Bankart variants, such as the Perthes lesion can cause anterior glenohumeral stability. This lesion occurs when the scapular periosteum remains intact but is stripped medially, while the anterior labrum is avulsed from the glenoid but remains partially attached to the scapula by the intact periosteum. The labrum may assume a coapted position and thus a Perthes lesion may be seen to better advantage with MRI in abduction and external rotation (ABER). A Perthes lesion is illustrated in the MR arthrogram image (FS T1WI) of Fig. 84.1. Here, contrast undermines the anterior labrum with stripping of the glenoid periosteum. There is not significant displacement of the former structure, and in distinction to the Bankart lesion (see Fig. 83.1B), the attachment of the labrum to the periosteum remains intact. Disruption of the scapular periosteal attachment is also characteristic of anterior labral ligamentous periosteal sleeve avulsion injury (ALPSA), which is a Bankart-type lesion in which the torn anterior labrum is displaced medially and rotated inferiorly, interposed between the avulsed periosteum and scapular bone (a “medialized” Bankart). Similar lesions may be seen with the posterior labrum and are referred to as POLPSA (posterior periosteal sleeve avulsion). Avulsions of the anterior band of the inferior glenohumeral ligament may occur at its humeral attachment (HAGL) although mid-portion tears are more common. On conventional MR, these lesions are difficult to detect in the absence of a joint effusion but may appear as foci of hyperintensity on T2-weighted images. MR arthrography may show extravasation of contrast material through the ligamentous defect at its humeral attachment. The inferior displacement of the ligament characteristically results in a J shape. Posterior HAGL lesions (PHAGL) are less common and consist of avulsions of the posterior band of the inferior glenohumeral ligament at its humeral attachment. Associated marrow edema or humeral bone avulsions may occur with both of these lesions and are sometimes referred to as BHAGL.

Fig. 84.1

Superior labral tears are also common, although these lesions do not result in instability. The detection of anterosuperior lesions may be difficult given the frequent presence of normal variants in this region, including the sublabral foramen (common) and Buford complex (less common). A sublabral foramen is an anatomic variant in which the anterosuperior labrum is

Runge, von Tengg-Kobligk, Heverhagen
not well-attached to the glenoid above the superior epiphyseal line of the glenoid. A sublabral foramen may be confused with a superior tear. Features suggesting tear over this anatomic variant include an irregular labral margin, abnormal labral hyperintensity, and separation between the glenoid and labrum. In the Buford complex, the anterosuperior labrum is absent and there is a thick, cord-like middle glenohumeral ligament attaching to the superior labrum directly. True tears of the superior labrum are best detected in the coronal plane, with arm placement in external rotation during scanning potentially aiding in detection. Tears involving the superior labrum, extending anteriorly and posteriorly are termed SLAP (superior labrum anterior posterior) lesions, of which multiple types have been described. The typical appearance is seen on the direct arthrographic MR images (FS T1WI) of Fig. 84.2A,B in which intraarticular gadolinium chelate undermines portions of the superior labrum up to the attachment of the biceps tendon without clear displacement of the anchor from the labrum.

Fig. 84.2

Type 1 lesions are characterized by labral degeneration and irregularity of the free edge margin. Abnormal labral signal without extension to the surface is generally considered indicative of labral degeneration rather than tear. Type 2 SLAP lesions consist of high SI extending to the labral surface, involving the biceps anchor. Type 2 SLAP lesions comprise a plurality of true labral tears. Superior labrum anterior cuff and peel-back lesions are subtypes of type 2 SLAP lesions, extending predominantly in the anterior and posterior directions, respectively. A meniscoid variant labrum at its free edge may be confused in appearance for a type 2 SLAP lesion. In the former, glenoid articular cartilage extends to the area of labral attachment, whereas in a type 2 tear, displacement of the labrum from the glenoid cartilage by 3-4 mm is frequently seen. Fraying of the labrum and synovitis are often useful ancillary findings indicating tear. Bucket-handle type superior labral tears without and with a concurrent tear of the biceps tendon constitute Type 3 and 4 lesions,

Runge, von Tengg-Kobligk, Heverhagen
respectively. Bucket-handle tears are typically well-visualized in the sagittal plane where three hypointense structures are seen—the two bucket-handle components and the biceps tendon. Bucket-handle tears may extend into the anterior or anteroinferior labrum. The latter case, in which a concurrent Bankart and SLAP lesion are present, constitutes a type 5 lesion. Type 6 SLAP lesions consist of radial or flap tears with anchor involvement. SLAP lesions extending anteriorly to concurrently tear the middle glenohumeral ligament constitute type 7 lesions, whereas tears resulting in posteriorly labral detachment are considered type 8 lesions. A type 9 lesion consists of concentric avulsion of the entire labrum around the glenoid. Coronal images best demonstrate detachment of the superior and inferior labrum, while axial images best display the remainder with the entire detached labrum often being visible on sagittal slices. Type 10 lesions concurrently involve the tendons of the rotator cuff via extension through the superior glenoid ligament. While not a cause of anterior instability per se, glenolabral articular disruptions (GLAD) are a cause of shoulder pain, consisting of partial anterior labral tears with defects in the adjacent articular cartilage. Fluid sensitive sequences demonstrate hyperintense chondral divots or flaps adjacent to the nondisplaced labral tear. On direct MR arthrography, contrast infiltrates the labral tear and extends into the cartilaginous defect.