90. Temporomandibular Joint Disease

The temporomandibular joint (TMJ) is a synovial joint between the mandibular condyle, glenoid fossa, and articular eminence of the temporal bone. MR reliably evaluates joint pathology and meniscal position in most patients, with the exception of those where metallic dental hardware limits image quality secondary to susceptibility artifact. Over half of patients have bilateral dysfunction, thus imaging is typically performed of both joints. SE T1WI are obtained in sagittal-oblique planes through the TMJ with both open and closed mouth views. FSE T2WI may be added if inflammatory disease, joint effusion, or meniscal degeneration is suspected. Coronal T1WI may be obtained to help identify meniscal displacement medially or laterally. Cine imaging may also be performed in incremental stages of mouth opening to allow for "dynamic" evaluation of joint motion.

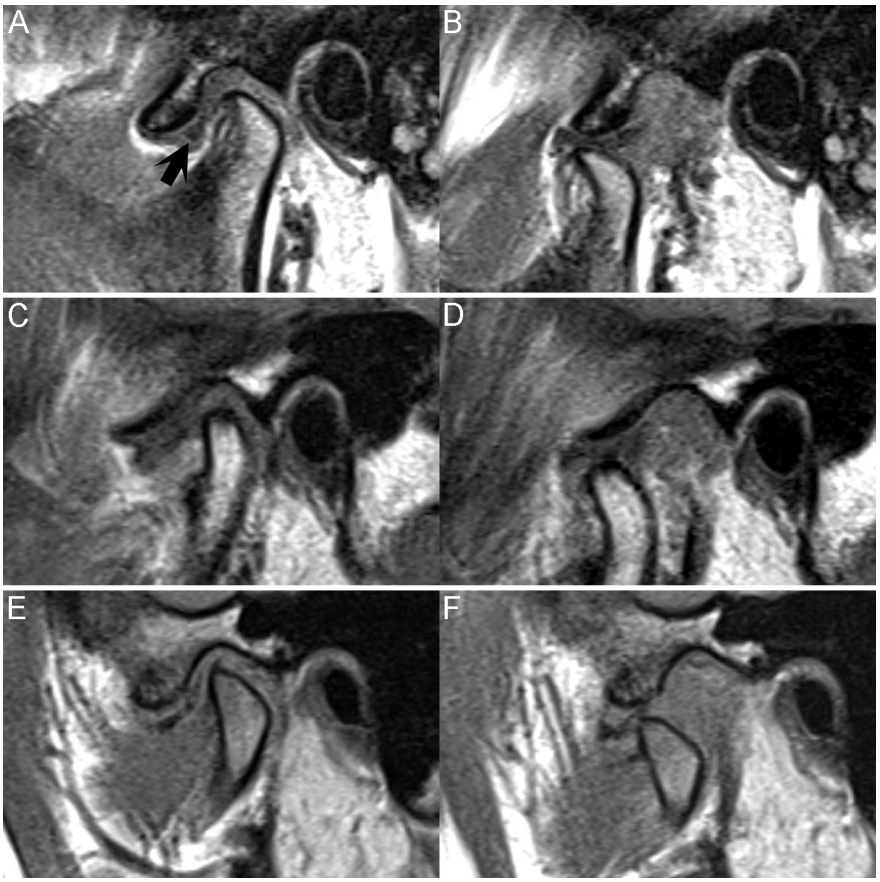

Fig. 90.1

The normal appearance of the TMJ is demonstrated in the (A) closed and (B) open mouth FSE T1WI of Fig. 90.1. The mandibular condyle is located anterior to the external auditory meatus with its head articulating with the glenoid fossa and articular eminence of the

temporal bone. The normal, biconcave meniscus is hypointense on both T1 and T2WI. When (A) the mouth is closed, the mandibular condyle lies centered in the glenoid fossa with the meniscus (black arrow) lying along its anterosuperior aspect. With (B) mouth opening, the condyle translocates anteriorly with the meniscus moving into the one- or two-o'clock position. The posterior portion of the meniscus is attached to the retrodiscal bilaminar zone which contains elastic tissue and fat and thus appears hyperintense to the meniscus. This hyperintensity is often lost in the presence of pathology. The bilaminar zone connects the meniscus to the temporal bone behind the condyle. The lateral pterygoid is located anterior to the meniscus and divides into superior and inferior heads. The former inserts onto the anteromedial aspect of the meniscus, applying traction to the structure to allow the anterior translocation of the condyle by the inferior belly of the lateral pterygoid. The most common cause of TMJ dysfunction is an anterior reducible dislocation of the meniscus, seen in the closed mouth FSE T1WI of Fig. 90.1C. Here the low SI meniscus is located anterior to the mandibular condyle with the condyle resting on the retrodiskal tissue. Figure 90.1 D demonstrates the reducibility of this dislocation: the condyle has translocated anteriorly under the posterior band of the meniscus, allowing it to assume a normal position in open-mouth views. Without such reduction, the anterior defect is termed fixed or nonreducible. Figure 90.1E,F demonstrate a fixed-type of dislocation with the meniscus again seen anterior to the mandibular condyle in (E) closed-mouth views. In (F) open mouth views, the low SI disk remains dislocated anteriorly to the condyle. Often a fixed, chronically dislocated meniscus will appear deformed or compressed due to repetitive trauma during opening and closing of the mouth. A so-called locked condyle occurs when mechanical obstruction prevents the anterior translocation of the condyle. As noted previously, medial or lateral displacement of the meniscus is often best-detected on coronal images. Chronically, disk perforations may occur, although this is better evaluated on arthrography than on MR. Myxomatous degeneration of the meniscal cartilage may also be seen, resulting in increased SI on T2WI. Ancillary findings such as degenerative changes within the superior body of the lateral pterygoid, particularly fibrosis, atrophy, and contracture, may occur. Resultant fatty replacement within the muscle leads to increased SI on T1WI. Thickening of the fascia within the inferior belly of the lateral pterygoid may also occur as a result of a chronic anterior disk displacement. A chronically dislocated disk results in the condyle articulating with the posterior attachment of the disk rather than with the disk itself, leading to inflammation and perforation of the posterior meniscal attachment. Osteoarthritis can occur, reflected by osteophytes and osseous deformities along the glenoid fossa and mandibular condyle. TMJ derangement may eventually lead to osteochondritis or even AVN of the mandibular condyle. Progression of MRI SI in condylar

AVN is similar to that previously described in the hip (see Chapter 85). Osteochondritis appears as a focal subarticular bone defect, often with central hypointensity surrounded by hyperintensity on both T1 and T2WI. Osteoarthritic changes within the TMJ are similar to those in other joints, and include condylar flattening along with articular fossa deformity. Accompanying subchondral sclerosis may be identified as low SI on T1 and T2WI, whereas edema-like SI indicates inflammatory changes within the joint. Other arthritides less commonly involve the TMJ. Detection of those of inflammatory etiology, in particular, is aided by the acquisition of CE T1WI in which inflamed synovium will enhance against a background of low SI joint fluid.