with negative Hounsfield units indicating fat. D. Subsequent CT of the abdomen performed two years later for another reason shows that the lesion is stable.

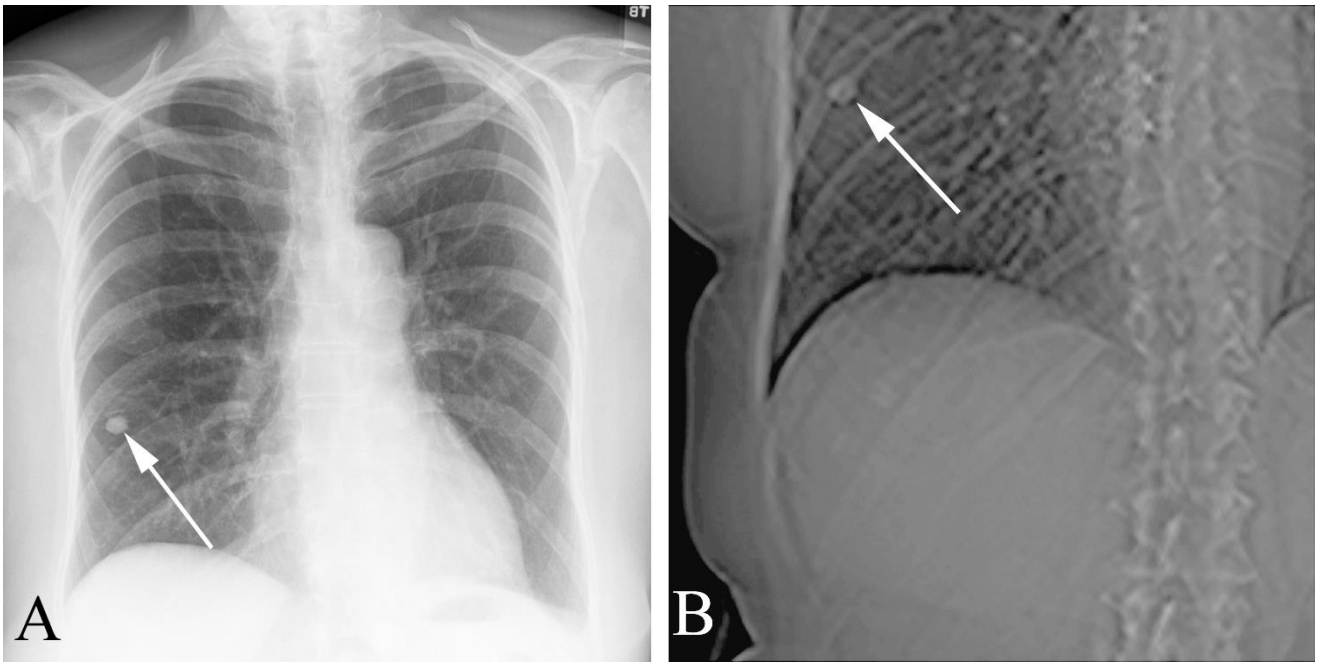

Figure 13. Chronic granulomatous disease in a 72 year old woman with an incidentally discovered pulmonary nodule. A. Chest radiograph of a 72 year old woman with leukocytosis following a total knee replacement shows a right pulmonary nodule (arrow). B. The patient had no old chest radiographs, but the scout film from a prior abdominal CT showed the nodule unchanged from three years earlier (arrow).

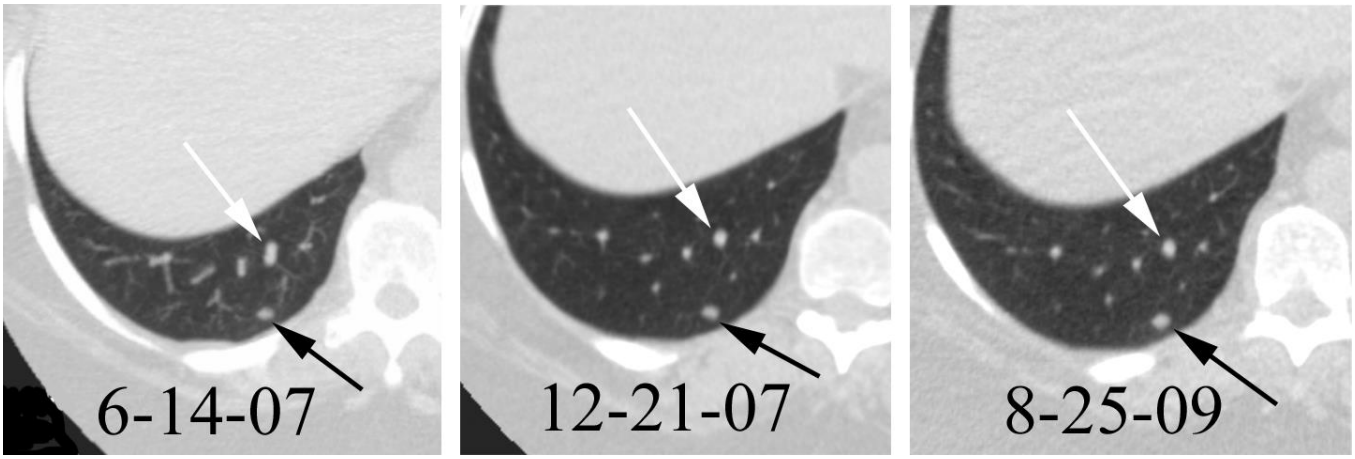

Figure 14. Chronic granulomatous disease in a 61 year old woman with incidentally discovered pulmonary nodules. Sequential studies show a small, stable nodule in the right lung base (black arrow). Note that on non-MIP studies, the nodules resemble vessels (white arrow). This is the same study as Figure 2, above, demonstrating the value of MIPs.

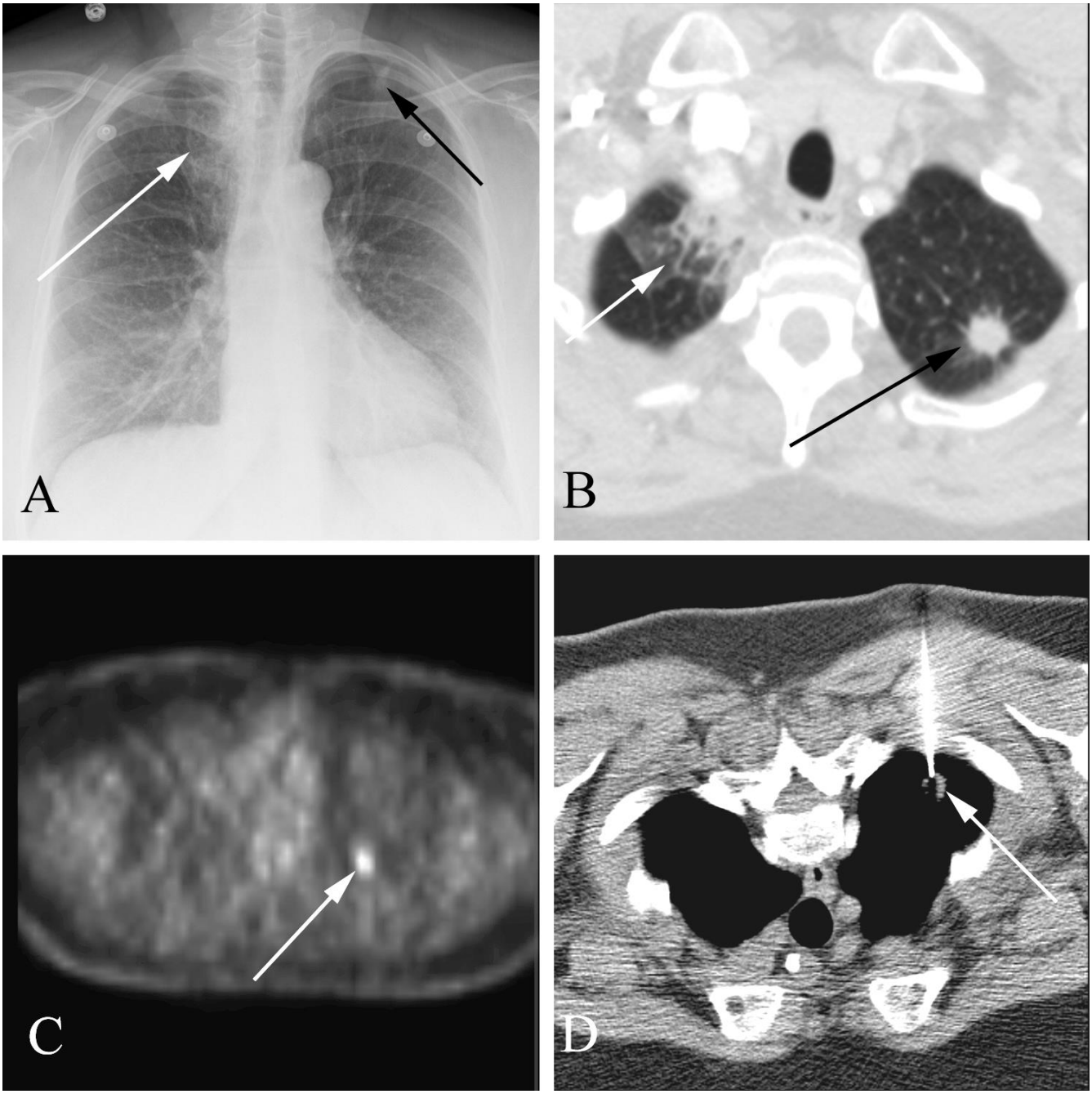

Figure 15. Adenocarcinoma in a 63 year old woman with pneumonia. A. Chest radiograph shows right upper lobe pneumonia (white arrow) and a contralateral left upper lobe pulmonary nodule (black arrow). B. CT study shows the pneumonia (white arrow) and the pulmonary nodule (black arrow). C. PET scan shows hypermetabolism at the location of the nodule (white arrow). D. A CT directed biopsy with the needle in the lesion (arrow). Adenocarcinoma was found at pathology.

SUMMARY

Most patients with cough and dyspnea will get a chest x-ray, and the main decision is whether and when to order a CT study. Common causes of cough are often treated empirically prior to proceeding to a chest CT. For patients with dyspnea, an urgent CT is performed if there are red flags for pulmonary embolism. Pulmonary nodule work-up is often performed with chest x-ray followed by CT (if no old films are available to demonstrate nodule stability), with biopsy/excision, sequential CT, or PET-CT depending on the circumstances of the patient and the size of the lesion.

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3 Silvestri RC, Weinberger SE. Evaluation of subacute and chronic cough in adults. UpToDate, accessed 9/14/09.

4 Hueston WJ. Respiratory problems. Chapter 26 in South-Paul JE, Matheny SC, Lewis EL (Editors), Current Diagnosis and Treatment in Family Medicine (2nd edition), McGraw Hill Medical, New York, 2008.

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7 Weinberger SE, Silvestri RC. Treatment of subacute and chronic cough in adults. UpToDate, accessed 9/14/09.

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Chest Pain

Donald L. Renfrew, MD

This chapter covers three main points designed to help you order the correct first test when evaluating patients with chest pain:

1. Virtually everyone with chest pain gets a chest x-ray.
2. Patients with suspected pulmonary embolism need emergent chest CT with contrast.
3. Imaging in suspected coronary artery syndrome depends on the clinical condition of the patient and risk assessment.

IMAGING OPTIONS IN CHEST PAIN

Primary care practitioners (PCPs) may order any of several exams in the evaluation of chest pain, including plain films, CT scans, nuclear medicine scans, magnetic resonance imaging scans, and ultrasound studies.

Plain films

See page 133 for a description of plain films of the chest. In addition to the chest x-ray (CXR), the PCP may also order rib detail films, which provide greater detail of ribs and demonstrate fractures that may be missed on plain films (Figure 1).

Computed Tomography

See page 131 for a description of computed tomography (CT) performed for cough and dyspnea. Typically, a standard, contrast-enhanced exam is obtained for these indications. As an alternative to the standard contrast-enhanced exam, CT may be timed to optimize enhancement of the pulmonary arterial tree. This is called a "chest computed tomographic angiogram" (Chest CTA), and the data from this technique is -processed to create specific views of the pulmonary arterial tree, usually in an oblique plane optimized for visualization of the main pulmonary arteries with maximum intensity projections (MIPs) (Figure 2). CT may also be used to measure the coronary artery calcium content, called "coronary artery calcification scoring" (CACS). For this study, the patient is hooked up to an EKG and a noncontrast CT scan is obtained with the data acquisition coordinated with the heart beat to minimize motion. Finally, more advanced equipment (at least 64-slice, by current recommendations), can acquire a "coronary artery computed tomographic angiogram" (CCTA), which is a map of the coronary arterial tree obtained with EKG gating and intravenous contrast (Figure 3). For 64-slice scanners, the heart rate must be below 70; many patients will require beta-blockers to achieve this low rate. CT scanners with more slices and faster imaging times can obtain diagnostic images with higher heart rates. Note that, at present, a chest CTA and a CCTA (of the coronary arteries) cannot be performed simultaneously with the same bolus of contrast material, although advances in scanning

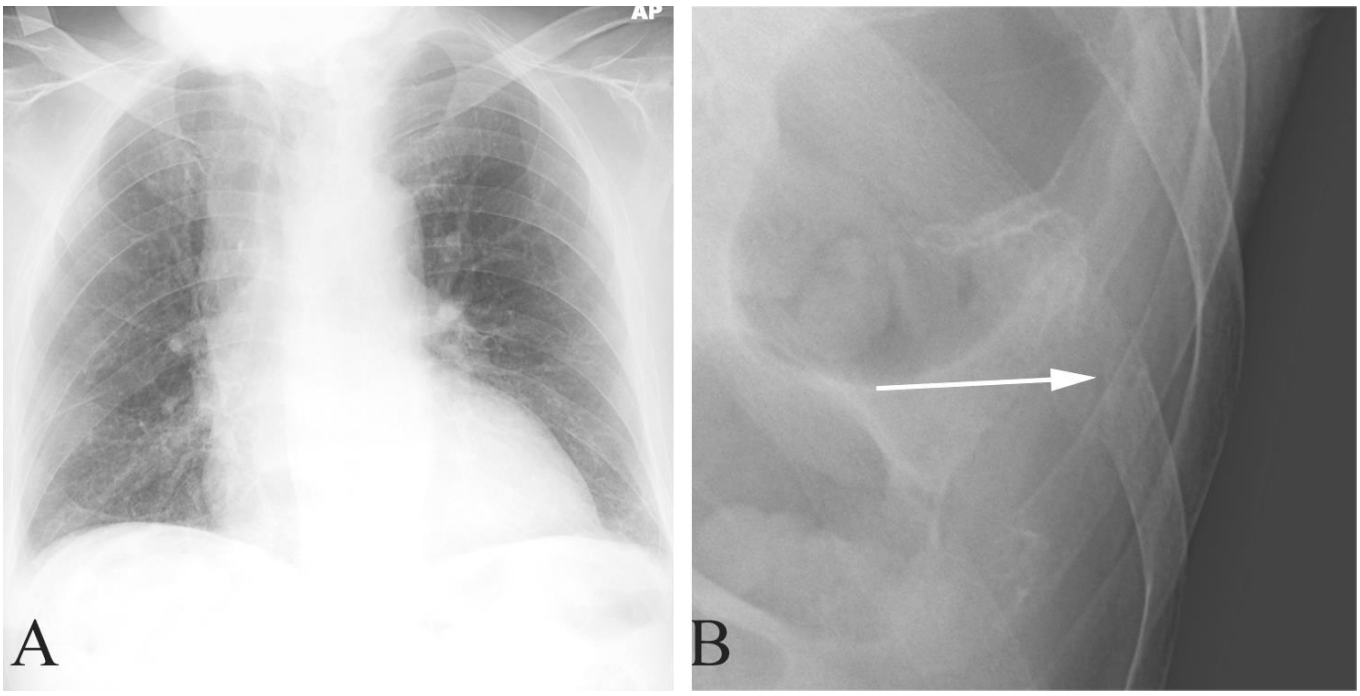

Figure 1. Rib fracture in an 86 year old with persistent chest pain following trauma, demonstrating the value of the rib detail series in demonstrating rib fractures. A. AP CXR (left) shows no abnormality. B. Rib detail film (right) shows a minimally displaced left 10th rib fracture, at the location of the patient's pain.

technology will probably soon allow the "rule-out triple" exam whereby a single study can evaluate for pulmonary embolism, aortic arch dissection, and coronary arterial disease. These techniques will be discussed in context below.

Nuclear medicine studies

Primary care providers may order ventilation-perfusion lung scans for patients suspected to have a pulmonary embolism who are allergic to contrast or have renal insufficiency. This study relies on the distribution of two different radioactively labeled substances: one is an aerosol used to evaluate ventilation, and the other an intravenous substance designed to be filtered at the smallest level of pulmonary vasculature and therefore to evaluate perfusion (Figure 4).

Nuclear medicine heart studies include infarct-avid imaging (now largely supplanted by serial enzyme evaluation) and myocardial perfusion studies, which may be performed at the time of the supposed cardiac event in equivocal cases[1], or, more typically, with a stress test in patients who have clinical features suspicious for coronary artery syndrome. For myocardial perfusion studies, a radioactively labeled material (typically tetrofosmin labeled with 99m-Technitium) is injected intravenously when the patient is at rest, and images of the heart are obtained. Later in the same day, or on a different day, additional radioactively labeled material is injected during cardiac loading (caused either by exercise or drugs). Images are obtained in both cases. Either planar or single photon emission computed tomographic (SPECT) images may be obtained; the latter are preferred as the test has higher sensitivity and the same specificity. Normal myocardium shows uniform uptake of radiotracer, infarcted areas show decreased uptake on both the rest and stress studies, whereas areas of reversible ischemia show normal activity at rest but decreased activity following exercise (see below).

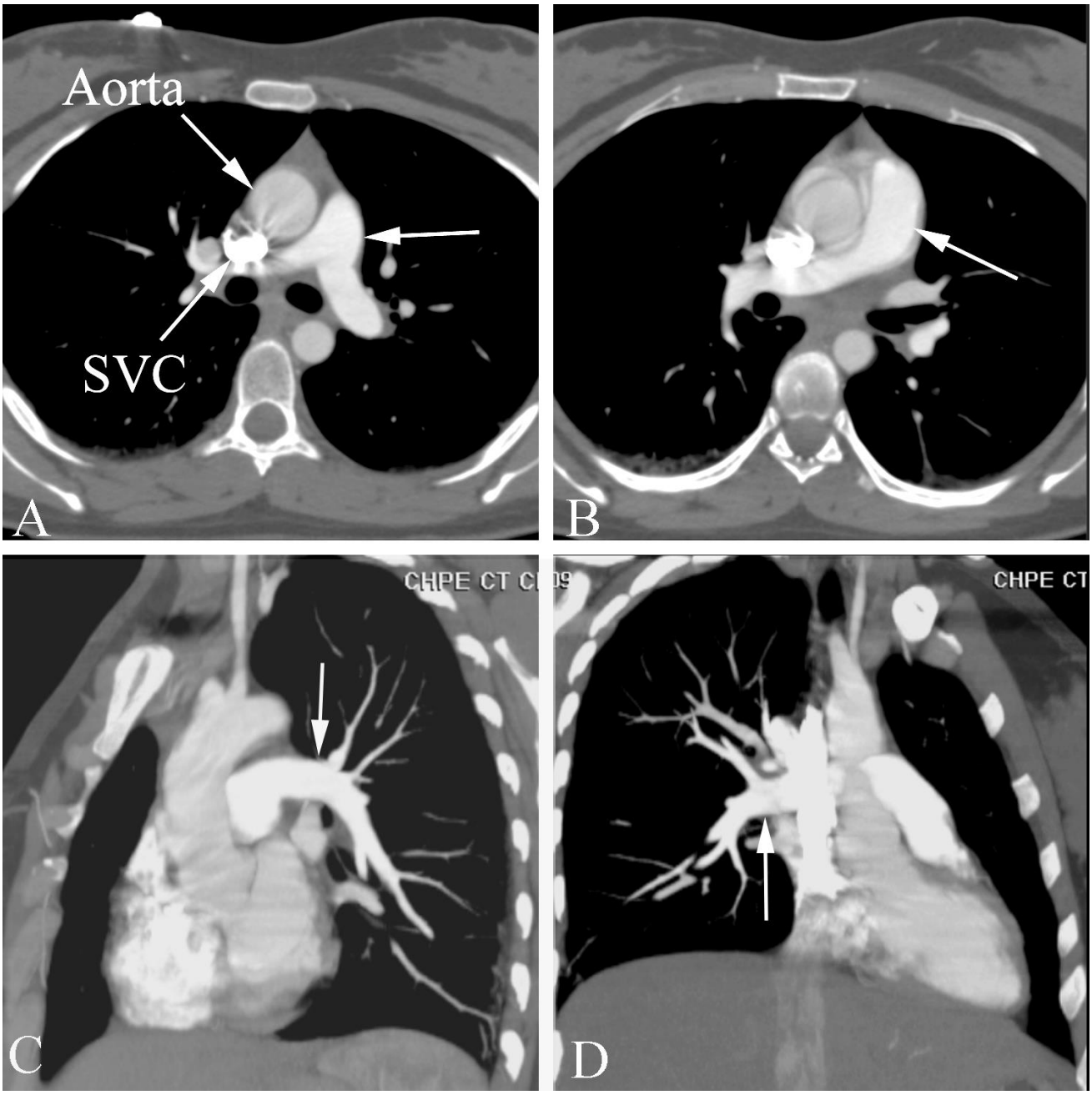

Figure 2. Normal chest CT angiogram in a 28 year old woman with acute chest pain. A. Axial contrast-enhanced CT study at the level of the main pulmonary artery (arrow) shows a normal appearance of the pulmonary artery. Note dense contrast coming into the heart through the superior vena cava. B. Axial contrast-enhanced CT at a slightly lower level shows that the main pulmonary artery (arrow) is approximately the same size as the ascending aorta. C. Coronal oblique reformatted contrast-enhanced CT study shows the left pulmonary artery (arrow) and branches to be free of filling defects.
D. Coronal oblique reformatted contrast-enhanced CT study shows the right pulmonary artery (arrow) and branches to be free of filling defects as well.

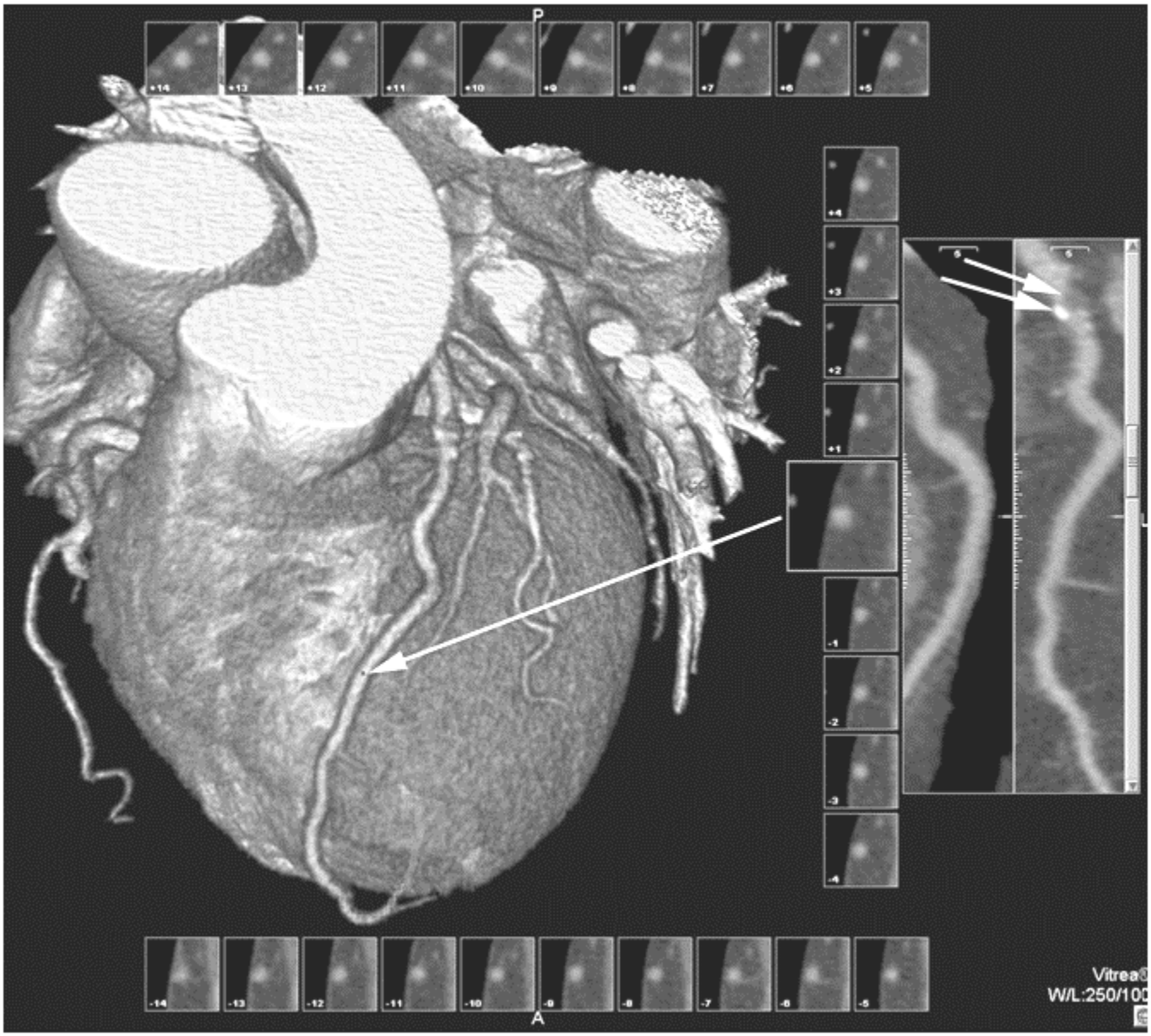

Figure 3. Computed tomographic coronary arteriography in a 52 year old with chest pain. This composite figure demonstrates a reconstructed view of the heart and coronary vessels, obtained following EKG-gated computed tomography. The left anterior descending (LAD) coronary artery is depicted along the surface of the left ventricle, with the long arrow connected to the associated cross-sectional image of the LAD. Images proximal to the level of the arrow are arrayed above the target location, and more distal locations below it. Along the right side of the image are two longitudinal reconstructions (at right angles) of the LAD. Note that in the right-sided image, there is a significant (>50%) stenosis of the proximal coronary artery (double arrow). Case courtesy of Dr. Marc Miller, Radiology Associates of the Fox Valley.

Magnetic resonance imaging

Despite years of intensive development and investigation, chest MRI remains a relatively infrequently performed method of cardiac imaging, with most uses restricted to patients who are allergic to iodine-containing IV contrast. However, this technique continues to evolve, and it is possible that cardiac MR may one day provide a single modality capable of evaluating the coronary arteries, cardiac valves, and myocardium in one exam. At this time, however, the technique is not routinely used in the evaluation of chest pain.

Echocardiography

Cardiologists interpret most echocardiograms. This examination is excellent for evaluation of the cardiac valves (including morphology, stenosis, and insufficiency), pericardium, chamber size, and wall motion, as well as obtaining some information about heart and great vessel pressures. It is the examination of choice for evaluating suspected acute valve insufficiency, for example, in a patient with chest pain and a new murmur. Since echocardiography is largely performed and controlled by cardiologists, it is not covered in this chapter.

CXR IN ALL PATIENTS WITH CHEST PAIN

Imaging of chest pain starts with a CXR. In most cases, a plain film does not reveal anything particularly helpful or diagnostic: Templeton et al[2] found that 23% of plain films obtained in patients with chest pain in the emergency room had an abnormality that influenced therapy, and the likelihood of finding a significant abnormality in a patient undergoing evaluation in an outpatient clinic is likely even less. Perusal of Table 1, a list of causes of chest pain in 300 patients presenting to a clinic with chest pain reveals why most plain films are unremarkable: the commonly encountered causes of chest pain seldom have any plain film manifestations. Nonetheless, there are a few relatively uncommon diseases that may cause chest pain and which have specific abnormalities on chest radiography.

Cause	Percent
Musculoskeletal, including costochondritis	36%
Gastrointestinal	19%
Cardiac	16%
- stable angina	10.5%
- unstable angina or MI	1.5%
- other cardiac	3.8%
Psychiatric	8%
Pulmonary	5%
Other	16%

Table 1. Causes of chest pain in non-emergent patients presenting to Michigan primary care practices. From: Klinkman MS, Stevens D, Gorenflow DW. Episodes of care for chest pain: a preliminary report from MRINET. J Fam Pract 1994; 38:345-352.

Pneumothorax, typically from a ruptured bleb[3], is one disease that plain films can diagnose without equivocation (Figure 5), although CT is better at demonstrating a small pneumothorax (Figure 1, Chapter 10, page 132). Patients with a pneumothorax will typically experience dyspnea in addition to chest pain, and are, of course, far more likely to present to an emergency room than an outpatient clinic. Pneumomediastinum (Figure 6) occurring with or without pneumothorax, and is an additional cause of chest pain which may be diagnosed on a CXR although, like a pneumothorax, the lesion is more easily seen on CT.

Rib fractures (Figure 1) may cause chest pain and while there is sometimes a history of trauma, such fractures may also occur

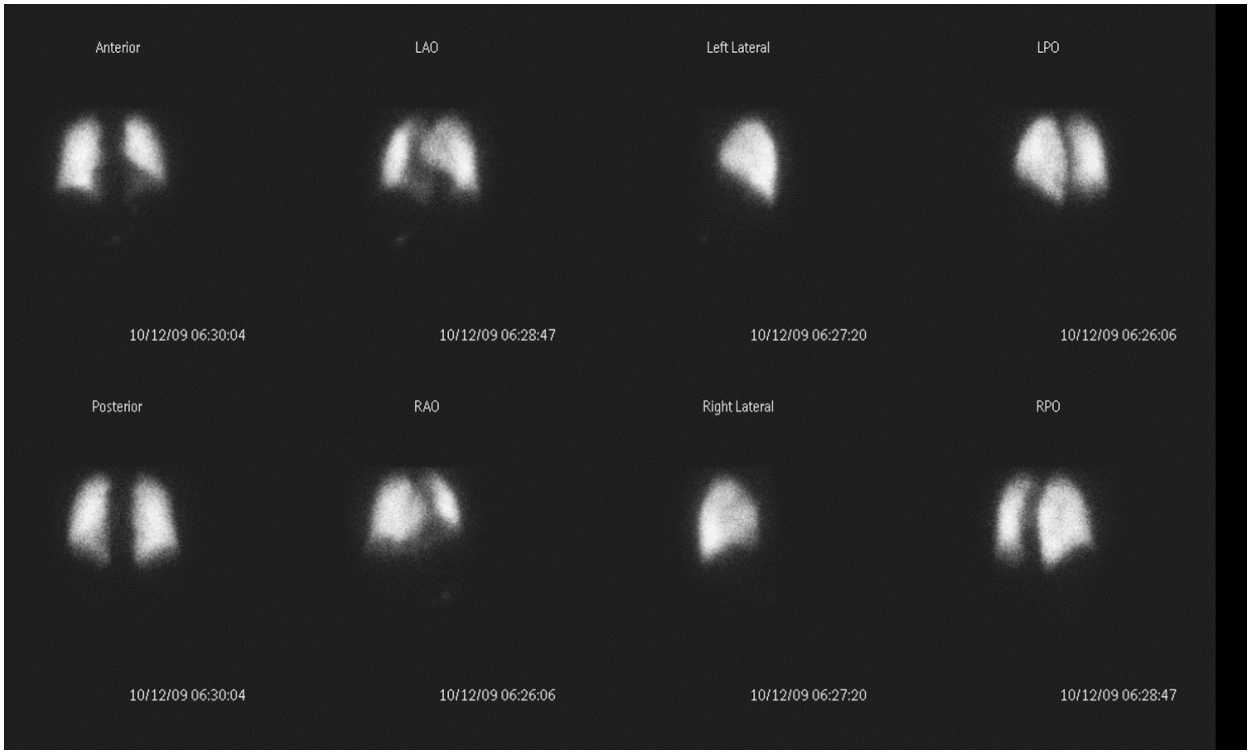

Figure 4. Normal perfusion study in a 70 year old woman with chest pain. The patient had an allergy to intravenous contrast material. The perfusion study shows normal perfusion to both lobes of the lungs on all projections. The ventilation study (not shown) was also normal.

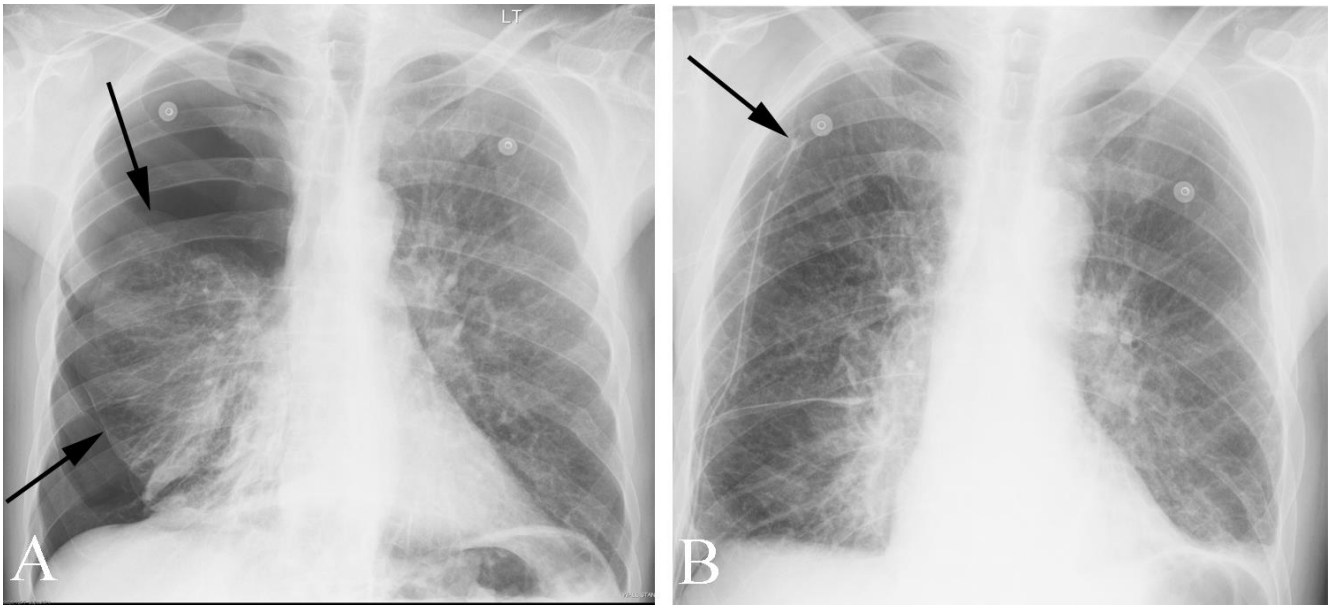

Figure 5. Pneumothorax in a 52 year old man with acute chest pain. A. CXR obtained in the ER shows a right sided pneumothorax (arrow). B. CXR following insertion of a chest tube shows re-expansion of the right lung, with the tube tip (arrow) in the superolateral thorax at about the 4th rib level.

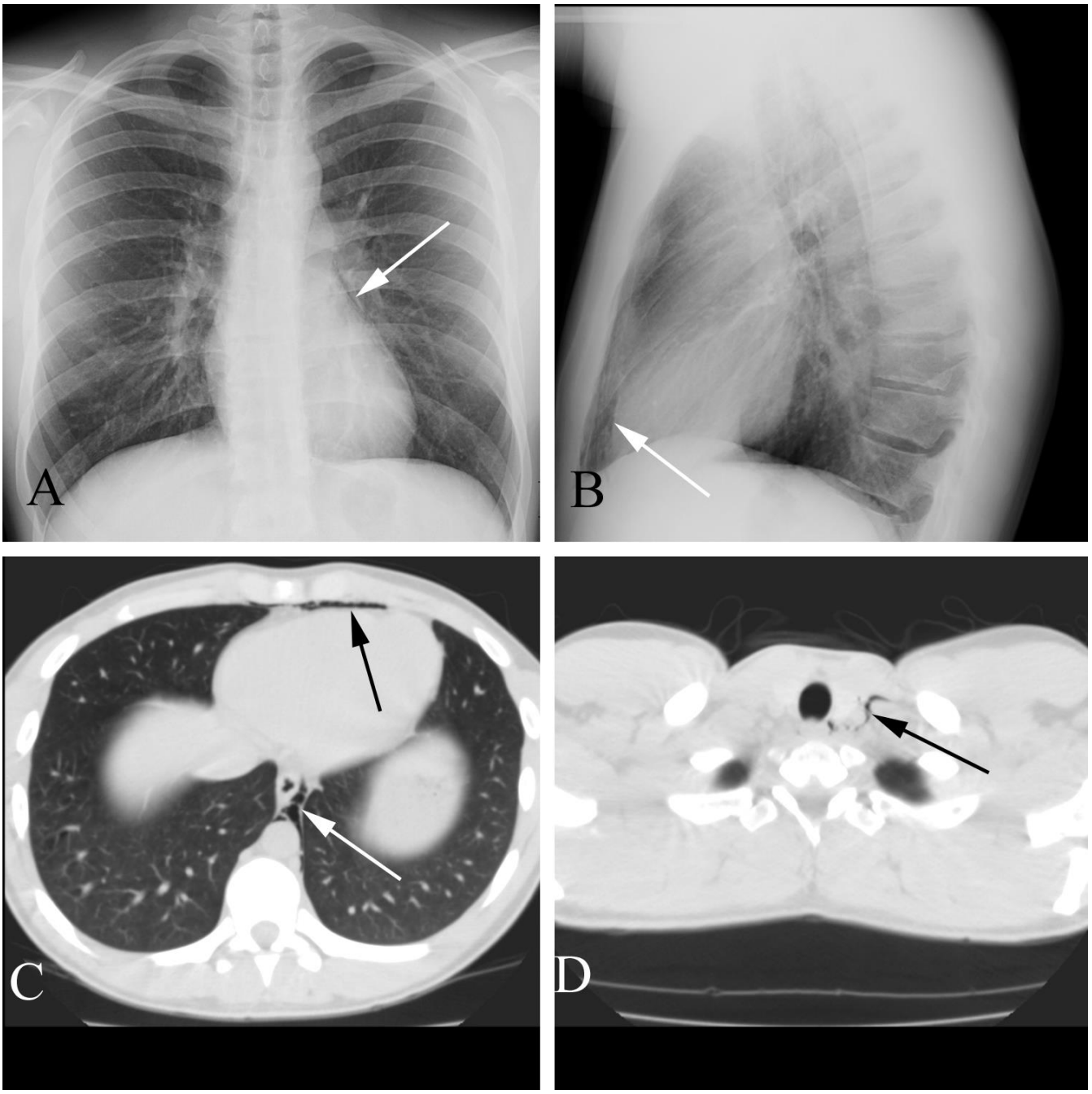

Figure 6. Pneumomediastinum causing chest pain in a 20 year old man. A. PA CXR shows only a subtle hyperlucency bordering the heart (arrow). B. Lateral CXR shows hyperlucency anterior to the heart (arrow). C. CT scan shows air anterior to the heart (black arrow) accounting for the lucency seen on the plain film, and air along the esophagus in the posterior mediastinum (white arrow). D. CT at the level of the thoracic inlet shows air dissecting along the left subclavian vessels (arrow).

secondary to prolonged or violent coughing, or sneezing. Note that, for the most part, rib detail films are more sensitive for the detection of rib fractures than is the routine CXR, but many rib fractures may escape detection because they are minimally displaced or occur at the costochondral junction. If a patient has focal reproducible tenderness of a rib, he probably has a rib fracture, and the important thing to exclude is any associated hemo- or

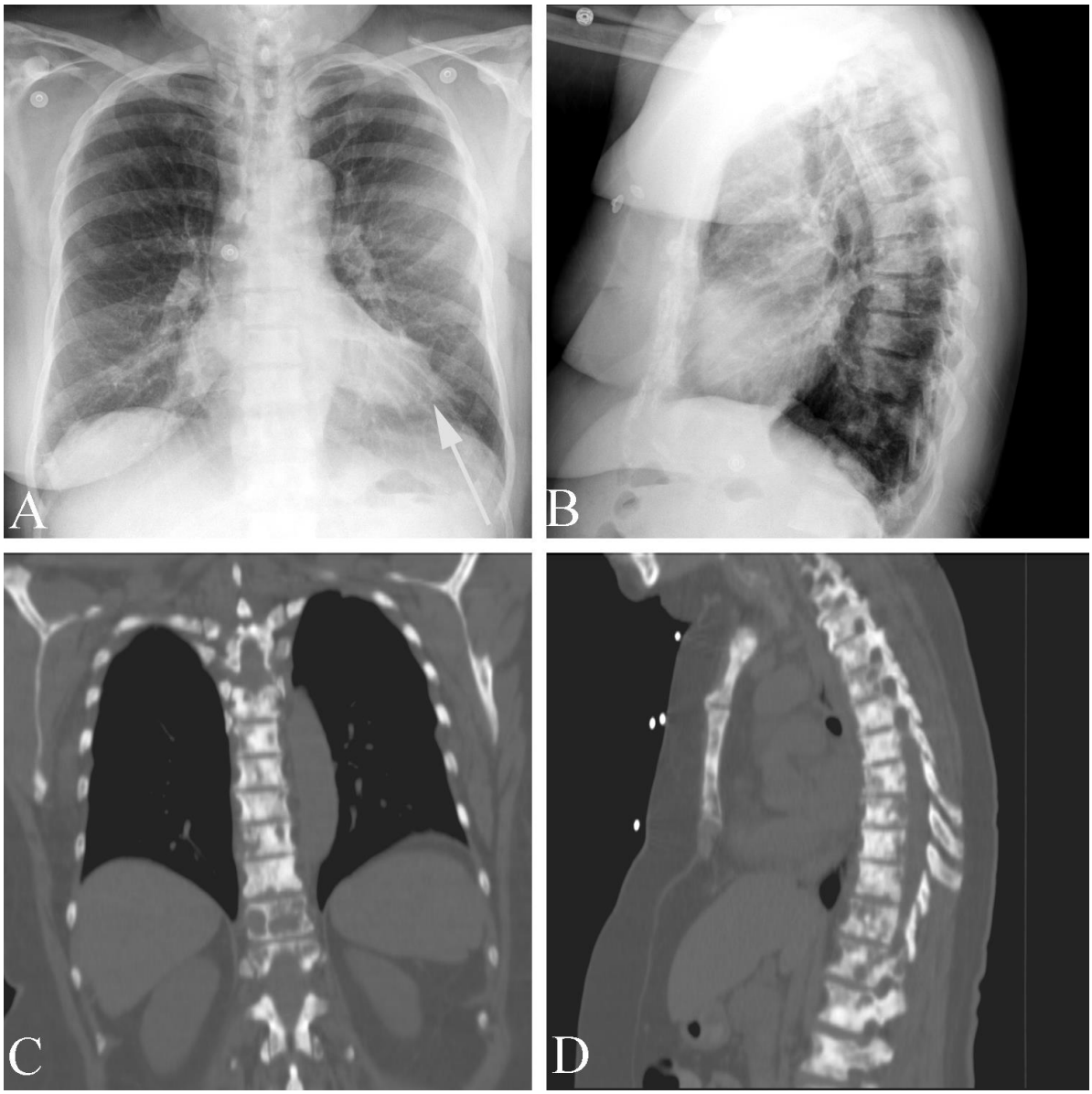

Figure 7. Metastatic breast cancer in a 70 year old woman with chest pain. A. PA CXR shows superior retraction of the left breast shadow (arrow). The patient had *not* had a mastectomy or any left breast procedure: the breast was retracted because of a tumor. B. Lateral CXR shows subtle patchy sclerotic change of the vertebral bodies. C. Coronal reformatted CT study demonstrates diffuse metastatic disease of the spine. D. Sagittal reformatted CT demonstrates diffuse metastatic disease of the thoracic spine and sternum.

pneumothorax, which may be done with the CXR. Another cause of chest pain which may be seen on a CXR (but which is better seen on a CT exam) is metastatic deposit to the skeleton (Figure 7). Other infrequently encountered causes of chest pain include painful arteriovenous malformation (Figure 9, Chapter 10, page 141) painful pneumonia (Figure 8), aortic dissection, esophageal rupture, and pulmonary hemorrhage (Figure 9). Note that many times, these diseases may have abnormalities on chest radiographs which are not specific and therefore require further evaluation, typically with CT. Even CT will usually provide a specific diagnosis only when correlated with all relevant clinical data.

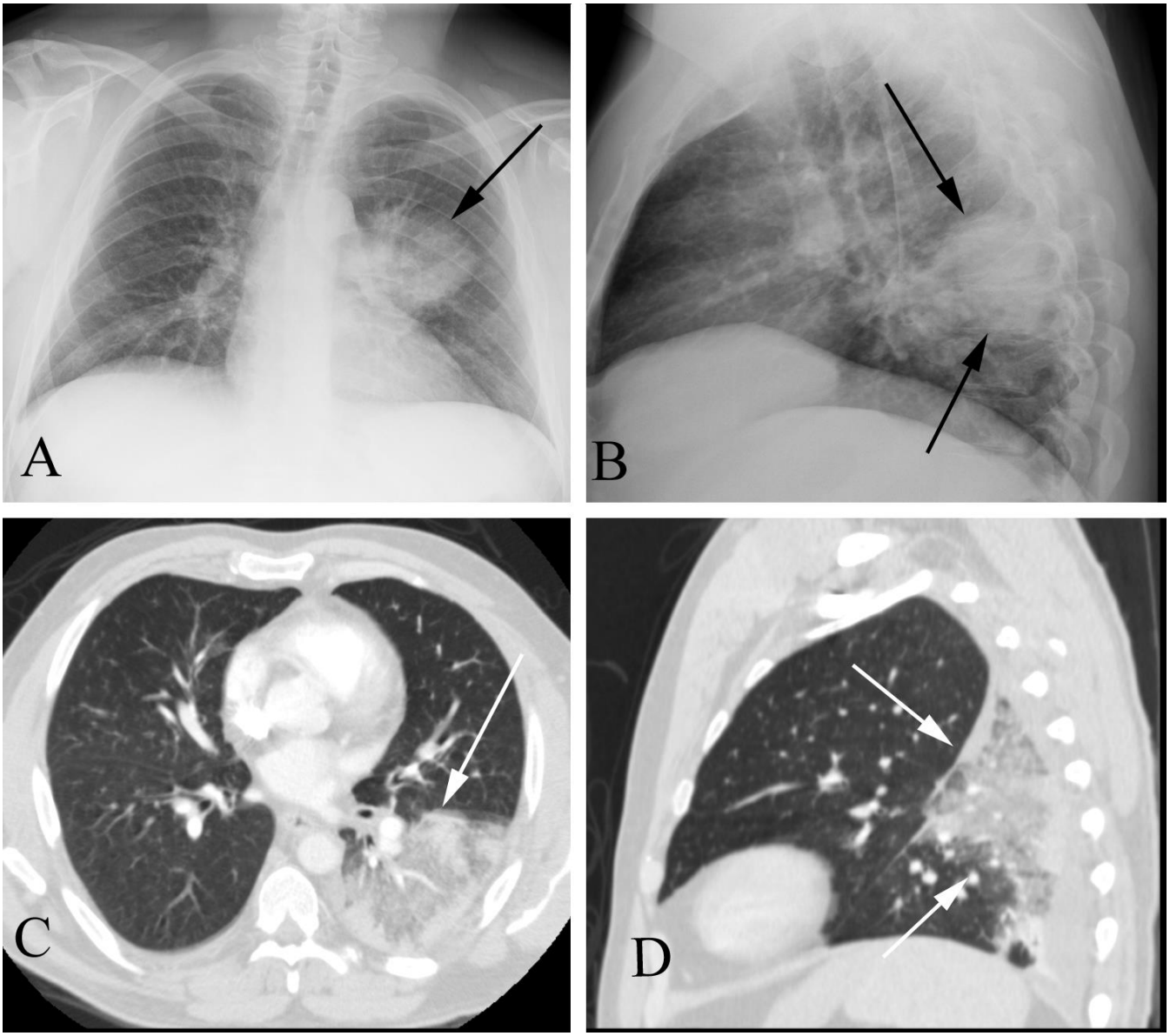

Figure 8. Pneumonia causing chest pain in a 47 year old man. A. PA CXR shows opacity in the mid left lung (arrow). B. Lateral CXR shows the opacity along the posterior chest wall (arrows). This is a so-called "round" pneumonia which mimics a mass. C. Axial CT shows consolidation in the left lower lobe (arrow). D. Sagittal reformatted CT shows consolidation along the posterior chest corresponding to the opacity seen on the plain film. Follow-up CXR (not shown) following treatment demonstrated clearing of the pneumonia.

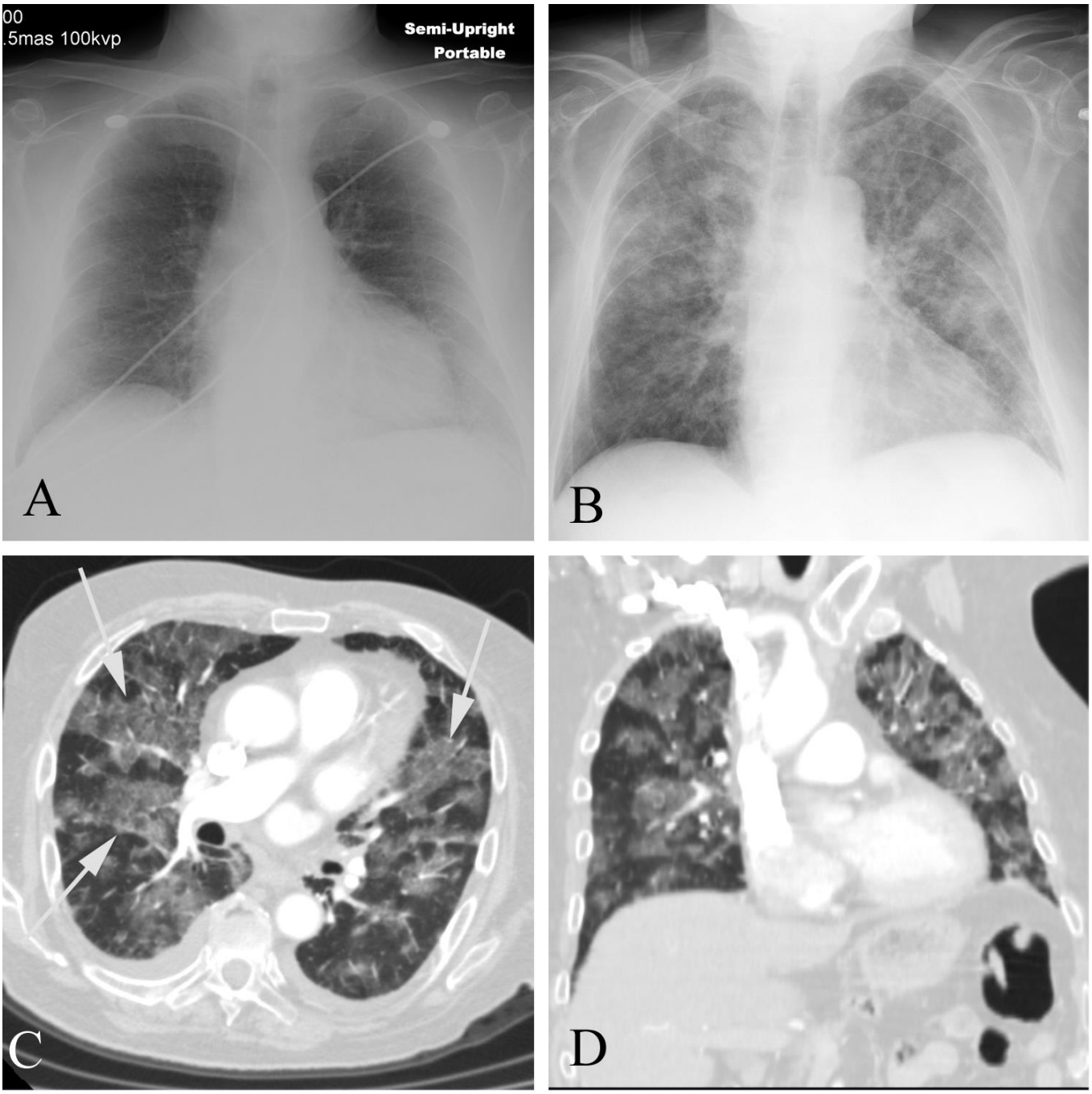

Figure 9. Pulmonary hemorrhage in a 75 year old man with chest pain. A. CXR taken three months before the onset of chest pain is normal. B. CXR done after onset of chest pain shows diffuse bilateral lung opacity. C. Axial CT shows extensive ground glass opacity (arrows). D. Coronal reformatted CT demonstrates ground glass opacity as well and demonstrates why the CXR had the appearance it did. Note that while the CXR and CT show striking abnormality, the findings are not specific and only correlation with the additional clinical features of anticoagulation and a sudden drop in hematocrit allowed the correct diagnosis.

SUSPECTED PULMONARY EMBOLISM

Pulmonary emboli frequently cause chest pain and remain a common cause of mortality, yet emboli often go undiagnosed. This follows from the fact that while the classic patient presents with an acute onset of severe chest pain and dyspnea, many patients have more subtle presentations[4]. One approach calls for scoring on the basis of what are known as the Wells criteria (see Table 2) and performing chest CTA if the likelihood of pulmonary embolism is high but obtaining a D-dimer if the likelihood of pulmonary embolism is low, in which case a chest CTA should still be performed if the D-dimer is elevated[5].

Variable	Points
Clinical signs and symptoms of leg DVT	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate > 100 BPM	1.5
Immobilization of more than three days or recent surgery	1.5
Previous PE or DVT	1.5
Hemoptysis	1.0
Malignancy	1.0

Table 2. Clinical Decision Rule for DVT. Likelihood of pulmonary embolism is considered high if the point total is more than 4. From: Wells PS et al. Derivation of a simple clinical model to categorize patients' probability of pulmonary embolism: increasing the model's utility with the SimpliRED D-dimer. Thromb Haemost. 2000; 83:416-420.

Regarding imaging, patients suspected to have a pulmonary embolism (like all patients with chest pain) will have a CXR. The CXR is therefore always obtained, and it is frequently abnormal, but, unfortunately, seldom helpful. Stein et al[6] found that while 84% of CXRs in patients with pulmonary embolism showed a variety of abnormalities, patients without pulmonary embolism had similar abnormalities at about the same rate. For example, 69% of patients with PE had pulmonary parenchymal opacity versus 58% without PE; 47% of patients with PE had a pleural effusion versus 39% without. Of course, a wide variety of diseases, many of which cause chest pain, may result in nonspecific CXR abnormalities such as pulmonary opacity and pleural effusion, making the CXR unhelpful in rendering the specific diagnosis of pulmonary embolism. As noted above, CXR is helpful in excluding certain other causes.

The mainstay of imaging diagnosis for suspected pulmonary embolism is now chest CTA (Chapter 10 Figure 2, page 134 and Figure 7, page 138). While pulmonary angiography was the reference standard in the diagnosis for years, CT is far more readily available, faster, cheaper, and less prone to iatrogenic mishap than is pulmonary angiography. CT allows quantification of the pulmonary emboli size, demonstrates their location, and diagnoses associated pulmonary infarction. In addition, CT allows an assessment of how severe the disease is: relative increases in right heart size (with a right ventricle: left ventricle ratio of more than one) or distention of the pulmonary artery (to greater than 30 mm) indicate severe disease with a worse prognosis[7] (Figure 10). These findings are associated with right heart failure, which is typically the cause of death in patients with large pulmonary emboli, and may indicate the need for emergent embolectomy. Finally, as when CT scanning finds alternative causes of flank pain in patients suspected of renal stone disease, CT scanning for chest pain may find such alternative diagnoses as pneumonia, cardiovascular disease, pulmonary fibrosis, and malignancy in patients with suspected pulmonary embolism[8].

For those who cannot undergo CT of the chest for evaluation of pulmonary embolism because of contrast allergy or renal failure, nuclear medicine ventilation-perfusion imaging (long a mainstay in the diagnosis of pulmonary embolism) is probably the best alternative. A normal perfusion study (Figure 4) essentially excludes pulmonary embolism, whereas gross mismatches of ventilation and perfusion (Figure 11) are diagnostic of pulmonary embolism. Unfortunately, many scans are indeterminate.

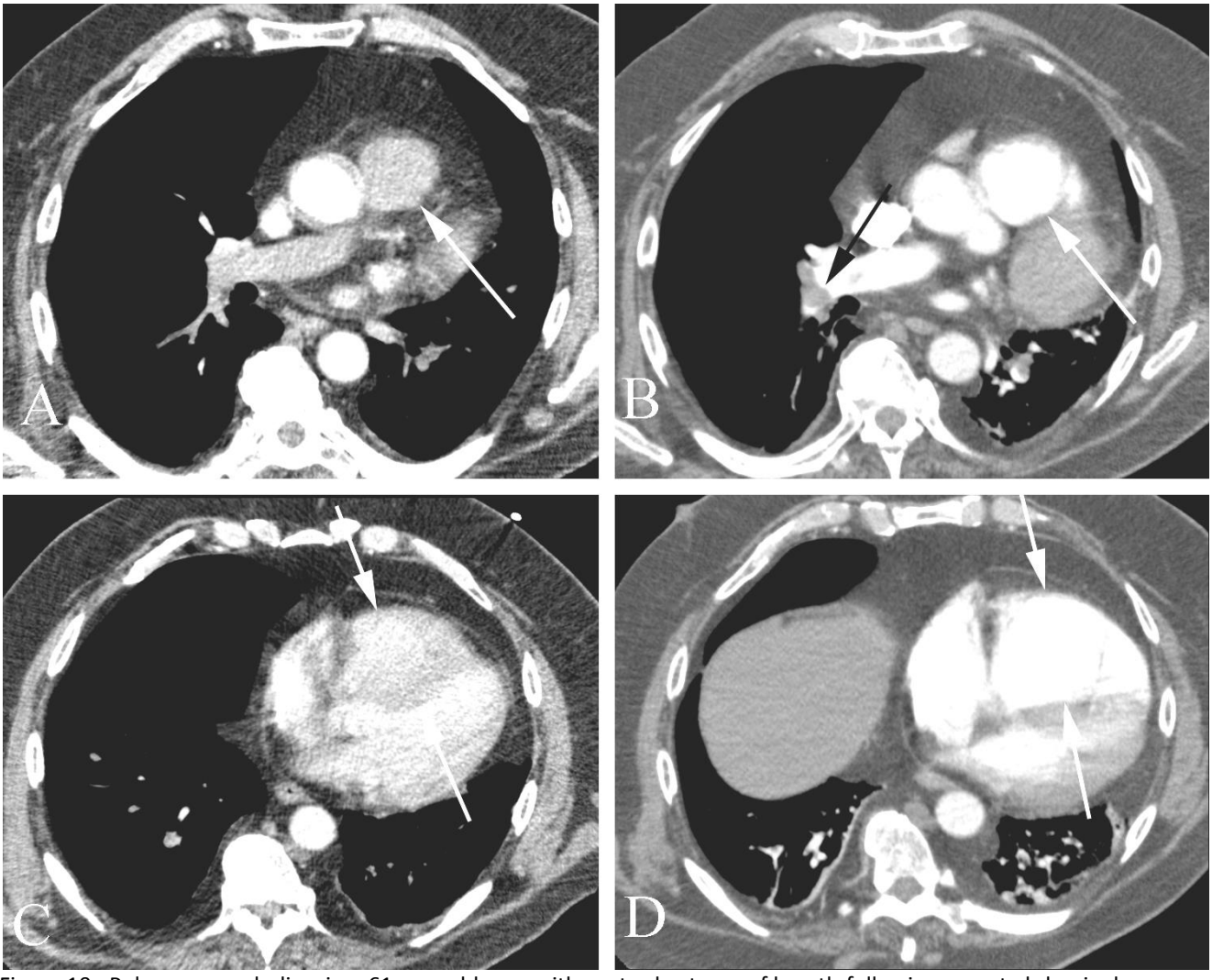

Figure 10. Pulmonary embolism in a 61 year old man with acute shortness of breath following recent abdominal surgery. A. Axial contrast-enhanced CT months before the patient's PE shows a normal sized main pulmonary artery (arrow). B. Axial contrast-enhanced CT shows a filling defect in the right pulmonary artery (pulmonary embolism) (black arrow) along with a dilated main pulmonary artery (white arrow). C. Axial contrast-enhanced CT at the level of the right ventricle months before the patient's PE shows a normal sized right ventricle (between arrows) D. Axial contrast-enhanced CT following the patient's pulmonary embolism shows a dilated right ventricle. An enlarged right ventricle is a poor prognostic sign in patients with pulmonary embolism.

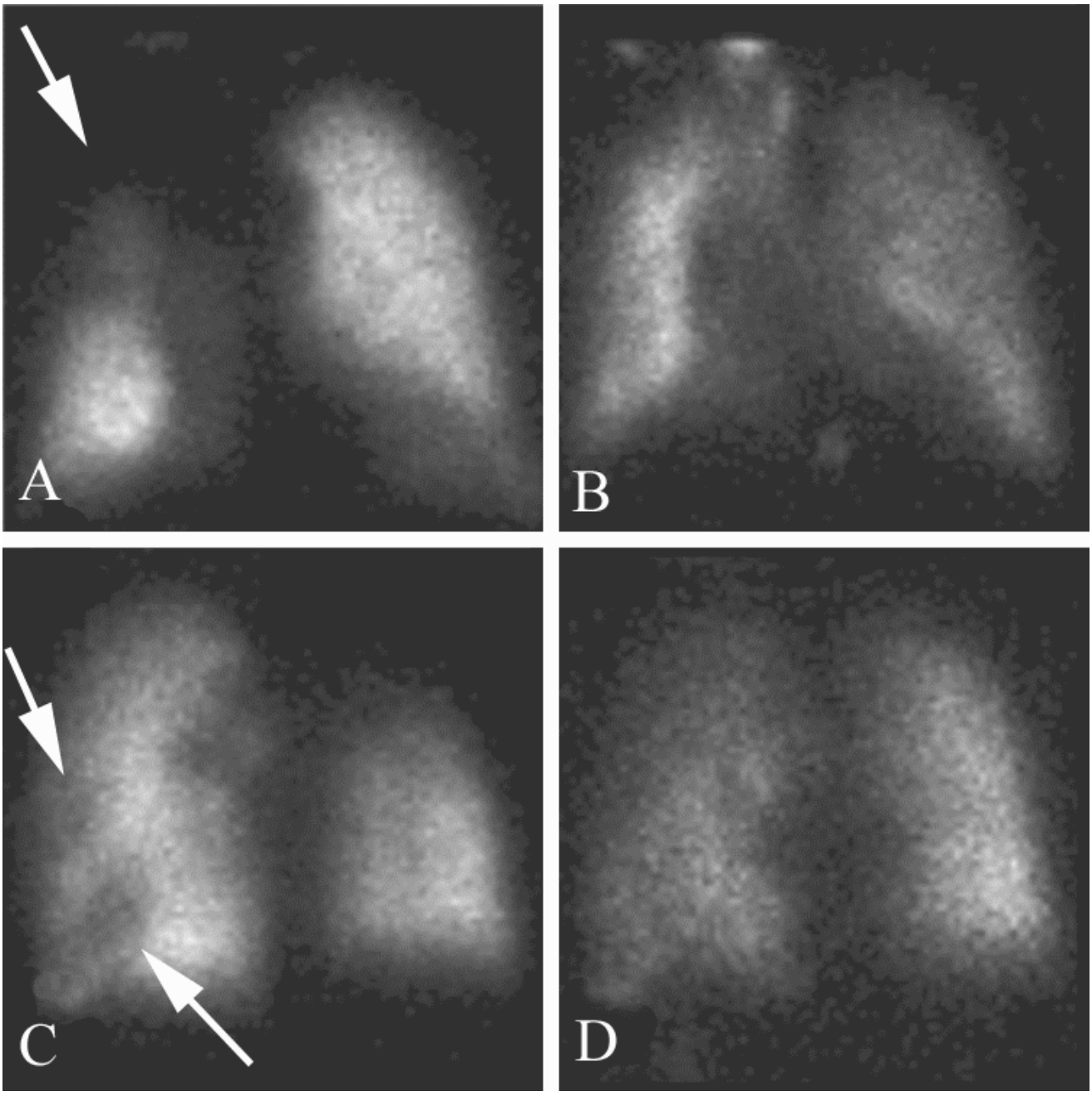

Figure 11. Pulmonary embolism in an 86 year old man with acute onset of shortness of breath and renal failure. A. Anterior perfusion study shows a focal area of decreased blood flow to the right upper lobe (arrow). B. Matched anterior ventilation study shows normal ventilation in the same region. C. Posterior perfusion study shows a focal area of diminished perfusion in the left lower lobe (arrows). D. Matched posterior ventilation study shows normal perfusion in the same region. Multiple mismatched ventilation-perfusion defects indicate a high probability of pulmonary embolism.

Another alternative to CT scanning is to perform bilateral lower extremity deep venous ultrasound examination (see page 179 for a discussion of this study), with the notion that since nearly all pulmonary emboli originate from the lower extremities, and since it is unlikely that all of the clot will break off to embolise to the lungs at one time, residual clot is likely to be found in the lower extremities. However, Turkstra et al[9] found a 3% false-

positive rate of diagnosis of DVT and a 70% false-negative rate. Note that technical aspects of lower extremity venous ultrasonography have evolved considerably since the publication of Turkstra et al (in 1997), however, and most departments would probably do better than these figures indicate. Subsequent studies by other authors have found much lower false negative rates[10],[11].

SUSPECTED CORONARY ARTERY SYNDROME

With coronary artery syndrome, the critical issues are:

1) Is a cardiac event (infarction) presently occurring?

2) Is the patient's chest pain secondary to coronary artery narrowing?

3) How likely is the patient to have a cardiac event in the future?

Acute myocardial infarction from ruptured/hemorrhagic plaque

Imaging of patients with suspected coronary artery syndrome, like imaging any patient with chest pain, starts with obtaining a chest radiograph. Even before the patient has the radiograph done, however, it is necessary to evaluate whether the patient is actively infarcting or not. Most patients with an active infarct will present to the emergency room rather than a clinic, and many of these patients are severely ill with, for example, hypotension or tachycardia. If you suspect acute myocardial infarction, the best course of action is to start an IV and draw blood for cardiac enzymes, obtain an immediate 12-lead EKG, have the patient chew a 325 mg aspirin, and arrange immediate transport to an emergency room, preferably in an ambulance equipped with a defibrillator[12].

The CXR can wait until the patient is in the emergency room. The clock is running in these patients: there are only 60 to 90 minutes or so to save ischemic myocardium undergoing infarction, so it is imperative that these patients be transported to an emergency room (or straight to the cath lab) as rapidly as possible.

What is happening in most of these patients is that plaque, which may have been years accumulating, has ruptured, resulting in acute occlusion of a coronary artery[13]. For these patients, while a chest radiograph will be obtained to evaluate for changes of acute congestive heart failure (Figure 12), the diagnosis will rely on EKG changes and/or cardiac enzymes, typically done sequentially until the diagnosis is secured. For those patients who proceed to the catheterization lab, imaging will be performed during catheterization, usually followed by stent placement or, if stents are not an option, emergency cardiac bypass surgery.

Angina from coronary artery narrowing

The drama of a patient presenting with an acute myocardial infarction is the exception, not the rule, for the patient seeing a primary care practitioner for evaluation of chest pain. Most patients with coronary artery syndrome have angina because of stenosis of the coronary arteries. To decide which study to order for evaluation of patients suspected to have chest pain on the basis of coronary artery stenosis, it is first necessary to evaluate the patient's risk. You must calculate the pretest probability of coronary artery disease by assessing their chest pain pattern and correlating the pain pattern with their age and sex (Table 3). Low risk patients (pretest probability of <5%) are unlikely to benefit from stress-EKG or perfusion imaging testing because a positive test is much more likely to represent a false positive result than to

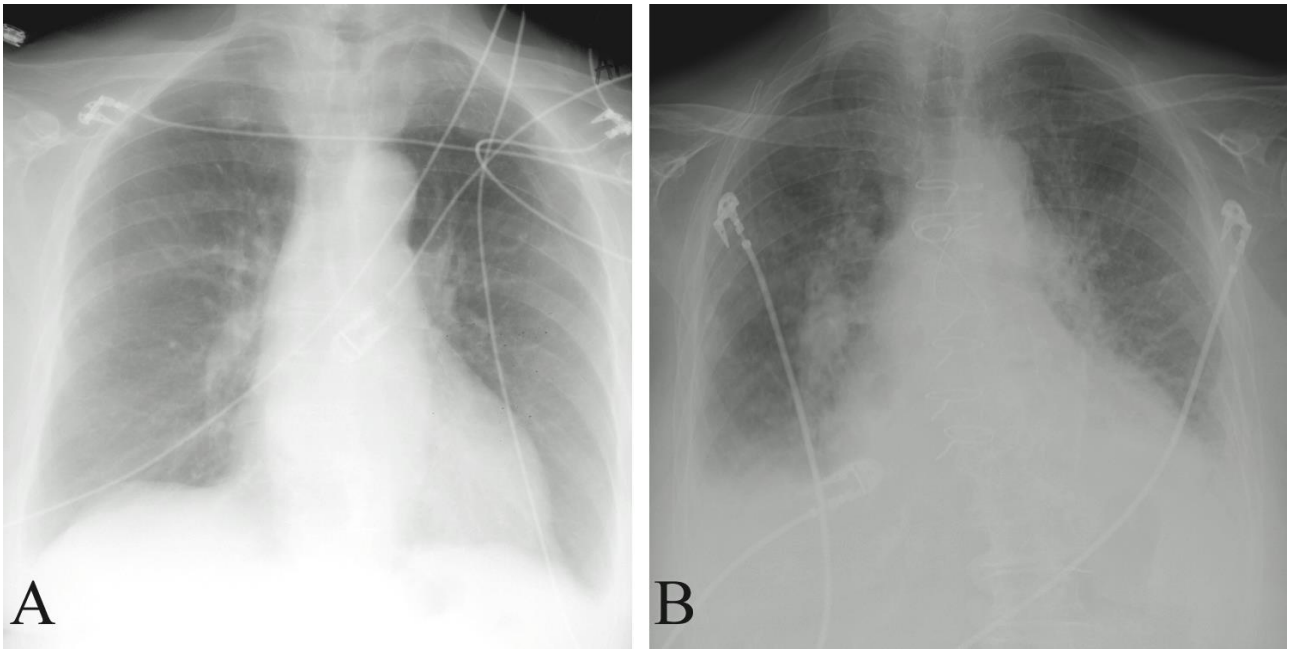

Figure 12. Florid congestive heart failure in 92 year old woman with chest pain from myocardial infarction. A. The baseline CXR from ten years prior to the patient's acute onset of chest pain shows a normal sized heart and normal pulmonary vessels. B. A CXR performed shortly after the onset of acute chest pain shows cardiomegaly, bilateral pleural effusions, and prominent pulmonary vasculature from congestive heart failure. The patient had acute abnormality on her ECG and abnormal cardiac enzymes.

represent true disease, and high risk patients (pretest probability >90%) are unlikely to benefit because a negative result is likely to be a false negative and these high risk patients should probably proceed to catheterization (or at least evaluation by a cardiologist) anyway[14]. The intermediate risk patients (pretest probability of between 25% and 75%; note that recommendations are less clear for patients with a pretest probability of heart disease between 5% and 25%, and for those with risk between 75% and 90%) should proceed to stress-EKG testing, or stress-EKG testing combined with myocardial perfusion imaging or echocardiography. The imaging component of the examination is typically added when there are baseline ECG abnormalities making interpretation of stress-induced changes problematic, when the patient is on digitalis, when there has been previous revascularization, or when a stress-ECG done without imaging produces equivocal results[15]. Note also that addition of imaging improves sensitivity in intermediate risk patients by about 20%[16]. SPECT myocardial perfusion images are interpreted by comparing the rest and stress images, usually with the assistance of a computer which will make a map of abnormally perfused areas (Figure 13). These abnormally perfused areas may be either fixed, indicating chronic ischemic change and scarring, or reversible, indicating myocardium that is at some risk for infarction. Either a positive EKG, or changes on a myocardial perfusion study, similar to a high pretest probability of coronary artery stenosis, usually indicates the need for coronary angiography or at least consultation with a cardiologist. With regard to imaging of the coronary arterial tree, CACS and CCTA are presently undergoing rapid evolution, and the inclusion of these tests in the algorithm for the work-up of suspected coronary artery syndrome depends on the availability of appropriate equipment, expertise, and

physician acceptance[17]. For patients with nonanginal chest pain, which is unlikely to represent coronary artery stenosis, coronary artery calcification scoring (CACS) offers an alternative to the stress test, or an additional test if the stress test is negative or equivocal. For the CACS to be helpful in this regard, it should be zero. In explanation: the score is based on the amount of calcium in the coronary arteries, and a lower score (less calcium) is better, with a score of 0 being ideal. Georgiou et al found that if the CACS is zero, the annual cardiac event rate was 0.6% per year over the next five years[18], and McLaughlin et al found only one cardiac event (in a cocaine user) within one month among 48 chest pain patients with a CACS of zero, whereas the 30 day event rate was 8% in those with a CACS of greater than zero[19]. On the basis of their results, McLaughlin et al felt that CACS excluded patients with nonanginal chest pain from further costly evaluation. If the CACS is not zero, or if direct anatomic visualization of the coronary arterial tree is desired, CCTA may be performed. The ordering, interpreting, and control of this modality continues to be controversial, in part because of the turf war it provokes between cardiologists (who have traditionally controlled most cardiac imaging modalities such as echocardiography, cardiac catheter angiography, and nuclear medicine myocardial perfusion studies) and radiologists (who have traditionally read all studies obtained on the CT scanner). From the perspective of the primary care provider, what matters is that this test is performed correctly and interpreted accurately, and at present the ability to perform the test is not widespread. If it is available, which patients should be sent for CCTA? Present recommendations (which are evolving with changes in technology) are: patients at intermediate risk for coronary artery disease (including those with equivocal stress tests), patients with known or suspected congenital or acquired coronary artery anomalies, and patients with coronary artery bypass grafts in whom it is not possible to engage the grafts during angiography[20].

Pretest Probability of coronary artery disease	Description	Testing
Low (<5%)	Asymptomatic or Women < 50 and Men < 40 with nonanginal pain	Typically none
Intermediate (25% - 75%)	Women > 50 with atypical angina or >30 and < 60 with typical angina Men > 60 with nonanginal pain, or > 30 with atypical angina	Stress EKG or stress EKG with MPI
High (>90%)	Men > 50 with typical angina	Cardiac catheterization

Table 3. Risk categories and test recommendations for coronary heart disease. Typical angina = Chest pain 1) with a typical quality and duration, 2) which is provoked by exertion or emotional stress, and 3) which is relieved by rest or nitroglycerine (all three); Atypical angina = Chest pain with two of the three characteristics; Nonanginal pain = chest pain with none or one of the three characteristics. Table modified from Garber AM, Hlatky MA. Stress testing for the diagnosis of coronary artery disease. UpToDate, accessed 10/7/09.

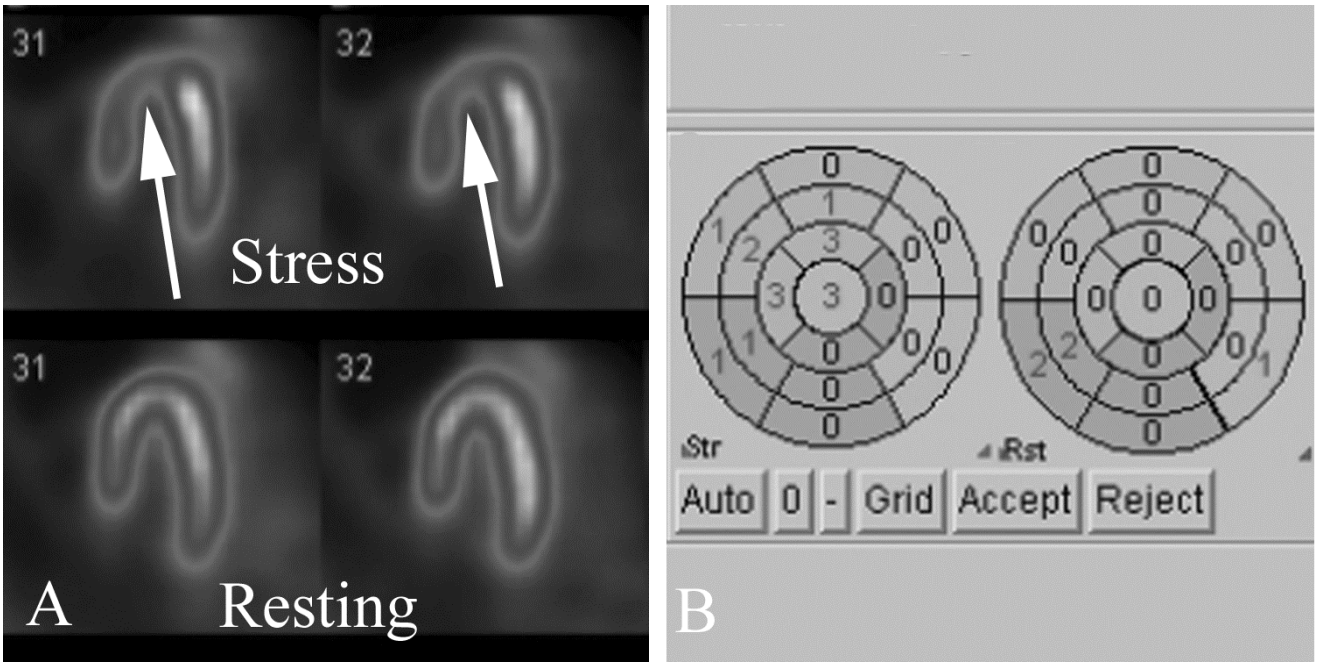

Figure 13. Positive myocardial perfusion study in a 64 year old man with exertional chest pain. A. Select vertical long axis views from a myocardial perfusion study show a defect along the septum extending into the apex (arrows) on two sequential images obtained during stress, with a normal appearance on the corresponding resting images. B. The computer generated map shows significant difference between the stress and resting radiotracer distribution (higher numbers imply lower perfusion), compatible with reversible ischemic changes. The patient underwent cardiac catheterization, with diagnosis (and stenting) of a left anterior descending coronary artery stenosis, with subsequent relief of symptoms.

Risk of future cardiac events

In evaluation of patients with suspected coronary artery disease, two separate risks are important: 1) the risk that a patient with chest pain has significant coronary artery stenosis (discussed above) and 2) the risk that a given patient will suffer a cardiac event (defined as death, infarction, or surgical/percutaneous intervention) during some specified time period. In the two prior sections, I discussed patients who are suspected of having a cardiac event (whose "risk" is 100%, *right now*!), and patients who are suspected to have coronary artery stenosis. Patients with stenosis have a high risk of future events, and are generally under the care of a cardiologist. In these two sets of patients, there is either plaque which has ruptured, or there has been demonstration of plaque which may rupture. Absent a current infarction or known stenosis, in patients with no chest pain, what is the risk of a future event?

Cardiac risk may be calculated using the age and sex of the patient, total cholesterol, HDL cholesterol, smoking status, systolic blood pressure, whether or not the patient is on antihypertensive medication[1], and serum cholesterol. This risk is generally reported in 10 year increments, with *low risk* defined as a less than 10% chance of cardiac event in the next 10 years, *intermediate risk* defined as between a 10% and 20% chance of cardiac event in the next 10 years, and *high risk* defined as a greater than 20% chance of cardiac event in the next 10 years. Note that those with diabetes and previous episodes of coronary artery disease are all at high risk.

[1] See on line calculator at http://hp2010.nhlbihin.net/atpiii/calculator.asp

What is the role of imaging in evaluating risk? Coronary artery calcification scoring (CACS) measures the calcium load within the coronary arterial tree. Such calcifications denote plaque and show a correspondence with risk for plaque rupture: the more calcification, the higher the risk of plaque rupture. As noted above, an Agatston score of zero is associated with a less than 1% rate of coronary artery stenosis[21] and a low zero risk of a cardiac event in the next 10 years. In fact, a study of over 25,000 patients demonstrated that the CACS predicted all-cause mortality independent of and more accurately than standard coronary artery disease risk factors[22].

Regardless of the additional and independent value of CACS in risk evaluation, the official recommendation of the American College of Cardiology Foundation and the American Heart Association is to use the CACS in patients with intermediate risk, to determine whether to treat such patients more aggressively (which generally means adding or changing drugs to lower the serum cholesterol level). Patients with a low Agatston score (typically zero) remain at intermediate risk, whereas those with a non-zero Agatston score are upgraded to the high risk category.

While the recommendation to order CACS for intermediate risk patients is certainly reasonable for the primary care practitioner to follow, note that many imaging facilities will perform CACS on a self-referral basis. Since many insurance companies do not cover the cost of this exam, and the patient pays out of pocket, this is an unusual example of the free market at work in medicine within the U.S. It is instructive to note that this has resulted in aggressive pricing (some would argue below cost) and direct patient marketing of this exam. For this reason, patients may choose to obtain a CACS without referral and then come to a primary care practitioner with a request to explain the results[23].

Probably the most reasonable thing to do in this situation is to explain to the patient the AHA's recommendation and then calculate the patient's risk. If the patient is at an intermediate risk by the calculator, great: the test was indicated anyway and you can advise/treat the patient as noted above. If the patient is at low risk and has a score of zero, or is at high risk and has a non-0 score, then the CACS has only confirmed what the risk calculator told you anyway and no change in treatment or prognosis is forthcoming. The CACS was a waste of money and needless radiation exposure. The other situations that may arise are when the CACS *conflicts* with the risk calculation: either a low risk patient has a non-0 CACS, or a high risk patient has a 0 score. Such findings may be reassuring to the high risk patient or motivating to the low risk one, but at present recommendations are to *not* change treatment of such patients[23]. Note that in regard to motivating patients to change behavior, O'Malley et al found that knowledge and even visual presentation of CACS information did not provide patients any additional motivation (or improve their compliance with treatment)[24].

SUMMARY

When imaging patients with chest pain, nearly all patients will first have a plain film. For patients with acute severe shortness of breath or who have other features strongly suggesting pulmonary embolism, an emergent chest CT should be obtained; nuclear medicine ventilation-perfusion imaging may be performed if the patient has renal failure or contrast allergy. Imaging of suspected coronary artery syndrome depends on the clinical condition of the patient and risk assessment.

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Vascular Imaging

Donald L. Renfrew, MD

This chapter reviews vascular imaging. The main points of this chapter are:

1. CT angiography has largely supplanted catheter angiography for diagnostic vascular studies.
2. Catheter angiography may still be used in those situations where therapy is necessary or detail beyond the current resolution of less invasive methods is necessary.
3. Specific recommendations for imaging vary with the anatomic location and clinical situation.

Note that the book has already covered many examples of vascular imaging: Chapter 1 discussed renovascular hypertension; Chapter 2 discussed scrotal varicocele; Chapter 3 discussed cerebral artery aneurysm and malformation; Chapter 10 discussed pulmonary embolism; and Chapter 11 discussed coronary artery disease. This still leaves a number of topics, which this chapter addresses, along with the promise in Chapter 4 (see page 47) to address vascular imaging in the evaluation of neurologic symptoms including TIA and stroke.

In general, the gold standard for vascular imaging has long been catheter angiography. However, diagnostic catheter angiography has largely been replaced, at least for screening purposes or initial diagnosis, by other methods. In general, *vascular ultrasound* is now widely used for screening and follow-up examination (where applicable), supplemented by *computed tomographic angiography* (CTA) or *magnetic resonance angiography* (MRA) when more definitive evaluation is required or when ultrasound cannot be used. CTA has the advantages of higher spatial resolution and is less subject to motion artifact. MRA using flow techniques can be performed *without* contrast material, although contrast-enhanced MRA tends to provide better images. Catheter angiography nowadays is often used to confirm (or disprove) results obtained from noninvasive vascular imaging, and to allow intravascular intervention.

Beyond these generalizations, specific recommendations for vascular imaging vary by body part, and will be covered below, first addressing the arterial tree and then the venous side of circulation. Although this is a book on the radiology of symptoms, vascular imaging is performed relatively frequently in asymptomatic individuals for screening purposes, and the chapter will cover this topic as well.

ARTERIAL STUDIES

Intracranial aneurysms

As noted above, Chapter 3 discussed *symptomatic* intracranial aneurysms. What about *asymptomatic* intracranial aneurysms? Such asymptomatic intracranial aneurysms may be discovered either incidentally, during imaging for unrelated symptoms, or because of screening. In both cases, current recommendations are to monitor small aneurysms annually for two to three years[1]. If the aneurysm grows in this interval, referral for consideration of intervention is appropriate; if it is stable, extending the monitoring interval to two to five years is appropriate[1]. "Small" in this context means less than 10 mm, although some authorities use 7 mm[1]. Of course, such screening must take into account the age and general medical condition of the patient with more aggressive management of younger and healthier patients.

Regarding screening for asymptomatic aneurysms, there is no role for such studies in the general population. For subsets of the general population with a higher risk of aneurysm, recommendations are generally *not* to screen: for example, recommendations are *not* to screen for genetic syndromes known to be associated with intracranial aneurysms, and *not* to screen patients with a single first-degree relative with an intracranial aneurysm which has bled[2]. Patients with two (or more) first-degree relatives with bleeding intracranial aneurysms, however, should be screened. The role of screening in adult polycystic kidney disease (which is associated with increased risk of intracranial aneurysms) is unsettled[2].

Aortic arch, carotid vessels, and intracranial vasculature: screening and in patients with ischemic symptoms

As noted in Chapter 4, in patients with neurologic symptoms, a facilitated work-up including MRI of the brain and either US, CTA, or MRA of the carotid arteries is recommended to evaluate for a causative lesion (Figure 1). The main purpose of this facilitated work-up is to find stenotic arterial lesions which will benefit from carotid endarterectomy. Current recommendations call for carotid endarterectomy (CEA) for patients with 70-99% stenosis, and for men (but not women) with 50-70% stenosis[2]; recommendations for women are different because clinical trials have shown less benefit for CEA compared to medical treatment in women with <70% stenosis. The facilitated work-up is important, because the shorter the delay between the symptoms and surgery, the better the outcome, with the best outcomes when the surgery is performed within two weeks of the neurologic event[3]. If ultrasound is the initial study for documentation of stenosis, an MRA or CTA is typically performed for confirmation of results, since US tends to overestimate stenosis and does not provide for the direct visual assessment of the stenosis as well as CTA or MRA (Figure 2). For symptomatic disease with less 50% stenosis, annual follow-up is recommended to document stability[3], with referral for further evaluation in the event of disease progression.

In general, expert groups including the United States Preventative Services Task Force, the American Heart Association, the American Stroke Association, and the American Society of Neuroimaging recommend *against* screening the general population for carotid stenosis[4]. Bruits of the carotid are a better indicator of general vascular disease (e.g., coronary artery disease, lower extremity arterial disease, and contralateral carotid disease) than they are of ipsilateral carotid stenosis (Figure 3). In men between 40 and 75 years of age where study of the carotids after auscultation of a bruit or a community screening program (done on a self-referral basis) has discovered a stenosis, CEA should at least be considered in those with a stenosis of over 70%[3] (Figure 4).

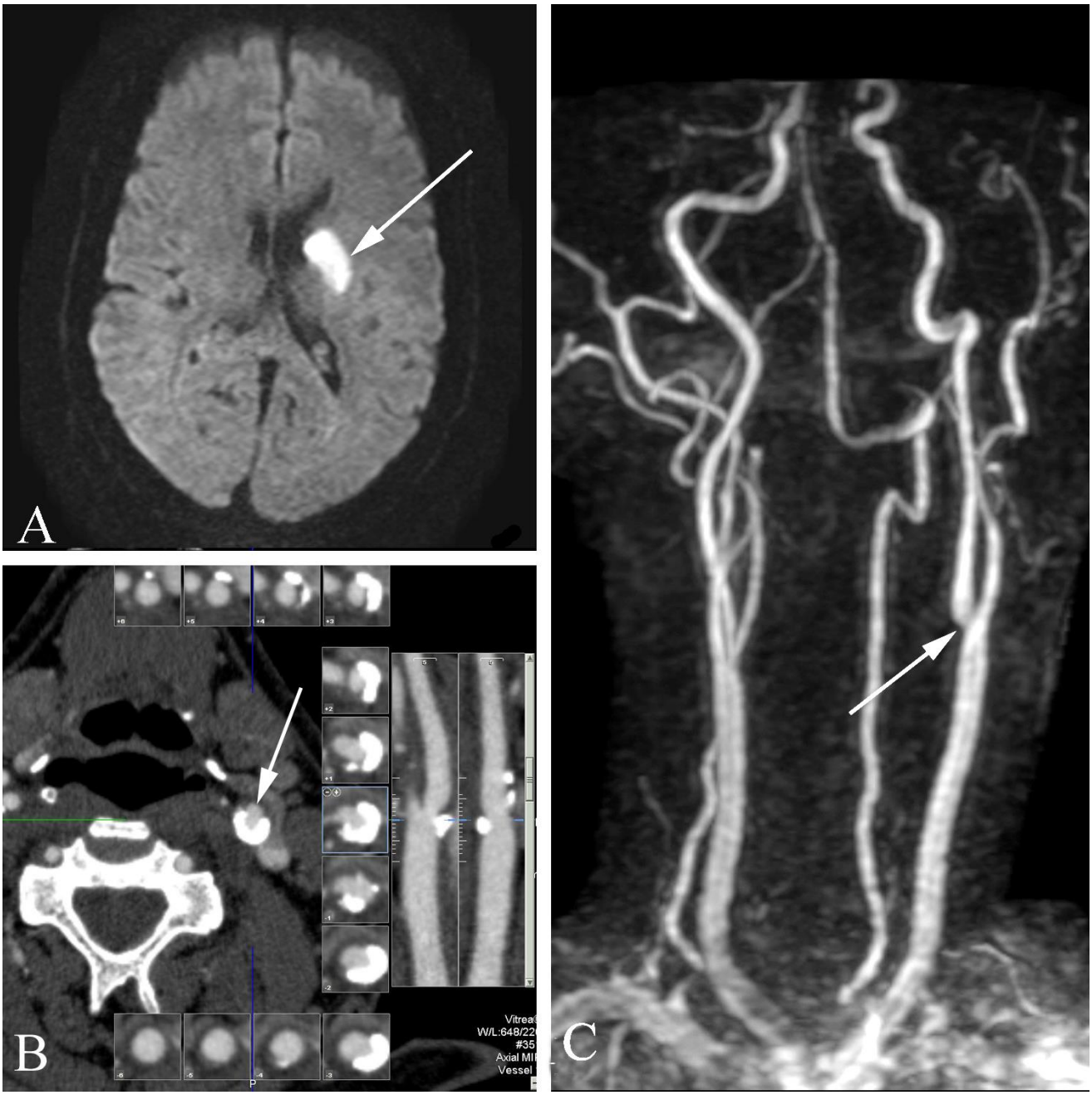

Figure 1. Less than 50% carotid artery stenosis in a 58 year old man with transient facial droop and aphasia. A. Axial diffusion weighted MR image demonstrates restricted diffusion in the left hemisphere adjacent to the ventricles (arrow) compatible with an acute cerebral infarction. B. CT angiogram through the left carotid shows an axial slice at the level of the proximal internal carotid artery (arrow) with axial images arrayed around the vessel from proximal to distal, with reconstructed long axis views at perpendicular projections on the right side of the panel, showing less than 50% stenosis. C. 3D maximum intensity projection MR angiogram confirms less than 50% stenosis of the left internal carotid artery.

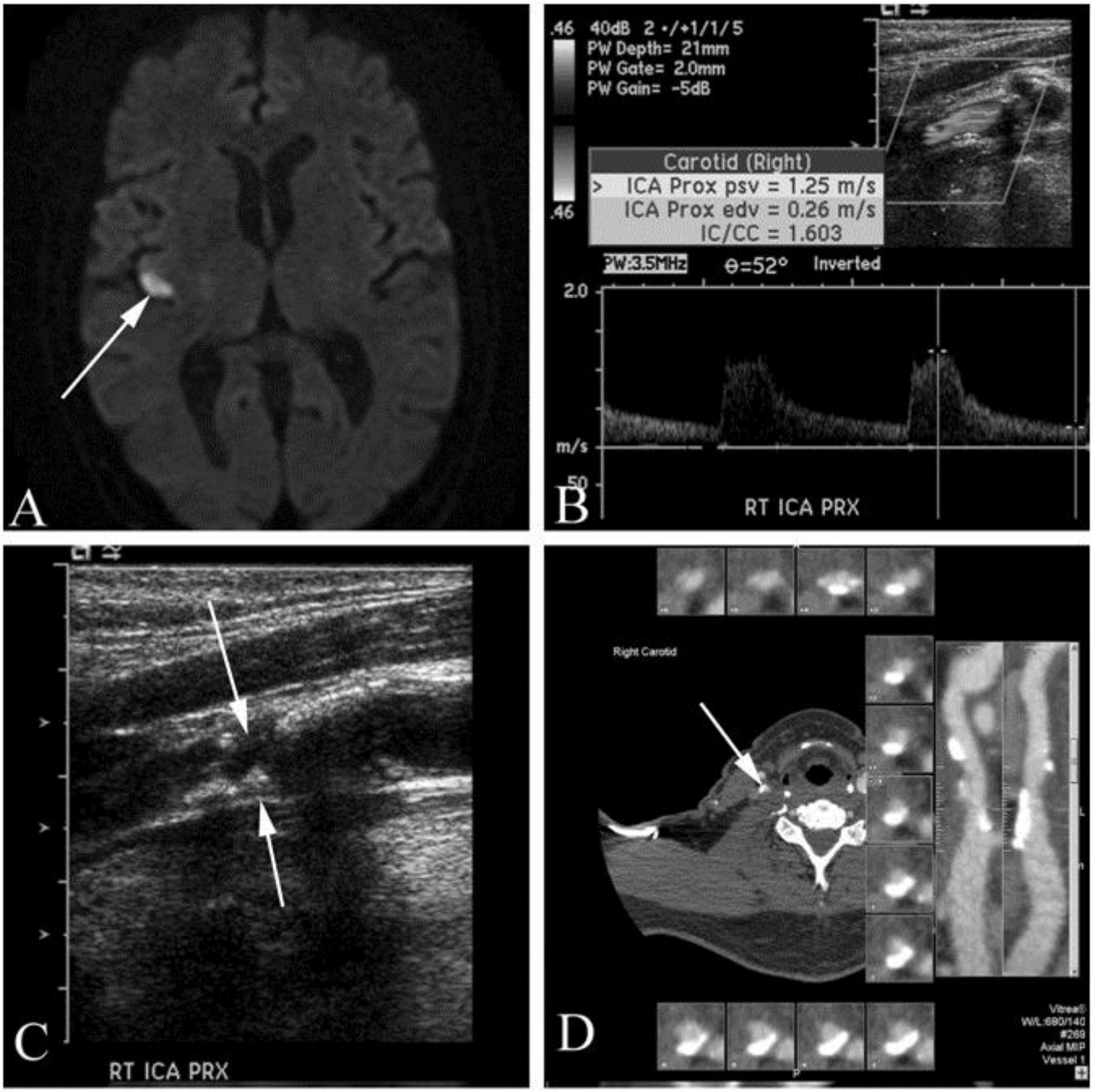

Figure 2. 50-70% stenosis of the right internal carotid artery in an 84 year old man with slurred speech and decreased strength in the right arm and hand. A. Axial diffusion weighted MR image shows a focus of restricted diffusion in the right hemisphere (arrow) compatible with an acute infarct. B. Right carotid ultrasound study shows peak systolic velocity (PSV) and internal carotid artery – common carotid artery ratio (IC/CC) compatible with only mild stenosis. C. Ultrasound at the

level of the bifurcation showing extensive atherosclerotic plaque making visual estimation of stenosis difficult. D. CT angiogram through the right carotid bifurcation (arrow) shows 50-70% stenosis along the proximal internal carotid artery.

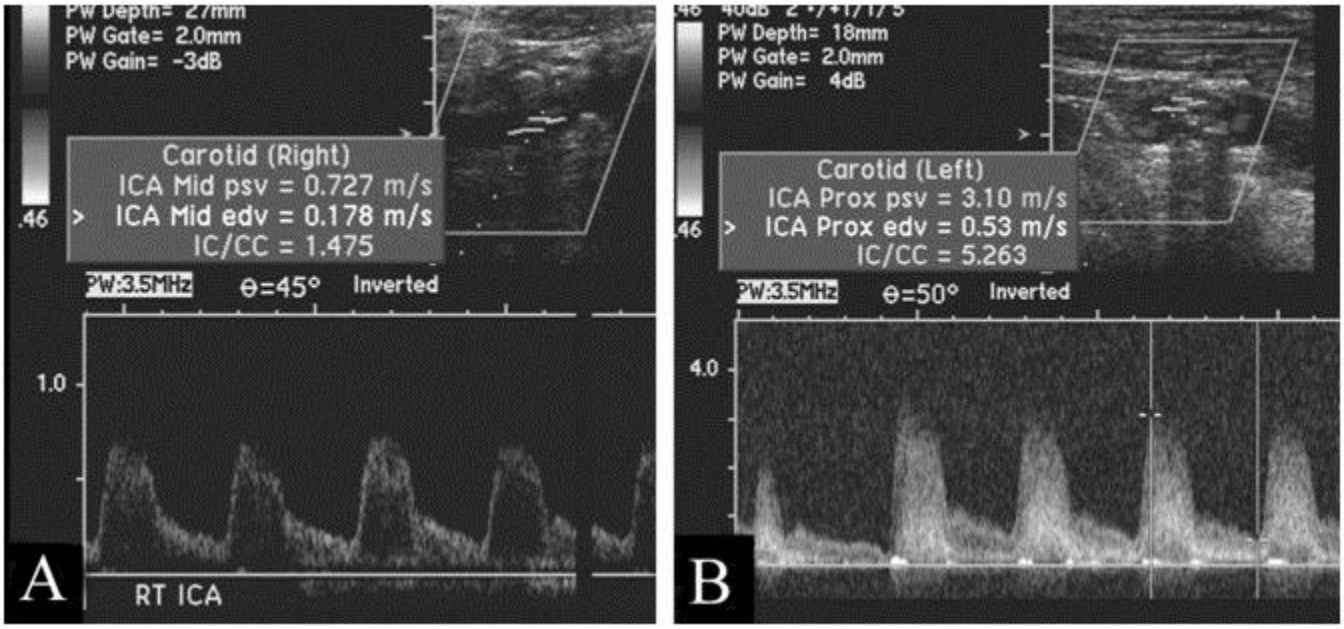

Figure 3. Carotid stenosis on the *left* side in an 83 year old woman with a *right* carotid bruit. A. Carotid US study (right carotid artery) shows normal peak systolic velocity (PSV) and internal carotid artery to common carotid artery PSV ratio (IC/CC) on the side of the bruit. B. Carotid US study (left carotid artery) shows abnormal elevated peak systolic velocity and internal carotid artery to common carotid artery PSV ratio compatible with greater than 70% stenosis on the side *opposite* the bruit.

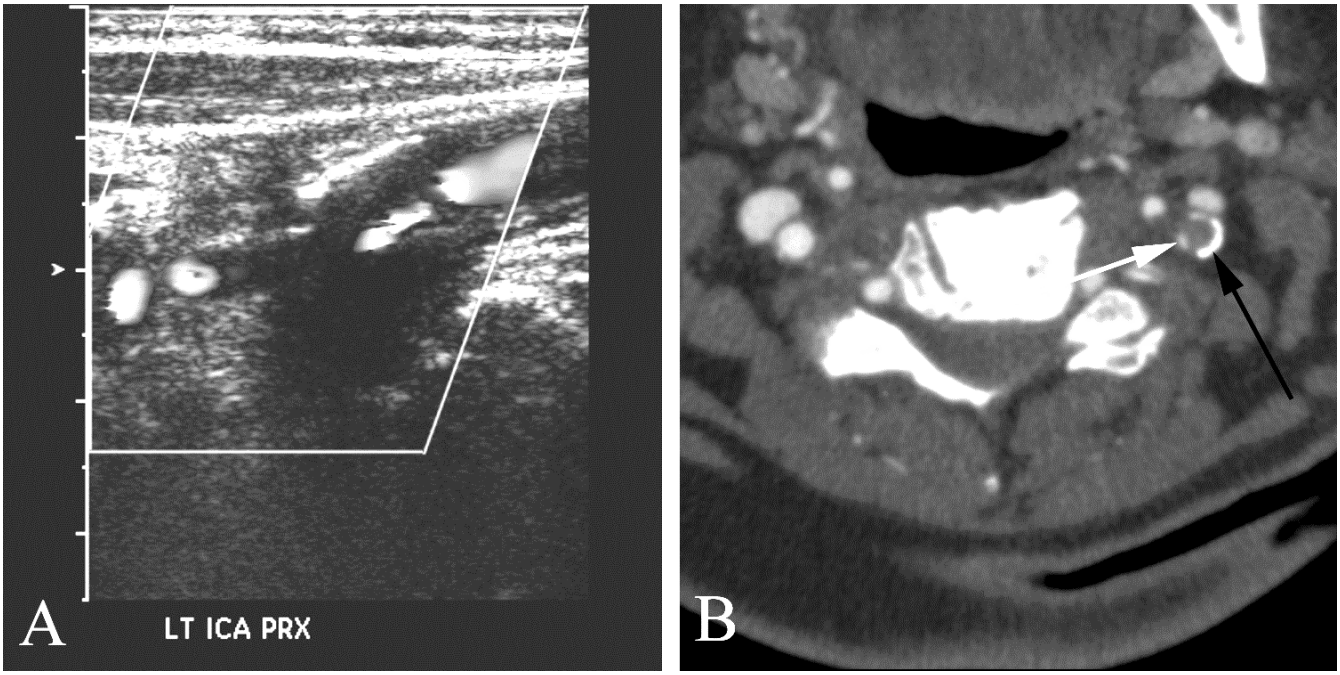

Figure 4. Severe left internal carotid artery stenosis in a 73 year old woman with an abnormal community screening result. A. Carotid US study shows dense calcification and severe stenosis of the proximal internal carotid artery. This was a diagnostic study done on the basis of the abnormal screening exam. B. Axial CT from a CT angiogram shows greater than

90% stenosis of the left internal carotid artery, with a trickle of flow (white arrow) along the periphery of a calcified arterial wall (black arrow). Other axial images and windowing confirmed that the lumen was open along the medial aspect.

Thoracic aortic aneurysm

Patients with symptomatic thoracic aortic aneurysms are generally direly ill because of rupture or dissection of the aneurysm. These patients typically come to the emergency room with severe chest pain. They will undergo emergent CT study to differentiate thoracic aortic dissection from pulmonary embolism (see pages 159-160). Most cases of dissection require emergent, hopefully life-saving, intervention. Some chronic thoracic aortic aneurysms may cause ongoing chest pressure (Figure 5).

Asymptomatic thoracic aneurysms are generally discovered during chest imaging for unrelated symptoms: for example, echocardiography, done for evaluation of cardiac function or valvular anatomy or chest x-ray examination for evaluation of cough and fever. Chest x-rays cannot differentiate aneurysmal dilatation of the aorta from tortuosity of the aorta, and so patients with an abnormal aortic contour need further evaluation with either chest CTA or MRA to define the aortic arch[5].

For patients with incidentally discovered, asymptomatic thoracic aneurysms, CTA or MRAFor patients with incidentally discovered, asymptomatic thoracic aneurysms, CTA or MRA allows evaluation of such important anatomic features as whether the aneurysm involves the ascending aorta, the descending aorta (with or without extension into the abdomen), the aortic arch and great vessels or some combination thereof. CTA or MRA also allow measurement of the size of the aneurysm, which is critical information since the risk of rupture is directly related to the size of the aneurysm: one study found that the five year risk of rupture was 0% for aneurysms less than 40 mm, 16% for aneurysms between 40 and 59 mm, and 31% for aneurysms greater than 60 mm[6]. Another study found that aneurysms over 60 mm had an annual 16% risk of dissection, rupture, or death[7]. Growing aneurysms also have an increased risk of rupture, even if smaller than 60 mm. As a result, general indications for surgery include: development of symptoms; diameter greater than 50 mm for ascending aortic aneurysms and 60 mm for descending aortic aneurysms, growth of greater than 10 mm per year, and evidence of dissection[8]. These indications lead to the following recommendations for follow-up of known thoracic aneurysms: 1) a repeat study in 6 months from aneurysm discovery, and, if stable; 2) annual studies thereafter to document stability. Sequential scanning using the same imaging modality (and even the same center or equipment, when possible) will likely yield the best results (Figure 6). When and if the above thresholds are crossed, surgical consultation is indicated.

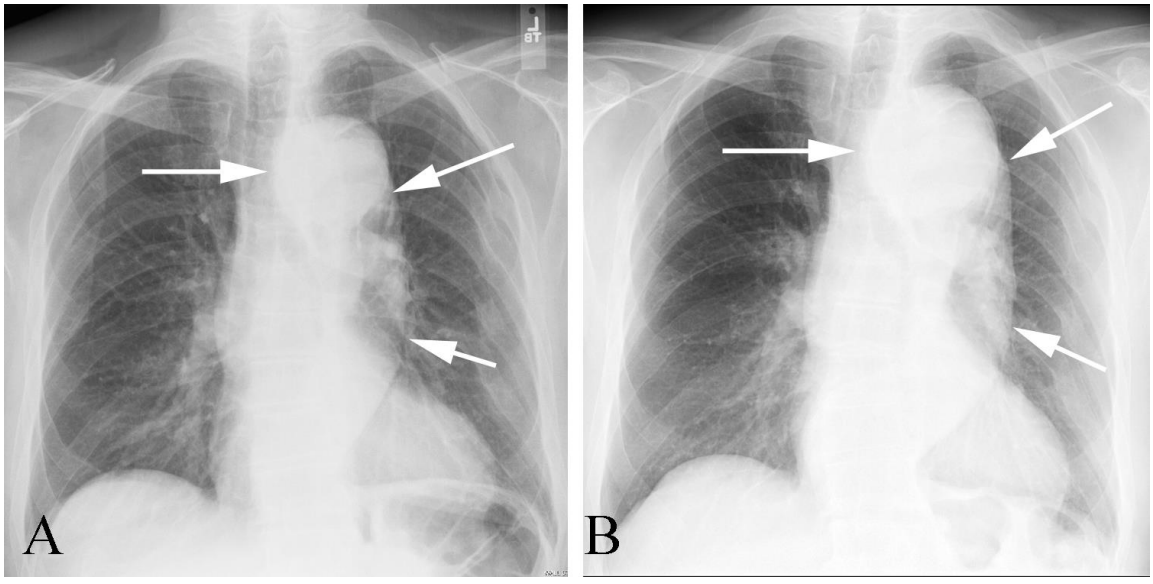

Figure 5. Enlarging thoracic aortic aneurysm in an 84 year old man with chest pressure. A. PA chest x-ray shows a dilated, tortuous aortic aneurysm (arrows). B. PA chest x-ray taken 18 months later (when the patient had a sensation of increasing chest pressure) shows that the aneurysm is larger (arrows).

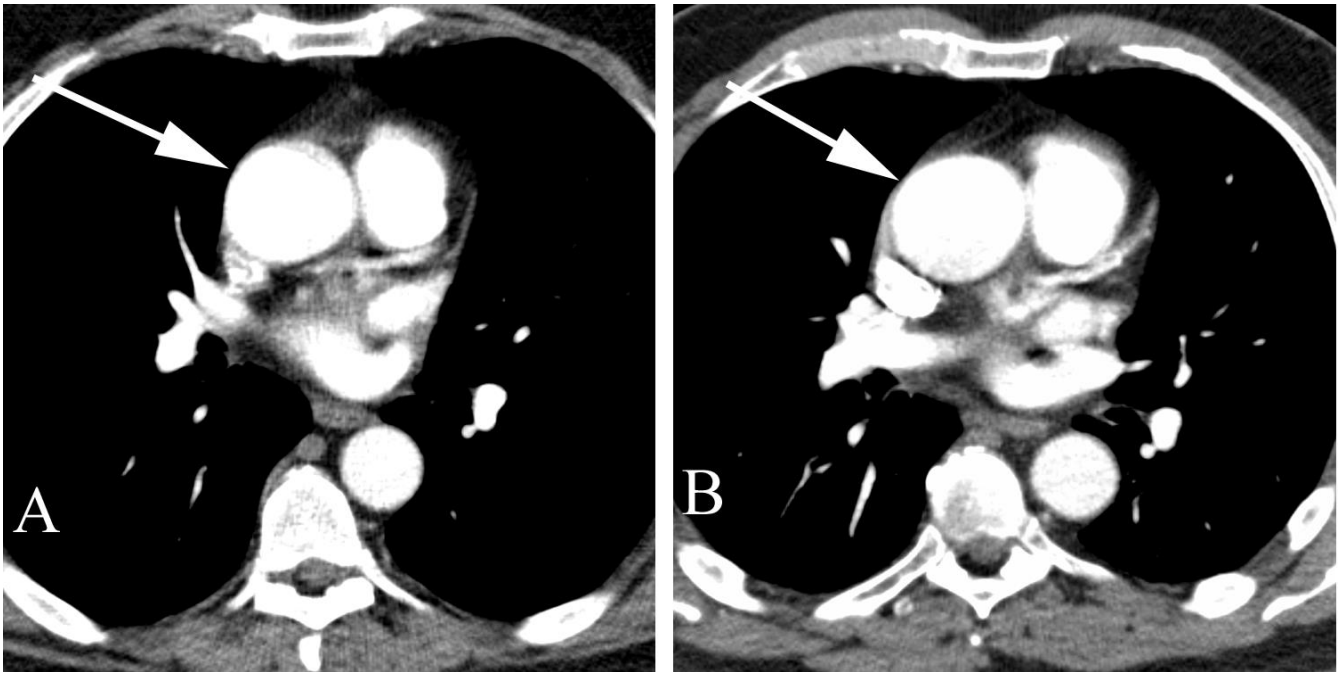

Figure 6. Stable thoracic aortic aneurysm in a 67 year old man with a known thoracic aortic aneurysm. A. Axial contrast-enhanced chest CT shows a thoracic aortic aneurysm which measured 51 mm. Annual studies were performed in follow-up. B. Axial contrast-enhanced chest CT done three years later shows an identical appearance and measurement. Interval annual studies (not shown) showed similar results.

Abdominal aortic aneurysm (AAA)

As with thoracic aortic aneurysms, patients with symptomatic abdominal aortic aneurysms are often direly ill and present to the emergency room with severe abdominal pain, if they don't exsanguinate first. These patients will undergo emergency abdominal CT followed by emergency surgery, if they are fortunate enough to survive. In general, symptomatic AAA's should undergo immediate evaluation by a surgeon[9]. Patients with AAA may also present with an otherwise asymptomatic pulsatile mass, in which case they should undergo US or CT evaluation.

Most AAA's are completely asymptomatic, however, and will be discovered either incidentally when imaging the abdomen for an unrelated abnormality or upon screening[10] (Figure 7). With respect to screening, patients may have an AAA after self-referral to a community screening program. Current recommendations call for screening any man greater than 60 years of age with a parent or sibling with an AAA, and for screening male smokers (or ex-smokers) between the ages of 65 and 75[11]. Aneurysms larger than 5.5 cm should be referred for surgical consultation. For aneurysms between 3.0 and 4.0 cm, US should be performed every 2 to 3 years, whereas aneurysms measuring 4.0 to 5.4 cm should be monitored every 6 – 12 months, with referral for surgical consultation if the aneurysm grows to greater than 5.5 cm, if the aneurysm grows more than 0.5 cm in a 6 month period[11], or if the aneurysm becomes symptomatic.

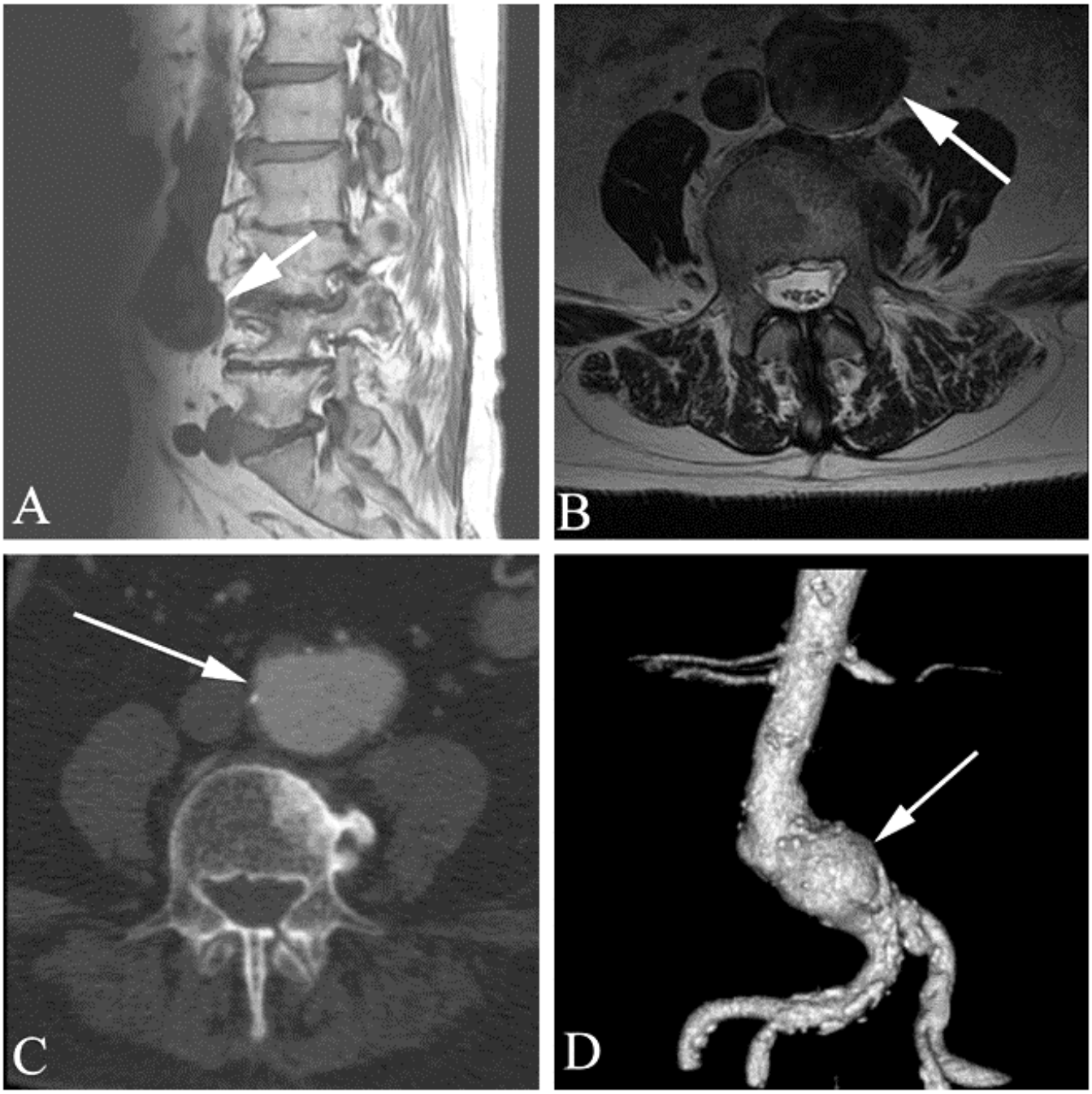

Figure 7. Abdominal aortic aneurysm incidentally discovered in a 74 year old with back pain undergoing lumbar spine MR study. A. Sagittal T1 weighted lumbar spine MR study shows an aneurysm of the lower abdominal aorta (arrow). B. Axial T2 weighted MR image through the L4 vertebral body level shows the aneurysm anterior to the lumbar spine (arrow). C. Contrast-enhanced axial CT angiogram shows the abdominal aortic aneurysm (arrow). D. Surface rendering based on CT data of the aneurysm, showing the location along the distal infrarenal abdominal aorta (arrow).

Other abdominal arteries (and veins)

Patients with chronic mesenteric ischemia or "intestinal angina" present with cramping abdominal pain following a meal (particularly a large fat-containing meal) which typically subsides approximately two hours after the meal[12]. These patients may have associated involuntary weight loss because of food avoidance caused by postprandial pain. CTA of the abdominal aorta and branches offers an excellent method of evaluation for suspected intestinal angina. Given the relatively rich anastomotic connections between the celiac artery, superior mesenteric artery, inferior mesenteric artery, and iliac artery branch vessels, stenosis or occlusion of only one branch rarely causes symptoms in the absence of simultaneous stenosis or occlusion of other branches, and therefore the diagnosis is usually made only when there are symptoms and stenosis or occlusion of at least two vessels[13].

Patients with acute mesenteric ischemia may have preceding symptoms of chronic intestinal ischemia, an "acute-on-chronic" situation where the patients have passed the threshold from intermittent to permanent symptoms. Patients with acute mesenteric ischemia usually have severe periumbilical pain[13]. Acute mesenteric ischemia may be caused by superior mesenteric artery embolism (50% of cases), superior mesenteric artery thrombosis (15-25%), mesenteric venous thrombus (5%) or nonocclusive ischemia (20-30%)[14], with each of these causes typically having a different clinical scenario (although the patients will all have abdominal pain regardless of the ultimate cause of the ischemia). Those with superior mesenteric embolism typically have cardiac disease with the embolism originating from the left atrium, left ventricle, or cardiac valves; those with acute thrombus usually have "acute-on-chronic" atherosclerotic disease of the mesenteric vessels with a prior history of intestinal angina (as noted above); those with acute venous thrombus may have a history of a hypercoagulable state (Figure 8), portal hypertension, abdominal infections, or abdominal trauma; and those with nonocclusive mesenteric ischemia (NOMI) are usually elderly patients, often with a cardiac condition being treated with drugs (e.g., diuretics) which reduce intestinal perfusion[14]. When acute mesenteric ischemia is strongly suspected, angiography is recommended since it is the reference standard for the diagnosis and may also allow for percutaneous intervention such as angioplasty[14]. When angiography is unavailable, or when the diagnostic suspicion is not high, CTA is an excellent alternative since it allows not only evaluation of the arterial tree but also evaluation of findings of associated bowel infarction (and other causes of abdominal pain).

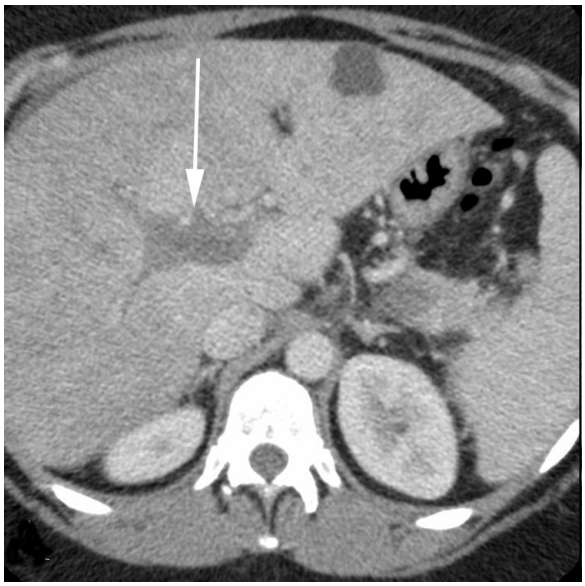

Figure 8. Portal venous thrombosis in a 34 year old woman on oral contraceptives with abdominal pain. Axial contrast-enhanced abdominal CT study during the portal venous phase study shows a clot filling the portal vein (arrow).

Evaluation of gastrointestinal bleeding typically relies on endoscopy. Occasionally, intermittent bleeding or bleeding with a low flow rate may be difficult to diagnose with endoscopy. In these cases, arteriography or CTA may be performed[14], but nuclear medicine studies allow diagnoses of lower rates of bleeding (even less than 0.5 mL/minute) and can reliably direct surgical intervention[15] (Figure 9).

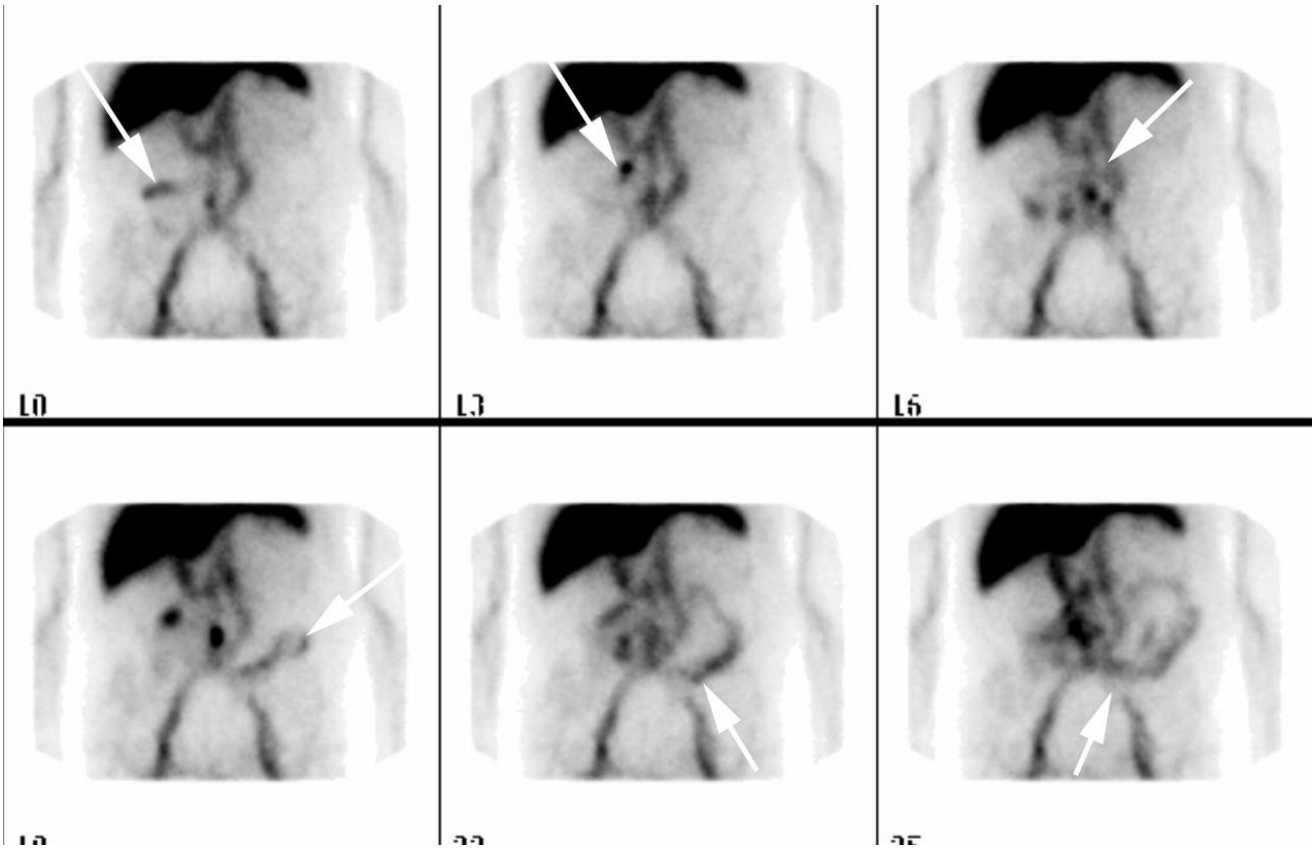

Figure 9. Gastrointestinal bleeding in a 75 year old. Sequential images (left to right, top to bottom) from a tagged red cell nuclear medicine examination demonstrate tracer in the right upper quadrant which on subsequent images shows a typical pattern of proximal small bowel distribution (arrows). The patient had already undergone upper and lower endoscopy without a clear source of hemorrhage; following the tagged red cell study he underwent repeat upper endoscopy which showed an ulceration along a duodenal diverticulum with active bleeding.

Lower extremity arterial evaluation

Symptoms of lower extremity peripheral arterial disease (PAD) include classic claudication, atypical leg pain, non-healing ulcers, and pain while at rest (when severe). However, up to 90% of patients with lower extremity peripheral arterial disease as determined by ankle-brachial ratio measurements may be asymptomatic[16]. For patients with symptoms, the initial diagnostic study is an ankle-brachial ratio determination, which involves comparison of the blood pressure at the ankle (using the posterior tibial or dorsalis pedis artery) with the higher of the right and left arm pressures: an ABI of <.90 is diagnostic of PAD (with pressures between .85 and .90 indicating mild arterial impairment, between 0.40 and 0.85 moderate arterial impairment, less than 0.39 severe arterial impairment), whereas an ABI of greater than 1.3 suggests calcified, noncompressible vessels which may also be a source of symptoms[17]. ABI readings between 0.91 and 1.0 are borderline and should be followed by an exercise exam wherein serial ABI calculations are made at 1-minute intervals following walking on a treadmill for five minutes at 2 mph on a 12% incline; ABI will typically remain stable or increase following exercise and decreases of 20% or more are diagnostic of PAD. For patients with symptoms and (either standard or exercise) abnormal ABI measurements who are candidates for revascularization, further evaluation can be performed with segmental pressure evaluation or CTA or MRA. Segmental pressure evaluation records the blood pressure at the ankle while inflating blood pressure cuffs at each of several lower extremity locations to better localize diseased vessels. Segmental pressure evaluation may be

supplemented by pulse volume recordings which are particularly helpful when extensive calcification makes vessels non-compressible. CTA and MRA can provide a visual "road map" of the lower extremity vascular tree documenting the location and quantifying the degree of arterial stenosis.

While screening for lower extremity PAD is not recommended for the general population, patients may self-refer to community screening programs and then come to the primary care practitioner with abnormal results. Indications for obtaining an ABI include evaluation of high risk patients, including those over 50 years of age who are smokers or diabetic[12].

LOWER EXTREMITY VENOUS STUDIES

Pain and swelling

Symptoms of acute deep venous thrombosis include pain and swelling of the calf. Other disease processes (e.g., muscle tear, lymphangitis, venous insufficiency, and Baker's cyst) may demonstrate similar clinical features, however, and distinction among these entities is important for patient management, since DVT can result in pulmonary embolism with significant associated morbidity and mortality. Typically, ultrasound examination consists of a combination of gray-scale imaging without and with compression where possible, color Doppler examination, and spectral Doppler examination with augmentation to assess for appropriate deep venous flow. Since reflux accounts for some cases of lower extremity pain and swelling even when there is no deep venous thrombus, it makes sense to routinely evaluate the saphenofemoral junction for reflux during Valsalva or during an upright position in those patients with lower extremity symptoms when the remainder of the study is normal. Documentation of either a filling defect (Figure 10) or lack of compressibility within the deep venous system is diagnostic of deep venous thrombosis. The examination will typically include compression evaluation of at least the popliteal and femoral veins*, and color flow images of the deep venous system including the calf to evaluate for filling defects. Note that clot may be isoechoic and therefore difficult or impossible to visualize without Doppler imaging and compression.

Once clot has formed, differentiation between persistent and recurrent deep venous thrombus by ultrasound is difficult[18]. Impedence plethysmography and MRI examination may be of benefit in these situations[19].

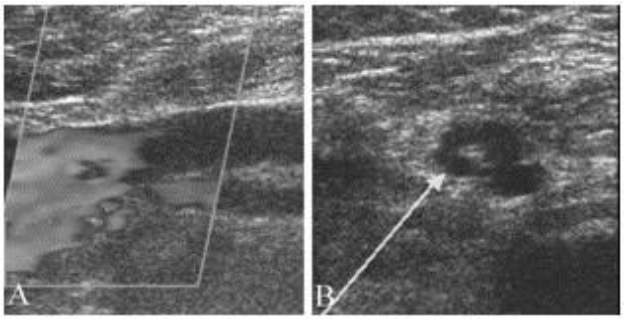

Figure 10. Deep venous thrombus in a 61 year old man with leg swelling and pain. A. Sagittal Doppler ultrasound study shows lack of filling in the distal femoral vein. B. Axial ultrasound study shows a filling defect within the lumen of the popliteal vein (arrow).

* Note that the preferred term for the vein formed by the continuation of the common femoral vein after the deep femoral vein has branched off, and connecting the common femoral vein to the popliteal vein, is the "femoral vein" and *not* the "superficial femoral vein" as indicated in some texts. The misleading term "superficial femoral vein" should be eliminated from use: the structure in question is part of the *deep* venous system and referring to the structure as "superficial" is a source of confusion which may have severe clinical consequences.

Varicose veins and valvular insufficiency

Patients with varicose veins from venous valvular insufficiency may have imaging performed of the deep *and* superficial systems prior to venous ablation procedures. Such evaluation typically includes the deep venous system for thrombosis (since valvular insufficiency and DVT may both result in leg swelling and pain) and documentation of the location and severity of venous valvular insufficiency. Such insufficiency is typically documented at the saphenofemoral junction by scanning during Valsalva or with the patient in an upright position. The examination can also document varicose veins and the caliber and depth of the saphenous veins (important for surgical planning).

SUMMARY

Modern imaging of the vascular tree typically involves a combination of ultrasound, CTA, and MRA. Catheter angiography is typically reserved for cases where therapy is necessary, or where detail beyond the current resolution of noninvasive vascular studies is necessary. Specific recommendations for imaging symptomatic patients and screening asymptomatic patients vary with the anatomic location and are reviewed above.

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Polyarthritis, Musculoskeletal Masses, and Osteoporosis

Donald L. Renfrew, MD

This chapter reviews imaging of "generalized" conditions of the musculoskeletal system. Chapter 14 deals with "particular" conditions of the musculoskeletal system. The three main points of this chapter are:

1. Evaluation of polyarthropathy relies on history, physical exam findings, and laboratory evaluation, with radiographs serving a minor, supporting role.
2. Most extremity masses are benign and not clinically significant. Imaging should be performed when malignancy is suspected or the cause is unclear on clinical evaluation.
3. Women over the age of 65 should have DXA to evaluate bone mineral density.

RADIOGRAPHS SERVE A MINOR, SUPPORTING ROLE IN EVALUATING POLYARTHRITIS

While this is a book on imaging, and while radiographs in arthritis may be dramatic and highly characteristic of specific diseases, imaging plays only a minor, supporting role in the initial diagnosis of polyarthropathy. Part of the reason for this is that while advanced rheumatoid arthritis (Figure 1), psoriatic arthritis, ankylosing spondylitis (Figure 2), calcium pyrophosphate dihydrate deposition disease (CPPD) (Figure 3), gout (Figure 4), and osteoarthritis (Figure 5) may have dramatic, obvious, and characteristic features, early in the course of these diseases (when the diagnosis is first made) the radiographic findings are usually much more subtle or even absent[1] (Figure 6).

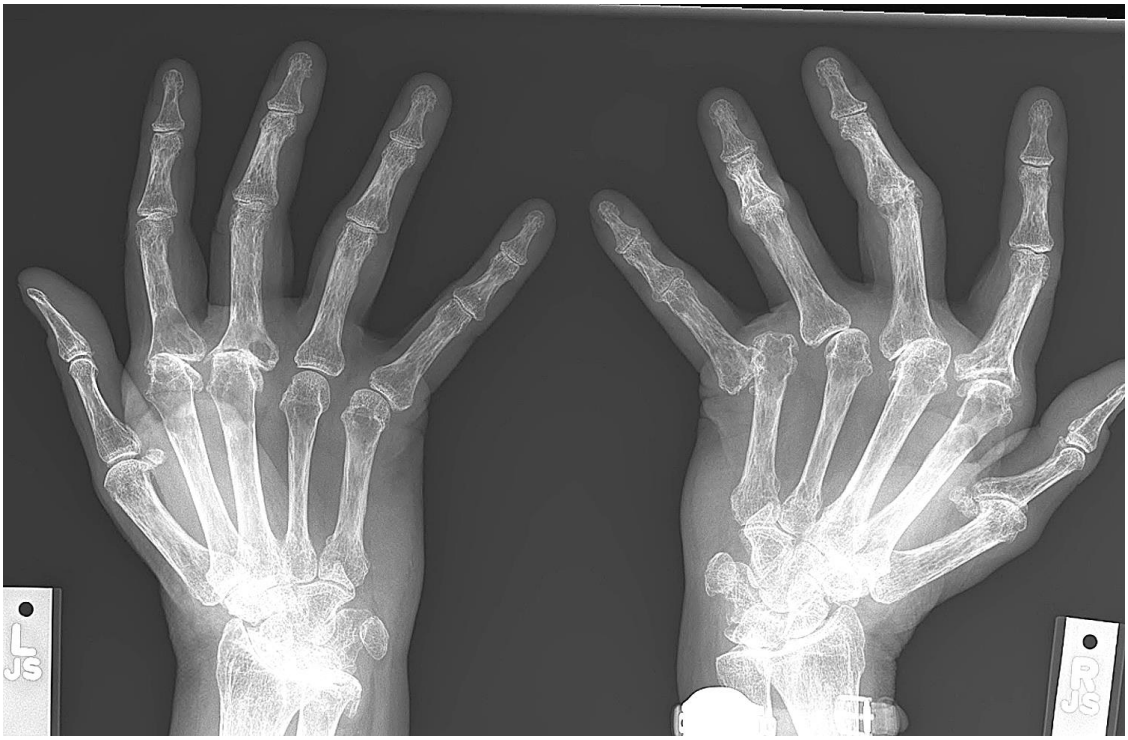


Figure 1. Severe rheumatoid arthritis in a 70 year old woman with chronic hand pain. "Ball-catcher's" view of the hands shows multiple classic features of rheumatoid arthritis including extensive multilevel metacarpophalangeal joint subluxation/dislocation, extensive erosions, demineralization, and carpal collapse.

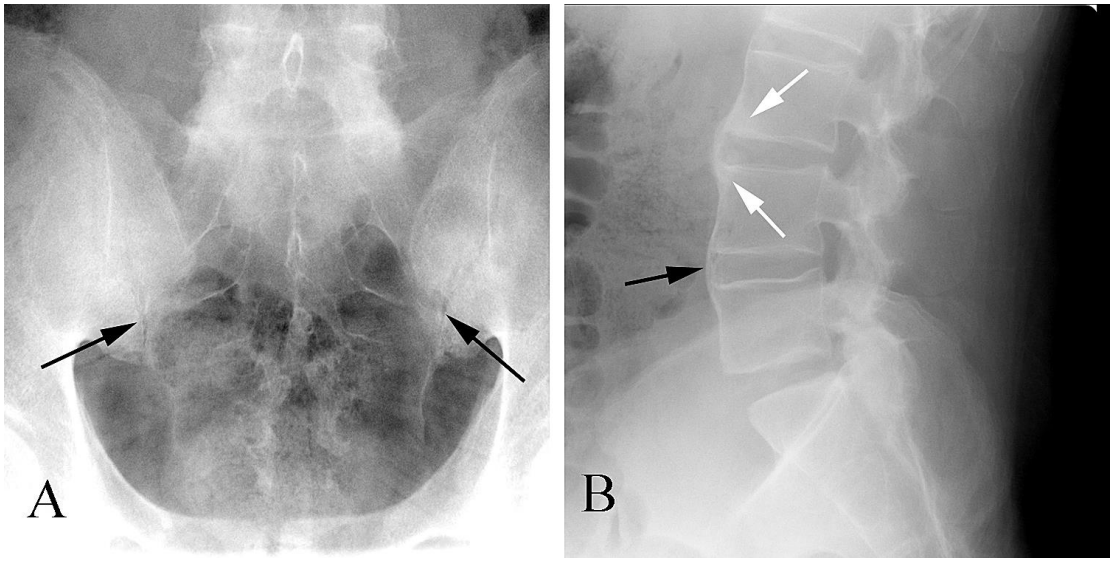


Figure 2. Ankylosing spondylitis in a 42 year old man with chronic back pain. A. AP plain film of the sacro-iliac joints shows erosions and loss of sharp definition along the inferior margins of the joints (arrows). B. Lateral plain film of the lower lumbar spine shows marginal syndesmophytes (black arrow) and "shiny corners" (white arrows) along the vertebral body margins.

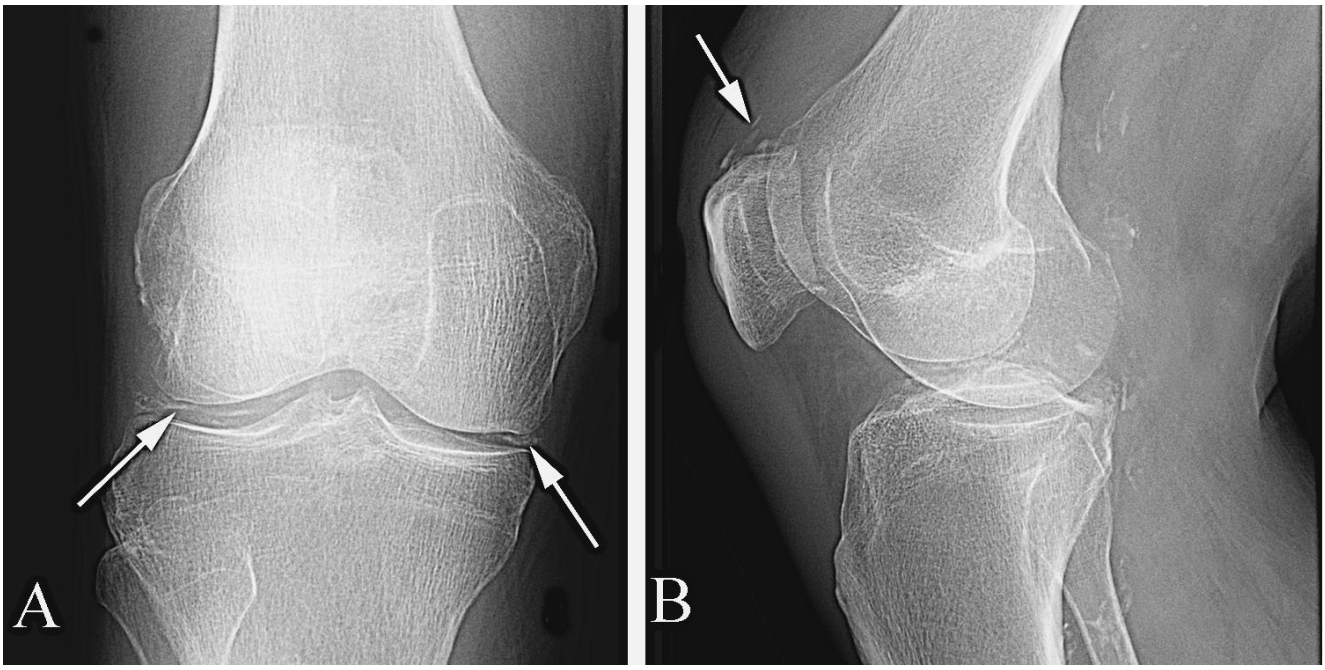


Figure 3. Calcium pyrophosphate dihydrate crystal deposition disease (CPPD) in a 75 year old man with knee pain. A. AP plain film shows chondrocalcinosis of both menisci (arrows). B. Lateral plain film shows a small joint effusion in the suprapatellar bursa as well as synovial calcification (arrow).

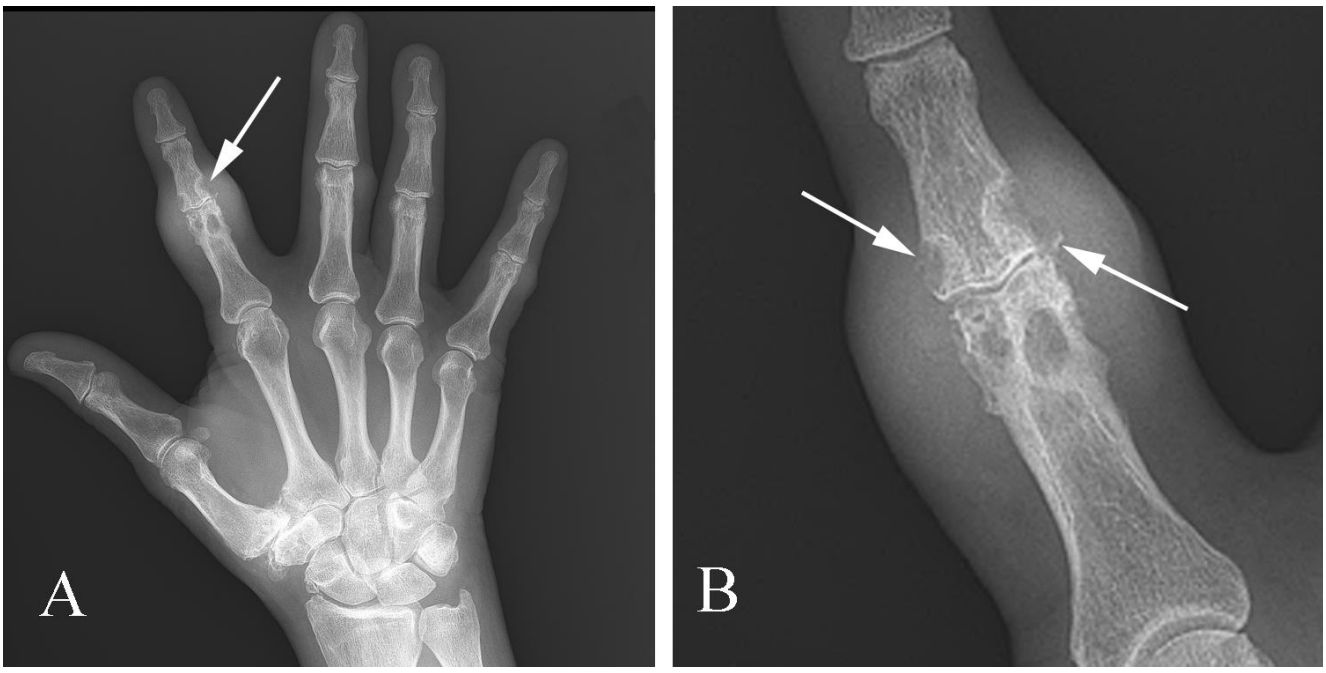


Figure 4. Gout in a 62 year old man with longstanding pain and swelling along the index finger proximal interphalangeal joint. A. AP plain film of the hand confirms soft tissue swelling of the index finger and shows several underlying cysts (arrow). B. AP plain film at a higher magnification confirms several cysts, which show overhanging edges (arrows) typical of gout.

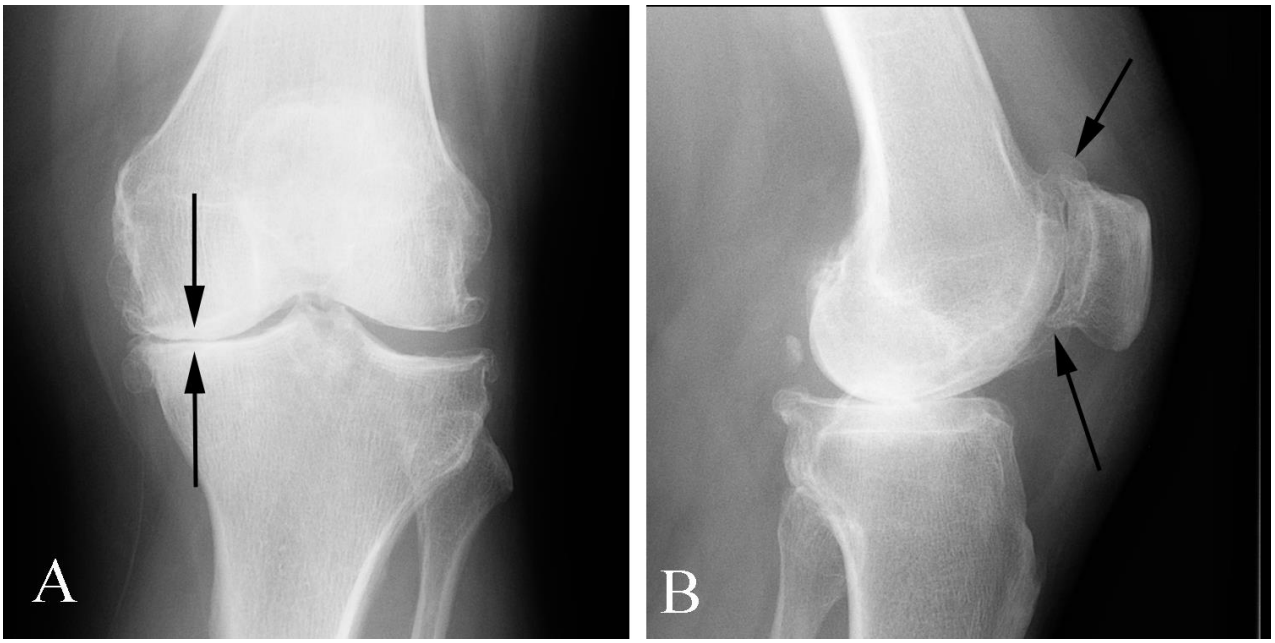


Figure 5. Osteoarthritis in a 58 year old woman with chronic knee pain. A. AP plain film of the knee shows osteophytic spurring, subchondral sclerosis, and medial compartment joint space narrowing (arrows). B. Lateral plain film shows extensive osteophytic spurring along the patellofemoral articulation (arrows).

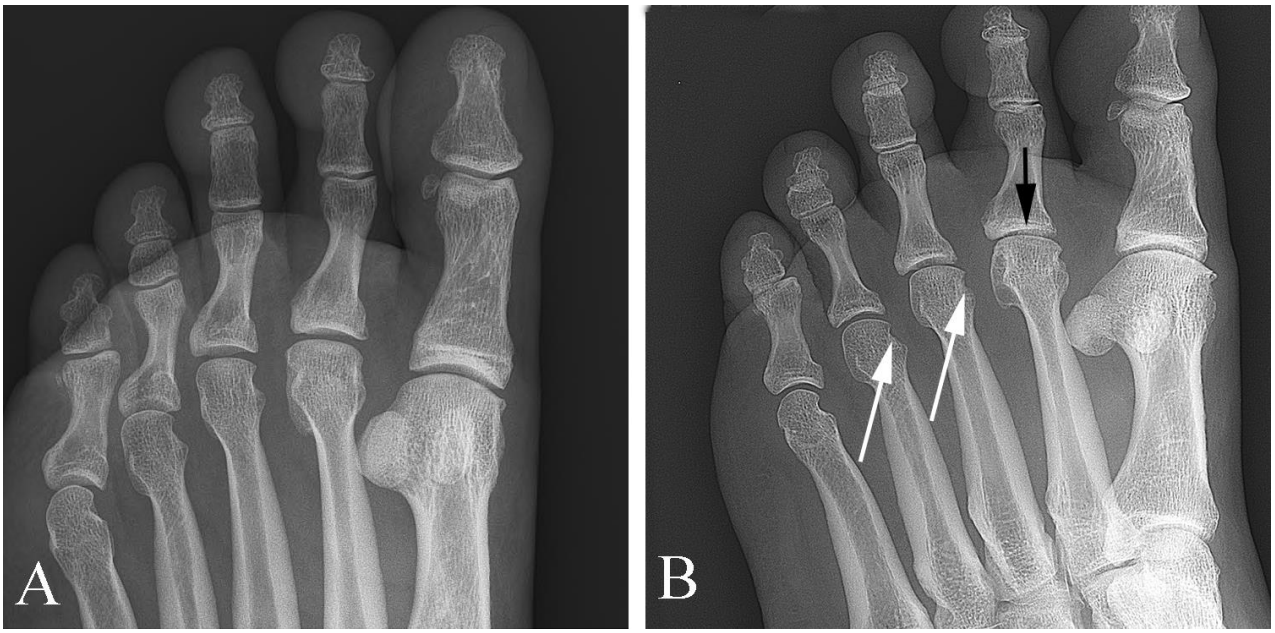


Figure 6. Early rheumatoid arthritis in a 49 year old woman with recent onset of morning stiffness and polyarthropathy. A. AP plain film (taken two years prior to the onset of symptoms because of post-traumatic pain) shows a normal appearance of the metatarsal bones and metatarsal-phalangeal joints. B. AP plain film (taken following the onset of symptoms) shows joint space narrowing at the second toe MTP (black arrow) and subtle erosions along the third and fourth metatarsal heads (white arrows).

The first step of evaluation in a patient with polyarthropathy needs to exclude the "must not miss" diagnosis of septic arthritis. Some of these patients may have a characteristic clinical history: a sexually active young woman with skin lesions (and gonococcal arthritis), a Wisconsin patient with a history of a tick bite (and Lyme disease), or a patient who has had a total joint replacement which now hurts following a skin infection elsewhere in the body (with hematogenous spread of organisms to the prosthesis). Most patients with septic arthritis and polyarthropathy will show at least some findings of systemic illness (e.g., fever and weight loss), with laboratory values of elevated white blood cell count (WBC), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Diagnosis typically relies on aspiration of joint fluid with demonstration of greater than 10,000 WBC/mcL consisting of at least 75% polymorphonuclear white cells[2]. Since the joint aspiration may show turbid, worrisome fluid but not yield a positive culture, blood cultures done at the same time may be helpful. Plain film findings typically lag well behind the clinical features of septic arthritis: early films show only a nonspecific joint effusion, while such dramatic features as destruction of cartilage or erosion of adjacent bone occur only late in the disease process (Figure 7).

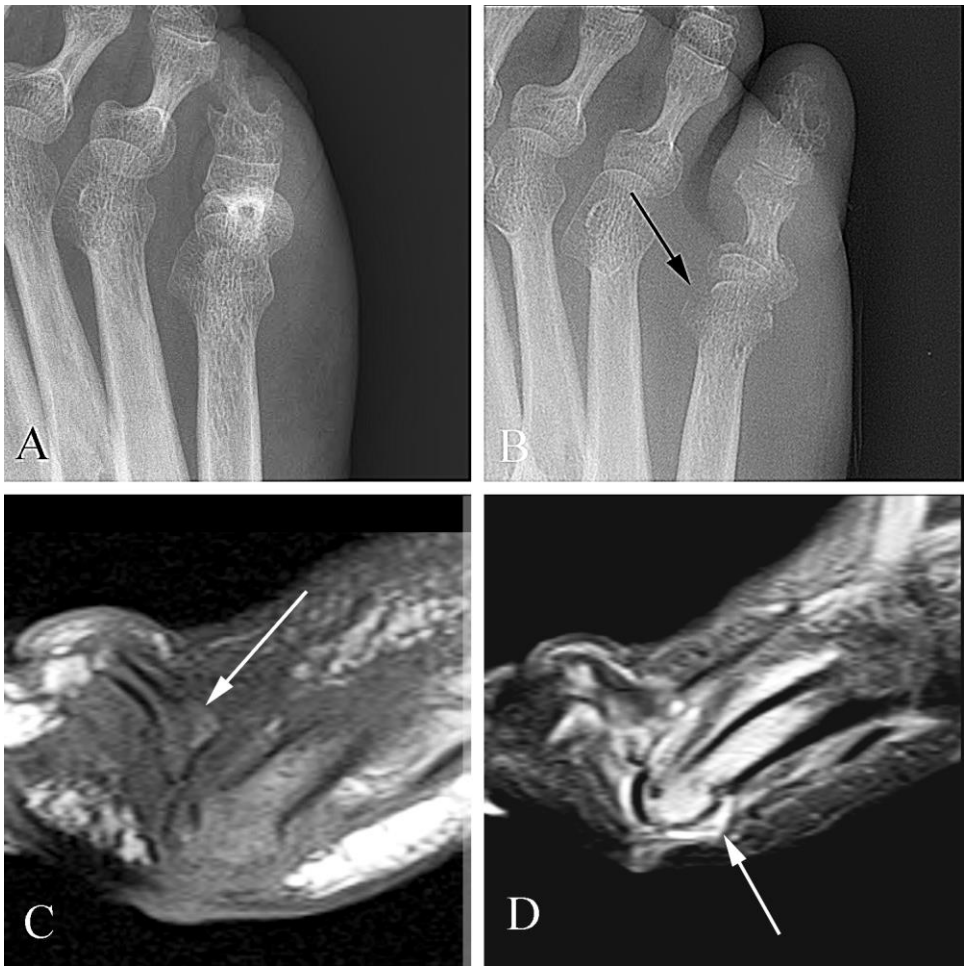


Figure 7. Septic arthritis and osteomyelitis in a 69 year old diabetic man with a draining ulcer along the base of the small toe metatarsal. A. AP plain film early in the course of the symptoms demonstrates an intact small toe metatarsal head. B. AP plain film six weeks later shows destruction of the metatarsal head (arrow) and proximal aspect of the proximal phalanx. C. Sagittal T1 weighted MR image shows dislocation of the proximal phalanx and decreased signal of the proximal phalanx (compare to the middle phalanx) and distal metatarsal, indicating osteomyelitis. D. Sagittal T2 weighted image shows fluid in the metatarsal-phalangeal joint (arrow) from septic arthritis.

If the history and physical examination do not strongly suggest septic arthritis, then multiple diagnostic considerations come into play. The ultimate diagnosis rests on a constellation of findings, since no one clinical feature, laboratory, or imaging test is definitive[3]. The usual first step is to determine whether the disease is inflammatory or not, which is established by the presence of morning stiffness (especially prolonged morning stiffness), and redness, warmth, and swelling of the afflicted joints. While crystal arthropathies (gout and CPPD) may cause inflammation, they typically present with monoarthropathy (see page 195) rather than polyarthropathy. Inflammatory polyarthropathies are much more likely to represent infections or postinfectious processes, or a rheumatologic disease, particularly rheumatoid arthritis, systemic lupus erythematosis (SLE), or psoriatic arthritis. In patients with inflammatory arthropathy, therefore, blood tests are helpful: rheumatoid factor and antibodies to citrullinated peptides may be positive in patients with rheumatoid arthritis, whereas antinuclear antibody is sensitive (but not specific) for SLE[1]. Note that experts caution against indiscriminate use of laboratory testing[3] since, for example, up to 25% of patients with rheumatoid arthritis may be seronegative, and many patients without rheumatoid arthritis will have a positive serum rheumatoid factor[1]. The American College of Rheumatology has provided diagnostic criteria[4] for the diagnosis of rheumatoid arthritis, with the presence of any four of the following seven features (generally present for at least six weeks) required for diagnosis: morning stiffness lasting one hour before maximal improvement; soft tissue swelling of 3 or more joint areas; arthritis of the hand joints; symmetric arthritis; rheumatoid nodules; serum rheumatoid factor; and typical radiographic changes of the hand and wrist. As noted above, the radiographic findings play a minor and supportive role, representing only one of seven features and not representing an absolute requirement for the diagnosis (the patient may have any combination of four of the seven features for the diagnosis).

To confuse matters further (at least radiographically) patients with psoriatic arthritis may present in any of at least three different fashions, one of which bears a strong clinical (and radiographic) resemblance to rheumatoid arthritis (the other two are dactylitis with a "sausage digit" and spinal arthritis[2]). While the characteristic skin changes and/or nail pitting of psoriasis precede (or occur about the same time as) the arthritis in 85% of cases, in 15% of cases the skin manifestations only occur after the onset of the arthropathy[2].

Noninflammatory polyarthropathy almost always represents osteoarthritis, in which case the diagnosis is usually straightforward since the clinical features including *lack* of morning stiffness, aggravation with motion, and improvement with rest, are relatively characteristic. Plain films may document joint narrowing, osteophytic spurring, subchondral sclerosis, and subchondral cyst formation (Figure 5).

MOST EXTREMITY MASSES ARE BENIGN AND NOT CLINICALLY SIGNIFICANT

Ganglia and nodules represent most soft tissue masses and are benign, of little clinical consequence, and do not require imaging[5]. Ganglia represent collections of cystic or gelatinous material located near a joint or tendon, and likely represent an outpouching of joint synovium or the tendon sheath containing thickened or solidified fluid, particularly after communication is lost with the parent structure. Ganglia typically transilluminate, and surgeons will usually resect such lesions on the basis of the clinical examination without imaging. Even prior to surgical resection, office methods including aspiration and injection of steroids should be performed, as this may be successful in over 80% of patients[6]. One location where imaging may be useful in the evaluation of ganglia is in the wrist, as MR may reveal occult ganglia in patients with chronic wrist pain[7] (see Figure 26 page 209) – although in this instance the patient really does not have a palpable lesion (as the ganglia are occult).

Soft tissue nodules arise in a variety of conditions including repetitive trauma, silicone injection, rheumatoid disease, sarcoidosis, and vasculitis.

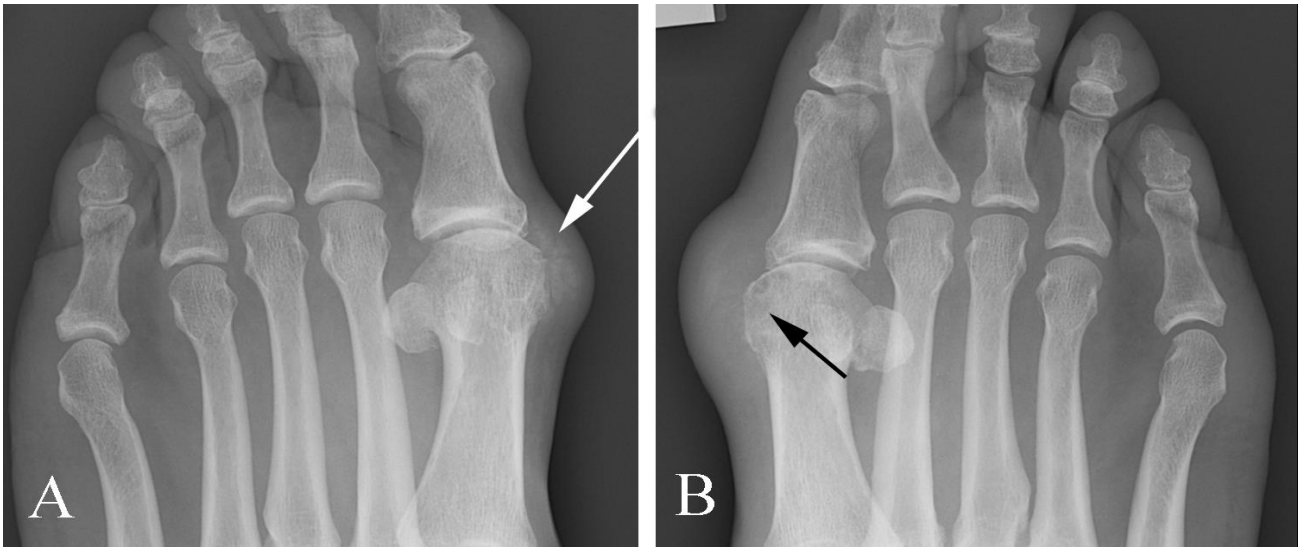


Figure 8. Gouty tophus in a 64 year old man with bilateral palpable lumps along the great toe metatarsal-phalangeal joints. A. Oblique plain film of the right foot shows a calcified soft tissue mass adjacent to the great toe metatarsal head (arrow). B. Oblique plain film of the left foot shows a tophus and also an underlying erosion of the great toe metatarsal head (arrow).

Nodules may also represent epidermoid cysts or gouty tophus (Figure 8). Multiple lesions tend to be infectious or inflammatory while solitary lesions may represent a noninflammatory nodule or tumor (Figure 9).

In cases where imaging is contemplated (again, not necessary unless the diagnosis is uncertain or malignancy is suspected), the three main imaging modalities to consider are:

Plain film evaluation

Plain film evaluation of bone tumors has traditionally been the method of choice for evaluation of suspected bone tumors, since it allows histologic characterization of many tumors (e.g., osteosarcoma, benign exostosis). In palpable lesions arising in soft tissue, plain films may also provide useful additional information by showing either calcification of the lesion or characteristic changes in the adjacent bone or joint allowing a diagnosis. Plain films may also show features of a lipoma or a cluster of phleboliths characteristic of hemangioma. Acute inflammatory processes may be associated with periostitis of the adjacent bone, while chronic indolent masses may produce smooth remodeling of the adjacent bone, usually a feature associated with slow-growing, benign lesions. Frequently, however, plain films obtained for a palpable soft tissue mass are unremarkable.

Ultrasound

Ultrasound is most helpful in those situations where it is necessary to differentiate a cystic lesion from a solid one, since ultrasound is nearly 100% accurate in this task. Ultrasound may also be helpful in distinguishing lesions with internal flow on color Doppler imaging (e.g. tumors) from those without internal flow (e.g. blood clots), and for demonstration of suspected vascular malformations. Unfortunately, distinguishing one histologic type of tumor from another is usually not possible with ultrasound, and evaluation of adjacent bones and joints for secondary helpful diagnostic features is not as easy as with plain films (see above).

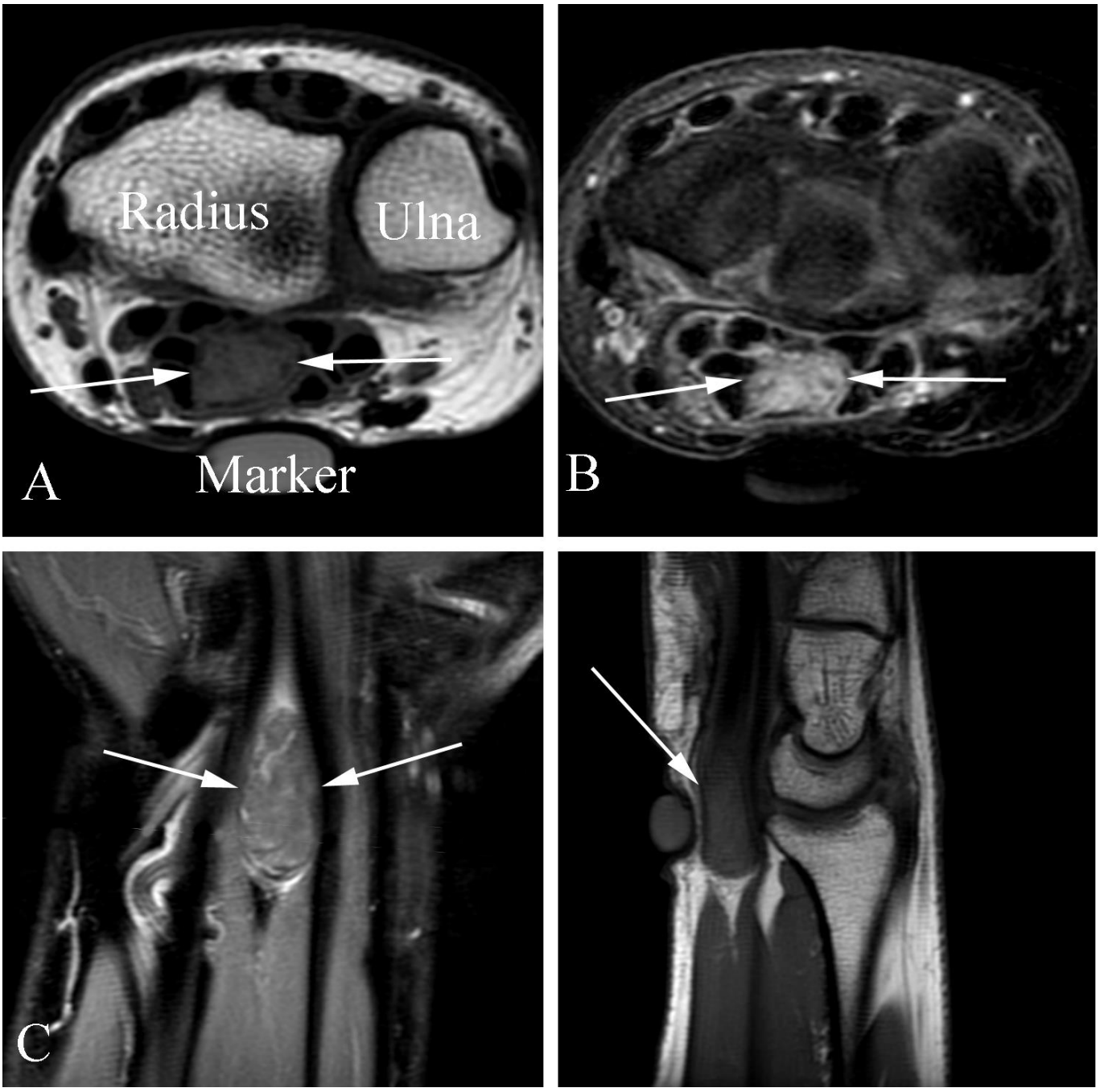


Figure 9. Giant cell tumor of the tendon sheath in a 50 year old woman with a palpable wrist mass. A. Axial T1 weighted MR study shows a soft tissue mass (arrows) between flexor tendons along the anterior aspect of the distal forearm near the radiocarpal joint. B. Axial T2 weighted image demonstrates the mass, which shows some T2 prolongation (increased signal intensity on the T2 weighted image). C. Coronal fat-suppressed proton density image demonstrates the mass (arrows) interposed between flexor tendons. D. Sagittal T1 weighted image demonstrates the isointense mass (arrow) deep to the marker, along the ventral aspect of the wrist superficial to the distal radius and the lunate bone.

Magnetic resonance imaging

Magnetic resonance imaging has supplanted CT in the evaluation of extremity soft tissue masses. It *may* allow a histologic diagnosis in the case of lipomas and allows near certainty in many other lesions (e.g. cysts and arteriovenous malformations). MR does not always allow histologic characterization, however, and in general cannot

determine with complete accuracy whether a lesion is malignant or benign[8]. However, using available tables breaking down tumor types by location versus age and comparing the frequently seen lesions with the imaging features, it is usually possible to provide a brief differential diagnosis which contains the correct diagnosis[9]. In addition, MR allows determination of important anatomic features of the tumor such as whether it is confined to its compartment of origin and whether it displaces or invades critical adjacent structures (for example, the neurovascular bundle). These factors figure into the orthopedic oncologist's determination of the optimal approach or even whether the tumor is resectable.

One important caveat regarding apparent primary musculoskeletal tumors: it is best to proceed with biopsy of these lesions only *after* consultation with an orthopedic oncologist. Surgery for malignancy involves the resection of any tissue which may have come into contact with (and been seeded by) the neoplastic tissue. In the event that the tumor is malignant (which you won't know until after the biopsy is performed), the orthopedic oncologist will need to resect the tract leading from the skin to the biopsy location. The surgeon will therefore want to direct the biopsy path to optimize results.

WOMEN OVER THE AGE OF 65 SHOULD HAVE DXA TO EVALUATE BONE MINERAL DENSITY

Osteoporosis is largely a silent disease without symptoms prior to (what may be a devastating) hip fracture. Indeed, fragility fractures of the spine are often asymptomatic and discovered when imaging the skeletal system for another purpose[10], for example, the thoracic spine fracture found on a chest radiograph (Figure 10) or the lumbar spine fracture found on an abdomen and pelvic CT (Figure 11).

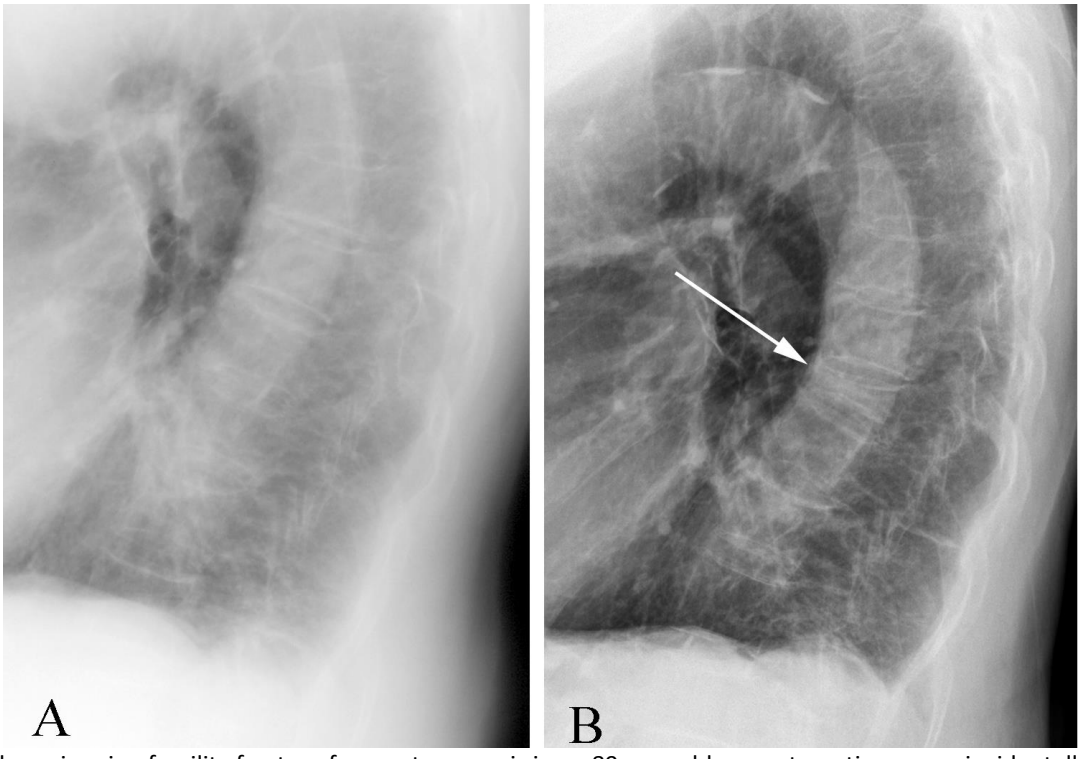
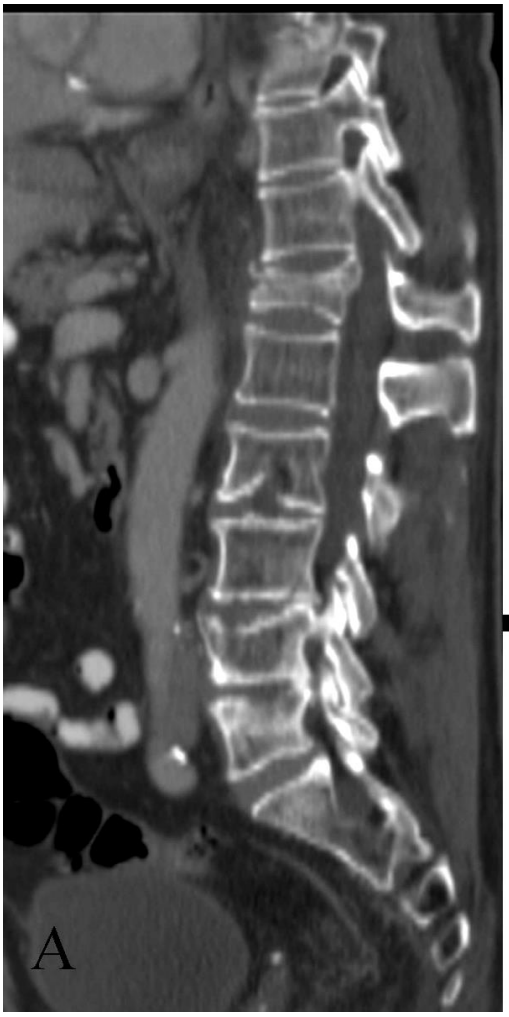
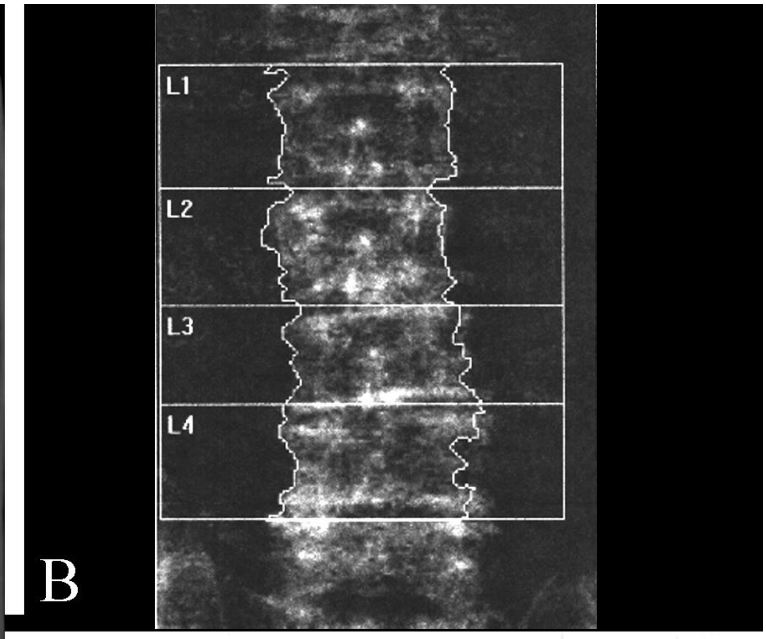


Figure 10. Thoracic spine fragility fracture from osteoporosis in an 89 year old asymptomatic woman incidentally discovered on a lateral CXR done for admission to a nursing home. A. Lateral from a prior CXR (cropped to show detail better) done 8 years previously shows normal vertebral body heights. B. Lateral from the admission CXR shows wedging of a thoracic vertebral body, classic for an osteoporotic compression fracture.





DXA Results Summary:

Region	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T - Score	Z - Score
L1	17.40	9.48	0.545	-4.2	-3.1
L2	16.49	12.17	0.738	-3.2	-2.0
L3	13.93	10.79	0.774	-3.0	-1.7
L4	17.87	14.52	0.813	-3.0	-1.7
Total	**65.69**	**46.96**	**0.715**	**-3.4**	**-2.2**

C

Figure 11. Incidentally discovered lumbar spine fragility fracture in an 81 year old man undergoing CT for abdominal pain. A. Sagittal reformatted CT examination shows T12, L2, L4, and L5 fractures. B. AP view of the patient's spine from his DXA study. C. Results summary from the DXA study showing osteoporosis.

At the same time, osteoporosis is an important disease to diagnosis. It is a widespread condition: in the USA there are over 1,500,000 fractures annually, including over 250,000 hip fractures[11], numbers which will only increase with the aging of baby boomers and the associated shift of the "demographic bulge" to a higher age. It is associated with significant morbidity and mortality: 50% of hip fracture patients will not be able to walk without assistance, 25% will require long-term assistance, and there is a 10-20% mortality rate in the six months following fracture[11]. It is a source of tremendous medical expense: estimates of cost were $10 billion in 1995[11]. Finally, there is effective treatment for the condition, with nonpharmacologic and pharmacologic therapy resulting in a considerable decrease in the risk of fracture[10].

Fortunately, there is an excellent tool for reproducible, cost-effective measurement of bone mineral density (BMD) with a minimum of radiation exposure: dual energy x-ray absorptiometry (DXA). While other methods of measurement of bone mineral density (for example, quantitative ultrasound and quantitative CT) exist[12], DXA is preferred because the World Health Organization reference data were obtained by DXA[13] and DXA measurements are incorporated into the WHO diagnosis and treatment guidelines. Prospective cohort studies have demonstrated a strong relationship between fracture risk and BMD

measured by DXA[14], and randomized trials have shown a reduction of fracture risk with drug therapy based on DXA results[15].

To determine which patient may benefit from DXA, it is necessary to assess multiple factors including age, sex, menopausal status, body mass index, cigarette smoking, family history of osteoporotic fracture in a first degree relative, and use of oral corticosteroids[16]. Women with elevated risk factors should be screened at least by age 60, and all women should probably undergo screening by age 65 even if they have no risk factors for osteoporosis[16]. Screening in men is less well established, but men account for approximately 20-30% of all hip fractures with an associated high mortality rate[16], so screening should probably be considered for men over the age of 75 and for those with a history of oral corticosteroid use, alcohol abuse, or hypogonadism[16].

Regarding which part of the body to study, the patient's overall risk of fracture may be estimated by measurement of any location, but fracture risk of a particular location (spine, hip, or forearm) is best estimated by measurement at that location[17]. Therefore, hip measurement best predicts the likelihood of a hip fracture (more likely to be disabling). However, spine measurements are more sensitive to both loss of bone mineral density and regained density following treatment[17]. Therefore, both hip and spine measurements are typically included in evaluation for osteoporosis. The report for a DXA study typically includes the BMD, a "Z-score" which is the number of standard deviations that the patient's BMD is from an *age matched cohort*, and a "T-score" which is the number of standard deviations that the patient's BMD is from *peak bone mineral density in a young person*. In addition, some reports may include a FRAX score, which is the 10 year likelihood of fracture based on a risk assessment tool developed by the WHO in 2008[12]. Further evaluation and treatment must, of course, be individualized, but triggers for treatment consideration include a T-score of < -2.0, or (if FRAX is available) a 10-year hip fracture risk of 3.0% or 10year overall fracture risk of 20%[10]. Note that using FRAX instead of the T-score as the basis of treatment will result in treating more older patients with higher (better) T-scores versus younger patients with lower (worse) T-scores because age is an independent predictor of fracture[12]. This makes sense, particularly considering that more fractures occur in *osteopenic* (defined as a T-score between -1.0 and -2.5) rather than *osteoporotic* (defined as a T-score of less than -2.5) patients, simply because there are so many more osteopenic patients than there are osteoporotic ones[12]. Nonetheless, for a *given* patient, decreases in BMD (and lower T-scores) are associated with increases in fracture risk. Note that a low Z-score (more than 2 standard deviations below the age-matched control group) should prompt investigation of an underlying cause beyond simply postmenopausal osteoporosis, e.g. glucocorticoid therapy or alcoholism[10].

Since up to one-sixth of patients taking bisphosphonates may continue to lose bone[10] following institution of therapy, follow-up studies should be performed, usually at a two year interval. Follow-up studies should typically be performed on the same machine, because it is not possible to evaluate changes in BMD unless cross-calibration has been performed[13]. In patients with a significant BMD decrease following treatment, further evaluation may include evaluation of therapy adherence, gastrointestinal absorption of medication, adequacy of vitamin D and calcium intake, or work-up for the development of another disease which may adversely affect bone mineral density[10].

SUMMARY

In patients with polyarthropathy, plain films play a minor, supportive role. Most extremity masses are benign, self-limited, and do not require imaging. Women over the age of 65 should have DXA to evaluate bone mineral density.

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Single Joint Pain

Donald L. Renfrew, MD

This chapter reviews imaging of symptoms confined to single joints within the musculoskeletal system. The main points of this chapter are:

1. Monoarthropathy may be a precursor of polyarthropathy.
2. Imaging should be tailored by which joint (shoulder, elbow, wrist, etc.) is symptomatic. When imaging is required, plain film evaluation should generally precede use of computerized tomography (CT), magnetic resonance imaging (MRI), arthrography, and nuclear medicine studies.

MONOARTHROPATHY MAY BE A PRECURSOR OF POLYARTHROPATHY

Since virtually any arthritis may initially present as a single joint abnormality, pain in a given joint may represent the first manifestation of polyarthritis[1]. In the absence of trauma, patient history and physical examination results will help to determine whether there are additional features (e.g., rash, fever, weight loss, etc.) suggesting a systemic process which may be manifesting as monoarthropathy. Other than trauma, infection (especially gonococcal) and crystal arthropathy (predominantly gout and calcium pyrophosphate dihydrate) are the main causes of monoarthropathy. Analysis of aspirated joint fluid is the key to making a diagnosis in most cases[1], and imaging typically does not add significant information. For further discussion of arthropathy, see Chapter 13.

IMAGING SHOULD BE TAILORED TO WHICH JOINT IS SYMPTOMATIC IN GENERAL, PLAIN FILM EVALUATION SHOULD PRECEDE USE OF CT, MRI, ARTHROGRAPHY, AND NUCLEAR MEDICINE

When imaging is used for evaluation of joint pain, plain films are the first step, as these are readily available, relatively cheap, and often straightforward to interpret. It is difficult to generalize regarding imaging recommendations when comparing the different joints in the body, since they are prone to different diseases. More complex (and expensive) imaging studies such as CT and MRI need to be tailored to the joint, symptoms, and suspected disease processes in each case.

Shoulder

Evaluation of acute post-traumatic shoulder pain starts with a plain film examination[2] for evaluation of fracture (Figure 1) and dislocation (Figure 2). If the plain film examination shows a complex fracture

requiring surgical fixation, CT may be obtained to evaluate the exact anatomy of the fracture, but the decision to order the CT is typically left to the orthopedic surgeon (Figure 3). If the plain film is negative but your suspicion of significant injury is high, order an MR study: the MR study will show both occult fractures (Figure 4) and any significant soft tissue injury (Figure 5).

Evaluation of chronic shoulder pain (either with or without a history of trauma) also starts with plain films. The plain film examination will demonstrate calcific tendinitis (Figure 6), chondrocalcinosis (Figure 7), and degenerative change (Figure 8). Note that as the shoulder is not a weight bearing joint, degenerative changes should raise the suspicion of underlying, causative pathology (e.g., crystal arthropathy, prior fracture, or longstanding full thickness rotator cuff tear). For patients with chronic shoulder pain requiring further evaluation after the plain film, MR is the study of choice. In younger patients, particularly athletes where shoulder instability is an issue, the MR should be done following an arthrogram, since labral tears are much easier to identify and assess following introduction of contrast material into the shoulder joint (Figure 9). For older patients, MR done without contrast material is usually adequate to exclude rotator cuff tear (Figure 10), although some pinhole tears may be difficult to diagnose without intra-articular contrast and some authors advocate intra-articular contrast for all shoulder MR studies[3]. Regarding full thickness rotator cuff tears, note that many older individuals may have asymptomatic full thickness rotator cuff tears: Sher et al[4] demonstrated that for *asymptomatic subjects* over the age of 60, 28% had partial thickness, and 26% had full thickness tears at magnetic resonance imaging. For patients between 40 and 60 years of age, 24% had partial thickness and 4% full thickness tears, whereas for those under the age of 40, only 4% had partial thickness and none had full thickness tears. A full thickness rotator cuff tear in a patient over 60 years of age may not be the cause of the patient's shoulder pain.

An additional clinical scenario to consider in shoulder evaluation is that of a patient who has had a prior rotator cuff repair. Rotator cuff repair, capsulorrhaphy, and acromioplasty may incite pain in the shoulder joint which is similar to pain from a torn (or retorn) rotator cuff [3]. MR is the usual method of choice for imaging evaluation of patients who have undergone rotator cuff repair. It is of note that such repairs are not necessarily water tight (and therefore will leak contrast at arthrography), but any large gap should certainly cause concern for a re-rupture.

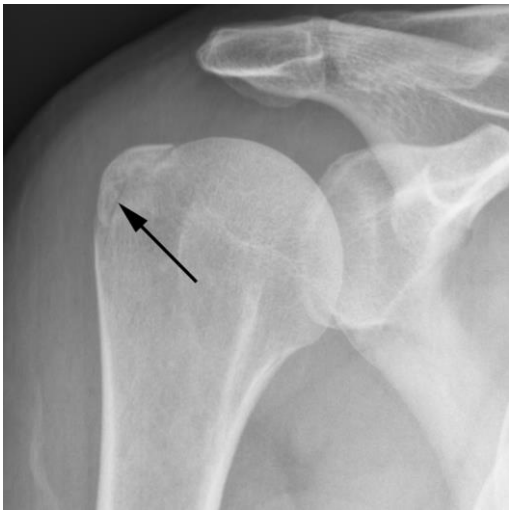

Figure 1. Humeral fracture in a 38 year old man with shoulder pain following trauma. AP plain film shows a fracture (arrow) through the base of the greater tubercle of the proximal humerus.

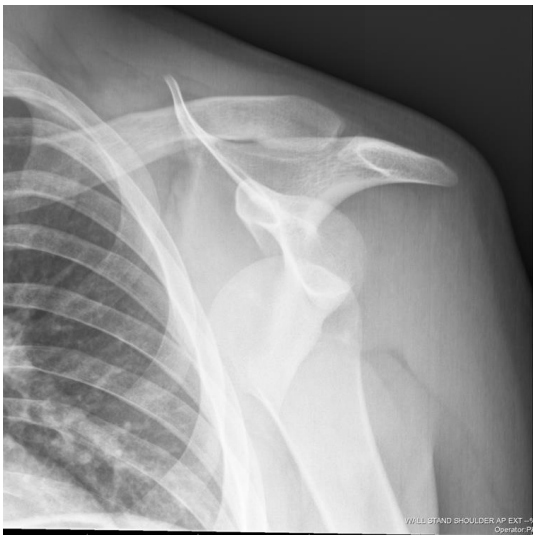

Figure 2. Shoulder dislocation in a 17 year old with pain following trauma. AP plain films shows a humeral head dislocation in the typical anterior, inferior location.

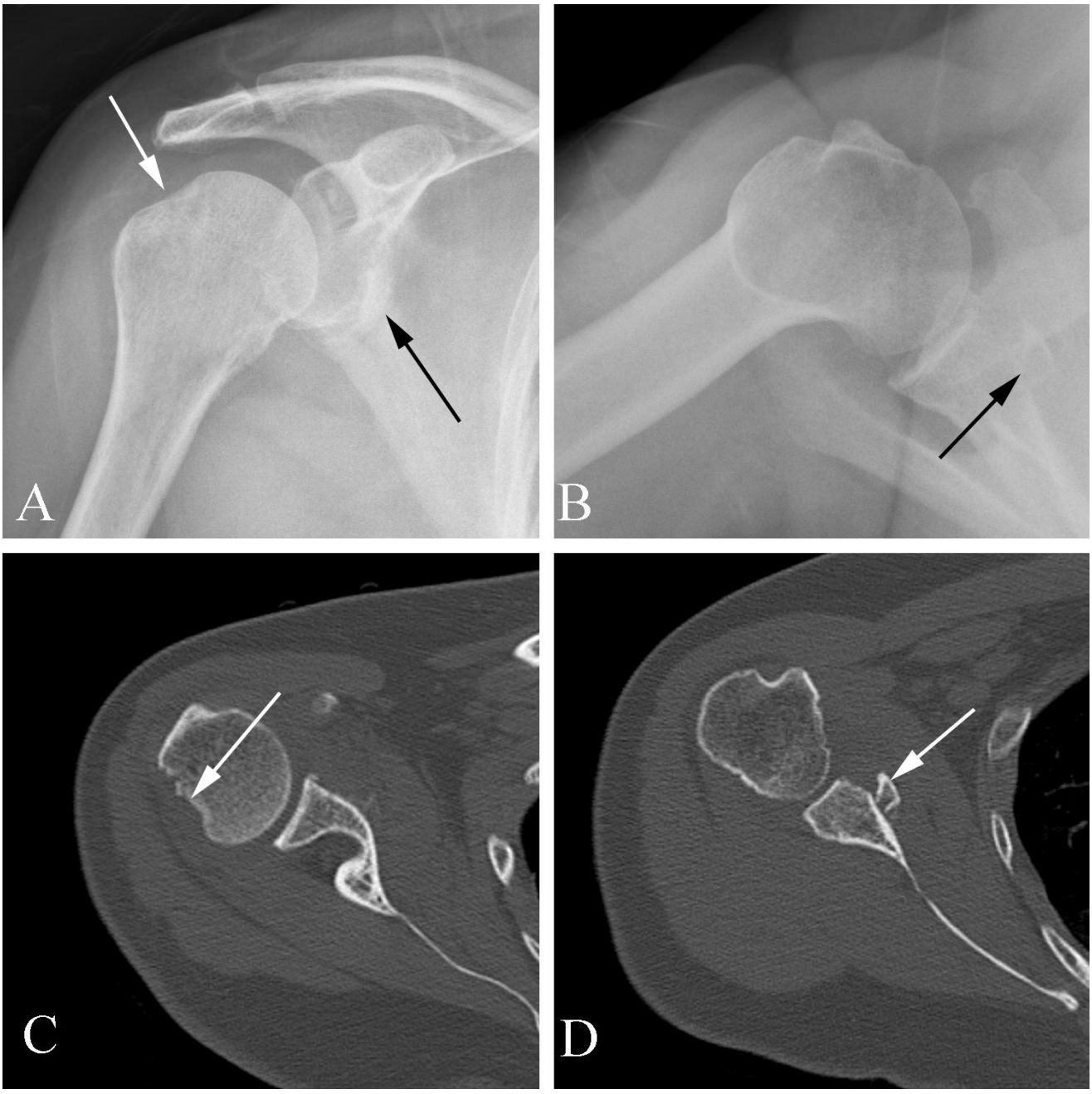

Figure 3. Complex shoulder fracture in a 65 year old woman with pain following trauma. A. AP plain film examination shows a "dent" in the superolateral humeral head (arrow), the Hill-Sachs deformity. B. Axial plain film examination shows discontinuity of the glenoid with a medially displaced bone fragment (arrow). C. Axial CT study at the level of the upper glenohumeral joint shows indentation of the humeral head (arrow). D. Axial CT study at the level of the lower glenohumeral joint shows a fracture of the anterior inferior glenoid rib with a medially displaced bone fragment. The imaging features are characteristic of a dislocation of the glenohumeral joint with associated fractures.

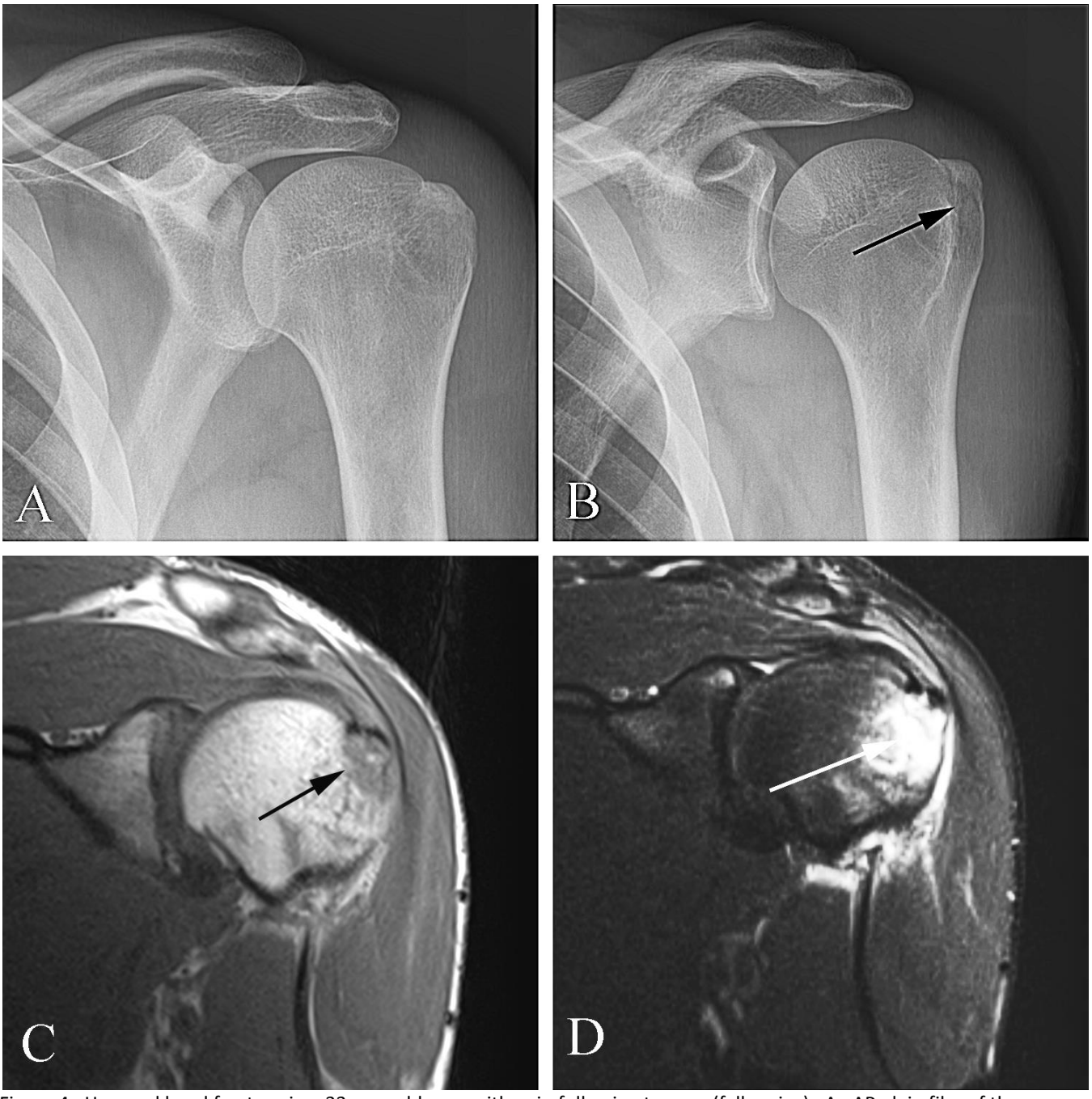

Figure 4. Humeral head fracture in a 33 year old man with pain following trauma (fall on ice). A. AP plain film of the shoulder with the patient's shoulder in internal rotation shows no displaced fracture. B. AP plain film of the shoulder with the patient's shoulder in external rotation suggests a possible subtle lucency (arrow) suggesting fracture, although the plain film features are not definitive. C. Coronal oblique T1 weighted MR study shows abnormal marrow and a fracture line along the base of the greater tubercle (arrow). D. Coronal oblique T2 weighted image confirms extensive marrow abnormality and a fracture through the base of the greater tubercle (arrow).

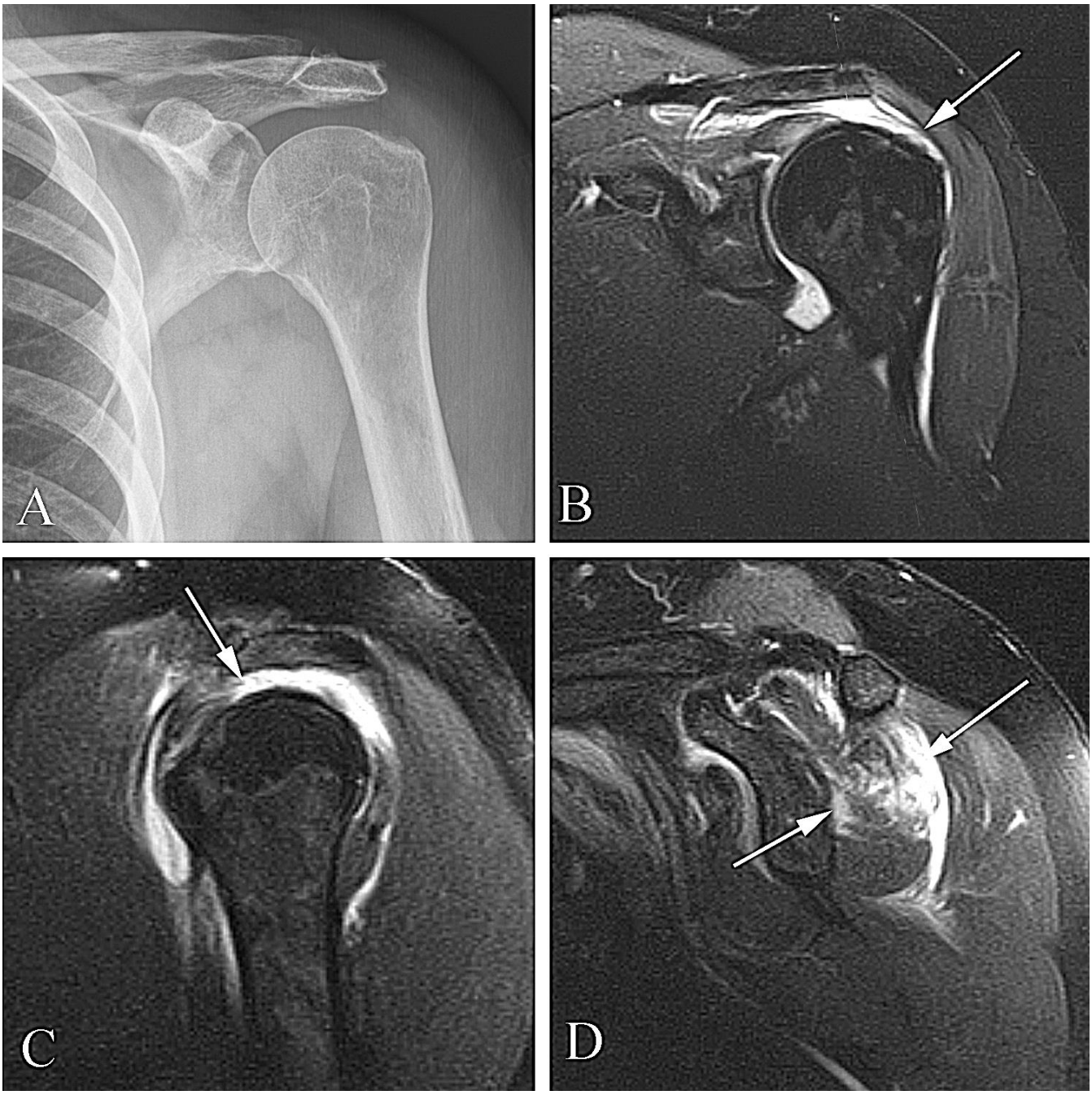

Figure 5. Acute rotator cuff tear in a 64 year old woman following trauma. A. AP plain film examination of the shoulder demonstrates no fracture. B. Coronal oblique fat-suppressed T2 weighted image shows a full thickness rotator cuff tear, with fluid along the normal course of the tendon deep to the deltoid muscle and superficial to the humeral head (arrow). C. Sagittal oblique fat-suppressed T2 weighted image demonstrates a large full-thickness rotator cuff tear (arrow) which involves both the supraspinatus and infraspinatus tendons. D. Sagittal oblique fat-suppressed T2 weighted image closer to the midline demonstrates extensive muscle tearing and interstitial increased signal (compatible with edema or hemorrhage) within the infraspinatus muscle (arrows).

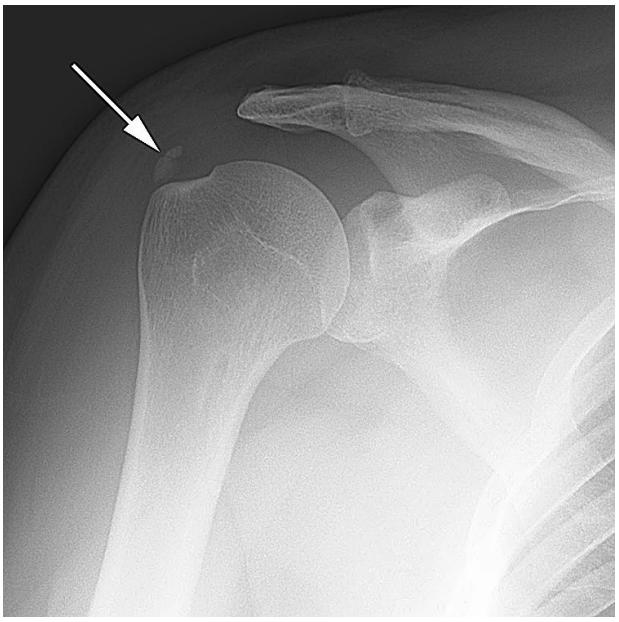

Figure 6. Calcific tendinitis in a 48 year old man with shoulder pain. AP plain film exam demonstrates calcification along the distal aspect of the supraspinatus tendon near its insertion on the greater tubercle (arrow).

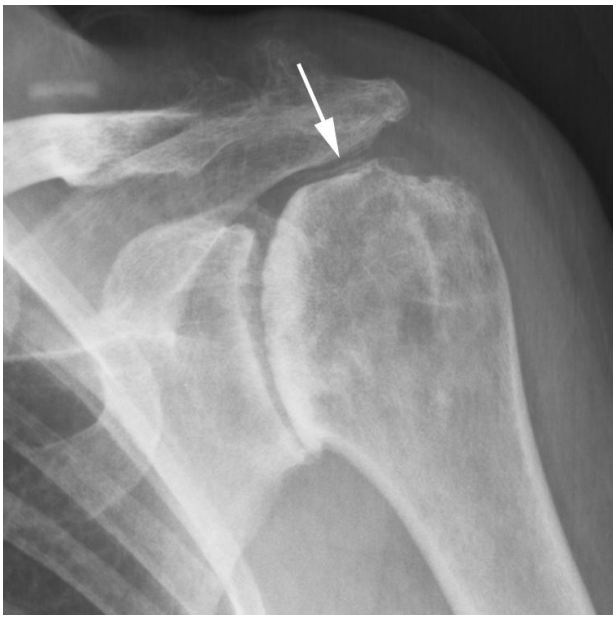

Figure 7. Chondrocalcinosis and degenerative changes of the glenohumeral joint in a 92 year old woman with shoulder pain. AP shoulder exam demonstrates subtle chondrocalcinosis of the humeral head articular cartilage (arrow), along with joint narrowing, subchondral sclerosis, and osteophytic spurring of the glenohumeral joint.

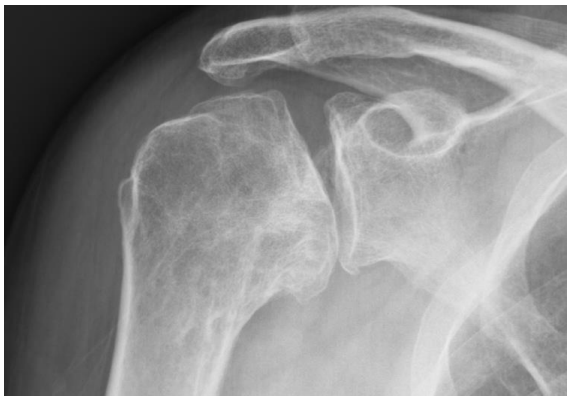

Figure 8. Degenerative change of the glenohumeral joint in a 47 year old man with shoulder pain who had a remote prior fracture of the humerus.

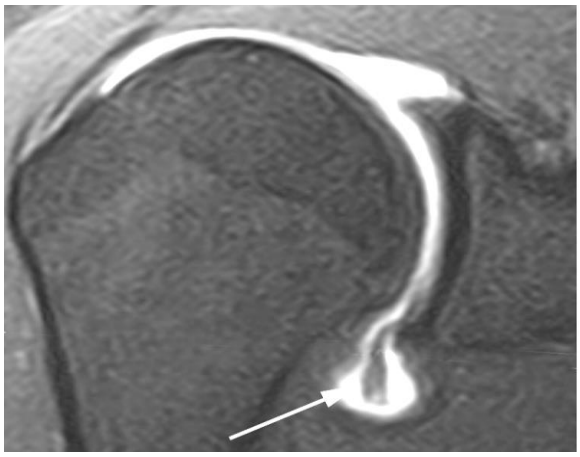

Figure 9. Labral tear/loose body in a 19 year old with repeated shoulder dislocations. MR arthrogram shows a large filling defect in the inferior recess (arrow).

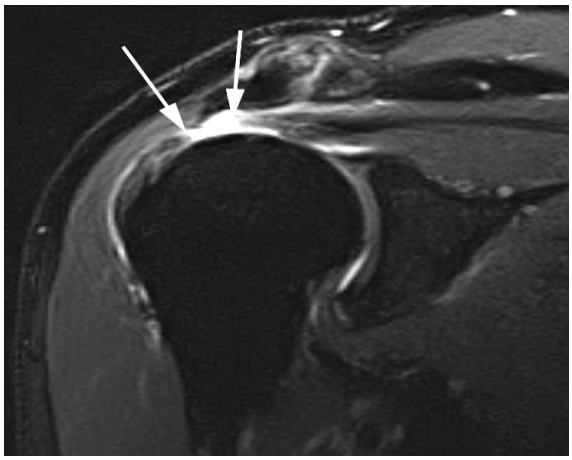

Figure 10. Full thickness rotator cuff tear in a 57 year old man with chronic pain and shoulder weakness. Coronal oblique fat-suppressed T2 weighted MR study shows a full-thickness defect in the supraspinatus tendon with retraction of the torn tendon margins (arrows).

Elbow

In patients with acute elbow pain following trauma, plain films should be obtained first. These will demonstrate not only cortical discontinuity but also the presence of a displaced fat pad, which is an indication of a joint effusion (Figure 11). In the setting of acute trauma, such an effusion typically represents a secondary finding indicating a nondisplaced radial head fracture. If the plain films show no fracture or effusion, MR may be of benefit, particularly if there are clinical features of a biceps tendon rupture (Figure 12) or other musculotendinous injury (Figure 13).

In patients with chronic elbow pain, imaging is usually not helpful[5]. Exceptions include evaluation of throwing athletes (particularly baseball pitchers) where MR-arthrography of the elbow joint may be of benefit to diagnose radial collateral ligament tears (Figure 14). MR of the elbow may also be of benefit to diagnose loose bodies within an effusion (Figure 15) and neurologic abnormalities centered at the elbow joint, where the ulnar nerve passes in close proximity to the distal dorsal medial humerus (Figure 16). Finally, while it is largely a clinical diagnosis, MR of the elbow can be helpful in confirming abnormalities of the origins of the common extensor and flexor tendons. A painful common tendon origin is called epicondylitis (a misnomer considering that the epicondyle is part of the humerus and is not inflamed). A painful lateral (extensor) tendon origin is also known as "tennis elbow", and a painful medial (flexor) tendon origin "golfer's elbow". MR will demonstrate abnormal signal of the tendon origins.

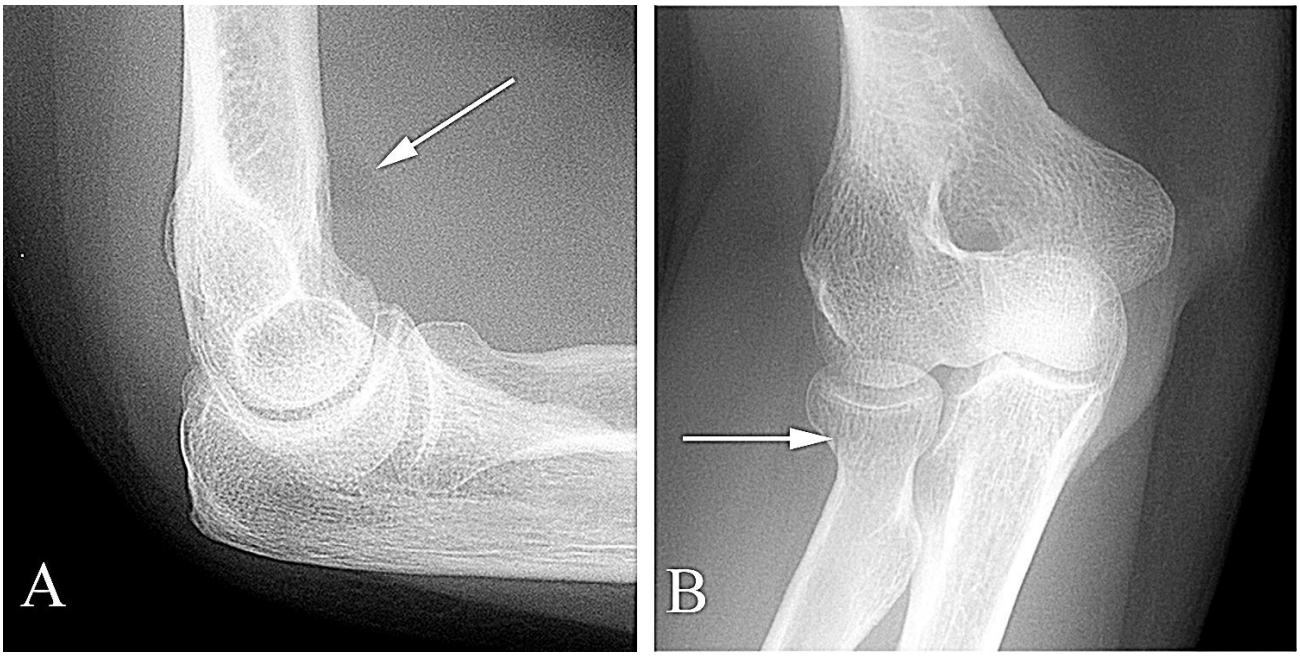

Figure 11. Radial fracture in a 51 year old woman who had pain following trauma. A. Lateral plain film of the elbow shows displacement of the fat pad from the anterior aspect of the distal humerus (arrow). B. Oblique plain film shows a subtle fracture through the radial neck (arrow). Elbow effusions following trauma are typically secondary to marrow and hemorrhage in the joint from a fracture.

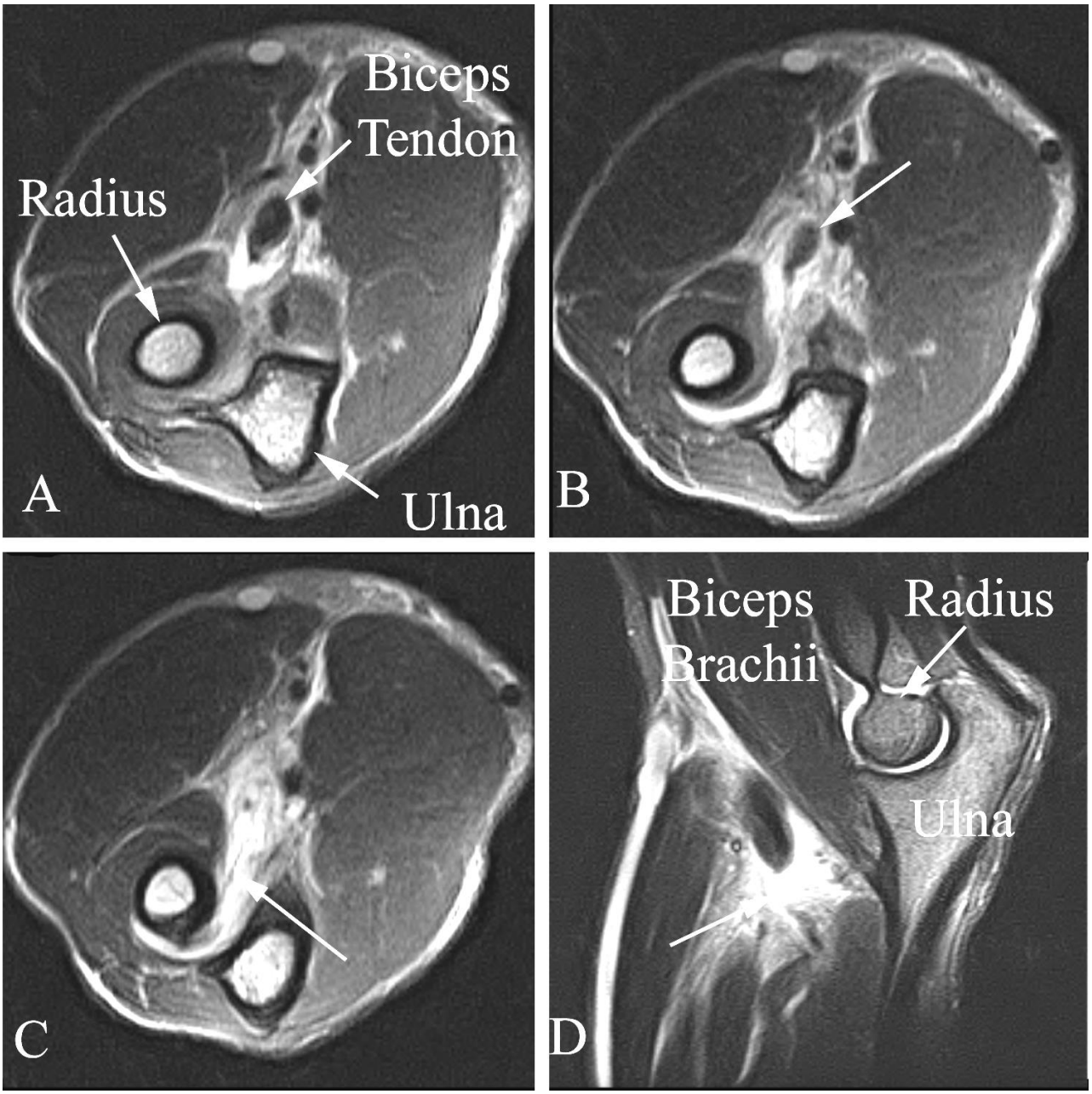

Figure 12. Biceps tendon rupture in a 47 year old man who had acute pain in the elbow following a lifting injury. A. Axial T1 weighted MR image demonstrates a swollen distal biceps tendon (arrow). B. Axial T1 weighted MR image slightly more distal in the arm demonstrates rapid tapering and an irregular appearance of the biceps tendon (arrow), particularly along its deep aspect. C. Axial T1 weighted MR image slightly more distally shows that the biceps tendon is absent (arrow) compatible with tearing and proximal retraction. D. Sagittal proton density MR image shows the tear through the distal biceps tendon (arrow) with retraction of the torn tendon margin.

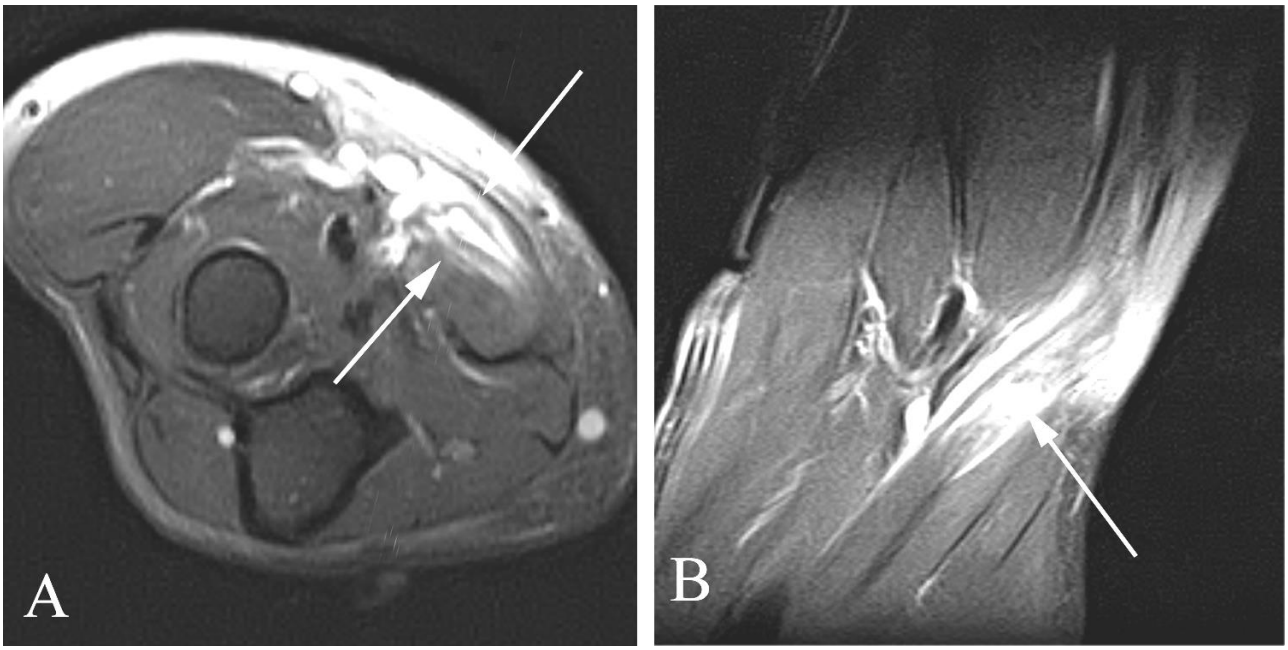

Figure 13. Pronator teres muscle tear in a 58 year old woman with pain following a golfing injury. A. Axial T2 weighted MR image demonstrates abnormal signal (arrow) in the pronator teres muscle. B. Coronal T2 weighted MR image shows a small focal hematoma (arrow) of the pronator teres muscle.

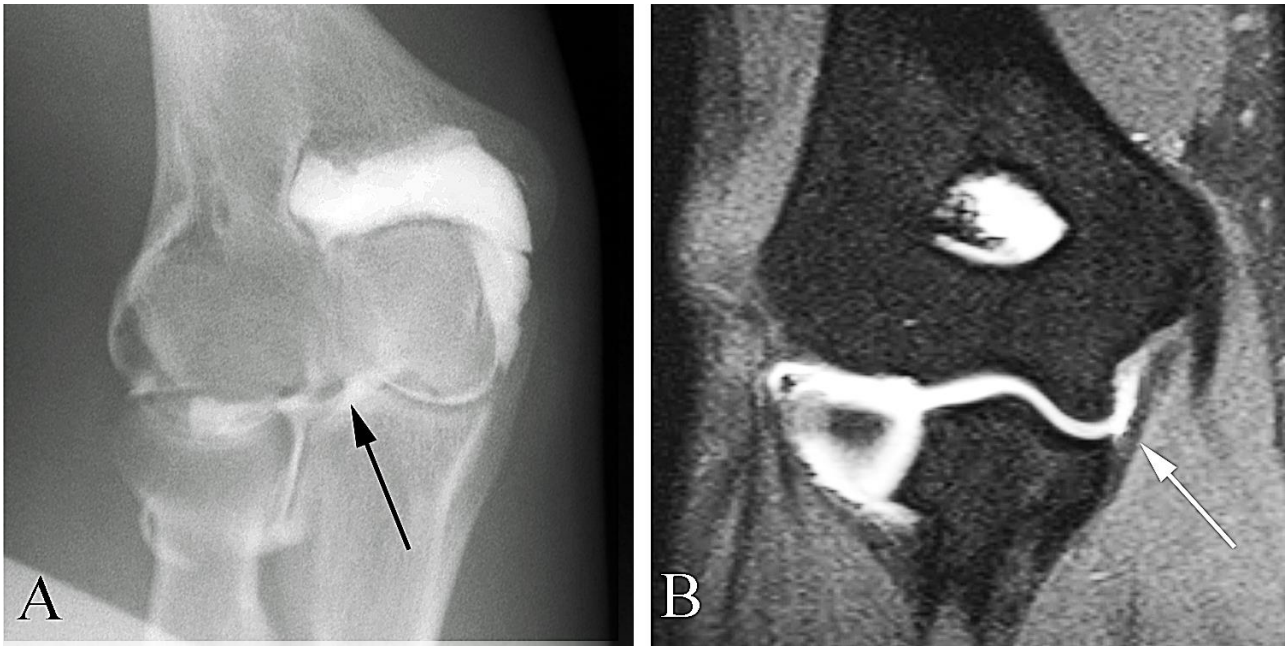

Figure 14. Intact ulnar collateral ligament in an 18 year old male pitcher with ulnar sided elbow pain. A. Arthrogram shows contrast material in the joint (arrow). B. Coronal T1 fat-suppressed image shows contrast material in the elbow joint, along with an intact ulnar collateral ligament (arrow). Note that the proximal aspect of the ligament was also intact and attached normally to the humerus but was out of the plane of section on this image.

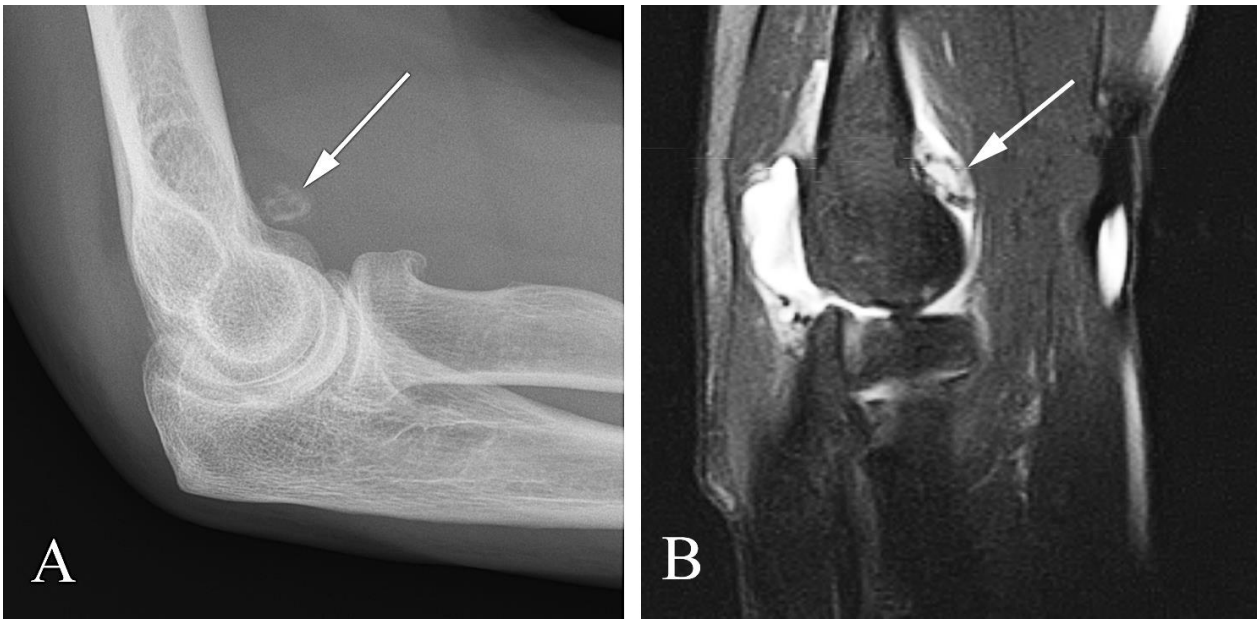

Figure 15. Loose body in the elbow joint in a 54 year old man with elbow pain. A. Lateral plain film shows an osseous fragment (arrow) projecting anterior to the distal humerus. B. Sagittal fat suppressed T2 weighted image demonstrates a joint effusion and the osseous fragment surrounded by elbow joint effusion fluid (arrow).

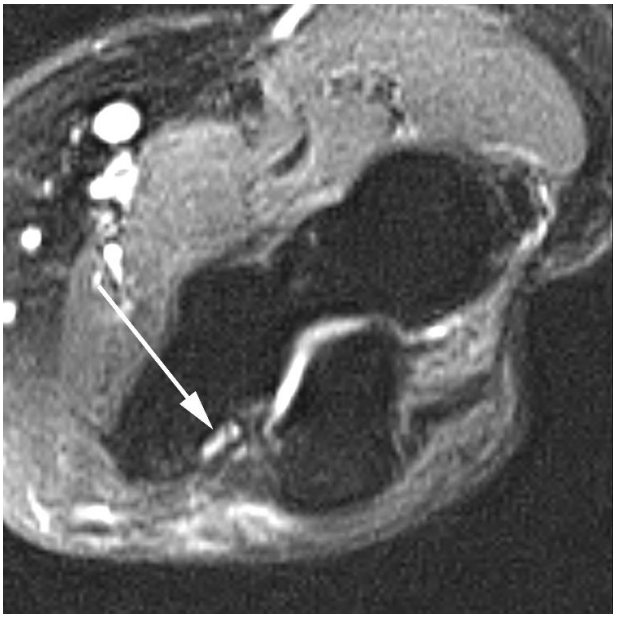

Figure 16. Ulnar nerve neuritis in a 50 year old woman with cubital tunnel syndrome. Axial fat-suppressed T2 weighted image shows abnormal increased signal in the ulnar nerve (arrow).

Wrist

As with the shoulder and elbow, plain films are the initial study of choice following acute injury of the distal forearm or wrist. Wrist trauma is relatively frequent because falls on an outstretched hand cause fractures of the distal radius (Figure 17) or carpal bones, either singly (Figure 18) or as a complex combination of lunate or perilunate dislocation (Figure 19). Fractures of the scaphoid are relatively common since this bone spans both the proximal and distal carpal rows. Regarding scaphoid fractures, plain films may be initially negative, in which case follow-up study following 10-14 days of immobilization, or immediate MRI, are beneficial. Scaphoid fractures – particularly through the proximal aspect – may go on to nonunion with avascular necrosis of the proximal portion of the bone secondary to the vascular supply (which enters the bone distally) being cut off by the fracture. As in other joint injuries, when plain films are negative and there is a high suspicion of fracture, either CT (Figure 20) or MR may be required to diagnose a fracture which is occult on plain film examination. MR may also demonstrate soft tissue injuries which cause pain but are not seen on plain films (Figure 21).

Plain films are also the best first step in the evaluation of chronic wrist pain, and may

demonstrate carpometacarpal (CMC) joint arthritis of the thumb (Figure 22). Plain films also may show variations of carpal anatomy including the ulnar-plus variant, where the ulna is significantly longer than the radius and which may be associated with ulnar abutment, and ulnar-minus variant (Figure 23), where the ulna is significantly shorter than the radius, which may be associated with avascular necrosis of the lunate. Plain films can also demonstrate secondary signs of De Quervain's tenosynovitis (of the short extensor and abductor tendons of the thumb) (Figure 24) as well as scapholunate advanced collapse (SLAC) wrist, or degenerative changes secondary to chronic scapholunate ligament tearing with proximal migration of the capitate bone (Figure 25). MR in patients with chronic wrist pain may demonstrate an otherwise occult ganglion of the scapholunate ligament[6] (Figure 26). MR is usually supplemented by arthrography to demonstrate triangular fibrocartilage and interosseous ligament tears (Figure 27). Similar to the case of shoulder rotator cuff tears (see above), the significance of wrist triangular fibrocartilage or interosseous ligaments tears in the middle aged and elderly is questionable.

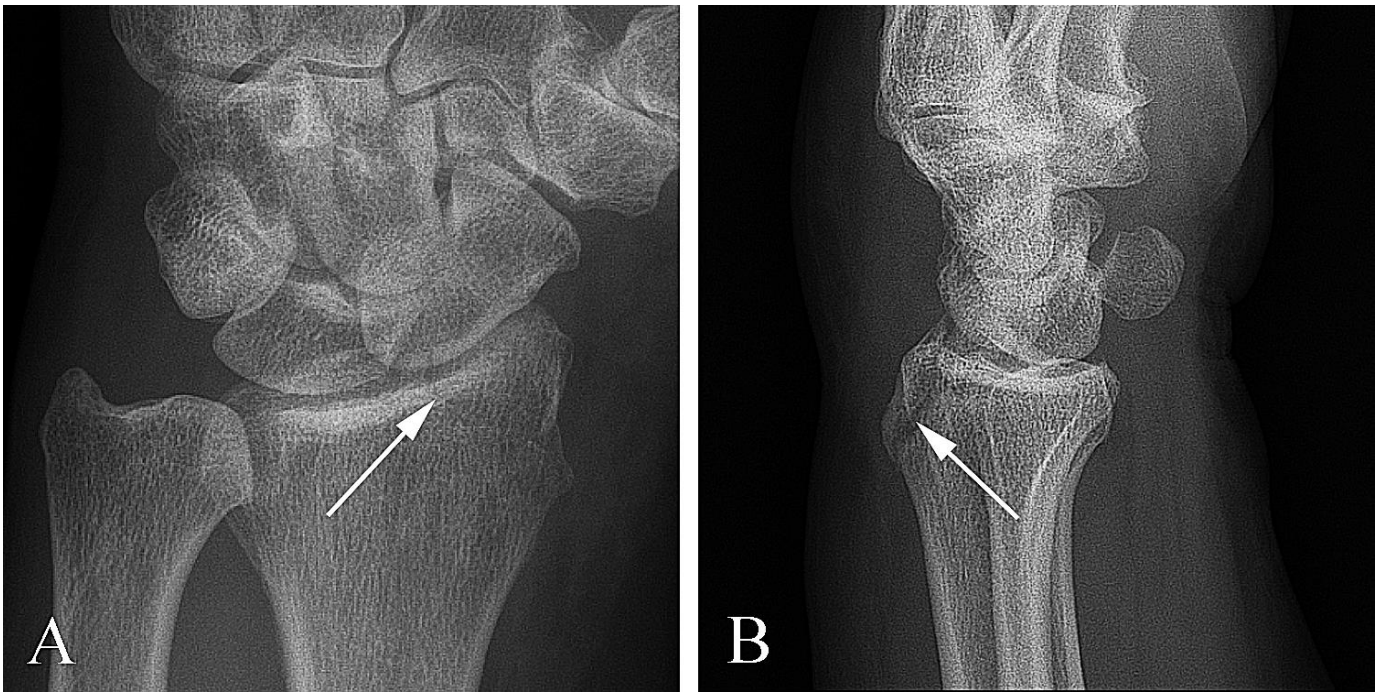

Figure 17. Radial styloid fracture in 48 year old woman with wrist pain following trauma. A. AP view of the wrist shows a subtle lucency along the base of the radial styloid (arrow). B. Lateral exam better shows the fracture as a lucency (arrow) and associated cortical interruption along the dorsal, distal radius.

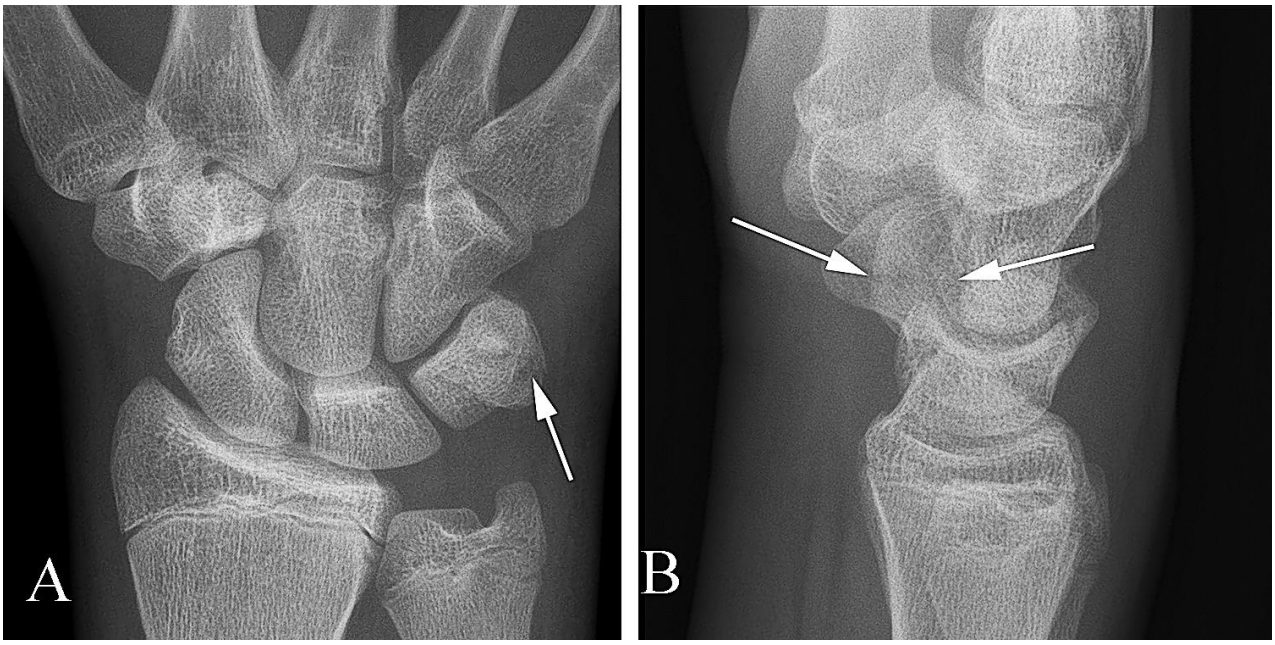

Figure 18. Pisiform fracture in an 18 year old man with pain following a fall on an outstretched arm. A. AP view of the wrist demonstrates a subtle lucency (arrow) through the pisiform bone. B. Lateral plain film examination confirms a fracture extending through the pisiform (arrows).

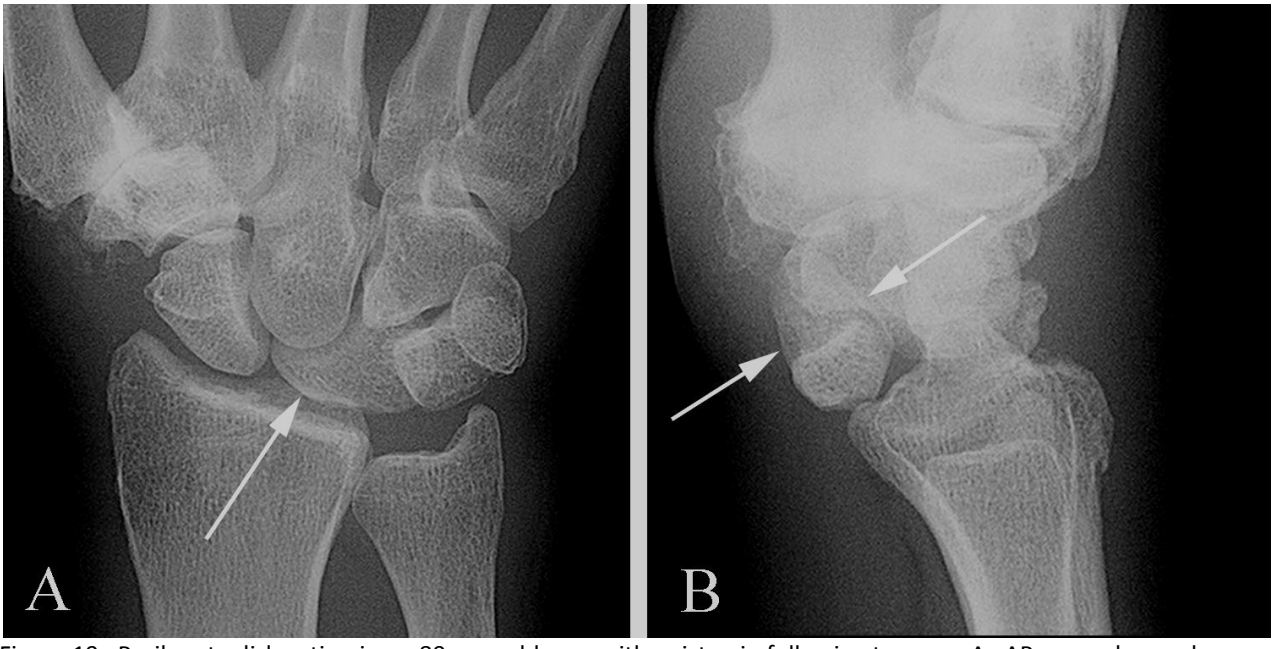

Figure 19. Perilunate dislocation in an 80 year old man with wrist pain following trauma. A. AP exam shows abnormal alignment of the lunate (arrow). B. Lateral exam shows anterior dislocation of the lunate which has been rotated ninety degrees from its normal orientation (arrows).

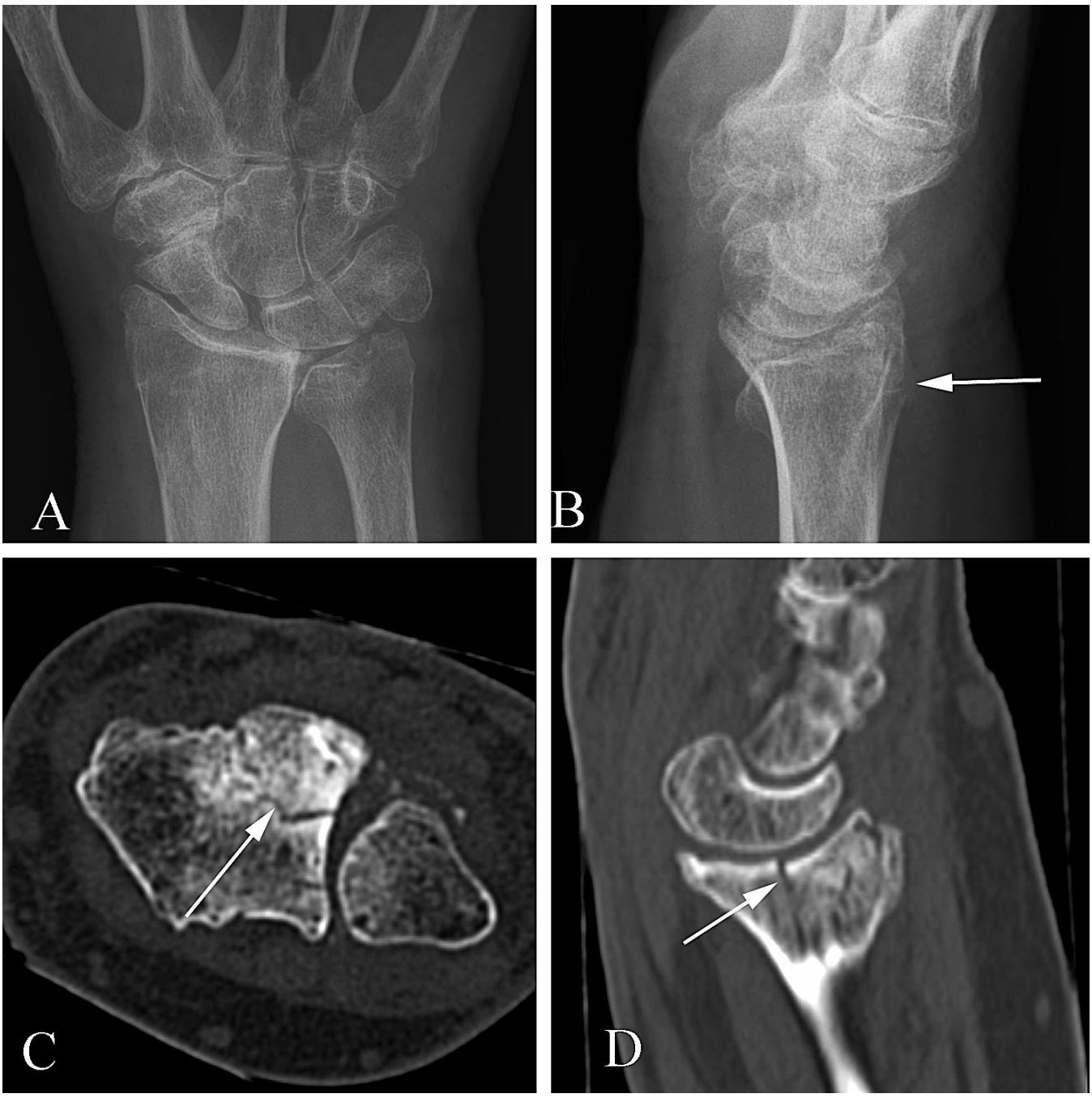

Figure 20. Distal radial fracture in a 79 year old woman with pain following trauma who has known rheumatoid arthritis. A. AP plain film shows chronic changes secondary to rheumatoid arthritis (including osteopenia and diffuse joint space loss) without obvious fracture. B. Lateral plain film examination shows possible interruption of the dorsal distal radial cortex (arrow). C. Axial CT study shows a fracture line extending to the articular surface (arrow). D. Sagittal reformatted CT shows an intra-articular fracture (arrow).

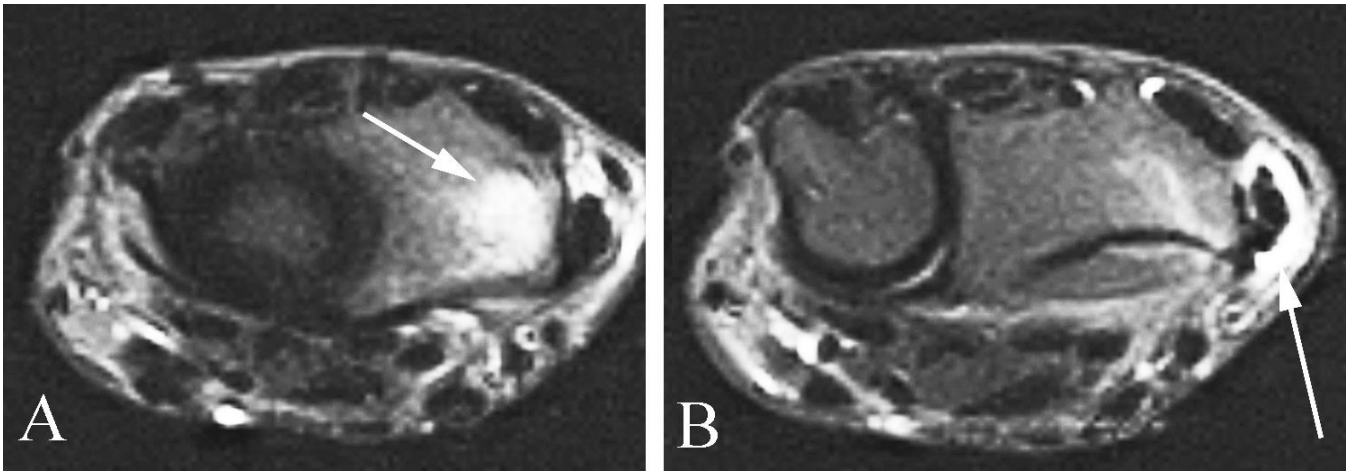

Figure 21. Bone contusion and tenosynovitis of the adductor pollicis longus tendon in a 56 year old woman with direct trauma to the lateral aspect of her wrist. A. Axial fat-suppressed T2 weighted image shows increased signal intensity in the distal, lateral radius (arrow) compatible with bone contusion. B. Axial fat-suppressed T2-weighted image slightly more distal shows fluid surrounding the adductor pollicis longus tendon (arrow) compatible with tenosynovitis.

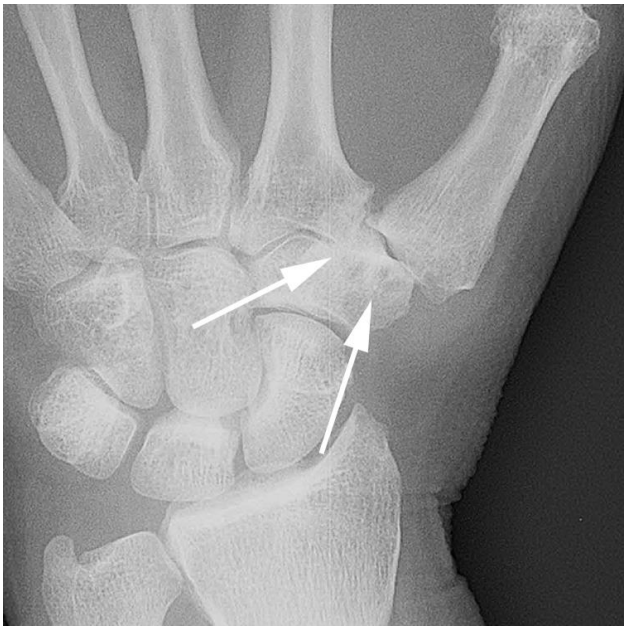

Figure 22. Thumb carpometacarpal joint osteoarthritis in a 69 year old with chronic pain along the base of the thumb. Oblique plain film exam shows joint space narrowing and osteophytic spurring along the thumb CMC joint.

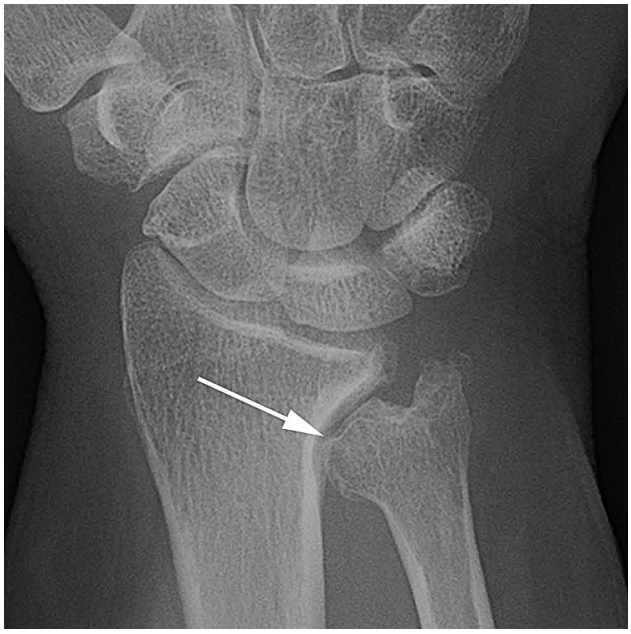

Figure 23. Ulnar minus variant with secondary degenerative changes in a 62 year old woman with chronic wrist pain. AP plain film examination shows that the ulna is considerably shorter than the radius, articulates abnormally with the distal radius, and that there is extensive secondary osteoarthritis along the distal radio-ulnar joint (arrow).

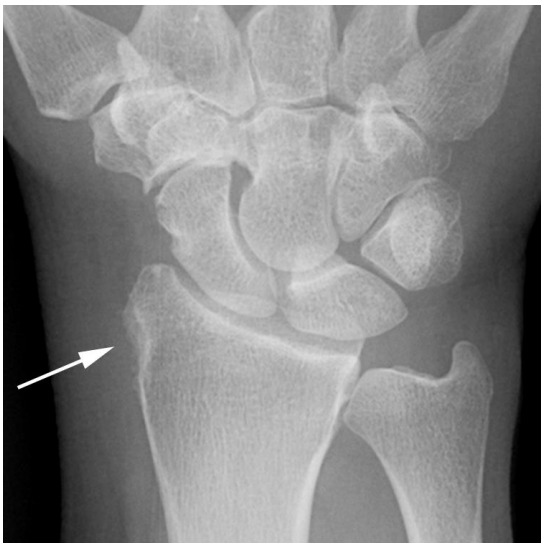

Figure 24. Secondary boney changes in De Quervain's tenosynovitis in a 56 year old with chronic radial sided wrist pain. AP plain film examination shows irregular periostitis along the base of the radial styloid (arrow).

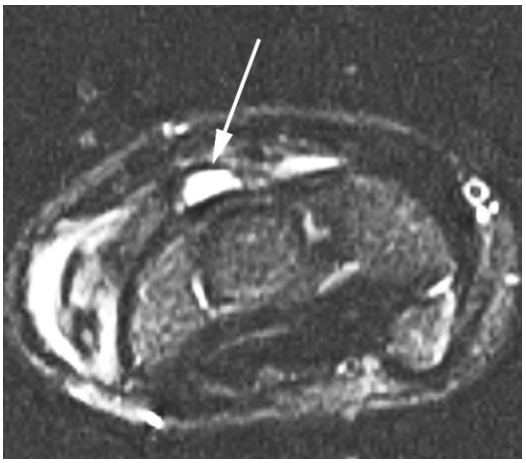

Figure 26. Ganglion cyst in a 16 year old volleyball player with chronic wrist pain. Axial fat-suppressed T2 weighted image shows a small mass which demonstrates marked T2 increased signal intensity along the dorsum of the carpus (arrow).

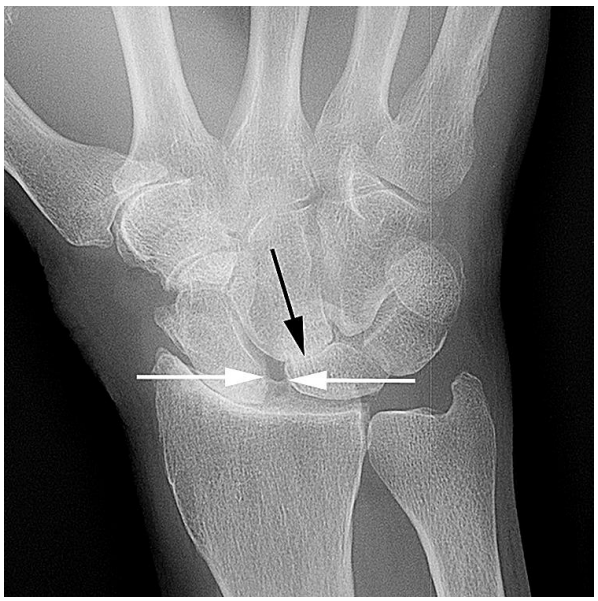

Figure 25. Scapholunate advanced collapse (SLAC) wrist in a 67 year old man with chronic wrist pain. AP plain film exam shows widening of the scapholunate interval (white arrows) and proximal migration of the capitate with elimination of the joint between the capitate and lunate (black arrow), along with degenerative changes between the scaphoid and radius.

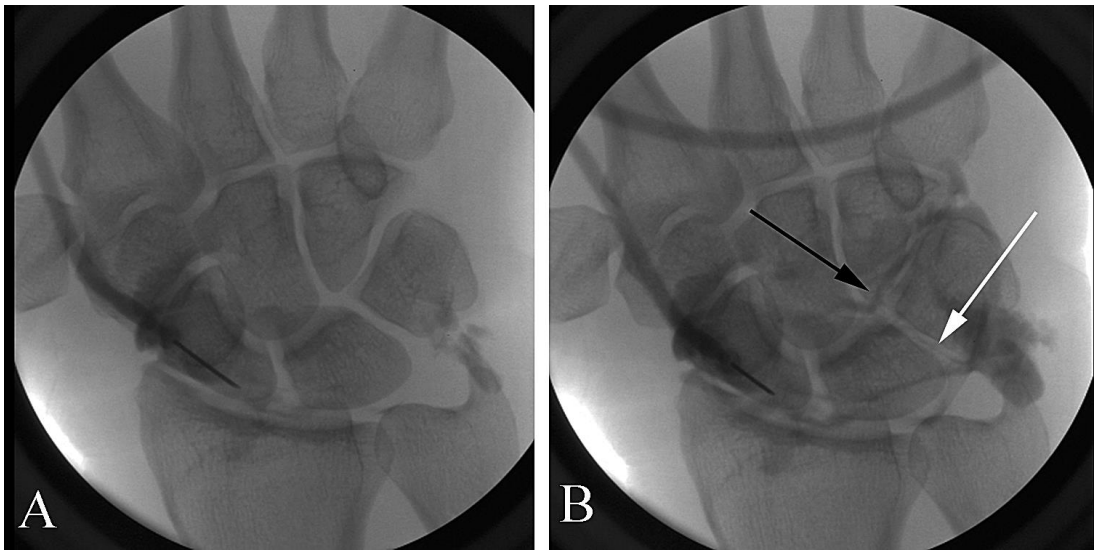

Figure 27. Scapho-lunate ligament rupture in a 52 year old with chronic ulnar sided wrist pain. A. Wrist arthrography during the early part of the injection with the needle along the radial aspect of the joint and contrast material pooling mostly along the radial side of the wrist. Note the lack of contrast material in the mid-carpal joints. B. Wrist arthrography later during injection, with contrast extending between the lunate and triquetrum (white arrow) into the mid-carpal joint (black arrow), diagnostic of a lunato-triquetral interosseous ligament disruption.

Hand

X-rays of the hands may be obtained following trauma, as the phalanges are the most frequently fractured bones in the body and x-rays can show fractures (Figure 28), dislocations, retained foreign bodies, as well as combinations of these abnormalities. CT may be helpful in the specific instance of intra-articular fractures through the base of the ring and small finger metacarpals, as the anatomy of the carpal-metacarpal junction on the ulnar side of the hand is complex with multiple overlapping structures making assessment of fracture position difficult. MR may be helpful to diagnose and distinguish between two types of thumb injury: the gamekeeper's thumb and the Stener lesion. In explanation: the ulnar collateral ligament (UCL) is an important stabilizer of the thumb metacarpophalangeal (MCP) joint. Chronic repetitive injury (said to occur from wringing the necks of game birds in England) or acute post-traumatic injury (most frequently against a planted ski-pole) may sprain or rupture this ligament. If the ligament is ruptured but nondisplaced (Figure 29) it may heal as long as it is held in position. This is the classic gamekeeper's thumb. There is an adductor aponeurosis which usually covers the superficial aspect of the MCP UCL. If this aponeurosis is also torn and the UCL displaced superficial to the aponeurosis, the injury is said to be a Stener lesion, which generally requires operative intervention for repair. Failure to diagnose UCL injuries may lead to debilitating, painful laxity of the thumb MCP joint.

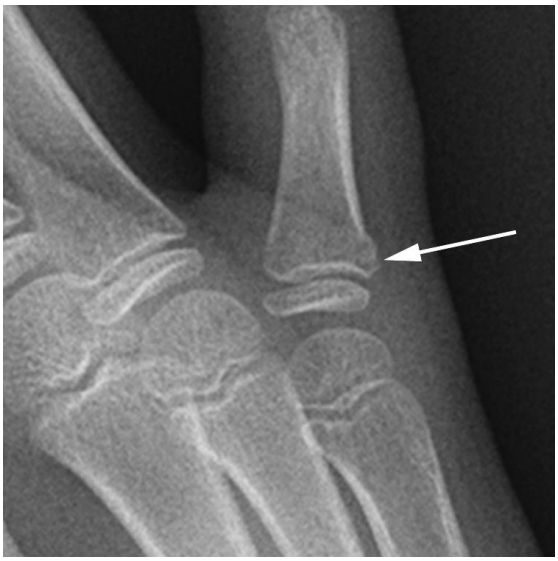

Figure 28. Proximal phalanx fracture in an 8 year old girl with pain following trauma. Oblique plain film examination demonstrates a minimally displaced Salter-Harris Type II fracture through the proximal, ulnar aspect of the proximal phalanx of the small finger (arrow).

Evaluation of chronic hand pain relies primarily on a clinical assessment of whether the pain is actually monoarticular or polyarticular, since the hand is frequently involved in polyarthropathy. Chapter 13 addresses the situation when the symptoms indeed involve multiple joints. If only one joint is involved, this may represent carpometacarpal (CMC) joint strain or arthritis of the thumb CMC, which can be differentiated with plain film examination[7] (Figure 22). Plain films are also helpful in evaluation of pain secondary to gout which has involved only a single joint (Figure 30). MR is rarely used for the evaluation of chronic hand pain unless there is a strong suspicion of tenosynovitis (particularly infectious) or retained foreign body.

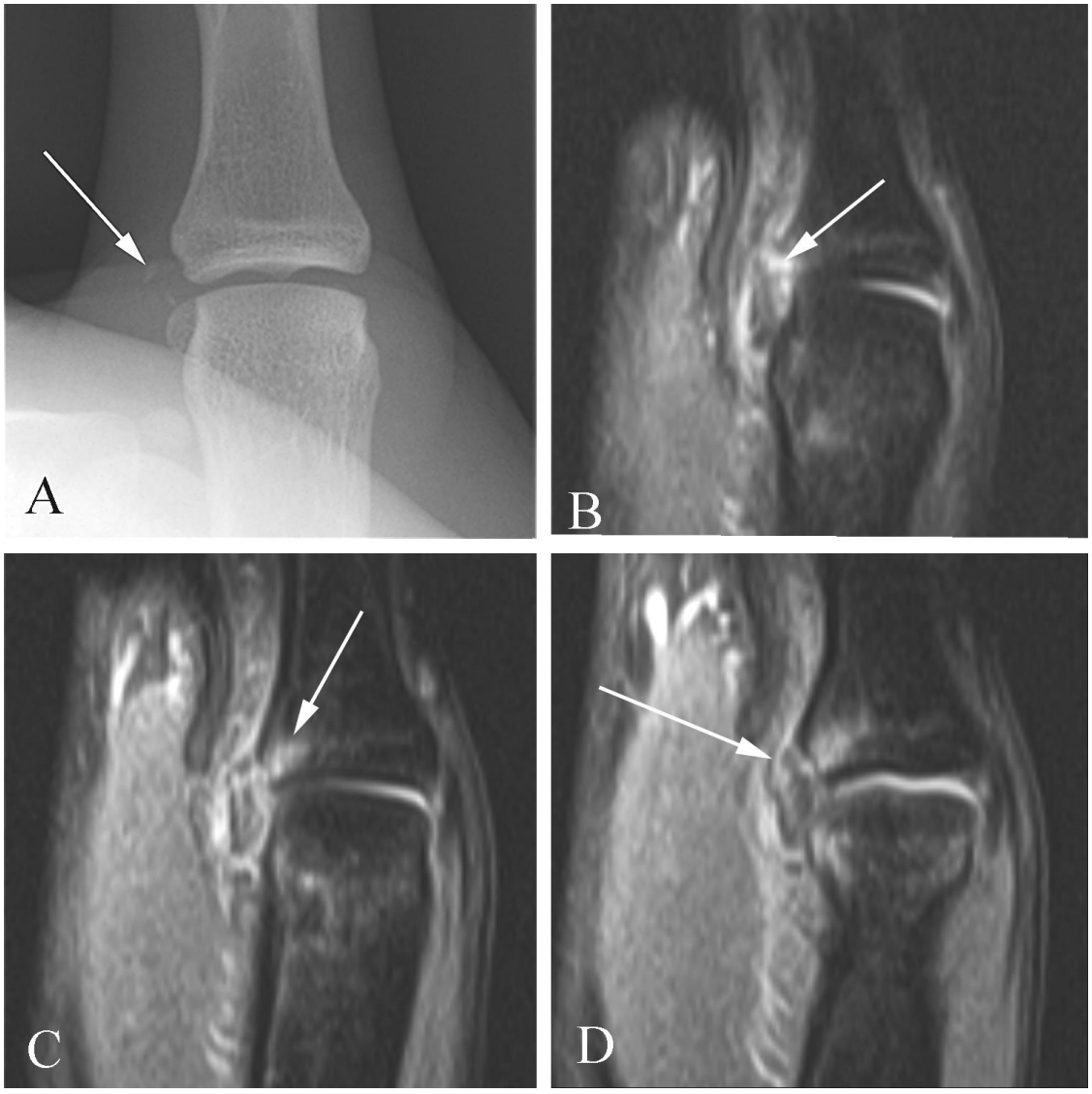

Figure 29. Gamekeeper's thumb in a 16 year old male with pain following trauma (football injury). A. AP plain film examination of the thumb demonstrates an abnormal fragment of bone projecting along the ulnar aspect of the joint (arrow). B. Coronal fat-suppressed proton density MR shows discontinuity of the ulnar collateral ligament (arrow) of the thumb MCP joint. C. Coronal fat-suppressed proton density image shows increased signal intensity compatible with contusion along the proximal, ulnar aspect of the proximal phalanx of the thumb (arrow). D. Coronal fat-suppressed proton density MR image shows that the overlying aponeurosis is intact (arrow).

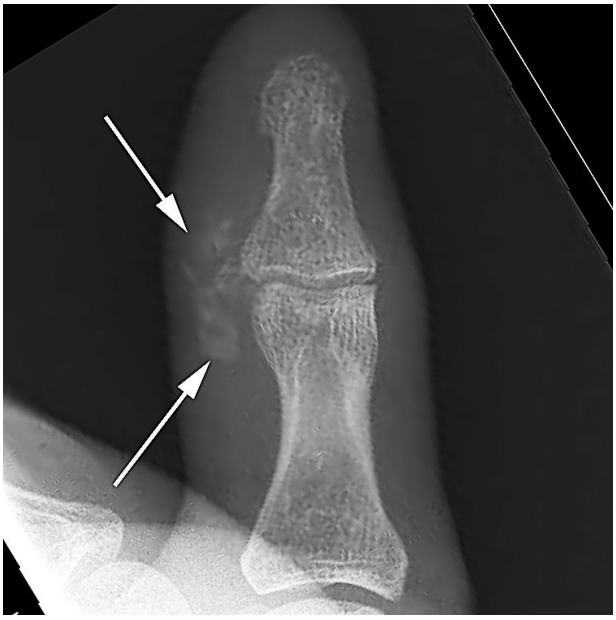

Figure 30. Gouty tophus in a 59 year old woman with acute thumb pain but no injury. AP plain film of the thumb shows calcified tophus in the soft tissues adjacent to the thumb interphalangeal joint (arrows).

Hip

X-ray examination is the study of choice for evaluation of the acutely traumatized hip[8]. Plain films will typically demonstrate fractures (Figure 31) and dislocations. In cases where the plain films are negative but persistent severe pain causes a high level of suspicion for fracture, either CT or MR may be performed. CT may be a better alternative when there is substantial trauma as in an MVA (Figure 32), whereas MR is actually more sensitive to subtle fractures[9] (Figure 33), and will detect soft tissue injuries not seen on plain films (Figure 34).

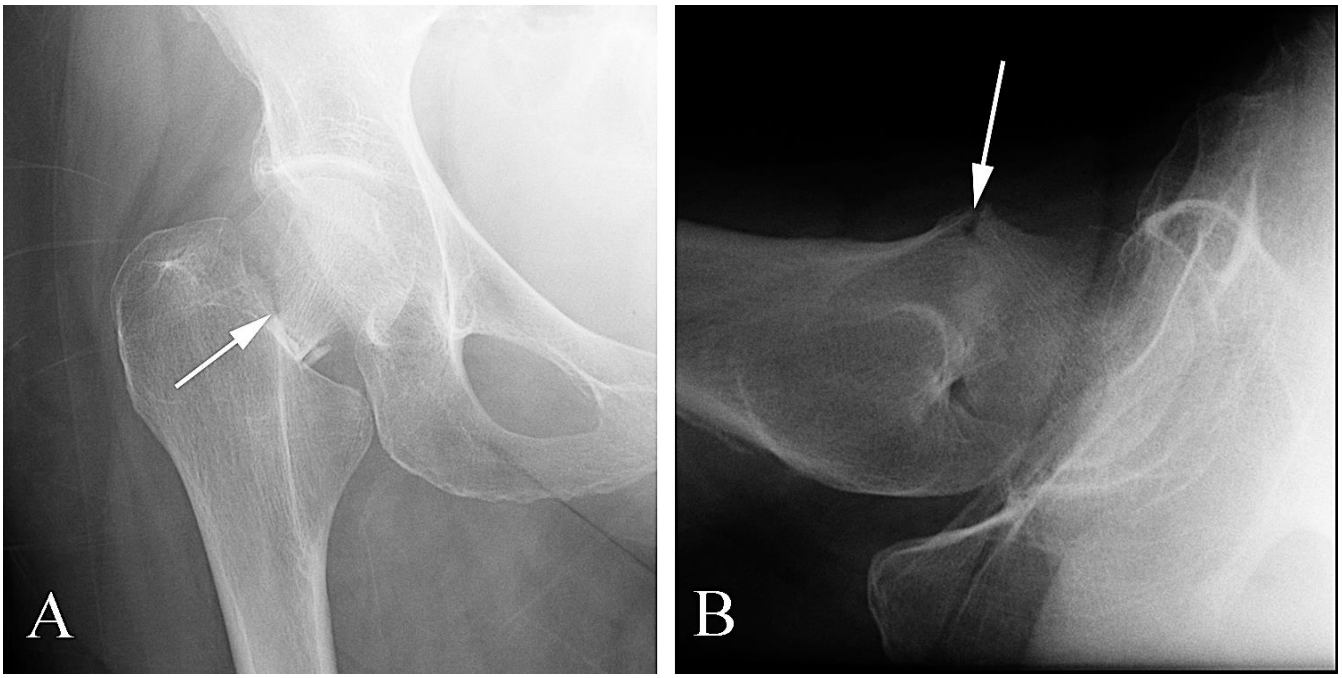

Figure 31. Proximal femur fracture in a 75 year old with pain following trauma. A. AP plain film of the hip shows a fracture through the base of the femoral neck (arrow) with associated shortening of the femur. B. Axial view of the hip shows anterior angulation of the fracture apex (arrow).

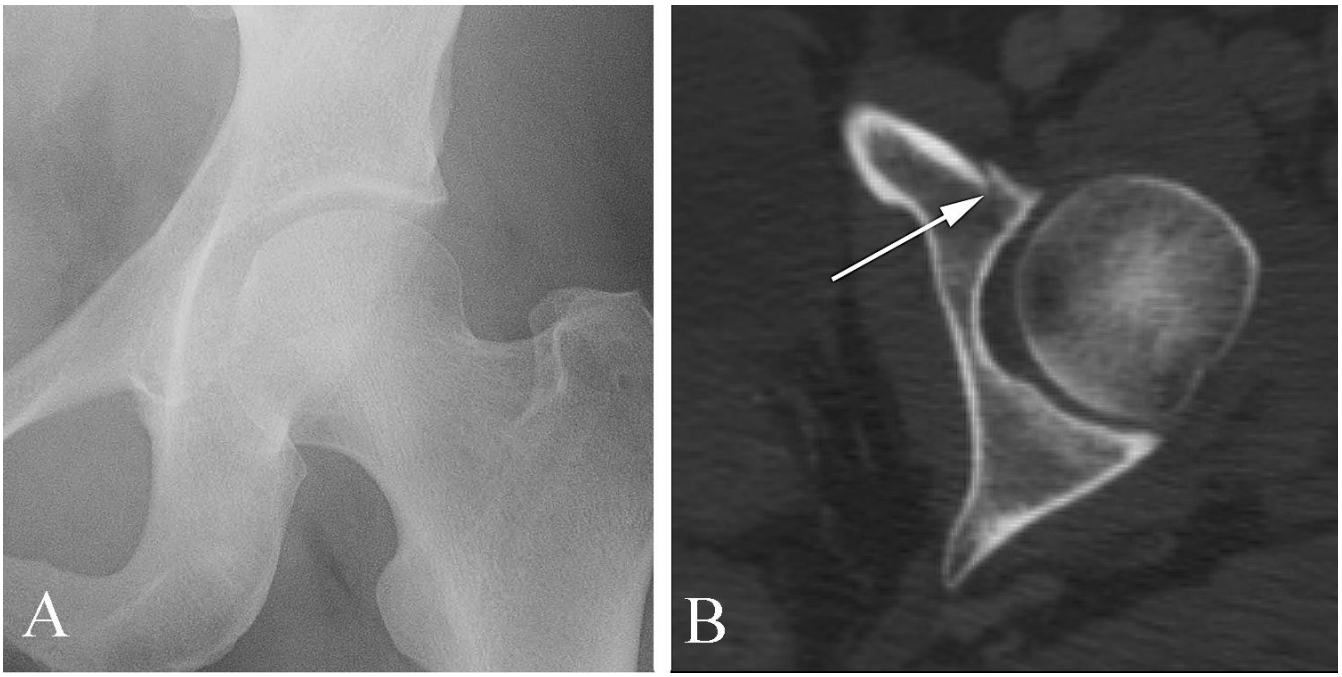

Figure 32. Acetabular fracture not visible on plain film but seen on CT in a 38 year old with pain following trauma. A. AP plain film examination of the hip shows no abnormality. The patient had persistent severe pain and therefore a CT was performed. B. Axial CT of the hip shows a fracture through the anterior column of the acetabulum (arrow).

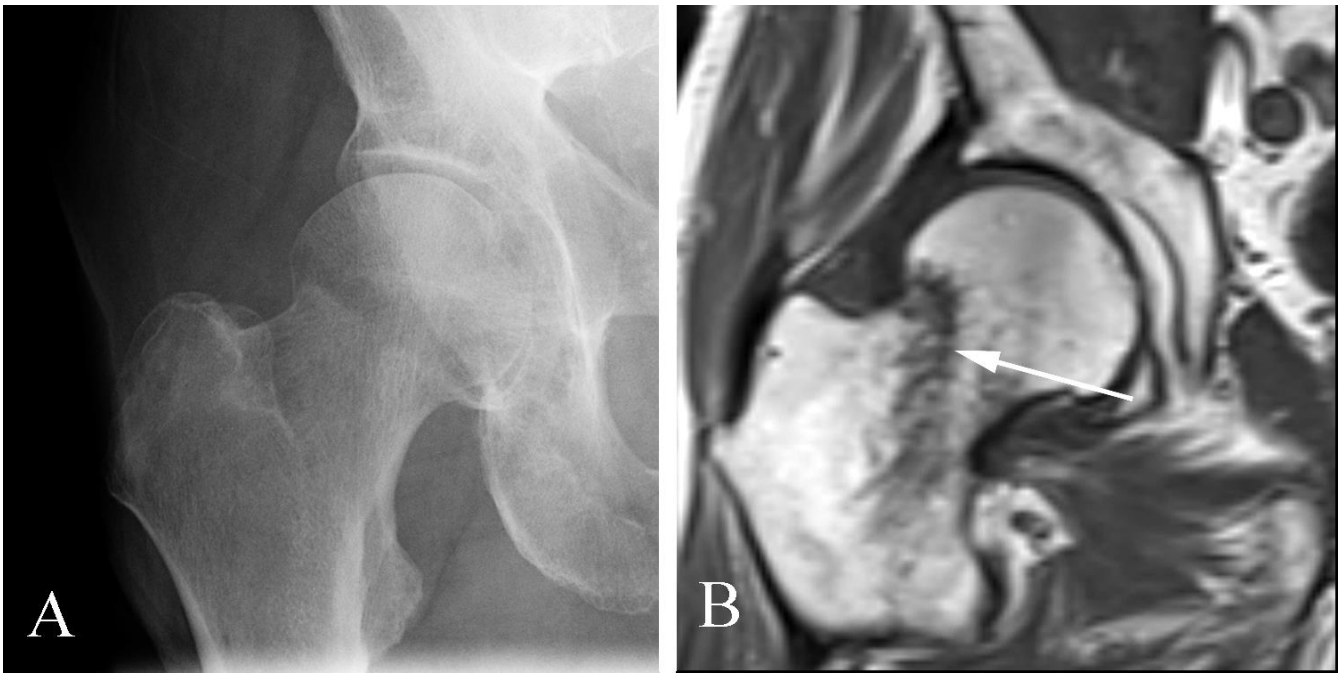

Figure 33. Hip fracture seen on MR in a 94 year old man with persistent pain following trauma. A. AP plain film of the hip shows no fracture. B. Coronal T1 weighted MR study shows a fracture line through the femoral neck (arrow).

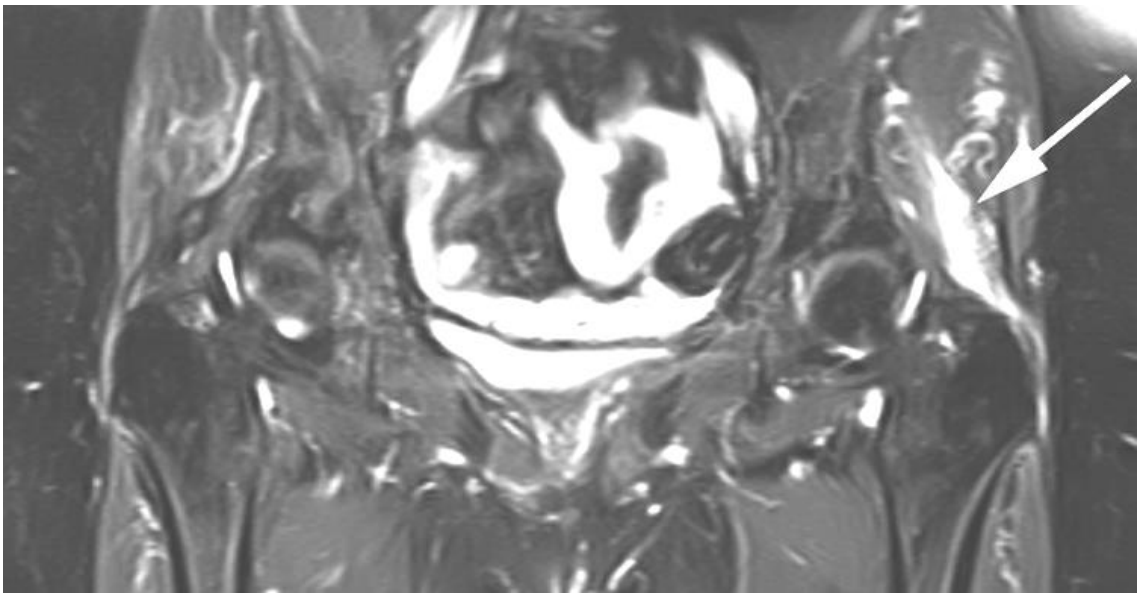

Figure 34. Gluteus medius muscle tear in a 75 year old woman with chronic hip pain. Coronal STIR image demonstrates abnormal increased signal in the left gluteus medius muscle (arrow) compatible with a muscle tear.

The first step in evaluation of chronic hip pain is also x-ray examination, which may demonstrate such causative abnormalities as osteoarthritis (Figure 35), CPPD, or gout (Figure 36). Plain films may demonstrate some processes which require further evaluation with MR, such as rapidly destructive osteoarthritis (Figure 37) or avascular necrosis (Figure 38). If plain films are unremarkable in patients with chronic hip pain, MR may demonstrate osteoarthritis (Figure 39) or avascular necrosis which is either subtle or not appreciated on plain film examination. In patients with symptoms of an acetabular labral tear (clicking or locking of the hip joint) MR arthrography is helpful to evaluate the internal structures of the joint including the labrum.

Patients with prostheses and following fracture fixation should also first undergo x-ray examination to evaluate possible dislocation (Figure 40), hardware complication (Figure 41), and dystrophic calcification (Figure 42). Such films are typically ordered at 1 year, 3 years, and 5 years (and then at 5 year intervals) by the operating orthopedic surgeon[10]. Painful prostheses which show no plain film abnormalities but for which there is suspicion of an infection can be aspirated; if imaging is required, the best course is performance of a nuclear medicine bone scan examination, followed by indium-labeled WBC evaluation: the combination of the two studies can help distinguish normal post-operative prosthesis changes from loosening and loosening from infection[10].

X-ray guidance is typically used for hip intra-articular injections of either lidocaine (for diagnosis) or steroids (for treatment) or combinations of the two, as injection without radiographic guidance is insufficiently accurate.

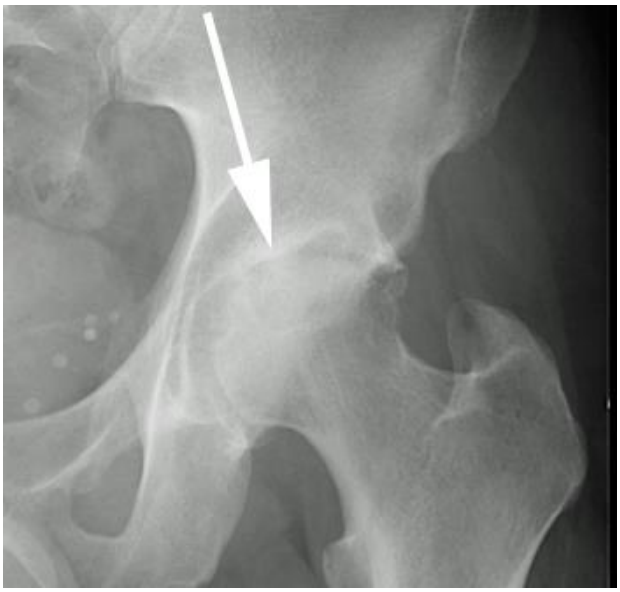

Figure 35. Osteoarthritis of the left hip in a 35 year old with chronic hip pain. AP plain film exam shows joint narrowing and subchondral sclerosis.

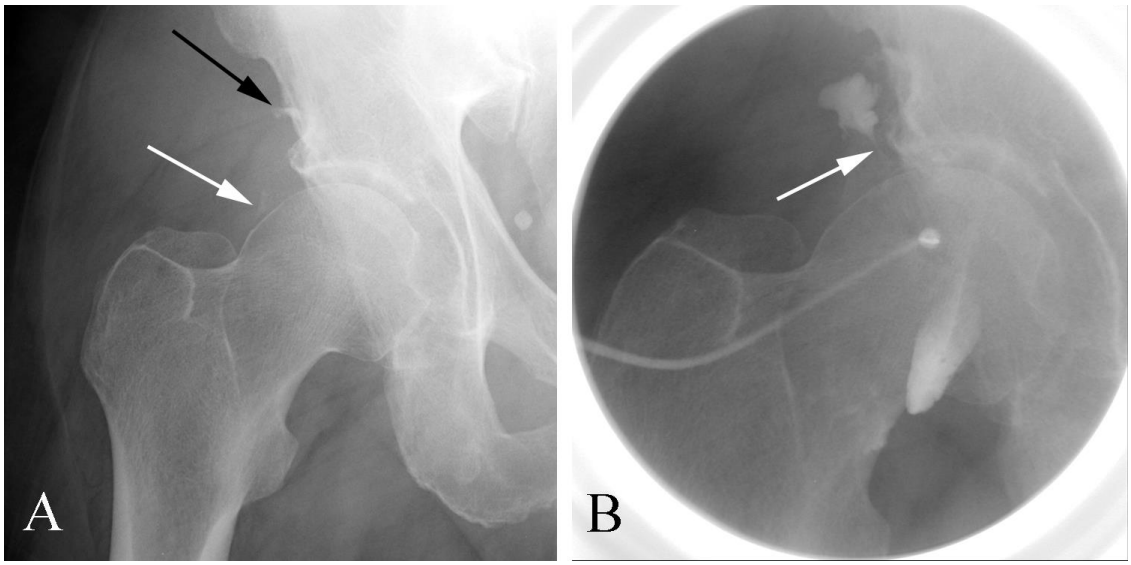

Figure 36. Gout of the right hip in a 70 year old man with chronic hip pain. A. Plain film examination shows calcification along the margin of the femoral head (white arrow). There is erosion with an overhanging edge along the lateral acetabulum (black arrow). B. Hip arthrogram shows contrast along the synovial extension adjacent to the acetabulum through a narrow connection along the femoral head (arrow).

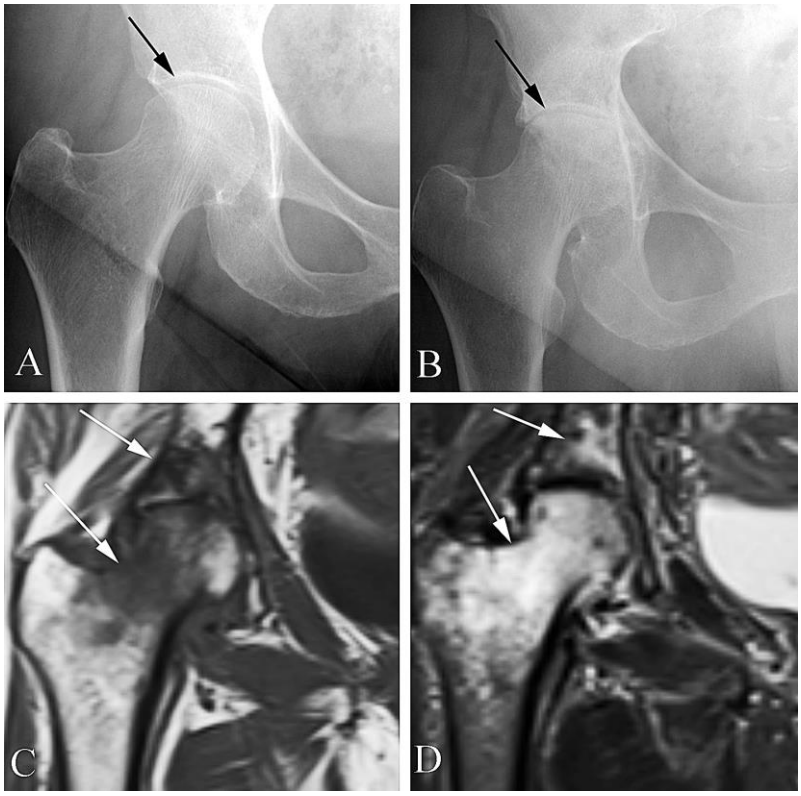

Figure 37. Rapidly destructive osteoarthritis of the right hip in an 80 year old woman with progressive hip pain. A. AP plain film at the onset of hip pain shows mild joint narrowing (arrow). B. AP plain film obtained six months later (with steadily increasing pain) shows marked progression of joint space narrowing (arrow). C. Coronal T1 weighted image shows extensive abnormal decreased signal intensity through the femoral neck and acetabulum (arrows). D. Coronal T2 weighted image shows abnormal increased signal intensity through the femoral head, neck, and acetabulum (arrows).

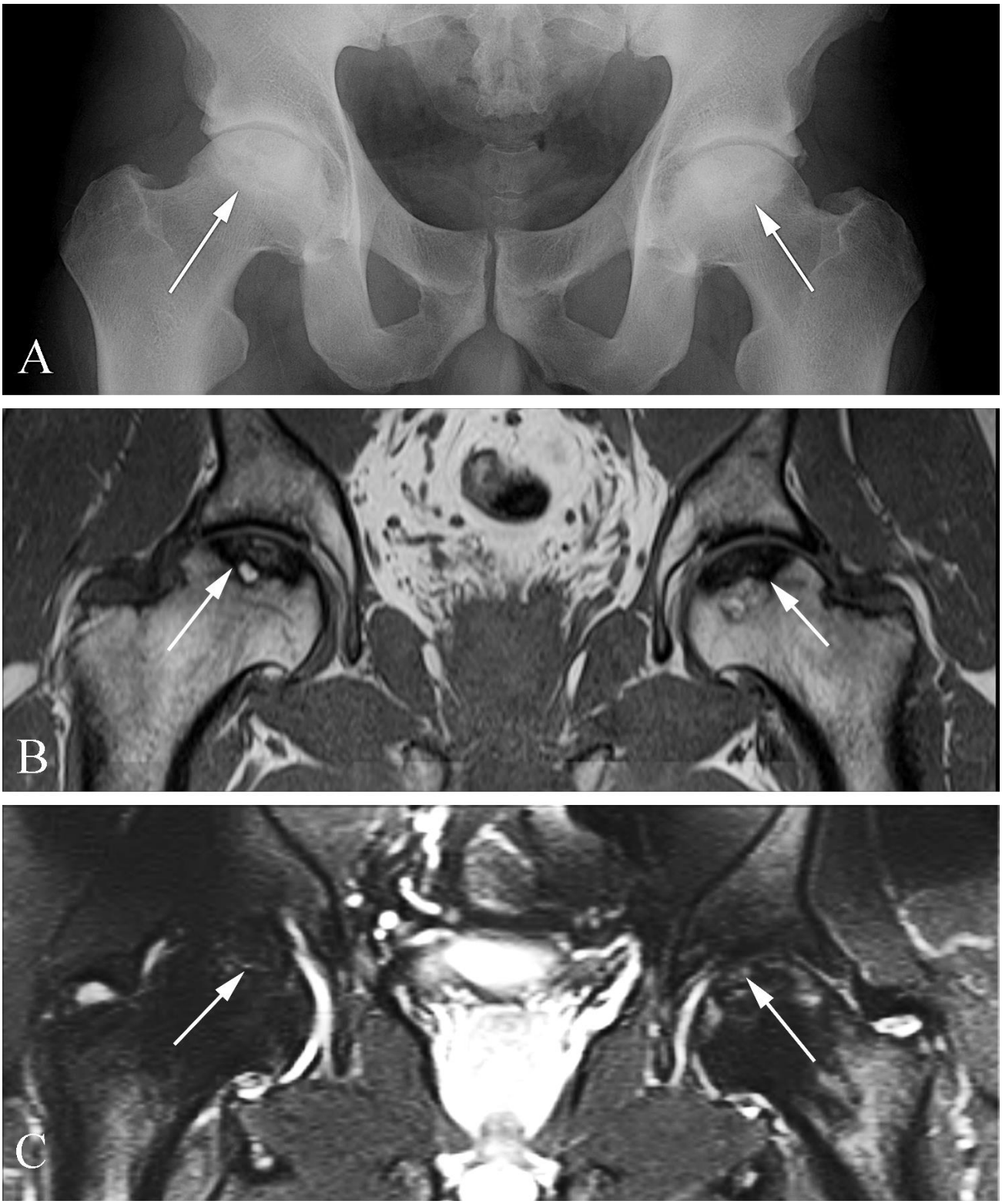

Figure 38. Avascular necrosis of the hips in a 38 year old man with chronic hip pain. A. AP view of the pelvis shows abnormal increased density (arrows) in both femoral heads. B. Coronal T1 weighted MR image demonstrates extensive decreased signal intensity (arrows) in the subchondral aspects of both femoral heads. Note mixed signal intensity at the interface between the normal signal intensity in the femoral necks and the decreased signal intensity in the heads. C. Coronal STIR image demonstrates mixed signal intensity in the femoral heads (arrows).

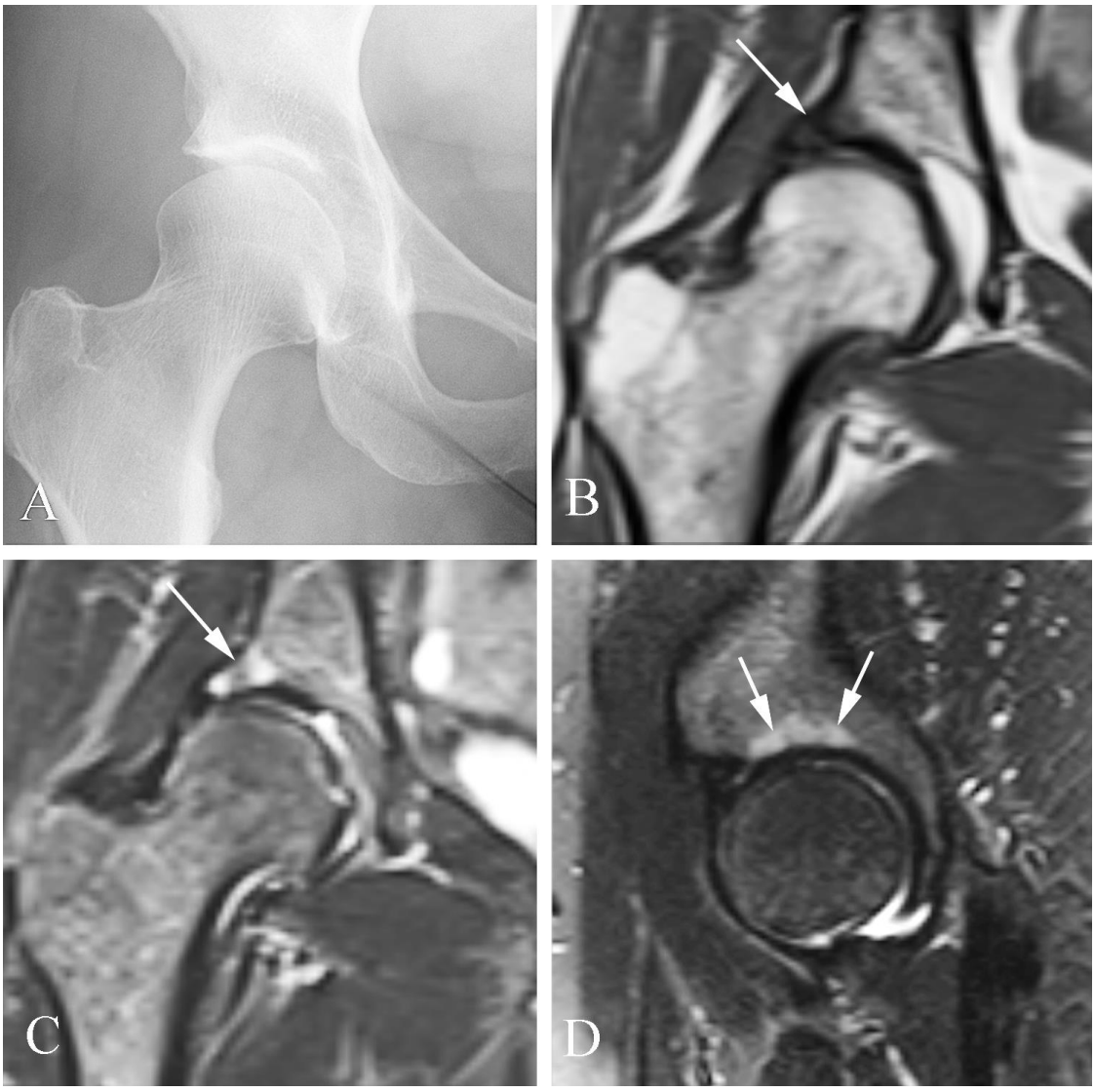

Figure 39. Early degenerative change seen on an MR study in a 50 year old woman with chronic hip pain. A. AP plain film of the hip shows no significant joint space narrowing, subchondral sclerosis, osteophytic spurring, or subchondral cyst formation. B. Coronal T1 weighted image demonstrates abnormal decreased signal intensity in the acetabulum (arrow). C. Coronal fat-suppressed proton density image demonstrates abnormal increased signal intensity in the acetabulum. D. Sagittal STIR image confirms abnormal signal intensity in the acetabulum.

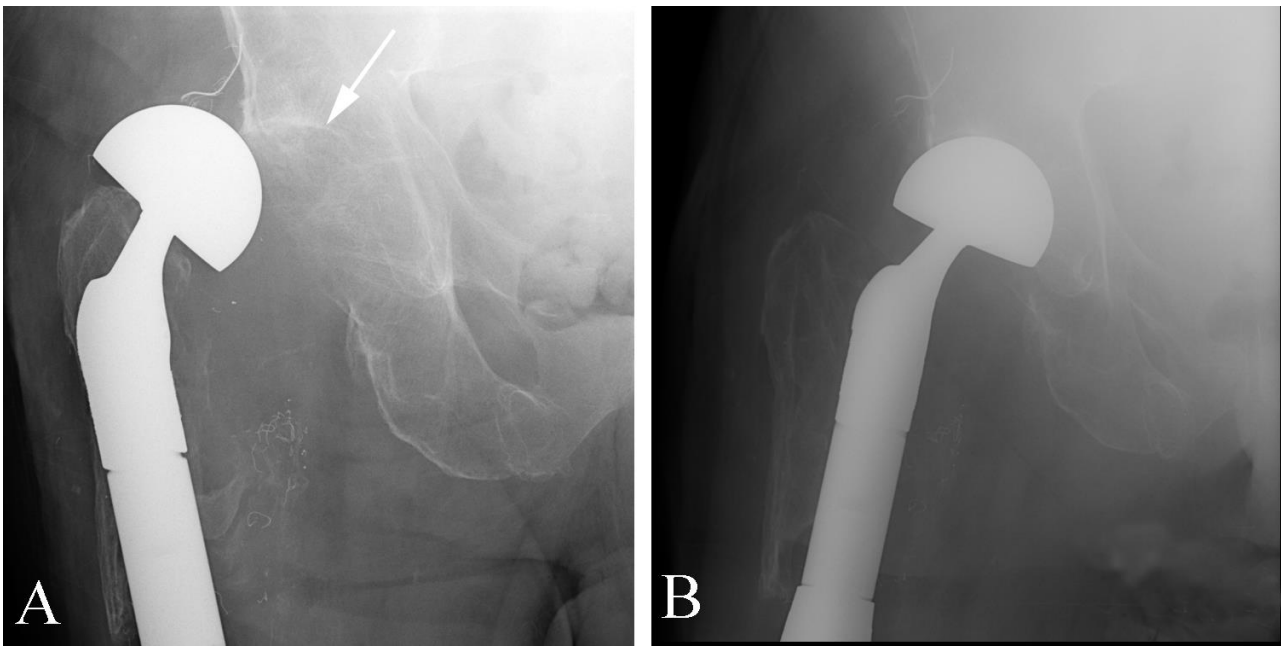

Figure 40. Dislocated hip prosthesis in an 87 year old with pain and hip deformity. A. AP plain film of the hip shows the femoral prosthesis displaced superolateral to the acetabulum (arrow). B. AP plain film of the hip following reduction demonstrates correct positioning of the prosthesis.

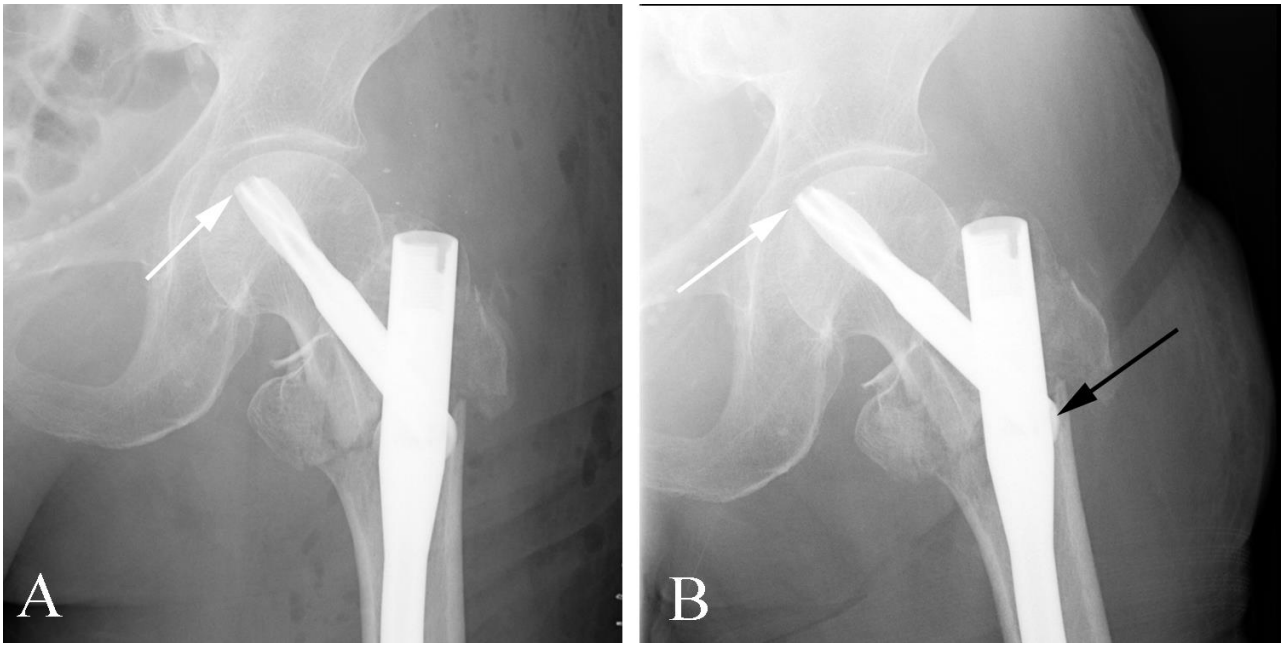

Figure 41. Complication of fixation device in an 82 year old woman s/p ORIF. A. AP plain film of the hip demonstrates the fixation device with an intramedullary rod and interlocking blade-plate with the tip of the blade-plate device projecting inside the femoral head (arrow). B. AP plain film shows migration of the helical blade plate through the femoral head cortex. Such migration is generally prevented in these devices because the device can "back out" of the medullary rod. In this case, the position of the device prevented "backing out" because the sliding, interlocking helical blade plate was covered by the margin of the femoral shaft (black arrow).

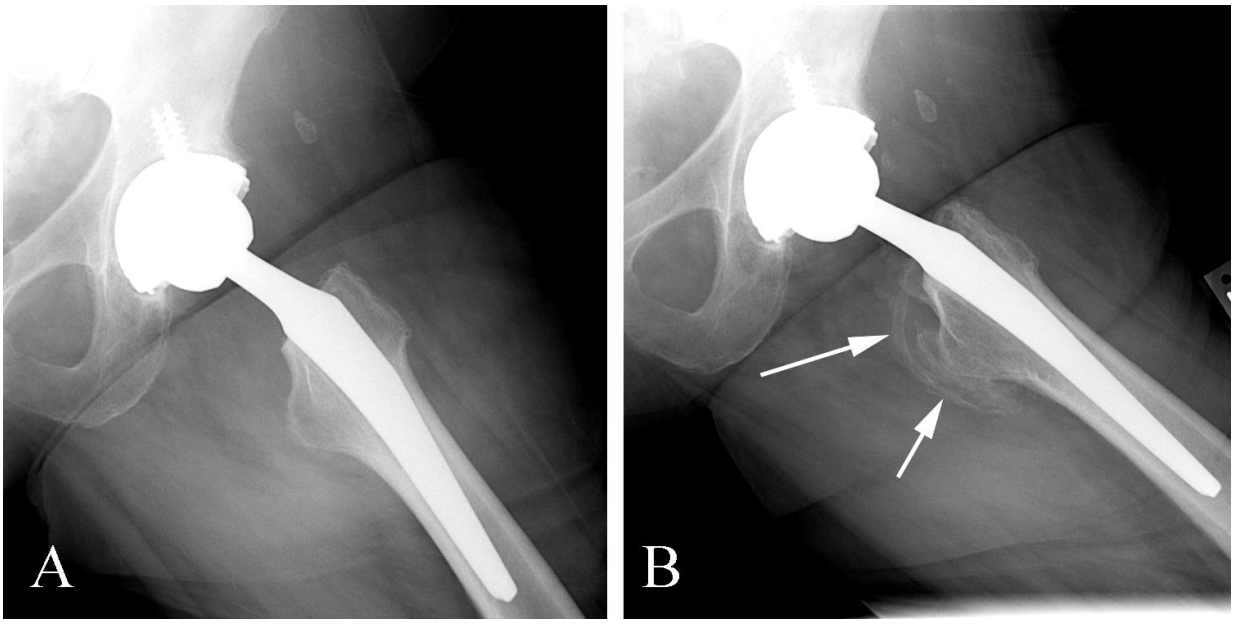

Figure 42. Dystrophic ossification in a 72 year old woman following hip prosthesis placement. A. Frog-lateral plain film of the hip following hip prosthesis placement demonstrates a normal post-operative appearance of the components and native bone and soft tissues. B. Frog-lateral plain film of the hip three months later demonstrates heterotopic bone formation along the hip joint margin (arrows).

Knee

As in other joints, x-ray examination is the first step in imaging the traumatized knee. Knee films may show and fully characterize the fracture, so that no further examination is necessary (Figure 43). Plain films may demonstrate a severe fracture that needs further evaluation with CT for surgical planning (Figure 44). Plain films may show a fracture that has a known associated significant ligamentous or other soft tissue injury, with an MR required for further evaluation (Figures 45 and 46). On the other hand, the plain films may show a nonspecific effusion, which suggests possible internal derangement and likely requires further work-up with MRI as well (Figure 47). If the plain films are negative and the patient has significant pain and/or instability, MR should be performed. MR has the ability to accurately characterize a wide range of injuries which may show no significant plain film findings, including: anterior cruciate ligament contusion and rupture (Figure 47); posterior cruciate ligament rupture (Figure 45); collateral ligament rupture (Figure 47); posterolateral corner injury; transient dislocation of the patella (Figure 46); radiographically occult fractures (Figure 48,); bone contusions; muscle tears (Figure 49); cartilage injuries; and meniscal tears (Figure 50). A negative MR effectively excludes significant osseous, cartilaginous, ligamentous, and tendinous injury.

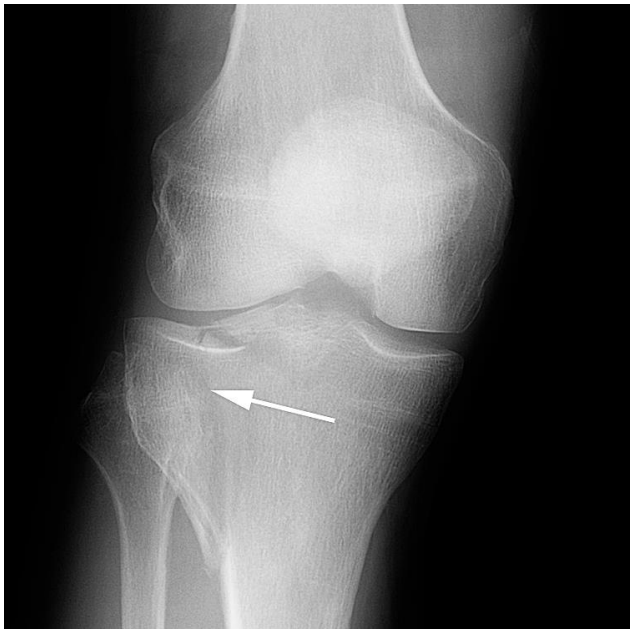

Figure 43. Tibial fracture in a 66 year old man with pain following trauma. AP plain film exam shows a parasagittal fracture through the lateral tibial plateau (arrow).

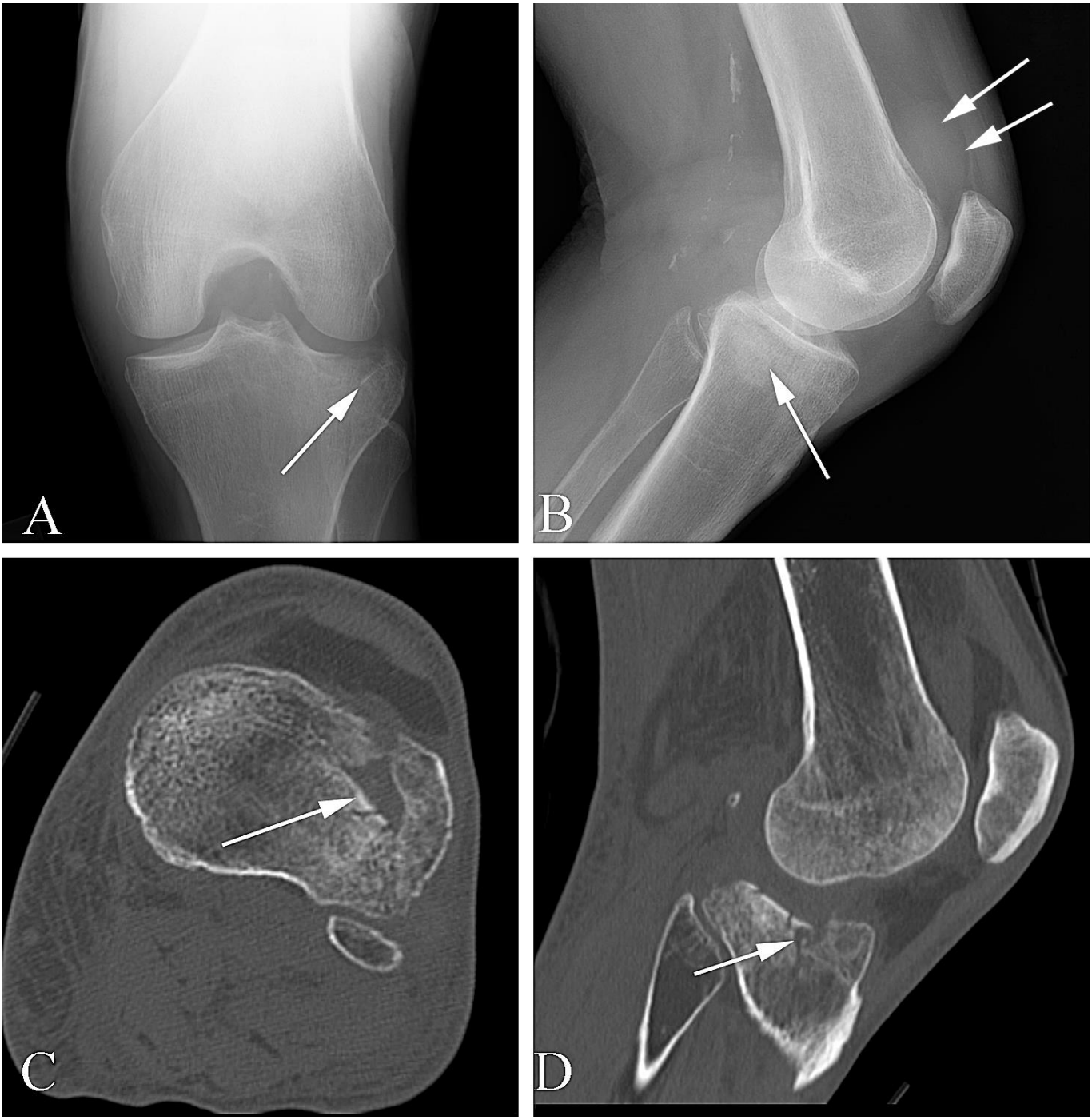

Figure 44. Tibial fracture in a 67 year old woman with knee pain following trauma. A. AP "notch" plain film of the knee shows a fracture along the lateral aspect of the tibial plateau (arrow). B. Lateral plain film of the knee shows an ill-defined density projecting at the level of the proximal tibia (arrow). Note the associated joint effusion superior to the patella (double arrow). C. Axial CT study shows a "hole" in the tibial plateau with rotation of the cortex out of its usual position (arrow). D. Sagittal reformatted CT study shows the tibial plateau fracture and documents the extent of depression and separation of fragments along the articular surface, as well as the number of fragments and their orientation (arrow).

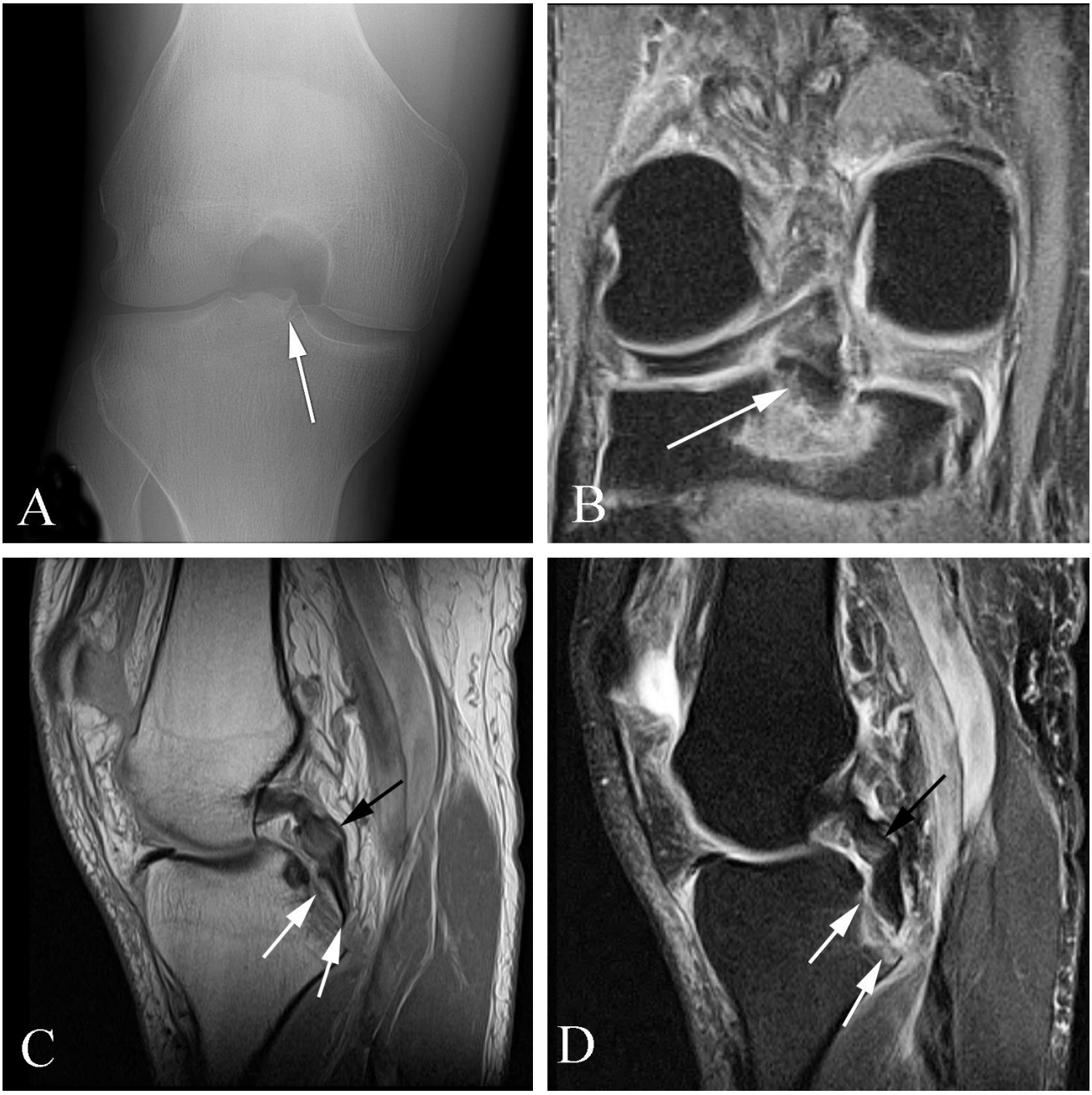

Figure 45. Fracture with associated soft tissue injury (posterior cruciate ligament avulsion) in a 49 year old man with pain following trauma. A. AP "notch" view of the knee shows a relatively subtle lucency in the mid portion of the tibial plateau. B. Coronal fat-suppressed proton density MR image shows a bone fragment of the tibial plateau (arrow) surrounded by a wide band of abnormal signal intensity. C. Sagittal proton density MR image shows separation of the posterior cruciate ligament from its insertion along the proximal, posterior tibia (white arrows). Note the abnormal thickening of the mid-portion of the posterior cruciate ligament compatible with tear (black arrow) D. Sagittal fat-suppressed T2 weighted image demonstrates abnormal signal intensity along the insertion of the posterior cruciate ligament (arrows).

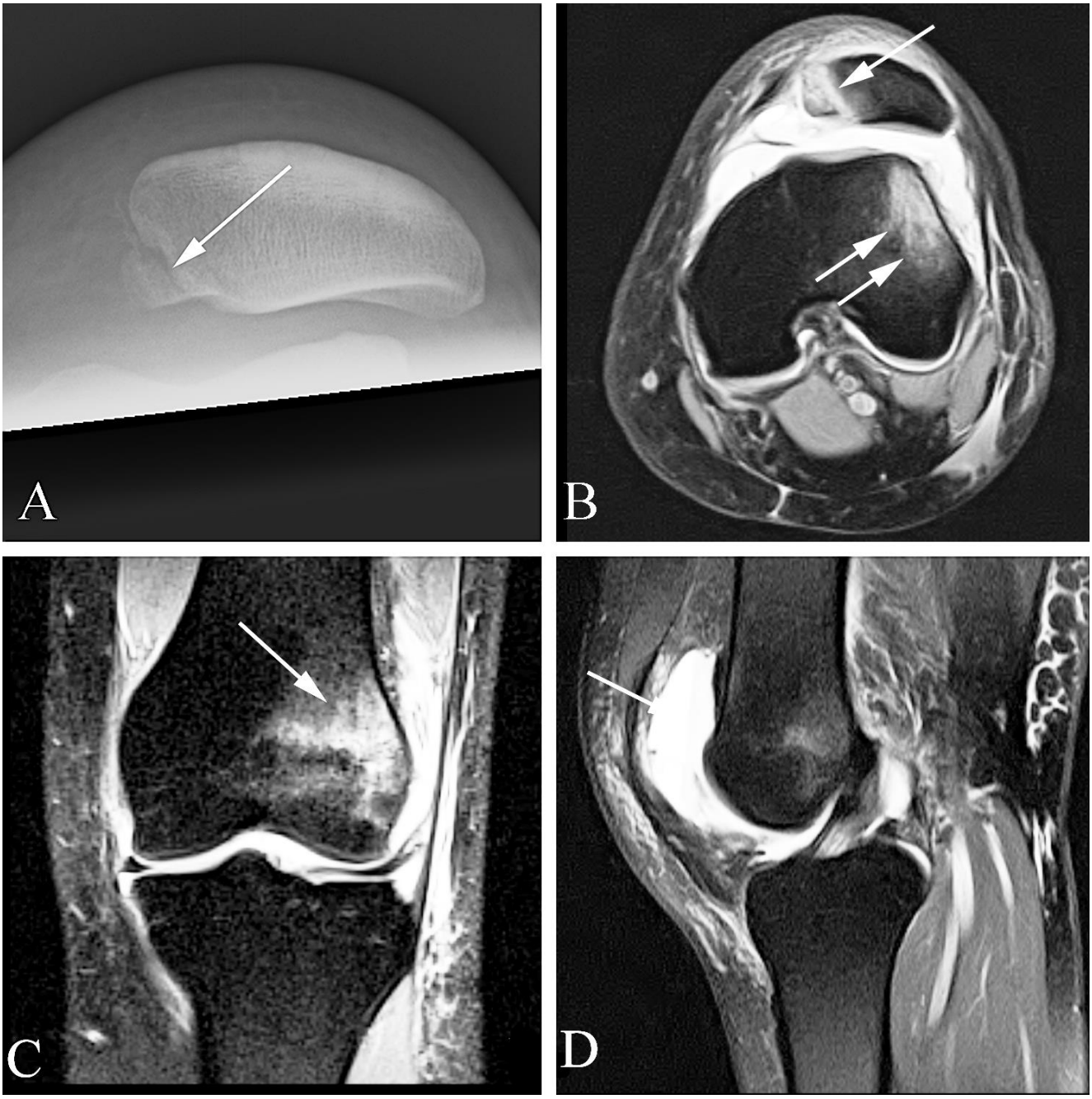

Figure 46. Patellar fracture from transient lateral patellar dislocation in a 22 year old woman with pain following trauma. A. Plain film axial (also known as a "sunrise") view demonstrates a fragment of bone along the medial patellar facet (arrow). B. Axial fat-suppressed proton density MR image shows the fracture (single arrow) along with abnormal increased signal intensity through the fracture fragment. In addition, there is extensive abnormal signal along the anterior, lateral aspect of the lateral femoral condyle (double arrows) from the associated contusion secondary to the transiently dislocated patella. C. Coronal fat-suppressed T2 weighted image also demonstrates the contusion along the anterior lateral femoral condyle (arrow). Note that this contusion is in a different location than that seen with an acute ACL tear. D. Sagittal fat-suppressed T2 weighted image demonstrates a fluid level in a knee joint effusion (arrow), indicating hemorrhage from the recent fracture.

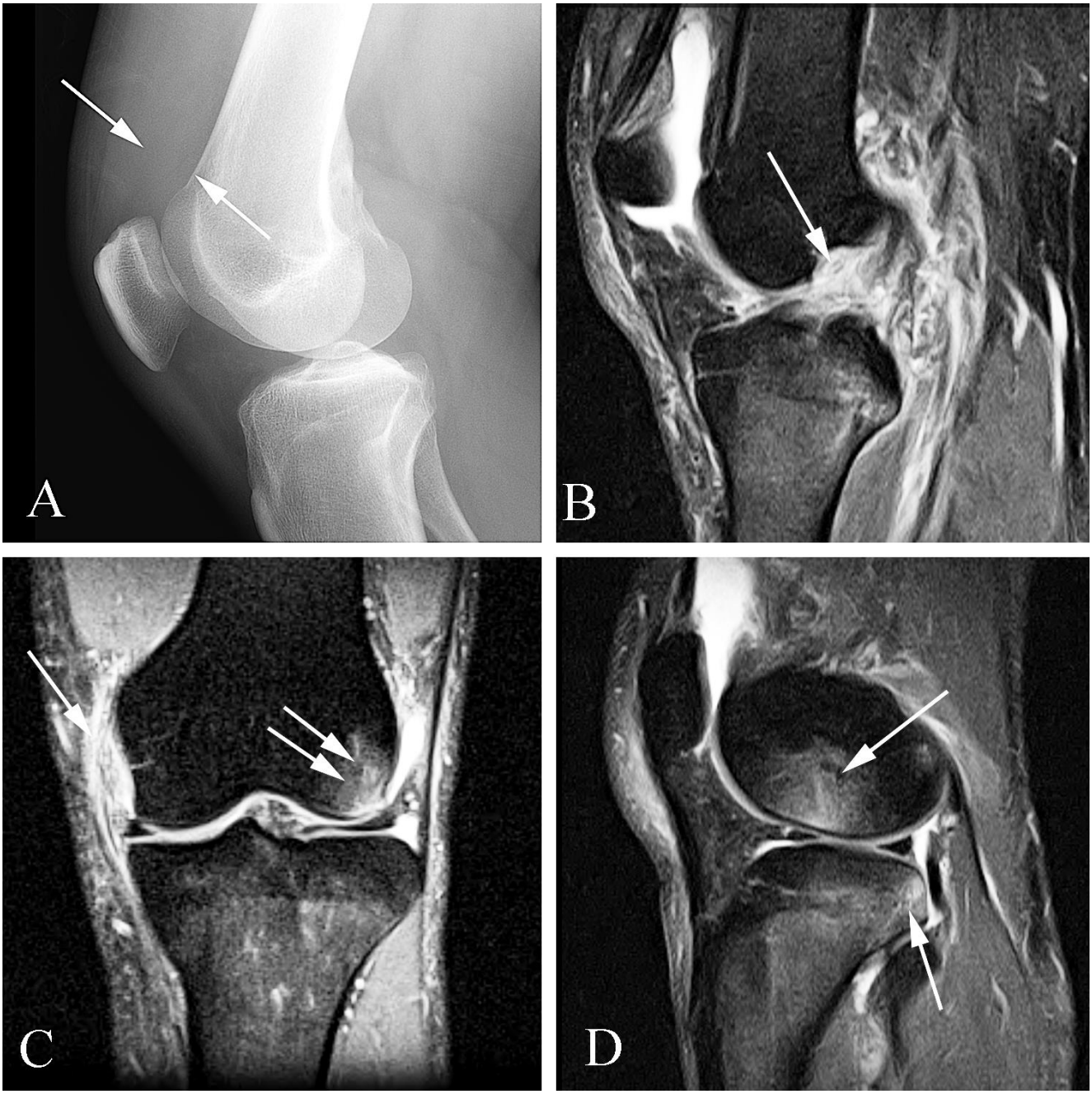

Figure 47. Knee effusion with associated ACL and MCL tears in a 33 year old man with knee pain. A. Lateral plain film shows fullness in the suprapatellar bursa (arrows), nearly always indicating a knee joint effusion. B. Sagittal fat-suppressed T2 weighted image demonstrates discontinuity of the anterior cruciate ligament (ACL) (arrow) secondary to an ACL tear. C. Coronal fat-suppressed proton density image demonstrates a tear of the proximal medial collateral ligament (MCL) (single arrow) along with abnormal increased signal in the lateral femoral condyle (double arrow) from bone marrow contusion. D. Sagittal fat-suppressed proton density image demonstrates so-called "kissing contusions" (arrows) of the lateral femoral condyle and posterior tibial plateau created by the pivot-shift injury which caused the patient's ACL and MCL tears.

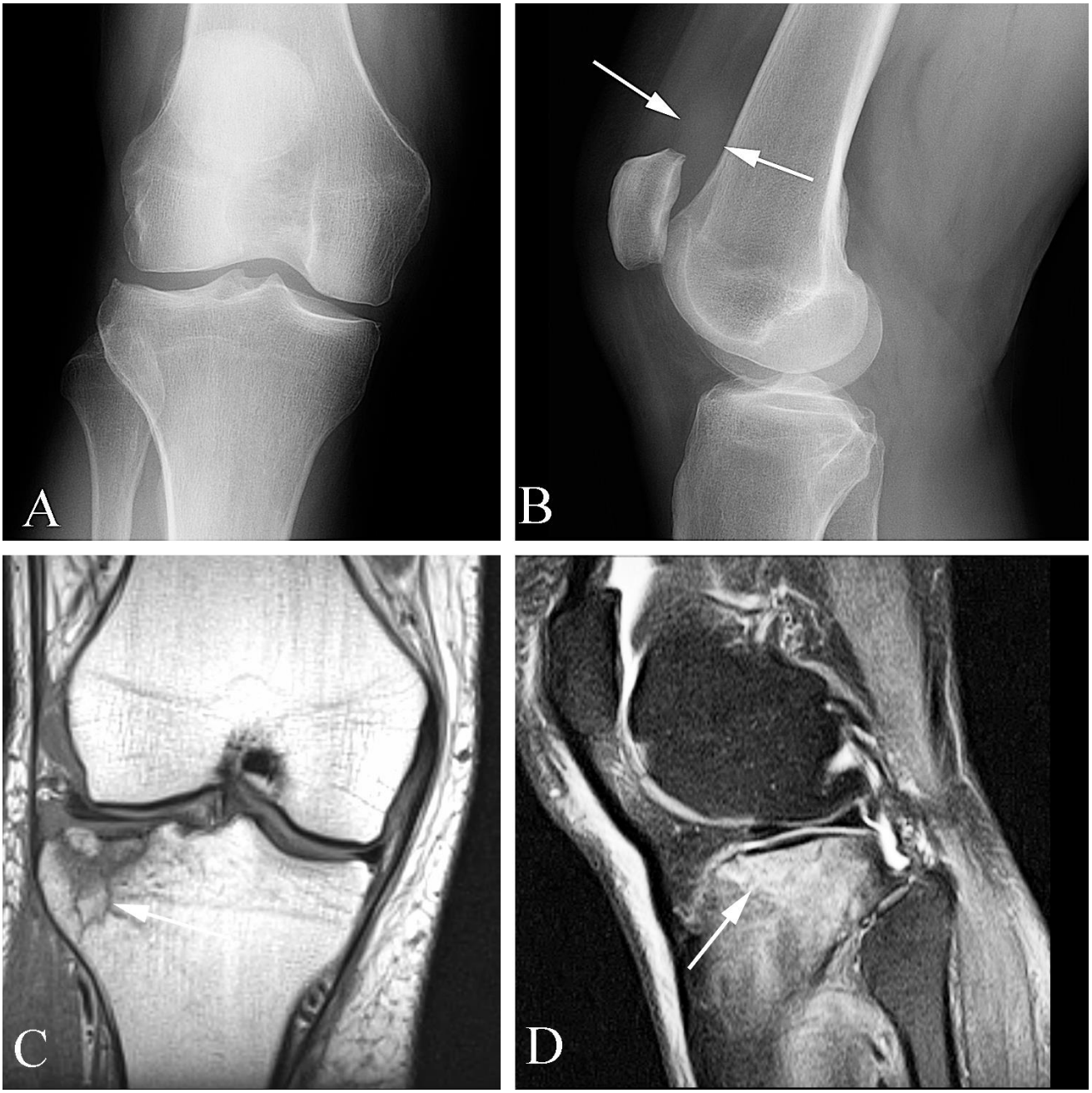

Figure 48. Radiographically occult fracture in a 62 year old man with pain following trauma. A. AP plain film of the knee shows no abnormality. B. Lateral plain film of the knee demonstrates fullness of the suprapatellar bursa (arrows), usually indicating a knee joint effusion. In the setting of acute trauma, this could indicate either a radiographically occult fracture, internal derangement (for example, a torn anterior cruciate ligament), or both. C. Coronal proton density MR image shows a combination of fracture (arrow) and contusion through the lateral tibial plateau. D. Sagittal fat-suppressed T2 weighted image demonstrates extensive increased signal intensity through the lateral tibial plateau (arrow) compatible with contusion.

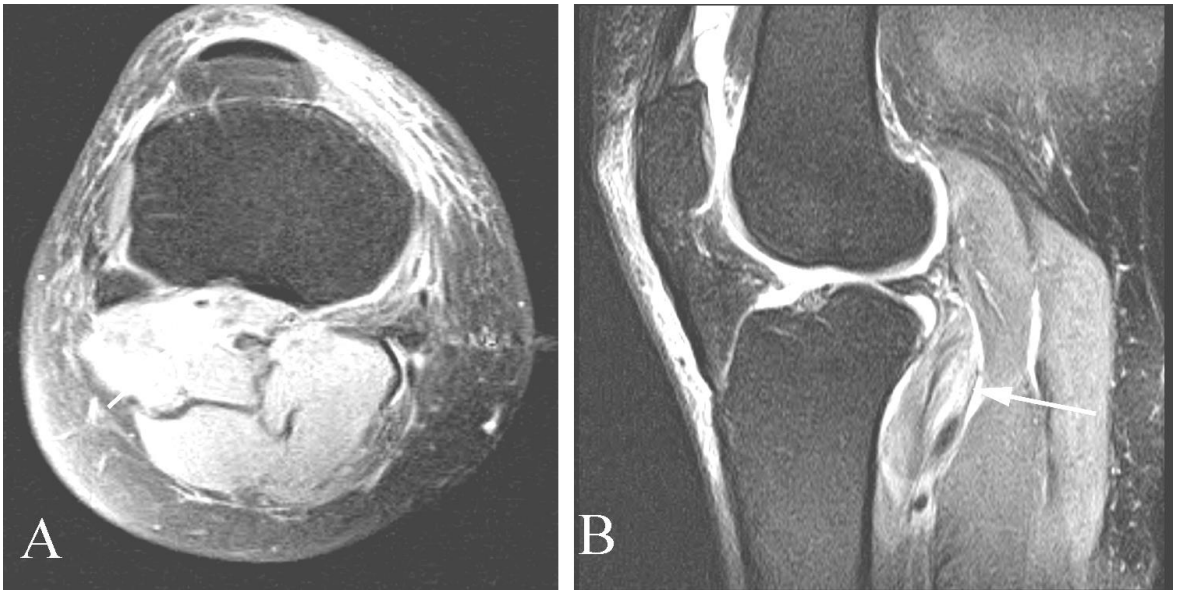

Figure 49. Popliteal muscle tear in a 48 year old man with pain following trauma. A. Axial fat-suppressed T2 weighted image shows abnormal signal and swelling of the popliteus muscle (arrow), compatible with a strain (partial thickness musculotendinous tear). B. Sagittal fat-suppressed T2 weighted MR imaging also demonstrates abnormal signal and swelling of the popliteus muscle (arrow).

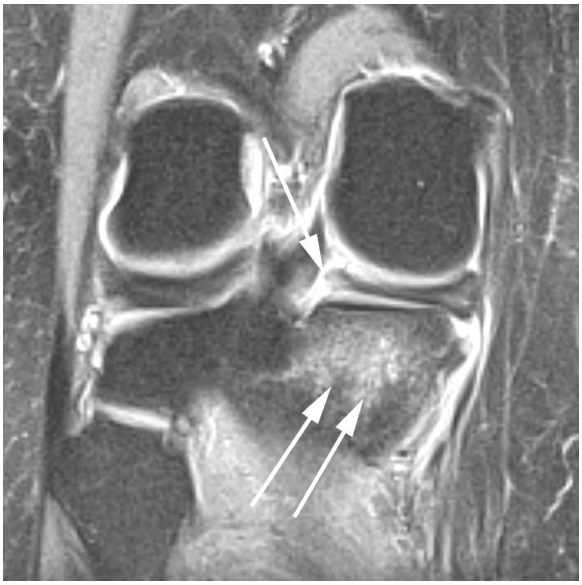

Figure 50. Meniscal tear and tibial contusion in a 68 year old woman with knee pain following trauma. Coronal fat-suppressed T2 weighted image shows both a tear of the dorsal root of the medial meniscus (single arrow) and a contusion of the lateral tibial plateau (double arrow).

Chronic knee pain should also be first imaged with x-rays, which may demonstrate causative osteoarthritis (Figure 5,Chapter 13, page 186) or chondrocalcinosis characteristic of CPPD crystal deposition disease (Figure 3, Chapter 13, page 185). Note that CPPD causes not only chondrocalcinosis, but often preferential degenerative changes of the patellofemoral articulation[11]. When further imaging of the knee joint is required because the plain film does not demonstrate a reasonable explanation, MR may be performed. MR of the knee in chronic pain may demonstrate meniscal tears (Figure 51), loose bodies (Figure 52) or such infrequently seen entities as spontaneous osteonecrosis of the knee (SPONK) (Figure 53) and symptomatic bipartite patella (Figure 54). Beware of obtaining an MR without a plain film, because it is often difficult or impossible to see chondrocalcinosis on MR and a knee MR may be incorrectly interpreted as showing osteoarthritis and meniscal degenerative change rather than CPPD crystal deposition disease (Figure 55).

The considerations that apply to hip prostheses and hardware apply to the knee as well: follow-up films will typically be obtained by the operating orthopedic surgeon, with sequential plain film evaluation serving as the primary method of detecting hardware loosening or fracture, with a combined bone/WBC nuclear medicine study required to evaluate for possible infection.

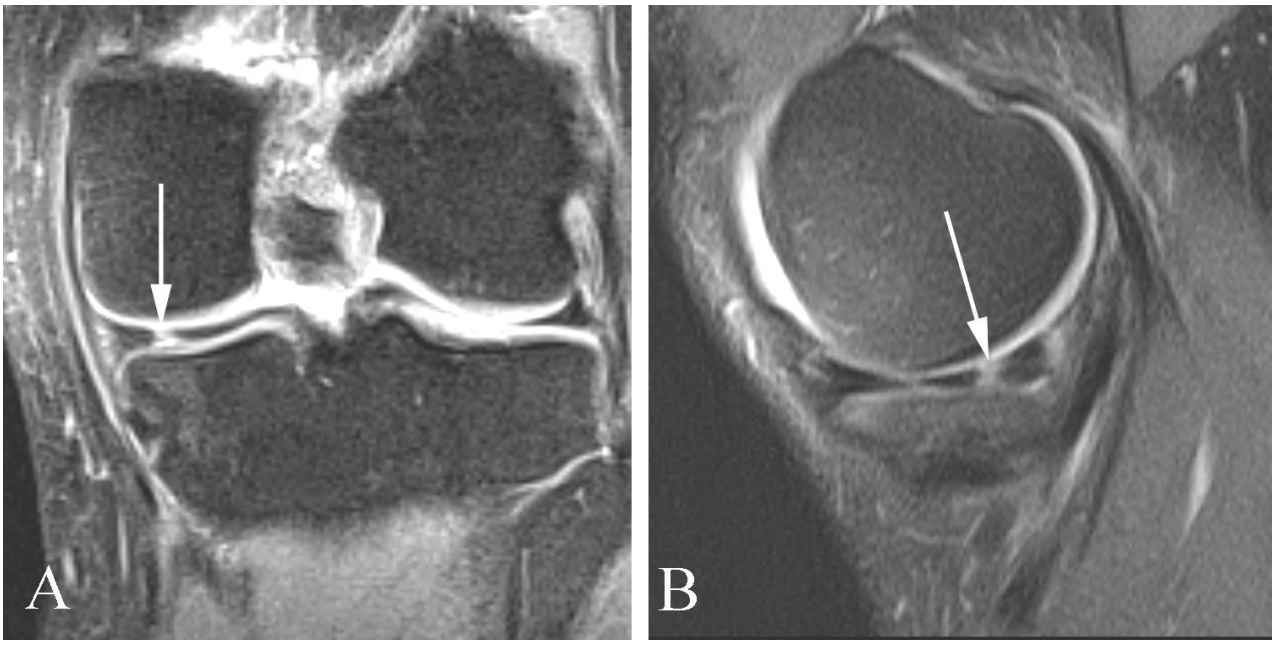

Figure 51. Meniscal tear in a 63 year old man with chronic knee pain. A. Coronal fat-suppressed proton density MR image shows a focal abnormality along the posterior horn of the medial meniscus (arrow). B. Sagittal fat-suppressed T2 weighted image (arrow) shows increased signal intensity extending through the meniscus from the femoral side to the tibial side (arrow), compatible with a full thickness meniscal tear.

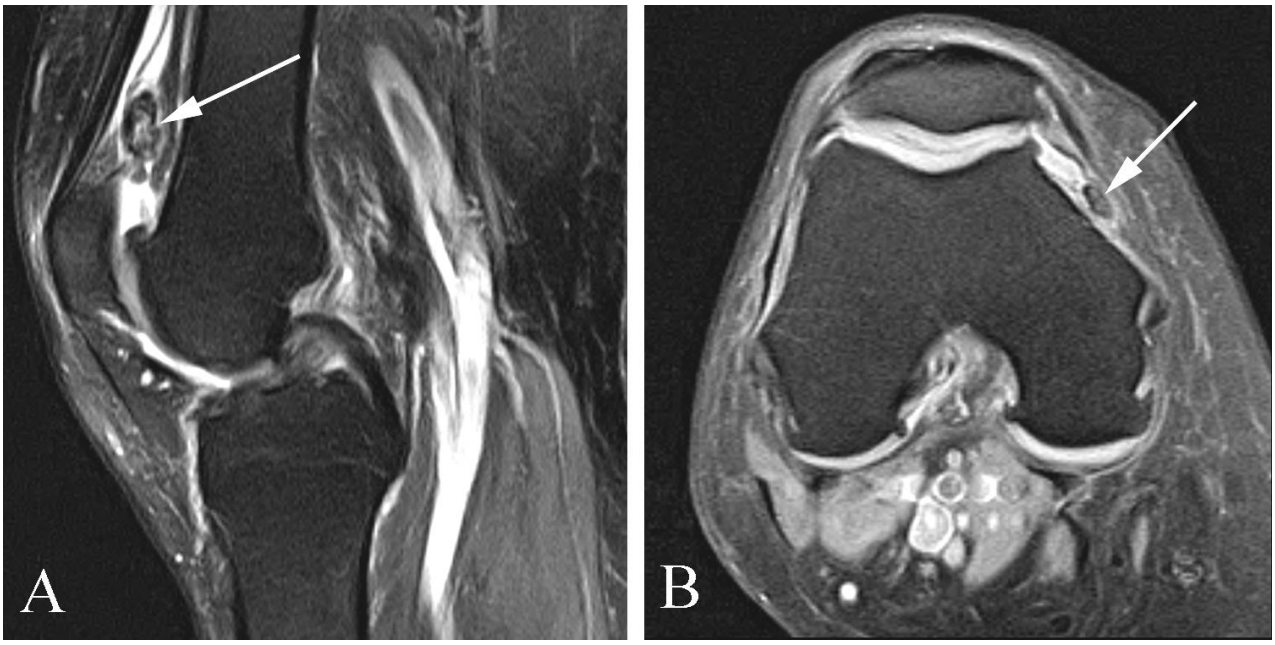

Figure 52. Loose body in the knee joint in a 56 year old woman with knee pain. A. Sagittal fat-suppressed T2 weighted image demonstrates an abnormality in the suprapatellar bursa (arrow). The lesion demonstrates mixed signal intensity compatible with an osseous fragment (with both cortical bone and bone marrow), and is surrounded by high signal intensity fluid. B. Axial fat-suppressed proton density image demonstrates another lesion along the lateral aspect of the joint (arrow). Note osteophytic spurring along the patellofemoral joint margins.

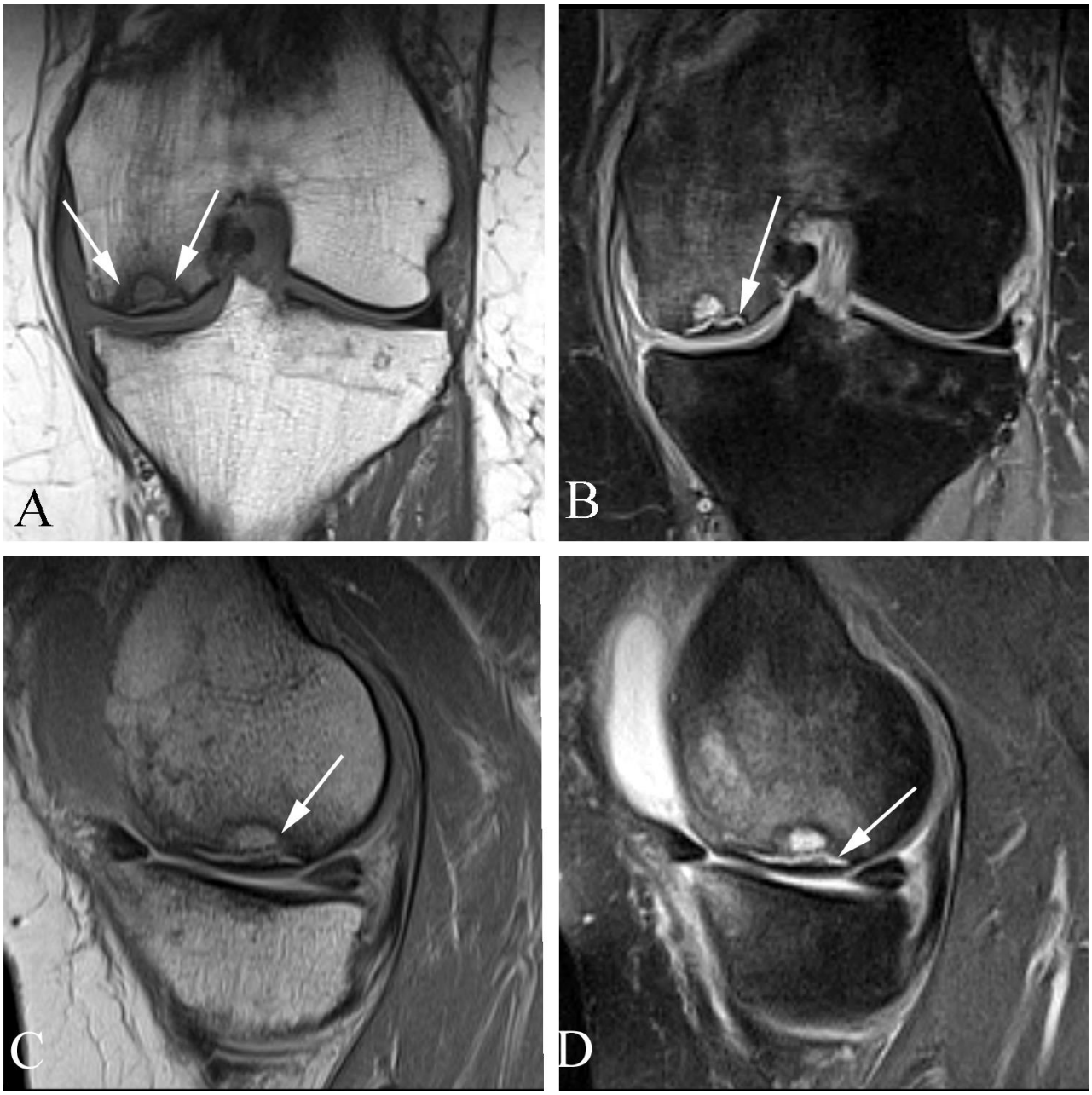

Figure 53. Spontaneous osteonecrosis of the knee (SPONK) in a 66 year old woman with chronic knee pain. A. Coronal proton density MR image shows focal signal abnormality along the medial femoral condyle (arrows). B. Coronal fat-suppressed T2 weighted image demonstrates extensive abnormal signal through the medial femoral condyle. Note the serpentine "double line" along the articular surface (arrow), characteristic of osteonecrosis. C. Sagittal proton density image shows the anterior to posterior extent of the osteonecrosis (arrow). D. Sagittal fat-suppressed T2 weighted image also demonstrates the "double line" of osteonecrosis paralleling the articular margin of the condyle (arrow).

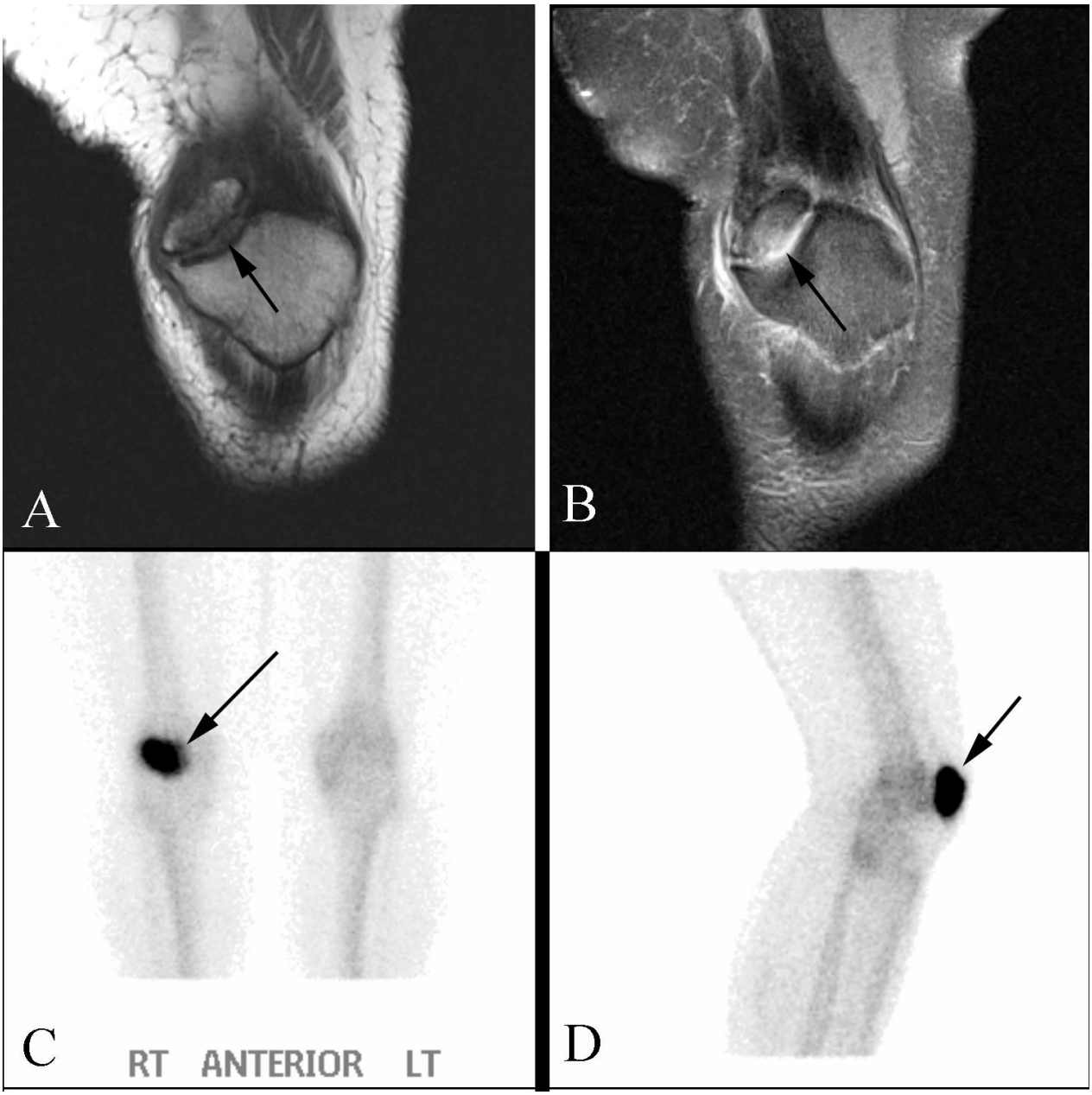

Figure 54. Symptomatic bipartite patella in a 41 year old woman with chronic knee pain. A. Coronal T1 weighted MR image demonstrates a cleavage plane between the body of the patella and a superolateral ossification center (arrow). Such an appearance is a relatively frequently seen (and usually asymptomatic) normal variation. B. Coronal fat-suppressed proton density image demonstrates increased signal intensity (arrow) along the interface between the patella and accessory ossification center. In asymptomatic patients, this interface will demonstrate *decreased*, not *increased* (as in this case), signal intensity. C. Frontal view nuclear medicine bone scan shows intense increased radiotracer uptake of the patella (arrow). D. Lateral view nuclear medicine bone scan confirms that the intense activity is in the patella (arrow) and not in the underlying femur.

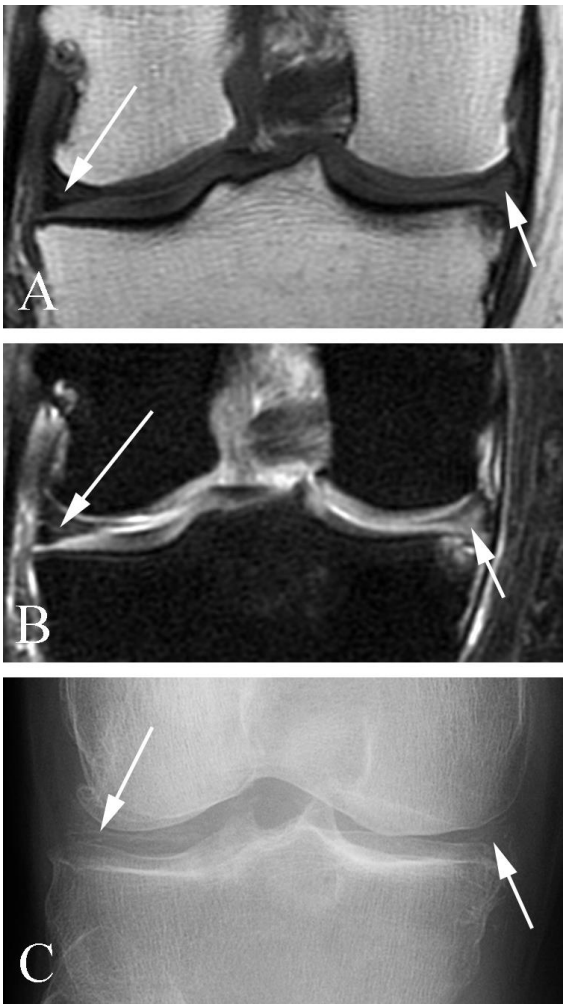

Figure 55. Chondrocalcinosis in a 71 year old woman with chronic knee pain, not seen on MR. A. Coronal proton density image shows mildly increased signal intensity in the menisci (arrows). B. Coronal fat-suppressed T2 weighted image also demonstrates increased signal intensity in the menisci (arrows). C. AP plain film examination shows extensive chondrocalcinosis (arrows).

Ankle

As in the other extremity joints, x-rays represent the first step in imaging the ankle. X-rays will demonstrate fractures (Figure 56) and fracture-dislocations (Figure 57) and will show ankle joint effusions which may be associated with fractures. For patients with negative plain films who have signs or symptoms of a radiographically occult fracture, MR is helpful. MRI of the traumatized ankle joint can demonstrate both fractures and ankle sprains. While imaging documentation of ankle sprains is not typically necessary, differentiation of a "regular" ankle sprain (involving the anterior talofibular, calcaneofibular, and posterior talofibular ligaments) (Figure 58) from a "high" ankle sprain (involving the distal tib-fib ligament) (Figure 59) may be important from a prognostic standpoint, given the necessity for athletes with a high ankle sprain to rehabilitate for longer prior to returning to play than athletes with a routine regular ankle sprain. MR can also detect radiographically occult fractures (Figure 60), and acute tendon ruptures.

For patients with chronic ankle pain, plain films may demonstrate osteoarthritis (Figure 61) or the rare condition of hypertrophic pulmonary osteoarthropathy (Figure 62). Plain films may also demonstrate either direct or indirect evidence of tarsal coalition in patients with painful flatfoot (Figure 63). CT is usually used for further evaluation of suspected tarsal coalition, given the superb bony detail of the complex articulations between the distal tibia and fibula, hindfoot, and midfoot, although MR may also demonstrate coalition (Figure 63). MR is more helpful in such soft tissue abnormalities as hindfoot sprain (Figure 64), tenosynovitis (Figure 65), tendon tears (Figure 66), bursitis (Figure 67) loose bodies within the ankle joint (Figure 68) and peroneus brevis tendon split (Figure 69).

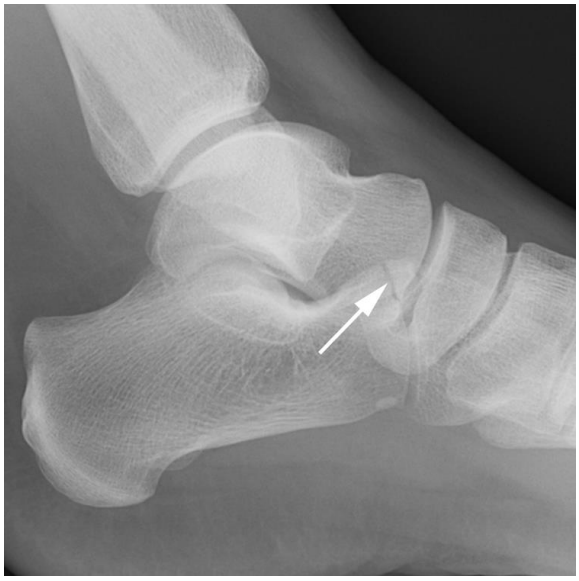

Figure 56. Anterior process fracture in a 25 year old woman with ankle pain following trauma. Lateral plain film of the ankle shows a fracture line (arrow) along the base of the anterior process of the calcaneus.

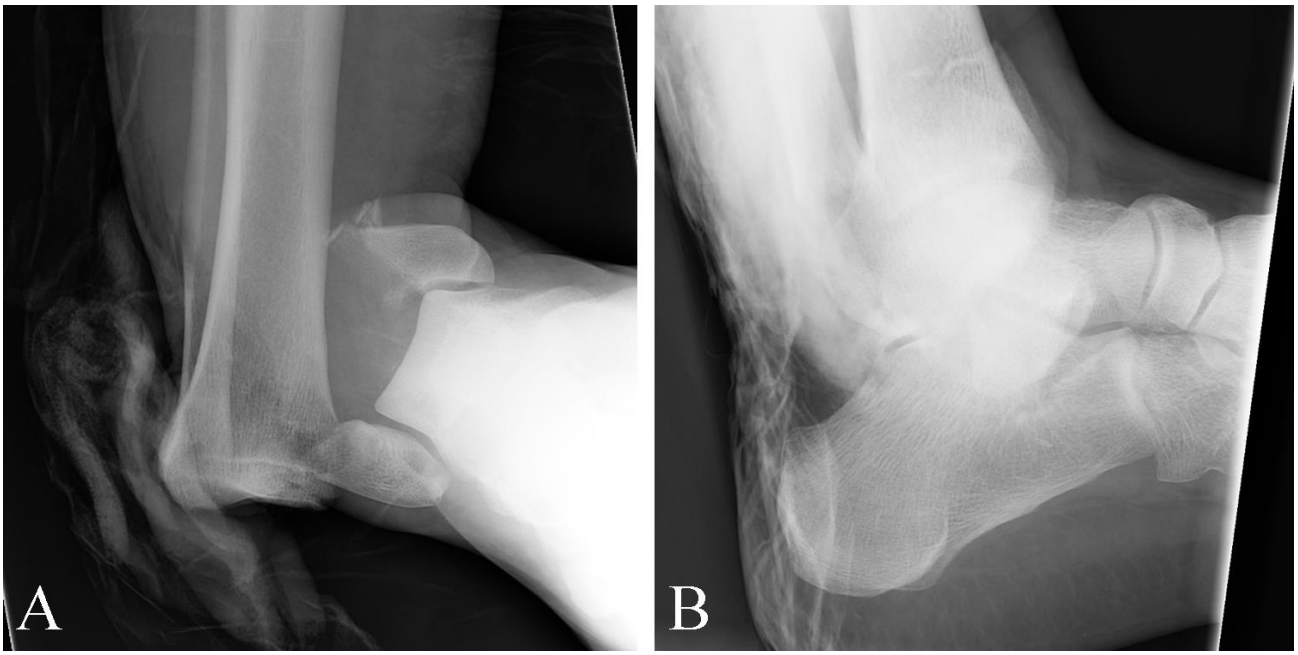

Figure 57. Fracture-dislocation of the ankle in a 46 year old man following a motor vehicle accident. A. AP plain film examination shows gross medial, proximal displacement of the talus. Note fractures of both the medial and lateral malleoli. B. Lateral plain film examination shows malalignment of the talus compared to the tibia, although this view clearly underestimates the true extent of derangement as seen on the frontal view.

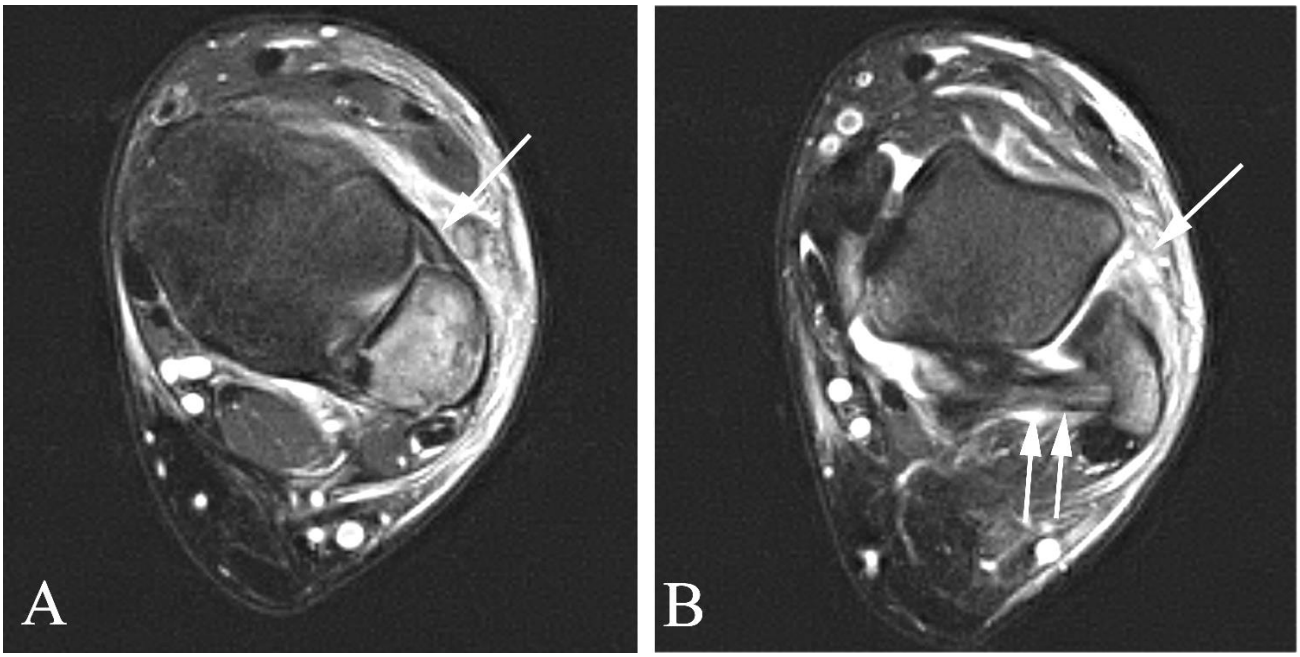

Figure 58. Ankle sprain (torn anterior talofibular ligament) in a 16 year old man with pain following trauma. A. Axial fat-suppressed proton density MR image shows a normal, intact distal syndesmotic connection (the distal tib-fib ligament) spanning the anterior aspect of the distal tibia and fibula (arrow). B. Axial fat-suppressed proton density MR image slightly inferior shows an intact posterior talofibular ligament (double white arrow). At the usual position of the anterior talofibular ligament, there is ill defined increased soft tissue density (arrow) without definition of the ligament itself, characteristic of a full thickness anterior talofibular ligament tear.

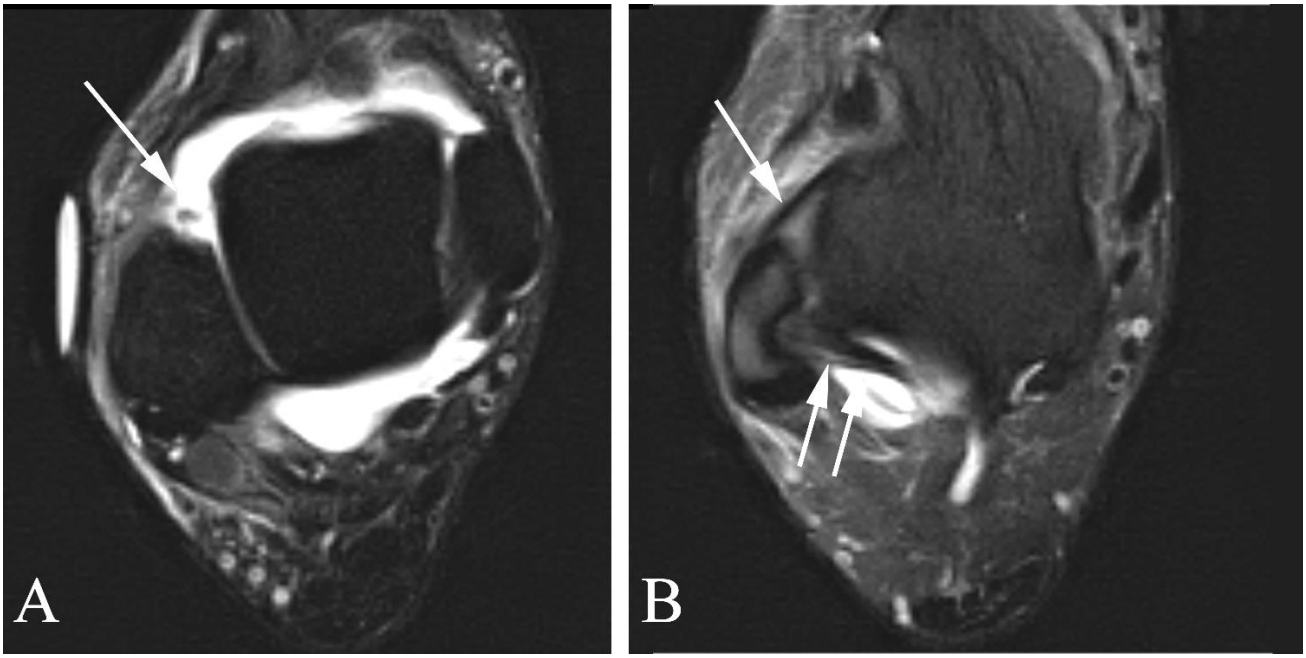

Figure 59. "High" ankle sprain in a 42 year old man with pain following trauma. A. Axial fat-suppressed proton density MR image shows lack of normal tissue between the distal tibia and fibula (arrow) because of a complete tear of the distal tib-fib syndesmosis. Note the large joint effusion with fluid (high signal intensity) along the anterior and posterior aspects of the talus. B. Axial fat-suppressed proton density MR image shows a normal anterior talofibular ligament (arrow) and posterior talofibular ligament (double arrow).

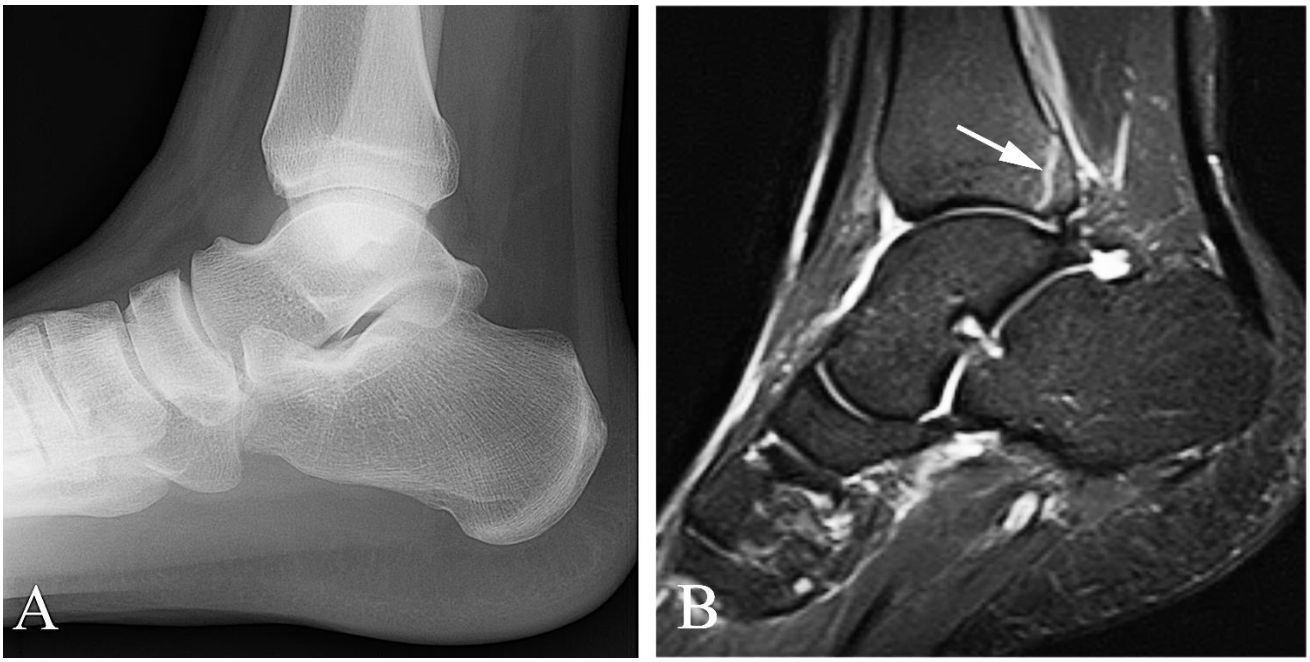

Figure 60. Radiographically occult fracture in a 28 year old man with pain following trauma. A. Lateral plain film examination of the ankle is normal. B. Sagittal fat-suppressed T2 weighted image demonstrates a fracture line (arrow) through the posterior, distal tibia (the so-called "posterior malleolus").

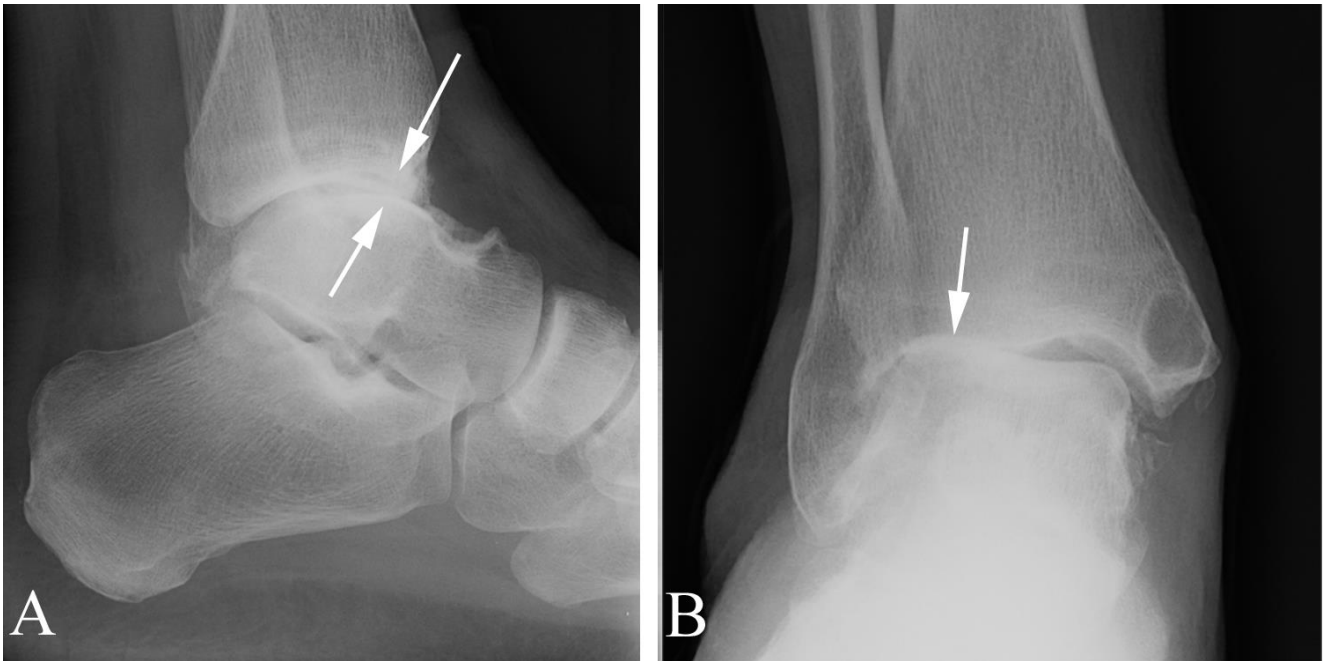

Figure 61. Osteoarthritis of the ankle in a 71 year old man with chronic ankle pain. A. Lateral plain film examination shows joint narrowing (arrows), subchondral sclerosis, and osteophytic spurring along the ankle joint. B. AP plain film also demonstrates joint narrowing, subchondral sclerosis, and osteophytic spurring (arrows). Note the large cyst in the medial malleolus.

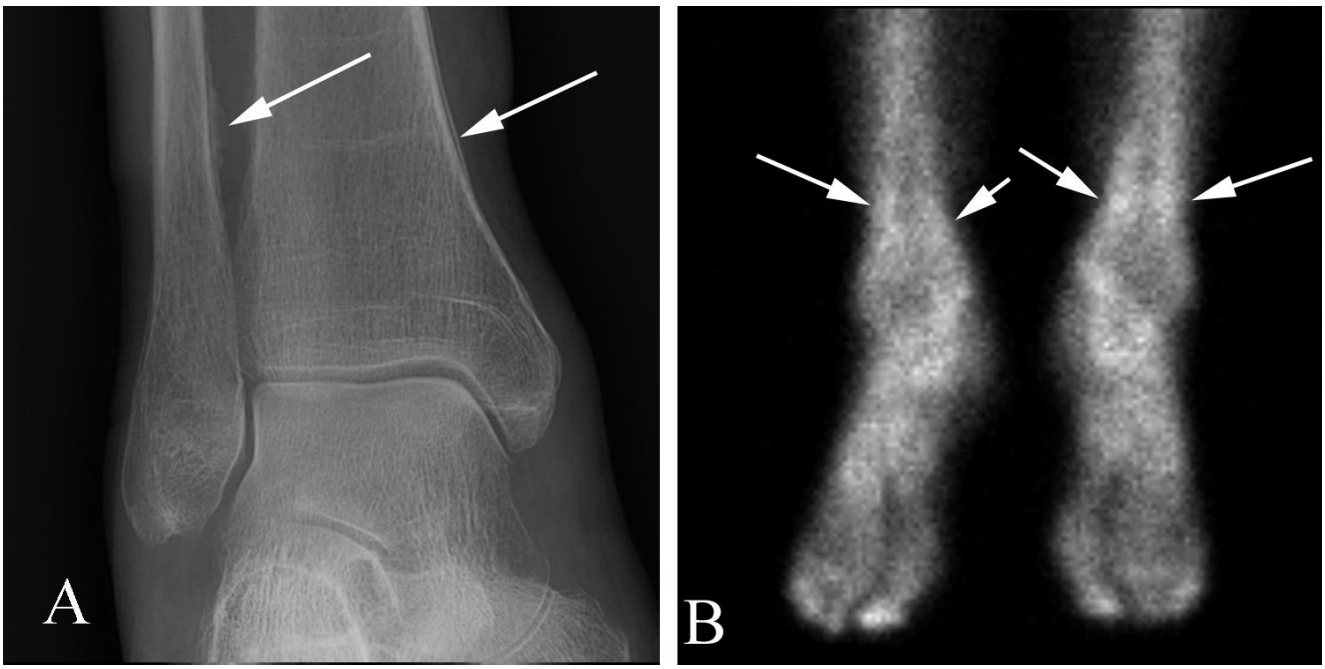

Figure 62. Hypertrophic pulmonary osteoarthropathy in a 74 year old woman with chronic ankle pain and lung cancer. A. Mortise view of the right ankle demonstrates diffuse periostitis along the distal fibula and tibia (arrows). The contralateral ankle (not shown) showed similar findings. B. Nuclear medicine bone scan shows diffuse increased radiotracer localization through both feet and ankles, including along the cortical margins of the distal tibia and fibula bilaterally (arrows). Chest CT (not shown) showed a large lung mass that proved to be cancer.

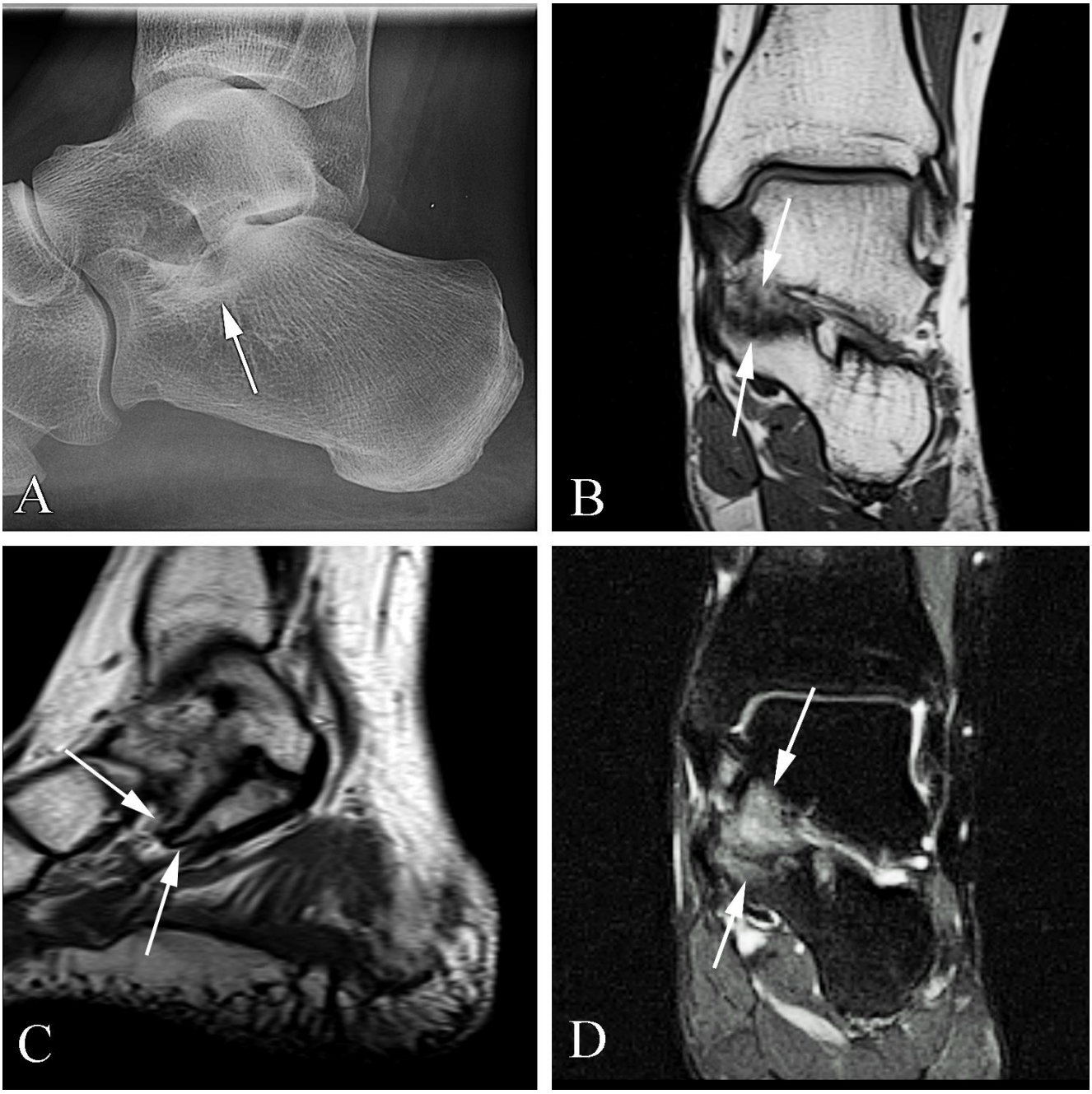

Figure 63. Subtalar coalition in a 32 year old woman with chronic ankle pain. A. Lateral plain film examination shows subtle increased density (arrow) along the margin of the subtalar joint. B. Coronal T1-weighted MR image demonstrates fusion across the medial subtalar joint (arrows) with a broad band of decreased signal intensity compatible with adjacent reactive change. C. Sagittal T1-weighted MR image through the level of the medial subtalar joint shows extensive spur formation along the joint margins (arrows). D. Coronal fat-suppressed T2 weighted image demonstrates increased signal intensity along the joint margins (arrows), compatible with reactive change.

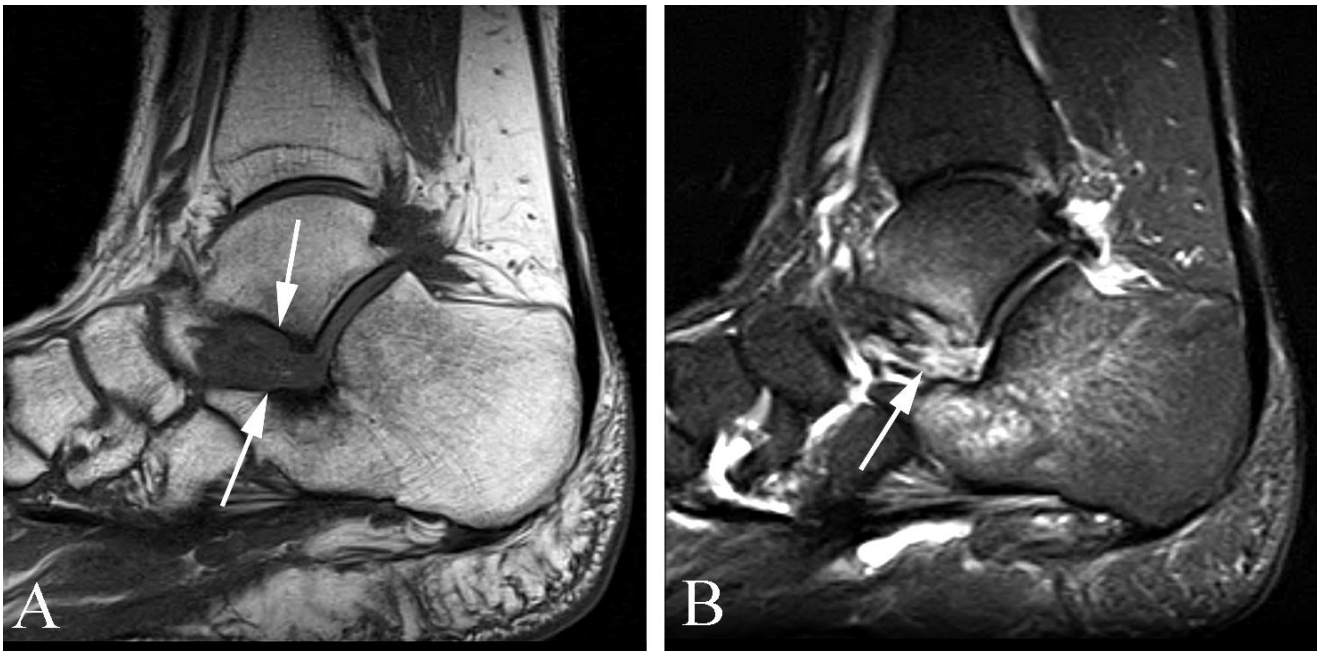

Figure 64. Hindfoot sprain in a 57 year old woman with remote trauma and chronic ankle pain. A. Sagittal proton density image demonstrates decreased signal intensity in the space between the talus and calcaneus (arrows), which is usually occupied by well-defined ligaments and fatty tissue. B. Sagittal fat-suppressed T2 weighted image demonstrates increased signal intensity in the space between the talus and calcaneus (arrow) as well as in the marrow along the adjacent talus and calcaneus.

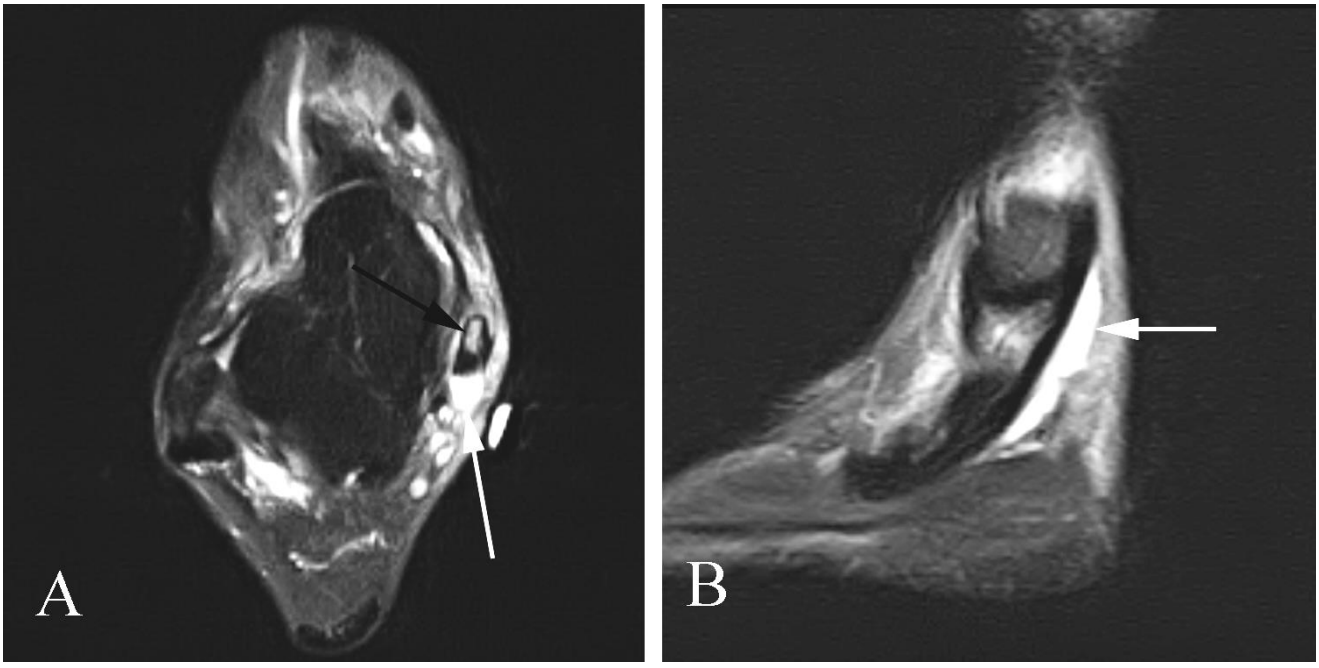

Figure 65. Posterior tibial tendon split and tenosynovitis in a 50 year old woman with chronic ankle pain. A. Axial fat-suppressed proton density image at the level of the ankle joint shows fluid along the posterior tibial tendon (white arrow) as well as abnormal increased signal within the tendon (black arrow). B. Sagittal fat-suppressed T2 weighted image demonstrates fluid along the tendon sheath (arrow).

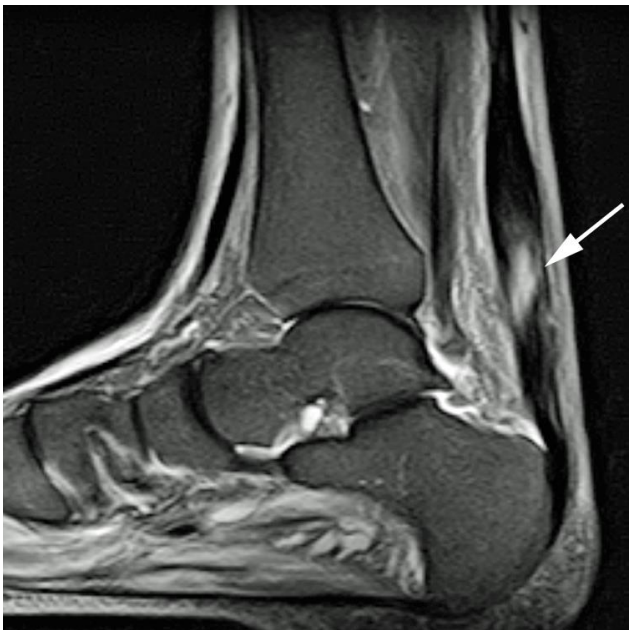

Figure 66. Partial thickness Achilles tendon rupture in a 72 year old man with diabetes and chronic ankle pain. Sagittal proton density MR shows swelling and abnormal increased signal in the distal Achilles tendon (arrow).

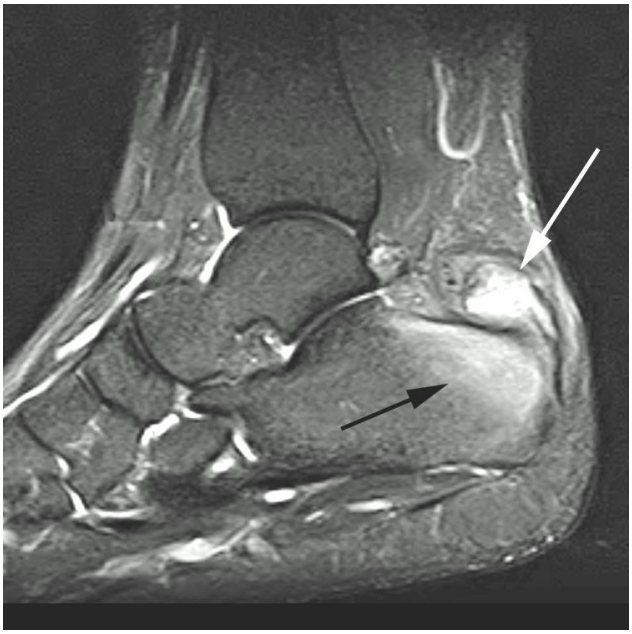

Figure 67. Achilles tendinitis and retrocalcaneal bursitis in a 27 year old woman with chronic ankle and heel pain. Sagittal proton density MR shows abnormal increased signal intensity in the calcaneus (black arrow) as well as focal abnormal signal in the fat superior to the posterior tuberosity of the calcaneus (white arrow).

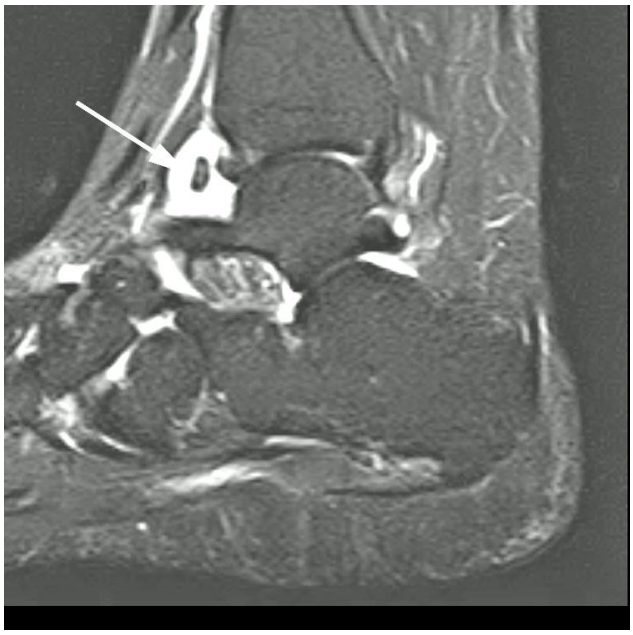

Figure 68. Loose body and degenerative change in a 55 year old woman with chronic ankle pain. Sagittal fat-suppressed proton density image shows a loose body (arrow) floating in effusion fluid anterior to the joint.

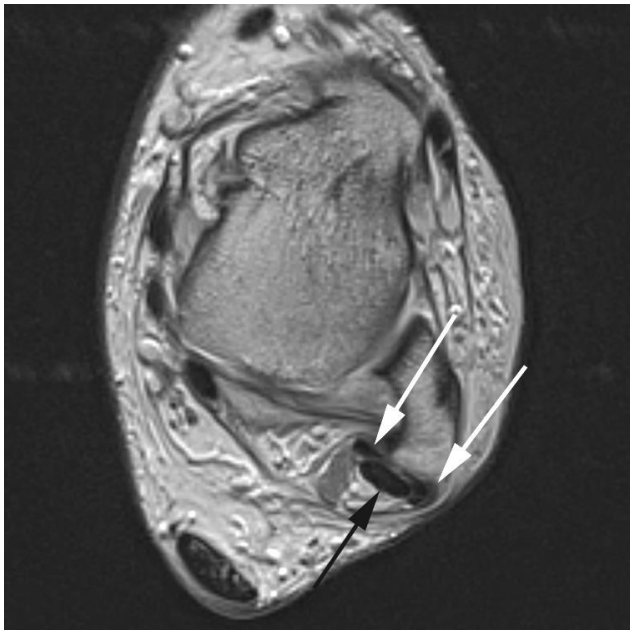

Figure 69. Peroneal tendon split in a 69 year old man with chronic ankle pain. Axial proton density image demonstrates the peroneus longus tendon (black arrow) against the posterior aspect of the fibula, with the split portions of the peroneus brevis displaced medially and laterally (white arrows).

Foot

As in the other joints of the body, plain film evaluation is the initial study of choice following acute injury. Radiographs will show fractures (Figure 70) and dislocations. Standing views with comparison to the other side are helpful to evaluate subtle degrees of subluxation at the base of the first and second metatarsal characteristic of a Lisfranc fracture (Figure 71), an injury which may lead to devastating long-term disability if missed. If plain films are unremarkable and the suspicion of fracture is high, CT may be helpful showing fractures of the overlapping bones within the midfoot (Figure 72).

Evaluation of chronic foot pain also begins with a plain film examination, which may document clinically obvious hallux valgus and cock-up toe deformity (Figure 73). Plain films may also diagnose stress fractures of the metatarsals (Figure 74), osteonecrosis of the head of the second metatarsal (also known as Freiberg's infraction), osteoarthritis (Figure 75), or gout (Figure 76). Diabetics frequently have foot problems secondary to peripheral neuropathy and vascular disease. Plain film examination is the first step in diagnosis of diabetics with foot symptoms. The neuropathy may lead to a denervation arthropathy of the midfoot known as a Charcot joint which is typically not particularly painful but results in swelling, warmth, and deformity including flatfoot (Figure 77). The vascular disease often leads to infection, and plain films may show osteomyelitis (Figure 78) but if infection is suspected MR is often performed regardless of the plain film results, both to diagnose the infection and to document its extent (Figure 79). Infection of the foot may also occur secondary to open wounds, particularly when associated with embedded foreign bodies.

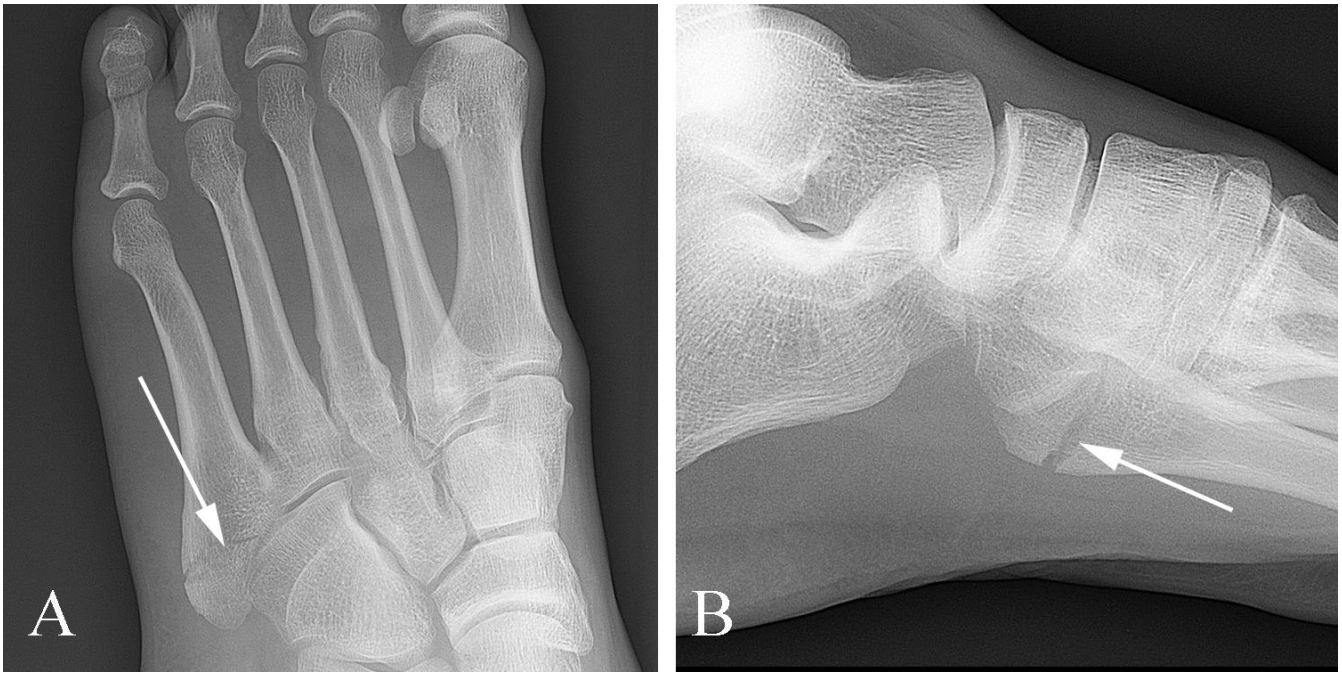

Figure 70. Fracture of the proximal aspect of the small toe metatarsal in a 57 year old man with acute foot pain following trauma. A. Oblique plain film of the foot demonstrates fracture lucency (arrow) through the base of the small toe metatarsal. B. Lateral examination also demonstrates the fracture lucency (arrow) along the base of the small toe metatarsal.

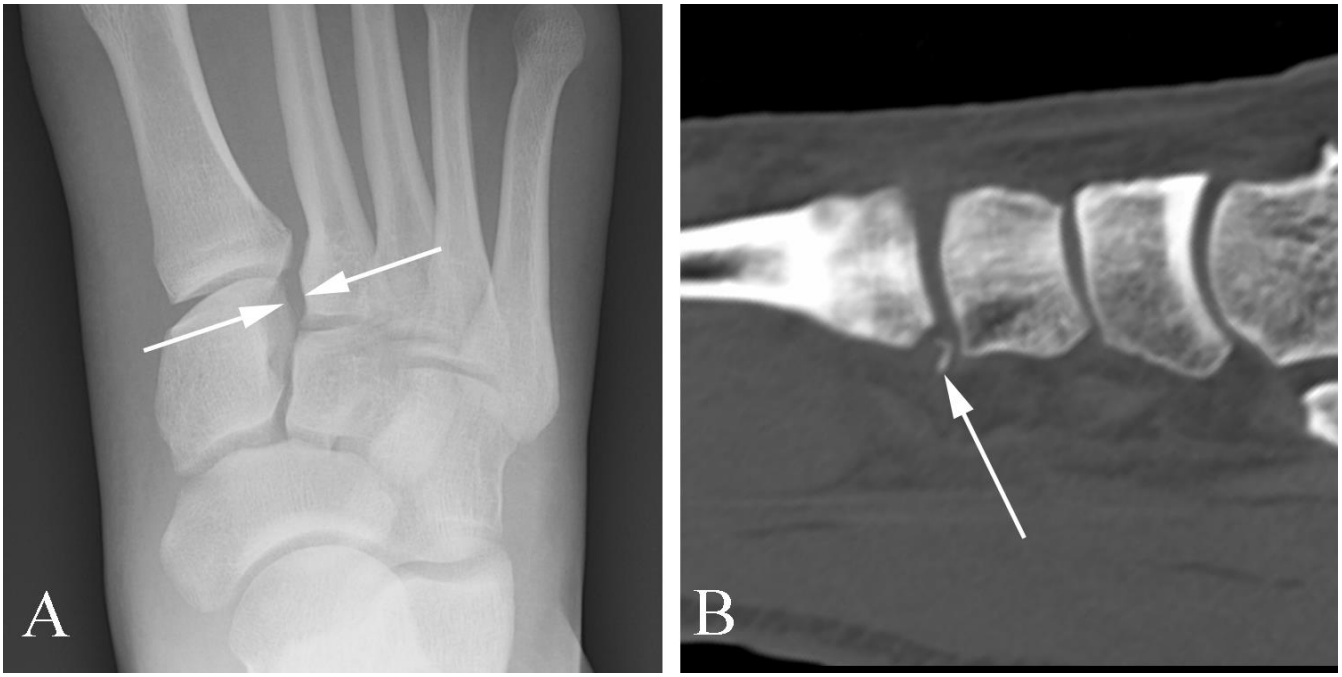

Figure 71. Lisfranc fracture in a 22 year old woman with acute foot pain following trauma. A. Oblique plain film examination of the foot demonstrates abnormal separation of the medial cuneiform and base of the second metatarsal (arrows). B. Sagittal reformatted CT study shows a small fracture fragment off of the second metatarsal base (arrow), compatible with avulsion of the plantar tarsal-metatarsal ligament.

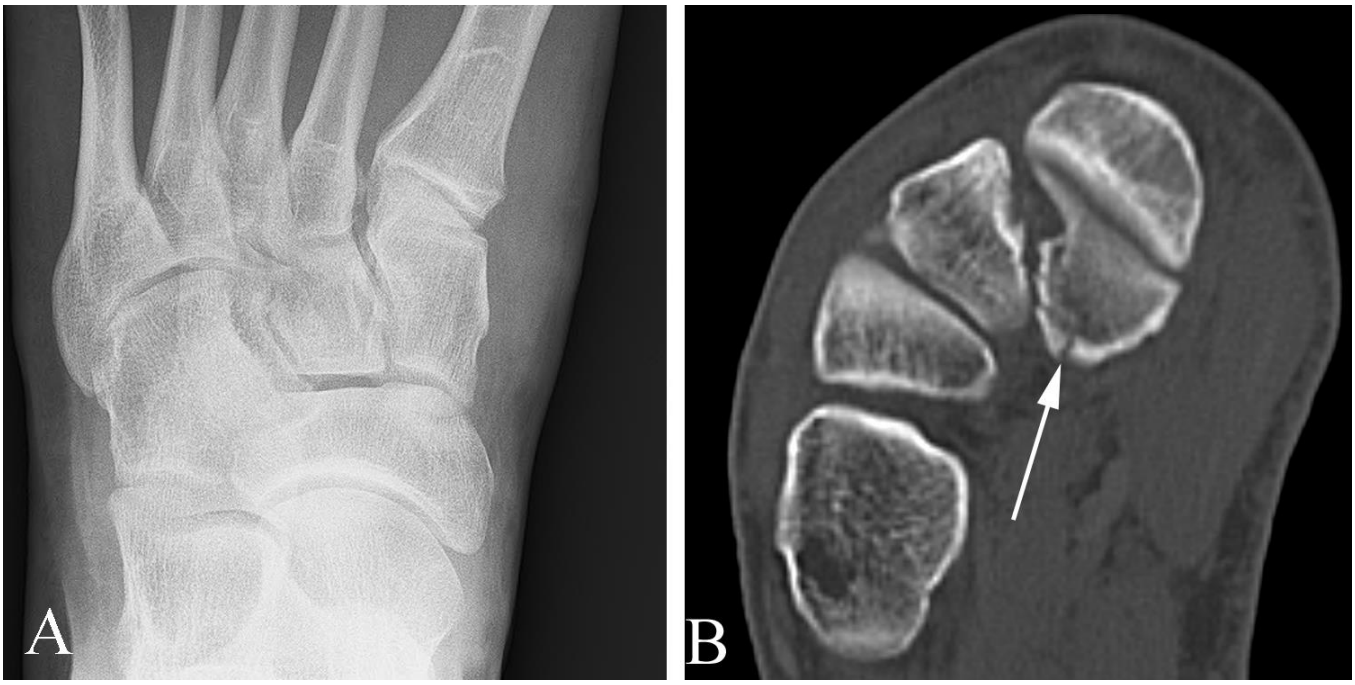

Figure 72. Occult (on plain films) cuneiform fracture in a 22 year old man with foot pain following trauma. A. Oblique plain film of the foot is normal. B. Axial CT examination shows a fracture through the medial cuneiform (arrow).

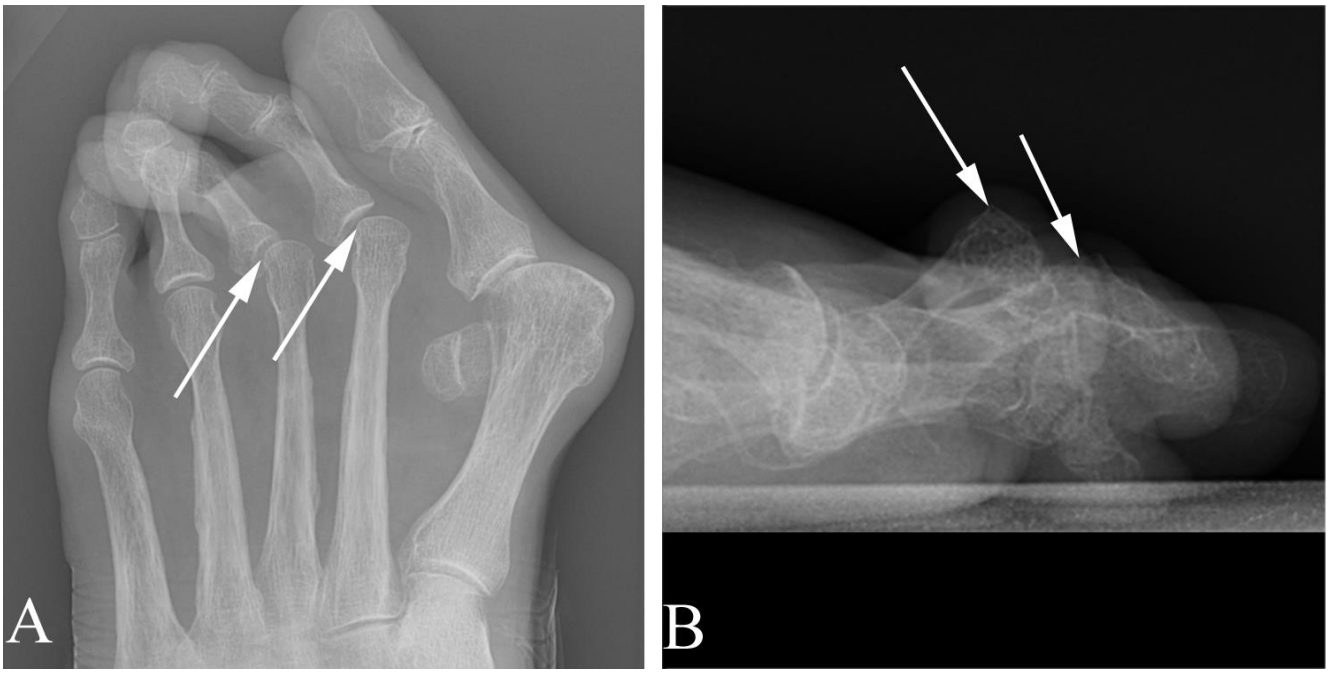

Figure 73. Both hallux valgus and cock-up toe deformities deformity in an 80 year old woman with chronic foot pain. A. AP view of the foot shows hallux valgus deformity of the great toe metatarsal-phalangeal joint as well as malalignment of the second and third toe MTP joints (arrows). B. Lateral examination shows extension of the metacarpal-phalangeal joints and flexion of the PIP joints of the second and third toes (arrows) also known as cock-up toe.

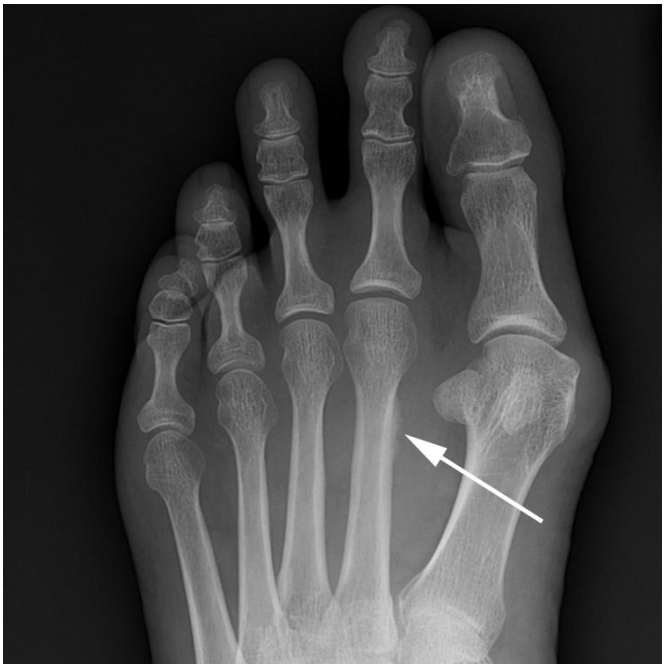

Figure 74. Stress fracture in a 56 year old with ongoing foot pain after taking a new job. AP plain film of the foot shows periostitis along the shaft of the second toe metatarsal (arrow), indicating a stress fracture.

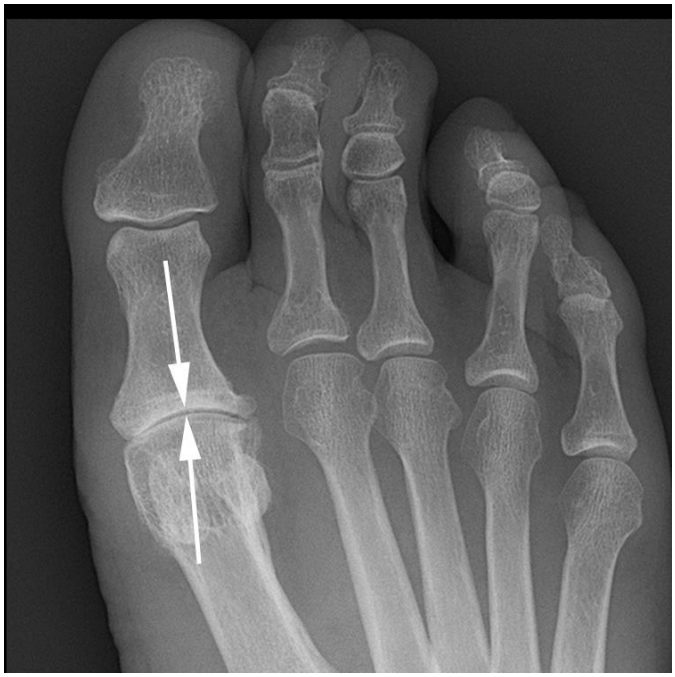

Figure 75. Great toe metatarsal-phalangeal degenerative change (hallux rigidis) in a 66 year old woman with chronic medial forefoot pain. AP plain film of the foot shows joint narrowing (arrows), subchondral sclerosis, and osteophytic spurring.

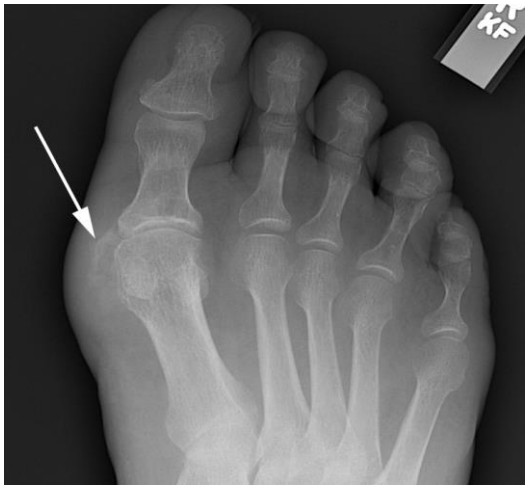

Figure 76. Gout with calcified soft tissue tophus in an 85 year old man with chronic medial forefoot pain. AP plain film of the foot shows calcified soft tissue (arrow) as well as swelling along the medial aspect of the great toe metatarsal-phalangeal joint.

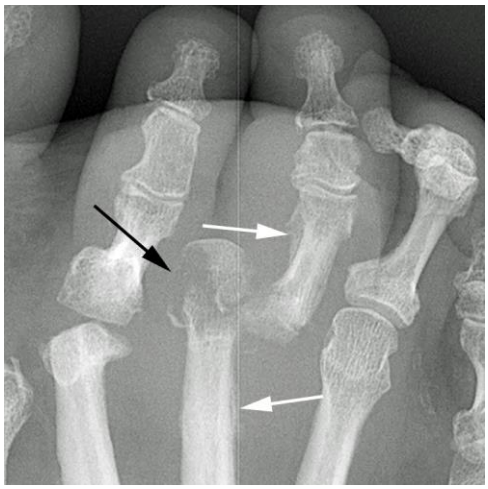

Figure 78. Osteomyelitis and septic arthritis seen on plain film examination in a 42 year old diabetic man. Detail from AP plain film exam of the foot shows periostitis along the third toe metatarsal and proximal phalanx (white arrows) and destruction of the metatarsal head (black arrow).

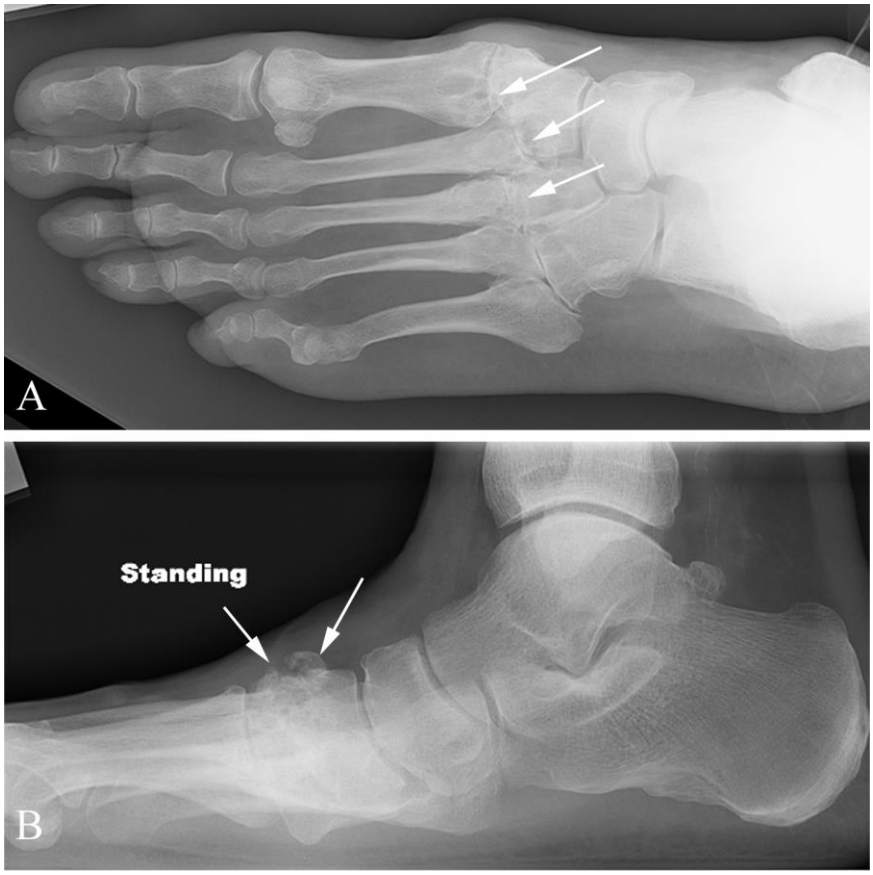

Figure 77. Charcot joint in a 68 year old with flat foot deformity and diabetes. A. AP view of the foot demonstrates joint narrowing and osteophytic spurring at the tarsal-metatarsal junction (arrows). B. Lateral examination shows pes planus and osteophytic spurring along the dorsum of the foot (arrow).

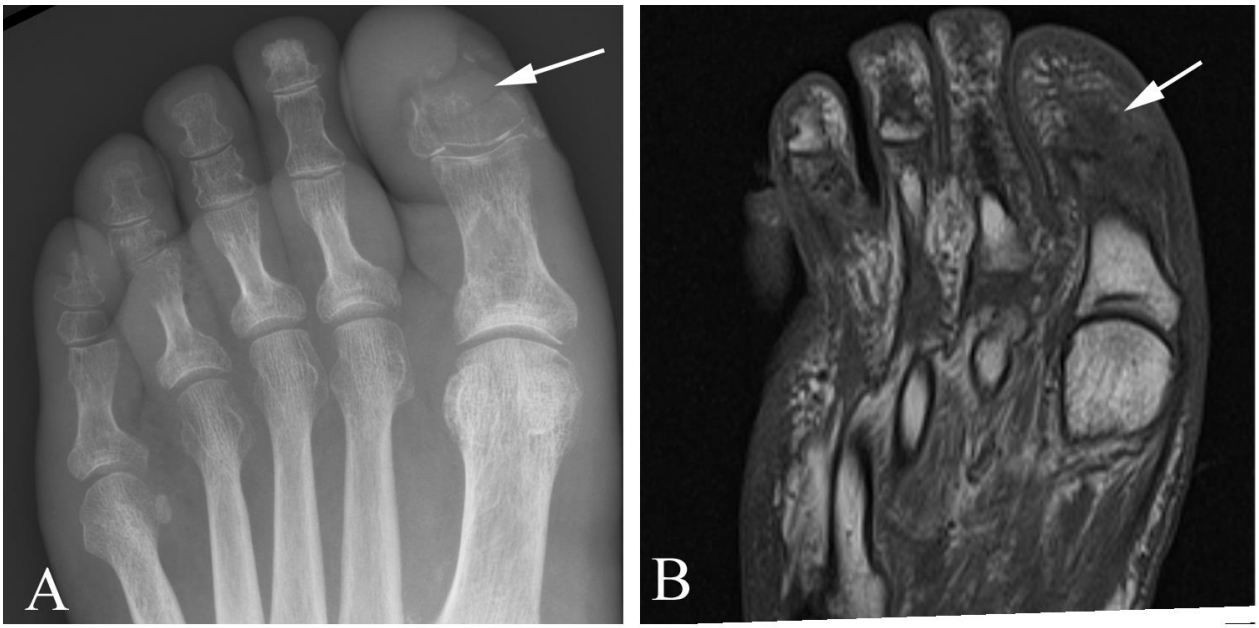

Figure 79. Osteomyelitis and septic arthritis in a 72 year old man with pain and a draining ulcer of the great toe. A. AP plain film examination of the foot demonstrates destruction of the distal phalanx of the great toe (arrow). B. Coronal T1 weighted MR image demonstrates abnormal signal throughout the distal phalanx (arrow) and through the distal aspect of the proximal phalanx, demonstrating the extent of the infection.

SUMMARY

This relatively long chapter has covered imaging of single joints. Acutely injured joints should generally undergo plain film evaluation if there is any suspicion of fracture. Further evaluation of acutely injured joints and evaluation of chronically painful joints depends on which joint is involved and to a certain extent on the disease process that the primary care provider feels is most likely to be causing the patient's symptoms, as noted in the above sections.

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Pediatrics

Donald L. Renfrew, MD

This chapter briefly reviews pediatric imaging. Because of the nature of the diseases of children who come to see a primary practice physician (or a general pediatrician), the number of radiology exams is typically far lower on a per-patient basis than in the adult population. Children tend to have different diseases than adults and this chapter deals with a few of the most common clinical scenarios when children are sent for imaging. This chapter is only a brief overview of five of the most frequently encountered and/or clinically important situations in which imaging may be important in pediatrics. For more in-depth discussion, and for additional topics, two excellent books are Lane F. Donnelly's *Fundamentals of Pediatric Radiology*[1] (less expensive and short enough to read in its entirety) and Hilton and Edward's *Practical Pediatric Radiology*[2] (more expensive and considerably longer)[3]. The main points of this chapter are:

1. A two-view chest x-ray (CXR) may help in the evaluation of a child with cough and fever or in a child with a fever and no localizing signs or symptoms.
2. A barium study or abdominal ultrasound may be performed in a neonate with persistent vomiting.
3. In a young child with bloody stools, intermittent abdominal pain, and a palpable abdominal mass, emergency surgical consultation should be sought. Plain films and US of the abdomen may be followed by an attempt at intussusception reduction.
4. CT examination of the abdomen and pelvis may be obtained for a child with RLQ pain, loss of appetite, nausea and vomiting, fever, and an elevated white count.
5. CT examination of the sinuses and orbits may be obtained for a child with a runny nose, fever, and headache, if accompanied by visual symptoms.

A TWO-VIEW CHEST MAY HELP IN THE EVALUATION OF A CHILD WITH COUGH AND FEVER OR IN A CHILD WITH FEVER AND NO LOCALIZING SIGNS OR SYMPTOMS

Chest radiography is widely used in the evaluation of pediatric patients with lower respiratory tract infections, although there is some controversy regarding the routine use of the chest x-ray and some studies have shown that it does not affect the clinical outcome of outpatient children with pneumonia[4]. The value of chest radiography is based on the ability to help distinguish between the various causes of pneumonia, to diagnose complications, and to exclude other causes of pneumonia symptoms.

With respect to the cause of pneumonia, while most pneumonia is viral, there are important age differences: in patients from 3 months to one year of age, 95% of pneumonia is viral, whereas for patients one to five years of age respiratory syncytial virus (RSV) accounts for approximately 50% of pneumonias. For school aged children (ages five to eighteen), the atypical pneumonias Mycoplasma pneumonia and Chlamydia pneumonia predominate[5]. Chest radiographs may be broadly categorized as showing one of three "patterns":

interstitial (with linear strands of density radiating from the hila, peribronchial cuffing, and "fat" hila) (Figure 1), alveolar or "air-space" (with dense white parenchyma, air bronchograms, a segmental or lobar distribution, with an accompanying pleural effusion) (Figure 2), and mixed (some combination of the two) (Figure 3). As a generalization, the interstitial pattern predominates in viral pneumonia, the alveolar pattern is seen with infection from typical bacteria (for example, Staphylococcus aureus and Streptococcus pneumonia), and the mixed pattern is seen with atypical organisms (Mycoplasma and Chlamydia). While true as a generalization, the chest x-ray does not do as good a job at this distinction as many radiology textbooks would have you believe, secondary to a number of factors including inter- and intraobserver variability of interpretation, overlap between the radiographic findings and causes, and the fact that radiographic findings may lag behind disease because of dehydration[6]. Therefore, the chest radiograph cannot be used in isolation to make the determination of whether a given patient's pneumonia is caused by a virus, typical bacteria, or atypical bacteria.

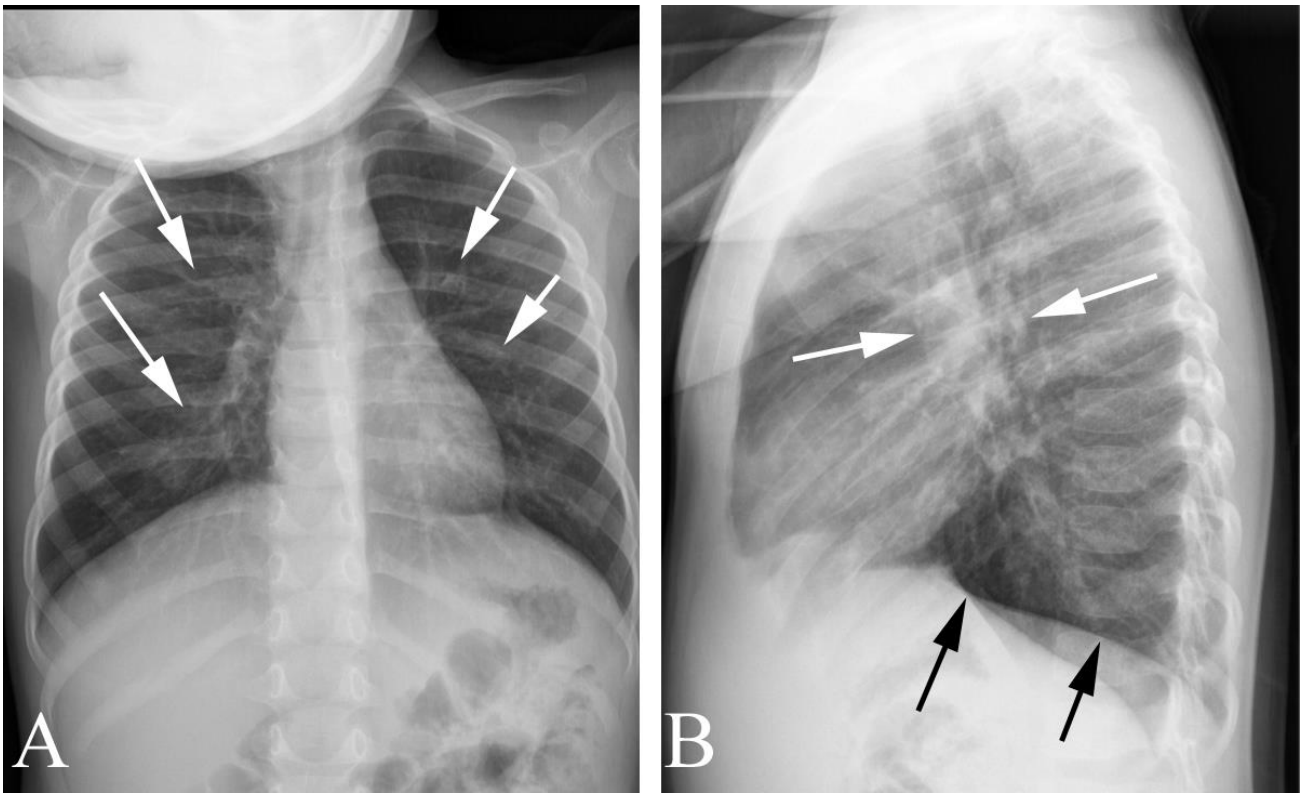

Figure 1. Interstitial pattern in a 2 year old with cough, shortness of breath, and likely viral pneumonia. A. Frontal chest radiograph shows streaky densities radiating from the hila (arrows). B. Lateral chest radiograph shows hyperinflation of the lungs with flattening of the diaphragm (black arrows) along with prominent hila (white arrows). The child's symptoms resolved without antibiotics.

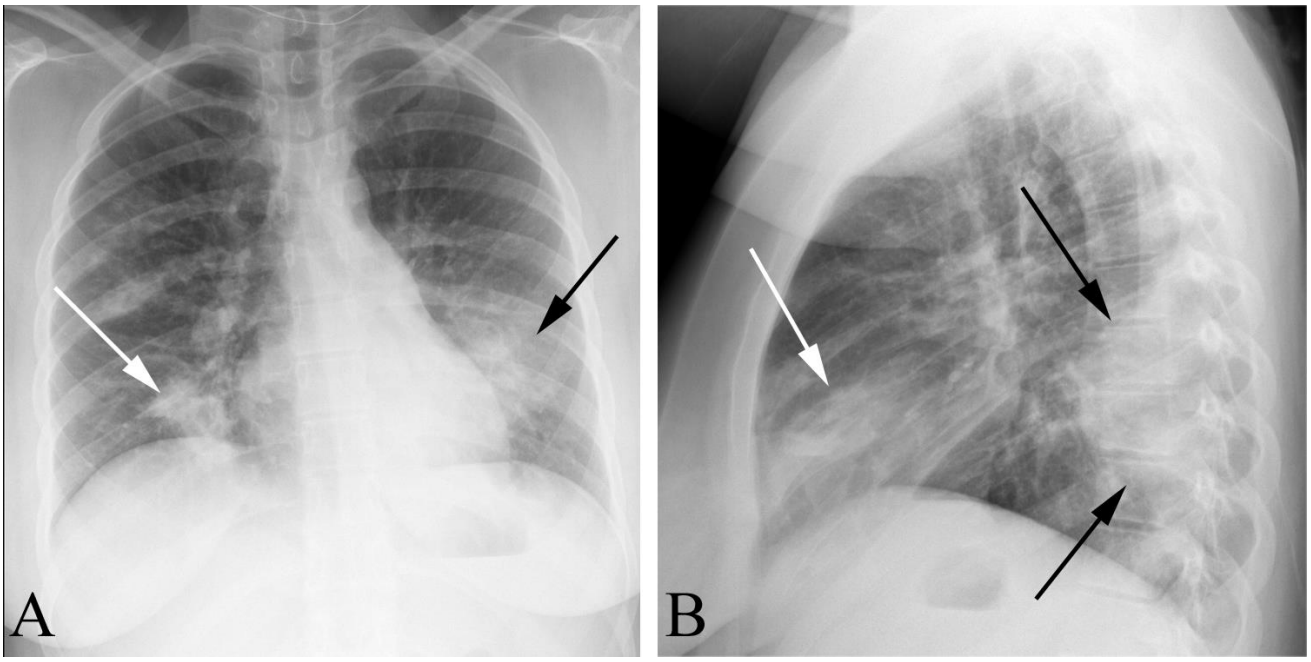

Figure 2. Alveolar pattern in a 15 year old with fever, cough, and bacterial pneumonia. A. PA chest radiograph shows focal abnormal densities in both the medial right base (white arrow) and along the heart border in the left base (black arrow). B. Lateral examination shows abnormal increased density overlying the heart (white arrow) and additional consolidation projecting over the mid thoracic spine (black arrows). Note that the vertebral bodies normally become more lucent from the apex to the base of the chest, whereas there is increased density of the mid-thoracic vertebrae in this patient.

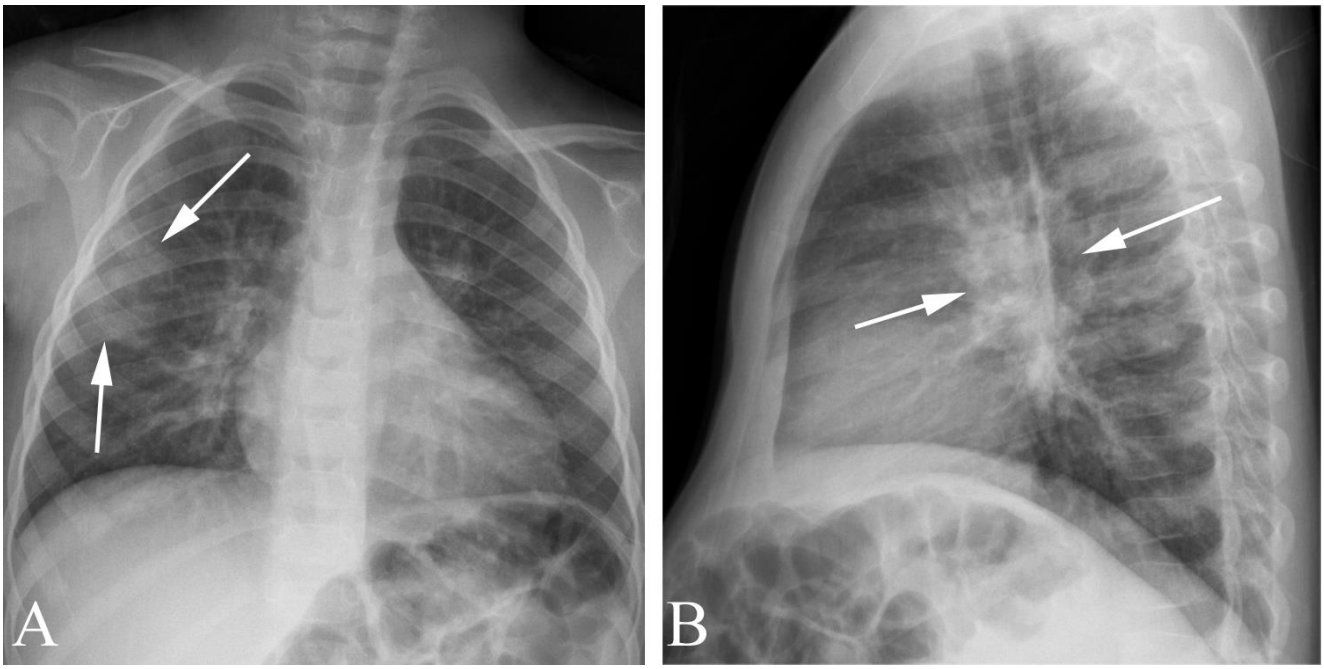

Figure 3. Mixed pattern in a 3 year old with fever, cough, and dyspnea. A. Frontal chest radiograph shows both prominent hila, streaky densities radiating from the hila, and abnormal opacity in the right mid-lung (arrows), only part of which may be accounted for by an overlying scapular shadow. B. Lateral exam confirms prominent hila (arrows). In such cases, it is difficult to tell if the vague pulmonary opacity represents atelectasis (which may accompany viral and/or atypical pneumonia) or a superimposed bacterial infection

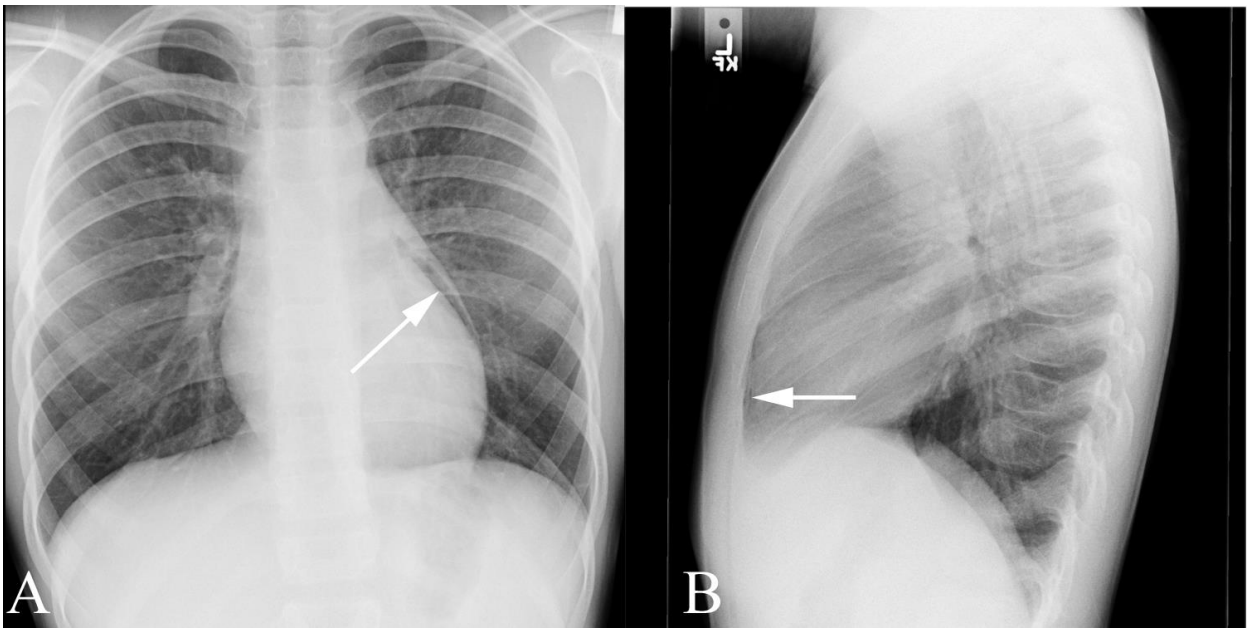

Figure 4. Pneumothorax in a 9 year old with asthma and cough. A. PA chest radiograph shows a lucency separating the normal thymus shadow from the heart border (arrow). B. Lateral chest radiograph shows a subtle lucency along the anterior chest wall (arrow) from pneumomediastinum.

The chest radiograph has more value in excluding such alternative explanations of cough and tachypnea as a retained foreign body (particularly when inspiration/expiration films are obtained) and congestive heart failure (for example, from viral myocardiopathy), and to diagnose complications of pneumonia such as development of a pneumothorax (from coughing) (Figure 4) pleural effusion, empyema, pneumatocele, necrotizing pneumonia, and lung abscess[5]. These performance features of the radiograph are particularly helpful in certain circumstances, such as when the child is severely ill and/or when the child is likely to be admitted to the hospital.

In those cases where the pediatric patient has a fever but no localizing signs or symptoms, a chest radiograph is helpful, since in one published report 26% of 146 children with fever and no clinical signs or symptoms of pneumonia (or other source of fever) with a white blood cell count of > 20,000/microL had radiographic evidence of pneumonia[7].

AN ABDOMINAL ULTRASOUND OR BARIUM STUDY MAY BE PERFORMED IN A NEONATE WITH PERSISTENT VOMITING

First, a few comments regarding vomiting in pediatric patients. As a generalization, it is best to involve the appropriate specialist (pediatric gastroenterologist, surgeon, or neurologist) earlier rather than later in cases where significant vomiting is occurring[8]. Features which merit particular concern include prolonged vomiting (greater than 12 hours in a neonate, greater than 24 hours in children under 2 years of age, and greater than 48 hours in older children); features of obstruction such as abdominal distension, visible bowel loops, marked change (increase or decrease) in bowel sounds, or bilious vomiting; features of a central nervous system cause such as vomiting without nausea particularly upon awakening or vomiting with a change in position; and features of adrenal crisis including hypotension out of proportion to the illness of the child or marked hyperkalemia[7].

In neonates with vomiting, the main considerations are normal spitting up, gastroesophageal reflux, idiopathic hypertrophic pyloric stenosis (IHPS), malrotation, and duodenal stenosis/atresia. The first two do not generally require imaging. If the child has most of the classic features for IHPS (presentation between 3 and 6 weeks of age, postprandial nonbilious projectile vomiting, desire to feed shortly following vomiting, and first-born male status) the best first step is probably ultrasound of the abdomen[9], performed in a fasting infant who is fed water or electrolyte-replacement solutions. The ultrasound study should demonstrate either a normal pylorus (Figure 5) or an abnormal pylorus (Figure 6) which may demonstrate abnormal wall thickness, length, or overall diameter or volume.

If the patient does not have the typical clinical features of IHPS, or if the ultrasound does not provide a satisfactory answer, then a UGI may be performed. This study should document prompt emptying of the stomach, presence or absence of gastroesophageal reflux, and (perhaps most importantly) the location of the ligament of Treitz (the transition from the duodenum to the jejunum). If the ligament of Treitz is not located in the left upper quadrant (to the left of the spine and superior to the duodenum), then malrotation (and associated risk for infarction of bowel), should be diagnosed.

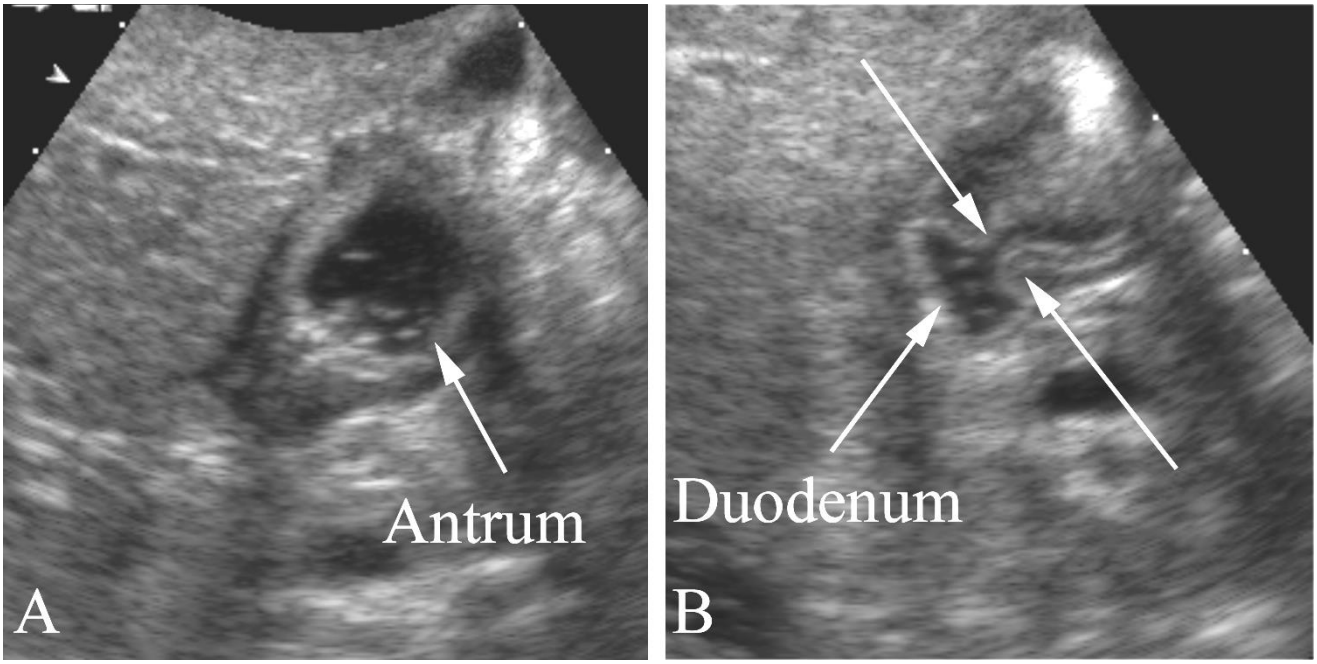

Figure 5. Normal pylorus in a 3 month old female infant with persistent projectile vomiting. A. Ultrasound examination through the mid-abdomen following oral administration of water/electrolyte solution shows the stomach antrum (arrow). B. Ultrasound examination obtained by constant observation of the antrum of the stomach following oral water demonstrates fluid in the distal antrum, pylorus, and duodenum. The arrows show the margins of the normal pylorus.

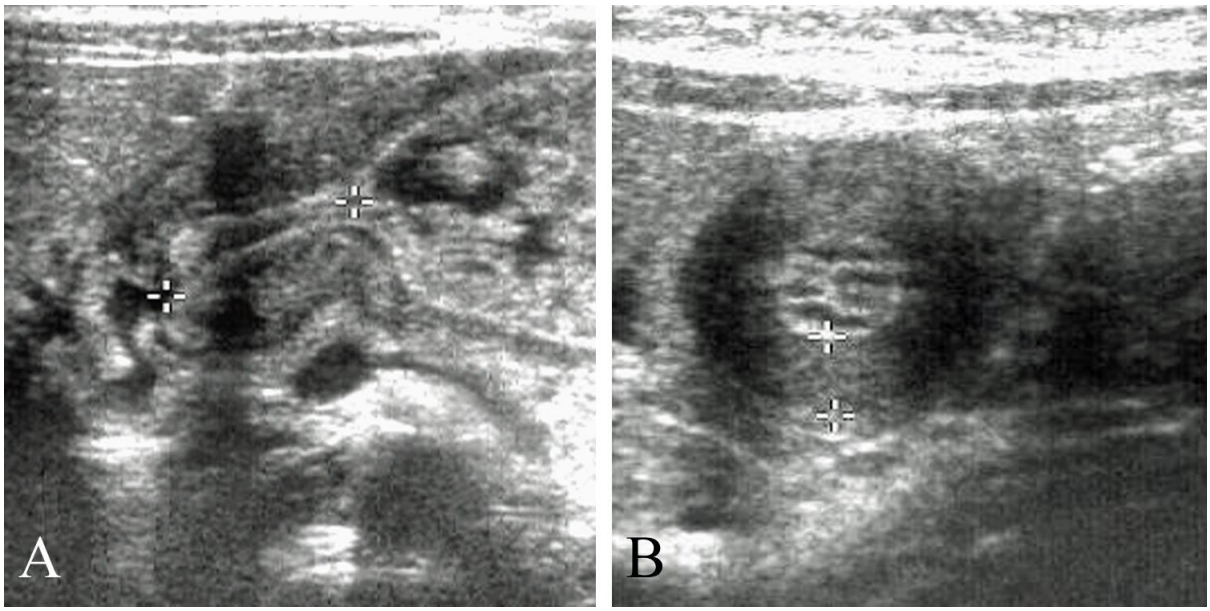

Figure 6. Idiopathic hypertrophic pyloric stenosis (IHPS) in a 2 month old child with vomiting and weight loss. A. Abdominal ultrasound with a longitudinal view of the pylorus (between calipers) shows an abnormally long and thick-walled pyloric channel. B. Abdominal ultrasound transverse view through the pylorus demonstrates an abnormally thick wall (calipers). Case courtesy of Dr. Jeffrey Zapolsky, Radiology Associates of the Fox Valley.

IN A YOUNG CHILD WITH BLOODY STOOLS, INTERMITTENT ABDOMINAL PAIN, AND A PALPABLE ABDOMINAL MASS, EMERGENCY SURGICAL CONSULTATION SHOULD BE SOUGHT PLAIN FILMS AND ULTRASOUND OF THE ABDOMEN MAY BE FOLLOWED BY AN ATTEMPT AT INTUSSUSCEPTION REDUCTION

Most (90%) of cases of intussusception will occur in the first two years of life, with the highest incidence between 5 and 9 months[10]. The classic presentation of pain, a palpable mass, and "currant jelly" stools[1] is seen less than 15% of the time[11]. Certainly, in cases where all of these features are present, and usually in cases where one or two of these features is evident in a child of the appropriate age, early surgical consultation is advised. Even if intussusception is diagnosed and treated in the radiology department, the child must be treated (prior to reduction) as if immediate surgery will be performed (with intravenous access and availability of a surgeon and operating room) because of the possibility of perforation during intussusception reduction[9].

In most cases, plain films and US (which may be ordered prior to surgical consultation in cases where there is a lower suspicion of intussusception) will be necessary for diagnosis. Plain films may document dilated small bowel loops from obstruction or even (usually indirectly) demonstrate a mass. Free air on the plain films is a contraindication to enema reduction, and typically patients with free air have a perforated viscus requiring operative intervention. Ultrasound usually shows a characteristic "bull's eye" or "coiled spring" appearance typical of intussusception. If there is no free air but there are ultrasound findings of intussusception, or if there is no free air but the clinical features are felt to be classic for intussusception, then enema reduction of the intussusception may be attempted. The method of reduction varies with local expertise and experience and may use either ultrasound guidance (with rectally administered air or saline) or fluoroscopic guidance (with rectally administered barium or water-soluble contrast material). As is

[1] This term applies to a bloody, mucus containing bowel movement which resembles currant jelly.

usually the case when many alternatives exist and one has not supplanted the others, there are advantages and disadvantages to each alternative, and there is likely a good reason (or several good reasons) that a particular radiology department chooses to treat intussusception in the manner that it does.

CT EXAMINATION OF THE ABDOMEN AND PELVIS MAY BE OBTAINED FOR A CHILD WITH RLQ PAIN, LOSS OF APPETITE, NAUSEA AND VOMITING, FEVER, AND/OR ELEVATED WHITE COUNT

For a discussion of appendicitis in adults, see pages 94-95 in Chapter 7. Many of the pediatric patients will be older than 5 years of age, and the diagnosis is often straightforward if all or most of the features of typical appendicitis are present, such as loss of appetite followed by nausea and vomiting, signs and symptoms of inflammation (peritoneal signs with an elevated white blood cell count), and pain localizing to the right lower quadrant. Imaging issues to consider in pediatric patients with possible appendicitis include a) whether to image; and b) if imaging is necessary, what modality to use. Some sources recommend early consultation with surgery[12], in which case the surgeons will make the decision of whether the clinical scenario is typical enough to proceed directly to surgery or if imaging needs to be performed.

In those cases where imaging is required, choices include ultrasound and CT. Ultrasound is recommended in those cases where local expertise is available in performance and interpretation of the exam. Ultrasound of the appendix is a demanding task, however, requiring considerable skill and extensive experience on the part of the ultrasound technologist and/or radiologist, and may not be available in all departments. As noted in Chapter 7, the exam is prone to false-negatives when performed by non-experts, and in this case a negative study cannot be relied upon to confidently exclude appendicitis. In these cases, CT needs to be performed anyway, and the US will only add time and money to the diagnostic evaluation. Furthermore, even in expert hands, if the ultrasound study is negative, or if the patient exhibits persistent features of appendicitis, repeat ultrasound or CT needs to be performed to confirm the apparent lack of appendicitis suggested by the initial ultrasound[13]. Ultrasound has a definite advantage in evaluation of female patients, however, in that it is the study of choice to evaluate gynecologic diseases (e.g., ovarian torsion, hemorrhagic ovarian cyst, pelvic inflammatory disease) which may mimic appendicitis.

When CT is chosen over ultrasound as the imaging method of choice, someone must decide how the examination should be performed. In all cases involving ionizing radiation, but particularly in pediatric cases involving ionizing radiation using CT, radiation dose becomes a factor in deciding how to perform imaging (see page 255). The technique should be optimized to use the lowest possible diagnostic dose of ionizing radiation (typically achieved by reduction in tube milliamperage and increased pitch with helical scanners). Of course, the more "passes" one takes through the abdomen and pelvis, and the larger area covered by those "passes" (covering both the abdomen and pelvis, versus covering only the pelvis), the more ionizing radiation is used for the study. Alternatives for scanning include:

1. Scan without any contrast material ("CT-KUB"). This alternative may be chosen when a ureteral calculus is a strong consideration (in addition to appendicitis). Advantages include an immediate scan without any oral, rectal, or IV contrast; disadvantages include possible lack of a fully diagnostic scan, sometimes necessitating a repeat scan (with more radiation exposure) after contrast has been given.

2. Scan with IV contrast only. The advantages of this alternative include rapidity and avoiding oral contrast. Disadvantages include that IV contrast may obscure small renal or ureteral stones (see Chapter 1) and that the distal small bowel is usually not well seen. With obstructing renal calculi, secondary signs (e.g., hydronephrosis and perinephric stranding) will usually indicate the diagnosis even if the stone itself is obscured.

3. Scan with oral and IV contrast. The disadvantages of this technique include that it takes longer (typically at least one hour, and often two, must pass before the terminal ileum and proximal colon contain enough contrast material for a diagnostic scan). Advantages include that it allows better evaluation of distal small bowel which may indicate an alternative diagnosis (gastroenteritis, Crohn's disease) to appendicitis as the cause of the patient's symptoms.

4. Scan with rectal contrast (with or without IV contrast). Advantages include high accuracy in diagnosis of appendicitis. Disadvantages to this alternative include lack of patient acceptance because of the necessity of performing a (water-soluble contrast) enema. In addition, while the large bowel (and frequently the appendix) is well seen and the technique is highly accurate in characterization of the appendix, contrast material may not freely reflux into the small bowel to evaluate for the alternative diagnoses mentioned in the previous paragraph.

As this brief review of scanning alternatives indicates, there are multiple considerations in play. Is it best to minimize radiation and maximize diagnostic yield, particularly when alternatives to appendicitis are under consideration? A single pass two hours after oral contrast administration with IV contrast will probably work best. Is it necessary to obtain an immediate diagnosis and is a ureteral calculus a strong consideration in addition to appendicitis? Immediate noncontrast scanning is the study of choice. Of course, such an immediate noncontrast study may be nondiagnostic and it may then require additional radiation, time, and expense to perform a study with oral or IV contrast. These considerations show why there is considerable variation in how radiology departments scan the patient suspected of appendicitis.

For patients with appendicitis, the imaging findings in pediatric patients are the same as those in adults: appendiceal swelling, a thick wall of the appendix which demonstrates abnormal contrast enhancement, and inflammatory changes in the surrounding periappendiceal fat (Figure 7). In patients where the process has progressed to the point where the appendix has ruptured, there is a disorganized appearance of the right lower quadrant because of leaked fluid and secondary inflammatory change (Figure 8).

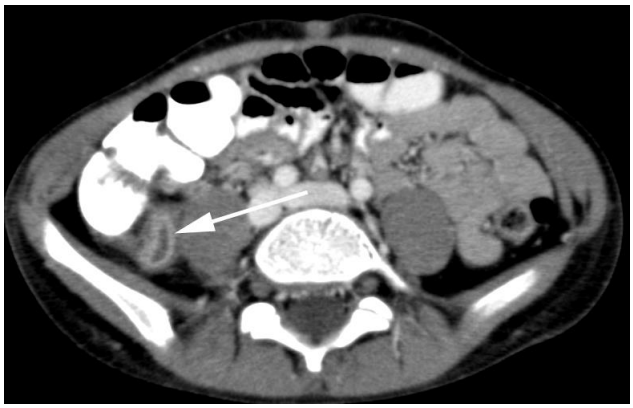

Figure 7. Appendicitis in a 6 year old boy with abdominal pain, nausea, and vomiting. Axial CT performed with oral and intravenous contrast material demonstrates a swollen, thick-walled appendix with marked contrast enhancement along the margins (arrow).

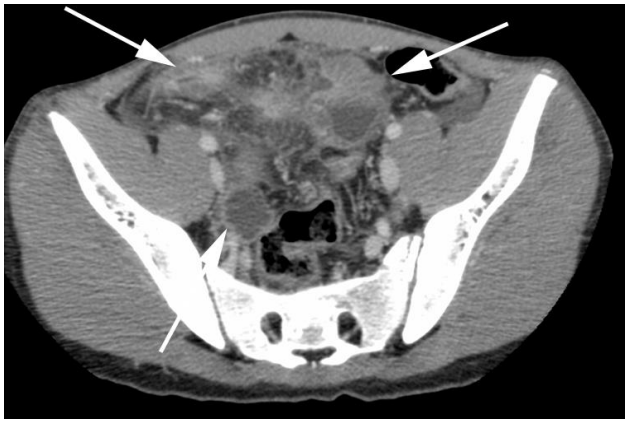

Figure 8. Ruptured appendix in a 16 year old male with abdominal pain for several days. Axial CT-KUB (CT performed without oral or intravenous contrast material) through the pelvis shows a disorganized appearance of the anatomy (between white arrows) in the right lower quadrant, with areas of inflammatory change within the peritoneum and focal areas of fluid representing abscesses/phlegmon, and no well-defined normal appearing bowel loops.

CT EXAMINATION OF THE SINUSES AND ORBITS MAY BE OBTAINED FOR A CHILD WITH A RUNNY NOSE, FEVER, AND HEADACHE IF ACCOMPANIED BY VISUAL SYMPTOMS

Sinusitis in pediatric patients generally manifests as a persistent runny nose, sinus congestion, sinus pain, and fever. Viruses cause most episodes of sinusitis, but approximately 10% of such viral infections are complicated by acute bacterial sinusitis[14]. When this occurs, the patient usually has either exacerbation of symptoms which were on the mend, or prolonged symptoms of between 10 and 30 days' duration. Since imaging (whether with plain films or CT) cannot distinguish between viral and bacterial sinusitis (both produce mucus membrane thickening and air-fluid levels), the American Academy of Pediatrics[15] and the American College of Radiology[16] do *not* recommend imaging.

There are, however, certain dreaded complications of sinusitis that require imaging. These complications involve spread of the infection from the sinuses to the orbits (orbital cellulitis) or cranial vault (septic cavernous thrombosis, meningitis, osteomyelitis of the frontal bone, epidural abscess, subdural empyema, and brain abscess)[13]. Such complications should be suspected in patients who develop orbital cellulitis, pain with eye movement, limitation of eye movement, double vision, vision loss, ptosis, proptosis, headaches, altered mental status, neurologic signs, or neck stiffness. In this case, a CT of the brain (including through the base of the maxillary sinus), performed using thin cuts (to allow multiplanar reconstruction) and contrast (to evaluate contrast-enhancing areas of inflammation) should be performed. MR of the brain (without and with contrast) is an alternative to CT in such cases. These cross-sectional exams will allow the distinction between, for example, preseptal (periorbital) cellulitis and orbital cellulitis. Such distinctions are critical to facilitate rapid treatment in patients with severe complications of sinusitis.

SUMMARY

This chapter briefly reviews a few of the more commonly encountered, or clinically important, scenarios in pediatric patients. For children with cough or fever, or with a fever and no localizing signs, a CXR may be helpful. In infants with persistent vomiting, abdominal US should probably be performed first if pyloric stenosis is suspected, with an oral contrast study if additional imaging is required. In a young child suspected to have intussusception, plain films and US may be followed at an attempt at reduction of the intussusception in the radiology department. For children with equivocal features of appendicitis, either US or CT may be used, although there are several variations in how to perform CT in these circumstances. In a patient with visual symptoms in addition to features of sinusitis, CT may be performed to exclude orbital extension of inflammation.

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Contrast Material and Radiation Exposure

Donald L. Renfrew, MD

This chapter reviews the damage done by radiology procedures secondary to injecting contrast material and exposing patients to radiation. The profession of radiology in general, and most radiology departments in particular, go to great lengths to limit this damage. Broadly speaking, the risk from contrast material and radiation exposure on a per procedure basis has decreased through the years, but as the number of procedures keeps increasing the global risk to all patients rises. In addition, for a given individual patient the risk may be significant. The four main points of this chapter are:

1. Contrast induced nephrotoxicity has likely been overestimated and, with current contrast materials, is quite low.
2. Idiopathic hypersensitivity reactions to contrast are rare and require rapid recognition and treatment.
3. Gadolinium-containing (MR) contrast should be avoided in patients with renal failure.
4. Radiation exposure has become a significant concern, particularly in young patients undergoing repeated CT scans.

CONTRAST INDUCED NEPHROTOXICITY HAS LIKELY BEEN OVERESTIMATED AND, WITH CURRENT CONTRAST MATERIALS IS QUITE LOW

To evaluate whether administration of contrast material has caused renal damage, it is necessary to have a measurement of renal function. While genuine creatinine clearance may be calculated by administering and then measuring excretion and serum values of inulin, iothalamate, iohexol, or DTPA[1], such measurements are not routinely used. Current clinical measurements, which are proxies of genuine renal function, have problems. Creatinine clearance requires the (generally impractical) collection of urine and represents the upper limit of the glomerular filtration rate (GFR) rather than GFR itself[1]. Serum creatinine, the most widely used measure of "function", varies between laboratories (by as much as .3 mg/dL) [1] and may increase after eating a large amount of protein (by as much as .2 mg/dL)[2]. It also varies with weight, age, and sex. In addition, small changes of serum creatinine (particularly when the creatinine is low, for example within the normal range) imply large amounts of damage[1]. Furthermore, changes in creatinine may lag behind renal damage by several hours or even days[1]. Thus, the significance of a serum creatinine value of 1.3 in a young, slender woman who had a creatinine of 1.1 yesterday is completely different than a creatinine of 1.3 in an elderly obese man who has had the same value for three years.

One method of compensating for some of the shortcomings of serum creatinine as a measurement of renal function is to *estimate* the glomerular filtration rate (eGFR). This may be done by means of the widely used Cockcroft-Gault or MDRD (Modification of Diet in Renal Disease Study) equations, which take into account age, gender, and body size. However, these equations are only valid if the serum creatinine is stable. Furthermore, in evaluation of acute renal disease, since age, gender, and body size will not change acutely, eGFR will simply reflect changes of serum creatinine anyway[2]. While serum biomarkers (e.g., cystatin C) show promise, none are in current clinical use[3].

Despite the difficulties of measurement of renal function, radiologists still must take renal function into account when administering contrast material. Radiology contrast material has undergone tremendous evolution in the past 50 years, as drug companies competed (and continue to compete) to bring less toxic products to the marketplace (see Table). The ionic, high osmolar contrast materials (HOCM) of yesteryear have largely been supplanted (in all but niche uses), at least in the United States, by nonionic, low osmolar contrast materials (LOCM). The latest product is an iso-osmolar contrast (IOCM). Claims about the relative nephrotoxicity of these different substances (many times driven by marketing) are sometimes conflicting and confusing.

Adding to the confusion is a recent set of articles which indicate that the risk of contrast induced nephrotoxicity may be overstated. Rao and Newhouse[4] reviewed over 3,000 articles regarding contrast induced nephrotoxicity and found only 40 dealing with intravenous injection of contrast material; only two of these contained a control group (not injected with contrast material), and these two studies found no difference (with respect to acute kidney injury) in the injected and non-injected patients. In a subsequent publication, Newhouse et al[5] reviewed the records of over 32,000 hospitalized patients who did not have contrast material but who did have sequential serum creatinine measurements taken, and found substantial variation (both increases and decreases) of serum creatinine measurements through time. The extent of these changes would have led to many of these patients being classified as suffering from "contrast induced nephrotoxicity", (as defined in published literature) had they been given contrast. A subsequent publication by Bruce et al[6] compared three groups: a control group, patients receiving LOCM, and patients receiving IOCM. At least with serum creatinine concentrations below 1.8 mg/dL the incidence of acute kidney injury was similar between the three groups. With higher serum creatinine, there was a difference between the particular LOCM used in this study (Iohexol) and the control group (with a small but statistically significant increase in serum creatinine in the LOCM group), but not between the IOCM (Iodixanol) and the control group.

So is the key to CIN the use of LOCM versus IOCM, with IOCM as safe as saline? Perhaps not: two large, randomized, multi-continent studies known as the IMPACT[7] and the PREDICT[8] study found no difference between another LOCM (Iopamidol, rather than Iohexol) and an IOCM (Iodixanol) in the rates of CIN in patients with elevated creatinine (IMPACT) and elevated serum creatinine and diabetes (an independent risk factor for CIN) (PREDICT). A meta-analysis by Heinrich et al[9] stated "Iodixanol [IOCM] is not associated with a significantly reduced risk of CIN compared with the LOCM together".

Ellis and Cohan offer an excellent summary of these issues in a "Perspective" article published in the AJR[2]. They note that given the frankly conflicting published results, opinion varies not only regarding the likelihood of CIN but also regarding prevention and treatment of CIN; indeed the recommendations offered by Ellis and Cohan are different than those offered by Rudnick and Tumlin in UpToDate[10].

Recognizing that this is an evolving topic, it should be noted that there is little controversy regarding the administration of LOCM in patients with normal renal function: it is assumed that these patients are at very low risk for CIN and no preventive measures need to be taken. In patients with elevated serum creatinine, radiology departments vary. Most will routinely evaluate serum creatinine, particularly above a cut-off patient age, to identify those who may be at risk for CIN. For those with an elevated creatinine (somewhat arbitrary), one alternative is to offer hydration (IV is

better than oral) with either normal saline or bicarbonate prior to and following the procedure. The (relatively benign, cheap, over-the-counter) antioxidant/mucolytic agent N-acetylcystine may also be administered prior to and following the procedure. The necessity and efficacy of these interventions remains controversial.

How does this knowledge help the primary care provider? You may want to check out how your radiology provider(s) deals with this situation, and confirm that they routinely use either LOCM or IOCM. In addition, you may inquire as to whether they routinely screen for renal insufficiency by checking serum creatinine, and whether they routinely pre-treat patients with an elevated creatinine with either hydration and/or N-acetylcystine. This knowledge may also help you when a radiologist (or radiology technologist) calls to ask you what to do because your patient, scheduled to undergo a radiology test which is usually performed with intravenous contrast, has an elevated creatinine. In patients who you know have elevated creatinine, you may wish to review with the radiologist the necessity of injecting contrast and review other diagnostic possibilities (noncontrast CT, ultrasound, noncontrast MR[1], and nuclear medicine options) to obtain the necessary diagnostic information. Finally, in patients who develop what appears to be CIN following contrast injection, it is reasonable to search the patient's history for possible alternative causes of acute kidney injury (either prerenal or acute tubular necrosis (ATN)) given the recent information about the relatively low likelihood of CIN. In those patients who have genuine CIN, it is usually a transient and short lived event: return to normal renal function is generally faster than with other causes of ATN.

[1] This list does *not* include contrast-enhanced MR, because the downside of injecting gadolinium based MR contrast (nephrogenic systemic fibrosis) is *far worse* than CIN.

Generic Name	Brand Name
Ionic, monomeric, hyperosmolal (>1400 mosmol/kg)	
Sodium iothalamate	Conray
Sodium diatrizoate	Isopaque
Ionic, dimeric, low-osmolal (600 mosmol/kg)	
Ioxaglate	Hexabrix
Non-ionic, monomeric, low osmolal (500 – 850 mosmol/kg)	
Iohexol	Omnipaque
Iopamidol	Niopan; Isovue
Ioversol	Optiray
Iopromide	Ultravist
Ioxilan	Oxilan
Non-ionic, dimeric, iso-osmolal (290 mosmol/kg)	
Iodixanol	Visipaque

Table. Contrast agents. The agents listed at the top of the table are older, higher osmolal and no longer routinely used in most radiology departments, having been replaced by low osmolal or iso-osmolal agents listed at the bottom of the table.

IDIOPATHIC HYPERSENSITIVITY REACTIONS TO CONTRAST MATERIAL ARE RARE AND REQUIRE RAPID RECOGNITION AND TREATMENT

Reactions to contrast material may be categorized as idiosyncratic hypersensitivity reactions (IHRs) and chemotoxic reactions[11]. Chemotoxic reactions include vasovagal responses, seizures, arrhythmias, and CIN (see above). These will not be discussed further.

IHRs manifest as pruritis, urticaria, angioedema, laryngospasm, bronchospasm, and hypotension and vary from mild (less than 10% with LOCM) to fatal (somewhere between 1:10,000 and 1:100,000). These reactions should be recognized immediately by whoever injects the patient with the contrast material. Treatment for contrast reactions should be done in the radiology department at the time of the reaction by personnel from the radiology

department, the emergency department, or the code team. Primary care practitioners will rarely participate in these events. Treatment is as for other hypersensitivity reactions, with reassurance and antihistamines for mild reactions and more drastic measures (including, but not limited to, IV fluids, oxygen, epinephrine, IV steroids, and pressors) for severe reactions. While the primary care provider may never witness or treat an IHR, the issue may arise for the primary care provider in two scenarios: patients with prior contrast reactions, and patients with asthma/atopy. In patients who have a "history of allergy to contrast material" it is important to first determine the exact nature of the "allergy". If the patient has had an obvious anaphylactic reaction requiring intubation, treatment with epinephrine, cardioversion, or hospitalization, it is best to completely avoid ever again using the specific instigating contrast. Whether an alternative contrast material should be given requires careful consideration and definite precautionary pretreatment, generally with oral steroids and antihistamines divided into several doses the day prior to and the day of the exam. If the patient has had a flushed sensation or nausea and vomiting in response to contrast injection, these are more likely a chemotoxic reaction rather than an idiopathic hypersensitivity reaction and do not place the patient at any additional risk for an IHR. Of course, this leaves a large group of patients that have had minor IHRs in the past with some sneezing or a few hives and itching. The approach to these patients varies from never administering contrast, to completely ignoring the event, and everything in between (including switching contrast and/or pretreatment with steroids and antihistamines), and there is no true consensus as to which is the correct approach.

The other scenario, patients with asthma/atopy, is less confusing: while these patients do bear some additional risk of an IHR (perhaps up to five times for asthmatics), no pre-treatment is generally recommended[11]. Such patients should be given LOCM or IOCM, but this is the nearly universal routine practice now anyway. Incidentally, despite the widespread misconception, a previous reaction to fish or shellfish is *not* an additional or independent risk factor for IHR, fatal or otherwise, from contrast injection, beyond the mild increased risk for anyone with allergies or asthma in general[12].

GADOLINIUM-CONTAINING CONTRAST SHOULD BE AVOIDED IN PATIENTS WITH RENAL FAILURE

What a difference a decade makes! A bit more than ten years ago, intravenous MR contrast compounds containing gadolinium were thought to be almost completely benign when administered in routine doses, and, indeed, were looked upon as a godsend for evaluation of the arterial tree and kidneys in patients with renal failure when there was a need to avoid iodinated contrast because of concerns about CIN (see above). We have progressed from the first case reports regarding nephrogenic systemic fibrosis (NSF) to the point where tort attorneys troll for cases on television in less than fifteen years.

What is NSF? It is a nasty disease consisting of thickening and hardening of the skin with expansion and fibrosis of the dermis that occurs *only* in patients with renal failure[13]. Almost all of these patients have been injected with one of the gadolinium containing MR contrast agents. Conversely, between 2.5% and 5.0% of those patients with renal failure (dialysis patients) will develop NSF. Since there is no way of predicting whether a given renal failure patient will or will not develop NSF (even though 2.5 – 5.0% do), and given the dreadful nature of the disease, there are very few circumstances where it makes sense to use gadolinium based contrast material in these patients. In those cases where it is deemed absolutely necessary to do so, use of gadoteridol (one of the many formulations of gadolinium possible) in the lowest dose, followed immediately by dialysis, is recommended[13].

For patients with an abnormal eGFR who are not in renal failure, there is no consensus regarding what to do, although the risk of NSF seems to be orders of magnitude lower for patients with an eGFR of greater than 30 mL/min compared to those in renal failure. There is probably little if any risk in patients with an eGFR of greater than 60 mL/min.

RADIATION EXPOSURE HAS BECOME A SIGNIFICANT CONCERN FOR CT STUDIES

The average amount of radiation exposure to the United States' population doubled between the early 1980s and 2006, due largely to increased use of radiology services, particularly the increase of CT scans from around 3 million in the early 1980s to 67 million in 2006[14]. Because of the ionizing effect of radiation, exposure to radiation increases the risk of cancer in the exposed patient, and some of these cancers may be lethal. Diagnostic radiology studies (unlike therapeutic radiation) almost never use enough radiation to cause direct effects such as hair loss or skin damage. One way of expressing the risk of radiology procedures is as the increased chance of a death caused by a cancer that has resulted from the radiation exposure. Note that the chance of death from cancer for a given patient with *no* exposure to radiation is, on average, 42%, or 420 per 1,000, and that radiation exposure adds to this risk[15]. Note also that there is no way to tell if a given cancer has developed secondary to administered radiation or some other cause.

So how is radiation measured, and how much does a patient receive during radiology procedures? There are a number of terms that may be used when measuring radiation exposure, but for our purposes we will use milisieverts (mSv). For the energy of radiation used in diagnostic imaging, 1 rad (radiation absorbed dose) = 10 miligray (another radiation absorbed dose) = 1 rem (roentgen-equivalent man) = 10 mSv[16]. Radiation doses vary with the type of procedure and the type of scanner[17]. Ultrasound and MR, of course, do not use ionizing radiation and the risk of death from cancer from use of these modalities is nil. As examples of radiation doses, chest radiographs and extremity radiographs generally result in *minimal* exposure (<0.1 mSv), pelvis radiographs and mammography in *low* exposures (0.1 to 1.0 mSv), single-pass abdominal CT and bone scans in *medium* exposure (1.0 to 10.0 mSv), and multiple-pass CT and whole-body PET in *high* exposure (>10 mSv). To relate the degree of radiation exposure to the likelihood of death from cancer caused by that radiation, it is necessary to extrapolate from data on atomic bomb survivors. In general, the known, *proven* risk increases linearly from doses of about 100 mSv upward, and risks below this level are based on a "linear, no-threshold hypothesis" that lower doses result in less additional cancer risk all the way down to zero radiation dose resulting in zero additional risk[18]. Compared to an adult of age 40, a child is 3-4 times *more* likely to develop a lethal cancer and an 80 year old 3-4 times *less* likely to develop a lethal cancer[15]. *As a generalization* (appropriate to within an order of magnitude): the *additional* risk (added to the background rate 420 cases per 1,000) of developing a lethal cancer from radiation exposure is approximately 1 per 1,000 per 10 mSv. Note that some experts[19] discourage discussing specific numbers with patients, preferring to use the terms "negligible" (<0.1 mSv), "minimal or extremely low" (0.1 – 1.0), "very low" (10-100 mSv), "low" (10 – 100 mSv) and "moderate" (> 100 mSv) to express the *risk* (not the radiation amount) when discussing the topic. For most radiology procedures (with the exception of multi-pass CT), the additional risk of developing a lethal cancer is less than 1 in 1,000. CT manufacturers are presently working diligently to reduce radiation doses, particularly in pediatric populations.

For pregnant patients, note that the risk of developing a lethal cancer within the fetus is less than that for a small child[20], and that there is no evidence of risk for fetal anomalies, intellectual disability, growth restriction, or pregnancy loss for doses less than 50 mSv. For higher doses, during the first 14 days of pregnancy there is an "all-or-none" phenomenon in which the fetus will die or survive without adverse sequelae[21]. During organogenesis (4 to 10 weeks after the last menstrual period), intrauterine growth restriction and congenital malformations may be seen (again, with doses of greater than 50 mSv). Note that fetal exposure (as opposed to maternal exposure) may be negligible in examination of maternal body areas other than the abdomen and pelvis. In general, ionizing radiation is to be avoided during pregnancy with alternative methods of diagnosis (e.g., US) preferred.

An excellent short review on this topic for both primary care practitioners and patients may be found at www.radiologyinfo.org under the tab "safety."

SUMMARY

Contrast induced nephrotoxicity has likely been overestimated and, with the current generation of contrast materials, is quite low. Idiopathic hypersensitivity reactions to contrast are rare but require rapid recognition and treatment. Gadolinium-containing (MR) contrast should be avoided in patients with renal failure. Radiation exposure, particularly with repeated CT scans performed in children, has become a significant concern.

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Imaging of the Pregnant Patient with Symptoms

Donald L. Renfrew, MD

Workup of pregnant patients presenting with symptoms depends on whether the symptoms represent a *complication of pregnancy* or represent disease *complicating the pregnancy*. See Table 1.

COMPLICATIONS OF PREGNANCY

Complications of pregnancy include miscarriage, ectopic pregnancy, molar pregnancy, placental abruption, intra-amniotic infection, uterine rupture, and severe pre-eclampsia with or without the HELLP syndrome[1]. See Table 2. Imaging for complications of pregnancy (other than pre-eclampsia and the HELLP syndrome) typically consists of pelvic ultrasound (Table 1), and additional imaging studies are seldom necessary.

For women with a positive pregnancy test (with or without symptoms), transvaginal pelvic ultrasound results will generally fall into one of four categories (see Rodgers et al):

1. Viable intrauterine pregnancy (IUP) at X age. The ultrasound exam shows a live intrauterine fetus (or fetuses) with age established by size on the ultrasound.
2. IUP at X age of unknown viability.
    a. *Probably normal (but too early to demonstrate fetal cardiac activity)*. The ultrasound exam shows an empty gestational sac without yolk sac or embryo, a gestational sac with a yolk sac but no embryo, or a gestational sac with a yolk sac and an embryo less than or equal to 4 mm in size with no cardiac activity.
    b. *Probably abnormal*. The ultrasound examination shows findings suspicious for, but not diagnostic of, pregnancy failure (e.g. no cardiac activity in a fetus measuring 5 mm, no fetal pole in a gestational sac measuring 20 mm).
3. Pregnancy of unknown location.
    a. With a normal appearance of the endometrium, possibilities include an early IUP, an occult ectopic pregnancy, and a completed spontaneous abortion.
    b. With an abnormal endometrium, the possibilities include a spontaneous abortion in progress (usually shows a thickened endometrium) or an early IUP (gestational sac vrs decidual cyst) or abnormal IUP.
4. Nonviable IUP at X age.
    a. There is a fetus with a crown-rump length of 7 mm or greater with no fetal cardiac activity.

[1] HELLP = **H**emolysis, **E**levated Liver **E**nzymes, and **L**ow **P**latelet Count

b. There is a gestational sac measuring 25 mm or greater with no fetus.
c. Sequential scans show no heartbeat 2 or more weeks after a scan with a gestational sac and no yolk sac, or 11 days after a scan with a gestational sac and a yolk sac.

In a young women with a positive pregnancy test and a live, intrauterine pregnancy, the likelihood of an additional, ectopic pregnancy is very small, except in patients who are taking fertility drugs or who have undergone in vitro fertilization. Patients with an ectopic pregnancy will fall into category 3 (above), with no IUP. The ultrasound study in women with ectopic pregnancy may show no additional abnormality, a hypervascular mass in the adnexa (most ectopic pregnancies implant in the fallopian tube), or (in rare cases) an extrauterine gestational sac and fetus.

Molar pregnancy may be accompanied by pelvic and abdominal pain, but the most characteristic feature is a disproportionate elevation of beta human chorionic growth hormone (beta HCG). Ultrasound typically shows a lack of a normal intra-uterine pregnancy with, instead, a vascular multicystic mass. Placental abruption is a difficult diagnosis, and ultrasound is relatively insensitive (see Glantz and Purnell). Ultrasound of the abdomen may be performed in pre-eclampsia and the HELLP syndrome to evaluate for free fluid in the abdominal cavity (a result of hemorrhage) or abnormalities of the liver (hemorrhage into the liver parenchyma or along the capsule).

DISEASE COMPLICATING PREGNANCY

Pregnant women may fall prey to any disease that nonpregnant women experience. Some of these diseases actually occur somewhat more frequently in pregnant women. Diseases complicating pregnancy (and the early post-partum period) include ovarian torsion, acute appendicitis, gallbladder disease, bowel obstruction, inflammatory bowel disease, pancreatitis, diverticulitis, cystitis, pyelonephritis, nephrolithiasis, pneumonia, gastroenteritis, and pulmonary embolism. These diseases generally produce symptoms as noted in Table 3, and for a discussion of the imaging evaluation of these symptoms in nonpregnant women, please see the appropriate chapters on abdominal pain, nausea and vomiting, chest pain, dyspnea, and flank pain. Imaging in pregnant women does *not* automatically progress as outlined in those chapters, however, secondary to concerns regarding maternal and fetal irradiation and contrast material injection. For additional discussion of these topics, please see "Chapter 16: Contrast materials and radiation exposure." Briefly, imaging of pregnant patients with symptoms of a disease complicating the pregnancy consists of ultrasound of the abdomen for patients with abdominal pain, US of the pelvis in patients with pelvic pain, and US of the lower extremities in patients with either leg swelling and tenderness along the course of the veins, or dyspnea and tachycardia suspicious for pulmonary embolism. Additional imaging is generally done only after evaluation by an obstetrician and in consultation with a radiologist. Use of ionizing radiation is avoided, if possible, because of the *stochastic* and *deterministic* effects of radiation. The stochastic effect presumably occurs even at very low doses of radiation, and results in an increased frequency of the development of malignancy following radiation exposure. This effect also occurs, of course, in nonpregnant patients, but in pregnant patients two issues increase the degree of concern over radiation exposure: 1) female breast tissue is much more sensitive to radiation during pregnancy; and 2) the ethical quandary of increasing the cancer risk of a fetus. While there are many factors that figure into the calculation

of the risk, as a generalization the lifetime risk of cancer increases approximately 0.1% per 10 mSv of exposure. This is roughly the dose of a CT study of the abdomen and pelvis using a circa 2000 era CT scanner (although the dose may be much lower with newer equipment and dose modification). Note that it is also roughly the dose *to the mother* for a chest CT, but that the infant dose in a chest CT is less than 1 mSv. Plain film exposure is generally much less than 1 mSv. This risk is generally felt to be reasonable in cases where a diagnosis is necessary (for example, with suspected appendicitis or pulmonary embolism) and no other options are available. There is a trend to use noncontrast MR in such situations as a substitute for CT in these circumstances.

Deterministic effects of radiation are linearly related to dose and have a definite threshold. Such effects occur only at much higher doses than produce stochastic effects[2]. Indeed, only extraordinary circumstances would lead to the degree of exposure (at least 50 mSv, and more likely greater than 100 mSv) necessary to produce deterministic effects on the fetus. These levels of radiation would only occur with multiple CT scans or procedures requiring prolonged fluoroscopy. Only severely ill patients would ever undergo these levels of medical radiation. If such a high level of exposure occurs during the first two weeks following conception, the fetus either survives intact or does not survive (an "all or none" effect). If the exposure occurs three to four weeks after conception, the likelihood of spontaneous abortion increases. If the exposure occurs in the 5^{th} to 10^{th} week following conception, the likelihood of possible fetal malformations increases; if the exposure between the 11^{th} and 27^{th} week, mental development is delayed or permanently damaged. All of these high levels of radiation also cause an increased risk of cancer with the general rule of approximately a 0.1% increase in risk per 10 mSv exposure.

In addition to avoiding the use of ionizing radiation, intravenous contrast is also best avoided in pregnant patients. Iodine containing contrast used for CT studies may suppress thyroid function in the fetus, and gadolinium containing contrast agents have shown teratogenic effects in animal studies. While both of these concerns are more theoretic than actual (see, for example, the paper by Bourjelly et al documenting a *lack* of fetal thyroid suppression following IV iodinated contrast), in most instances injection of contrast is avoided if possible. Certainly, any imaging work-up requiring ionizing radiation and/or intravenous contrast material which *can* be postponed until after delivery *should* be postponed until after delivery.

[2] Models of exposure vary, but most authorities assume that even tiny amounts of ionizing radiation are associated with an associated (tiny) increase in the likelihood of future development of cancer.

Table 1. Imaging of the Pregnant Patient with Symptoms

Indication	Imaging Examination
Pelvic pain with vaginal discharge and suspected complication of pregnancy	Ultrasound of the pelvis.
Flank pain +/- hematuria and suspected urinary calculus	Ultrasound of the kidneys and pelvis. MR urogram for problem cases. See "Flank pain" documents at www.symptombasedradiology.com for further details including a discussion of imaging in patients who are *not* pregnant.
Abdomen or pelvis pain with anorexia, nausea and vomiting, and suspected appendicitis	Ultrasound of the pelvis. MR of the abdomen and pelvis for problem cases. See "Abdominal pain" for further details including a discussion of imaging in patients who are *not* pregnant.
Chest pain or dyspnea and suspected pulmonary embolism	Ultrasound of both lower extremities to search for deep venous thrombosis. If negative, one of the following three studies will likely need to be performed, depending on local practice patterns: 1) noncontrast magnetic resonance angiography of the pulmonary arteries; 2) computed tomographic angiography of the pulmonary arteries; or 3) a nuclear medicine perfusion study of the lungs. See "Chest Pain" and "Dyspnea" for further details including a discussion of imaging in patients who are *not* pregnant.

Table 2. Complications of Pregnancy: Causes, Clinical Features, and Imaging Findings

Cause	Clinical Features	Imaging Findings
Miscarriage	Pelvic pain and vaginal bleeding	Lack of live intra-uterine fetus; lack of heartbeat with fetal crown-rump length of than 7 mm or more; lack of fetus with gestational sac mean diameter of 25 mm or more; failure to progress on sequential scans.
Ectopic pregnancy	In vitro fertilization or fertility drugs; abdominal pain; vaginal bleeding	Lack of live intra-uterine fetus; adnexal mass with hyperemia; ectopic gestational sac with live fetus (rare)
Molar pregnancy	Abdominal pain; hyperemesis; disproportionate elevation of betaHCG	Lack of normal intra-uterine fetus; endometrial multicystic mass
Placental abruption	Pelvic pain and vaginal bleeding; uterine contractions	Separation of the placenta from the uterus
Intra-amniotic infection (chorioamnionitis)	Fever; abdominal pain; leukocytosis; tachycardia; uterine contractions	A shortened cervical canal is a risk factor for chorioamnionitis.
Uterine rupture	History of prior cesarean delivery or uterine pregnancy, peritoneal irritation, vaginal bleeding	Free fluid in the peritoneal cavity; discontinuity of the uterus
Severe Pre-eclampsia and the HELLP syndrome	Abdominal pain; hypertension; hemolysis; elevated AST and LDH; low platelets	Complications including hepatic infarction, hematoma, or rupture. Placental abruption.

Table 3. Diseases Complicating Pregnancy: Causes, Clinical Features, and Imaging Findings

Cause	Clinical Features	Imaging Findings
Ovarian torsion	Pelvic pain on the side of the torsion; elevated WBC count	Cysts, swelling, or diminished flow (on color Doppler) of the ovary
Acute appendicitis	Right lower quadrant pain; anorexia; elevated WBC count	Swollen appendix; peri-appendiceal fat stranding
Gallbladder disease	Intermittent right upper quadrant pain; fever and leukocystosis	Gallstones; gallbladder wall thickening; pericholecystic fluid
Bowel obstruction	Prior abdominal surgery; abdominal pain; new onset nausea and vomiting after the first trimester	Distension proximal to, and collapse distal to, the obstruction; abnormality at the transition point (e.g., hernia)
Inflammatory bowel disease	Abdominal pain; altered bowel movements; fever; weight loss	Bowel wall thickening; fat stranding
Pancreatitis	Upper abdominal pain; elevated amylase and lipase; elevated WBC count	Pancreas swelling and peripancreatic fat stranding; focal fluid collections in or adjacent to the pancreas
Diverticulitis	Abdominal pain and fever; elevated WBC count	Diverticulae; pericolic fat stranding; free air or free fluid in the peritoneal cavity
Pyelonephritis	Flank pain; fever; pyuria	Hyperemia of the kidney; hydronephrosis with debris
Nephrolithiasis	Flank pain; hematuria	Renal stone in the collecting system
Pneumonia	Abdominal or chest pain; fever; cough; dyspnea	Lung consolidation; pleural effusion
Pulmonary embolism	Acute chest pain; dyspnea; decreased oxygen saturation	Filling defect in the pulmonary artery; abnormal lung perfusion
No imaging findings		
Cystitis, gastroenteritis		

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