Diagnosis and staging of various internal derangements is by far the most common purpose of MR imaging of the knee. By virtue of their superior spatial/anatomic resolution, 3 T magnets provide the most accurate and detailed depiction of chondral, subchondral, and ligamentous injuries. At our institution, a standard internal derangement protocol for the knee includes axial proton density with fat saturation (TE 35/TR 3500, 4-mm slice thickness, and 384 × 512 matrix), sagittal proton density without fat saturation (TE 22/TR 3300, 2.5-mm slice thickness, and 384 × 512 matrix), sagittal T2 with fat saturation (TE 65/TR 5500, 2.5-mm slice thickness, and 384 × 512 matrix), coronal T1 without fat saturation (TE 15/TR 550, 3-mm slice thickness, and 288 × 384 matrix), and coronal T2 with fat saturation (TE 65/TR 3500, 3-mm slice thickness, and 256 × 512 matrix).

♦ Cartilage

Patellofemoral dysfunction accounts for a majority of anterior knee pain. Whether from true chondromalacia or adverse biomechanics with patellofemoral instability, patellar cartilage lesions are easily evaluated on axial imaging. FSE proton density sequences, with or without fat saturation, provide excellent resolution of the cartilage surface interface with surrounding joint fluid or adjacent femoral cartilage. Figure 70–1A is a non-fat-saturated sagittal image that shows the normal cartilage-fluid signal contrast between the patellar superior pole cartilage and suprapatellar...
recess joint fluid (small arrow). Chemical shift artifact likely contributes to the conspicuousity of the cartilage-fat interface of the femoral trochlear cartilage and Hoffa’s fat pad (large arrow) as the frequency encoding direction is anterior-posterior. The proton density sequence has the advantage over cartilage dedicated GRE or spoiled GRE techniques (Fig. 70–1B) in that it also better depicts fine detail of the intrinsic and extrinsic ligaments and capsule of the knee. The arrow in Fig. 70–1B shows the location of a bone bruise associated with a recent anterior cruciate ligament (ACL) tear. The cartilage of the femoral lateral condylar notch and posterior tibial plateau appears normal.

In Fig. 70–2A, the superficial tangential zone of collagen in the surface layer of the patellar cartilage can be seen as a discrete low signal line that is often not resolved
with 1.5 T imaging. Note the uniform gradation of fluid signal intensity from the transitional layer to the deeper radial layer. The calcified layer is generally not distinguishable from the subchondral bone plate in normal cartilage. Where intact cartilage surfaces abut, the tangential zones are correspondingly twice as apparent due to the apposition of these two dark/low-signal lines. **Figure 70–2B** is an axial proton density image through the patellofemoral joint of a 15-year-old female that illustrates accentuation of the tangential zone contact of the medial patellar facet and femoral trochlea. **Figure 70–2C** shows the same phenomenon within the medial femorotibial compartment, this time on a non-fat-saturated sagittal proton density image. The arrow indicates the location of a subtle disruption of the tangential zone by a superficial chondral lesion with fraying. Note the absence of the fine dark line that normally delineates the cartilage surface. There is also a shallow fissure along the leading edge of the defect. **Figure 70–2D** is a coronal T2 fat-saturated image where the arrow again shows the double tangential zone contact. Compare this to the appearance of the tangential zone along the sharply curving surface of the lateral femoral condyle (arrowhead) where the line is normally not apparent. The fine dark line of the tibial plateau interface with joint fluid of the lateral femorotibial compartment opposite the arrowhead is a good example of a single tangential zone interface with fluid. Accentuation of the black lines of the double tangential zone contact helps to maintain the sensitivity for detecting even superficial cartilage lesions at 3 T. Poor visualization of this line due to magic angle and/or volume averaging effect can occur in predictable locations where the curvature of the articular surface abruptly changes—usually at the margins of the superior and inferior poles of the patella and margins of the femoral condyles as in **Fig. 70–2D**. However, this is generally not a source of confusion in the patellofemoral joint given that most patellar cartilage pathology occurs at or near the apex between the medial and lateral facets and throughout the lateral facet. Sagittal sequences generally show the margins of the superior and inferior poles with better clarity, as they are more perpendicular to the cartilage and subchondral plates of the poles. Patellar lesions limited to these locations can be detected along with the corresponding injuries to the quadriceps and patellar tendons and Hoffa’s fat pad attachments by use of the sagittal sequences. **Figures 70–3A through 70–3D** show a case of chronic patellar tendinosis associated with patellofemoral instability from underlying lateral femoral condylar and trochlear hypoplasia in a 36-year-old male. Note the shallow trochlear groove (normal sulcus angle 146 ± 5 degrees) and dominant lateral patellar facet with rounding of the patellar apex (arrow) in **Fig. 70–3A. Figure 70–3B** is an AP radiograph with the knee semiflexed showing the transverse constriction of the hypoplastic lateral condyle (arrows) and the widening of the lateral compartment joint space (arrowhead). **Figure 70–3C** is a sagittal proton density of the same case. The arrowhead shows a focal chondral lesion of the central patellar cartilage with fraying and at least one deep fissure superiorly. The small arrows show small inferior pole patellar enthesophytes while the large arrows point out edema and thickening of the proximal and distal patellar tendon. The fat-saturated axial T2 in **Fig. 70–3D** better shows the thickened fibrotic fibers (arrowheads) of the patellar tendon with centrally located amorphous elevated fluid signal (arrow) possibly reflecting mucoid change from chronic inflammation.
Thinning of the articular cartilage can be graded in groups of 1 though 4 with each group representing an additional 25% of thickness lost. **Figure 70–4A** is an axial proton density image with fat saturation where the arrow indicates a focal superficial chondral lesion of the medial patellar facet. Note the loss of the tangential zone dark line and adjacent patch of fibrillated cartilage just anterior to the arrow. **Figure 70–4B** is a sagittal proton density image without fat saturation of the same lesion. The arrow again shows the interface of joint fluid and the fibrillated cartilage.

As normal patellar cartilage is between 3 and 4 mm in thickness, a grade-2 lesion with between 25 and 50% thinning is generally easily distinguished from a grade-3 lesion with between 50 and 75% thinning. **Figure 70–4C** is another axial proton density image with fat saturation that shows an obliquely oriented fissure extending to half of the cartilage thickness. The obliquity of its dissection through the upper layers
of the cartilage suggests that rather than eventually penetrating the calcified layer, this lesion will go on to become a cartilaginous flap. The adjacent arrowhead indicates a small violation of the tangential zone with locally increased intrasubstance fluid signal. **Figure 70–4D** is a sagittal proton density image from the same case showing the open surface of the fissure and inferior oblique dissection (arrow). Engagement of the patella in the sulcus terminale of the femur during extension will open the fissure and propagate the lesion.

Full-thickness lesions that extend to the subchondral plate always result in subchondral edema initially and subchondral fibrocystic degeneration of bone eventually. Full-thickness fissures will have a focal flame-shaped area of edema while larger areas of exfoliation will have edema throughout the width of the exposed subchondral plate. An exception to the latter would be an acute/hyperacute chondral shear injury where cartilage separates along its deep calcified layer leaving...
the bony subchondral plate intact. Figure 70–5A is a sagittal T2-weighted image with fat saturation that shows a minute plume of subarticular edema (arrow) associated with an acute medial femoral condylar impaction injury that focally disrupted the adjacent subchondral plate. Figure 70–5B is a coronal T2-weighted image with fat saturation where the arrow indicates the apex of a cartilaginous flap within a more chronic chondral defect of the medial femoral condyle. The arrowhead marks the edge of the chondral defect. Note that the edge of the defect is rounded rather than frayed and shows uniformly low signal, likely representing chondral fibrosis. Marginal spurring attests to chronic osteoarthritis whereas medial compartment marginal edema indicates elevated stress distribution from an incompetent medial meniscus and worsening varus angulation. Contrast the appearance of this chronic chondral lesion to the acutely torn and flipped chondral flap of the medial patellar facet in Fig. 70–6A. This is an axial proton density image with fat saturation in an adolescent male who suffered a direct blow to the medial anterior patella. The small arrow indicates a fold in the tangential zone fibers of the cartilage that remain connected to the sheared fragment. The large arrow indicates a focal band of subchondral trabecular impaction. Superficial to this there is a disruption of the subchondral plate. Note the loose fibers of the cartilage transitional zone in between the arrows. There is increased fluid signal in the frayed edges of this defect as apposed to low signal fibrosis found in chronic lesions. The white arrowhead shows the torn fibers of the medial patellofemoral ligament/retinaculum attachment on the medial patella whereas the black arrowhead shows the stump of the torn medial patellar plica. Figure 70–6B is an example of full-thickness cartilage exfoliation of the entire lateral patellar facet as shown on routine axial proton density with fat saturation. The subchondral plate
is intact. Small plumes of subchondral edema are scattered throughout the length of the defect. The long arrow shows the smooth and slightly fibrotic edge of the apical extent of the patellar defect while the arrowhead shows the sharp nonfibrotic edge of a newer focal full-thickness defect in the superior medial trochlear ridge of the femur. The small arrow shows the transcortical segment of a normal perforator vessel entering the supratrochlear portion of the femoral metaphysis. No cartilage is normally present medially at this level.

Superficial fraying, particularly at the patellar apex is common. Figure 70–7 is a common arthroscopic appearance of the patellofemoral joint in which there is fine fibrillation of the cartilage surface at the patellar apex and lateral facet (arrows) and a matching area in the sulcus terminale of the femoral trochlea (arrowhead). A focal superficial defect of the cartilage can occasionally lead to intrasubstance edema, or imbibing of joint fluid into the cartilage, resulting in bulging of the overlying surface and an increase in the thickness of the cartilage locally. These lesions are soft to arthroscopic probing and are referred to as chondral blisters. They are equivalent to grade-2 chondromalacia.
Femorotibial cartilage lesions are common in older individuals as a result of osteoarthritis, meniscal degeneration, and incompetence, and in anyone with ACL deficiency, meniscal tear or significant impaction injury. Acute osteochondral impaction fractures, chronic repetitive osteochondral injuries (osteochondrosis dissecans), and subchondral insufficiency fractures are all well demonstrated with the routine internal derangement protocol at 3 T.