Radiology of Shoulder Prostheses

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ABSTRACT

This article is chiefly limited to the routine radiographic evaluation of shoulder arthroplasties and guidelines to their interpretation. Other imaging modalities for the evaluation of joint replacements are discussed elsewhere in this issue. The basic types of shoulder reconstructions and some of the shoulder replacement designs in previous and current use at The New York Orthopedic Hospital at Columbia Presbyterian Medical Center are illustrated, along with the indications and contraindications of the various types of reconstructions. Their complications and pertinent anatomy are included.

KEYWORDS: Shoulder arthroplasty, shoulder replacement

The glenohumeral joint, the most mobile in the body, owes its versatility to its musculotendinous envelope. Once this relationship is significantly compromised, complete surgical restoration of function is difficult to accomplish, albeit not from want of effort. An early attempt was made in Greek mythology when Tantalus sought favor with the gods by offering them Pelops, his son, as a tender morsel. All recoiled from the feast except Demeter, who, distracted by the loss of her daughter Persephone, absentmindedly consumed Pelops' left shoulder. Zeus punished Tantalus and chastised Demeter, who then created a splendid ivory replacement for Pelops, thereby atoning for her sin and accomplishing the first shoulder arthroplasty (Fig. 1).

Human shoulder reconstructions had no such divine origins or results. Gluck, in 1891, implanted various joints with pumice. Péan in 1894[1] initially replaced a tuberculous shoulder with a hard rubber ball as a head joined to a platinum stem and glenoid with a metal plate and screws (Fig. 2). Within 2 years, a chronic sinus led to a proximal humeral fibular graft secured by kangaroo tendon. Fibular grafts then replaced deficient proximal humeri with intact rotator cuffs, and Jones used gold foil to line arthritic surfaces.[2–5] Methylmethacrylate, previously used for skull repair, superceded pumice, which Charnley then used to implant total hips.[6] Neer pioneered modern shoulder arthroplasty with prosthetic replacements for osteonecrotic or severely fractured heads and proximal humeral deficiencies. He then used glenoid liners and polyethylene components to expand indications for glenohumeral degenerative disease.[7–9]

Two basic total shoulder designs evolved, unconstrained and constrained, with the former becoming increasingly common. Fewer than 10,000 hemiarthroplasties or total shoulder arthroplasties performed annually in the early 1990s rose to 17,500 in the late 1990s.[10] Unconstrained designs still yield the most reliable results with respect to pain relief, functional restoration, and prosthetic longevity in over 90% of patients[11] Neer's techniques continue to be used at New York Orthopedic Hospital–Columbia Presbyterian Medical Center with new design modifications continuing to evolve.[12]

ANATOMY

The shoulder joint's primary function, to position the hand in space, is achieved with a "mismatch." Only a small segment of a larger, rounded, posteromedially and

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Figure 1 The first ''divine'' shoulder arthroplasty. Demeter atones for devouring Pelops' shoulder by crafting a solid ivory replacement while Zeus supports the patient.

upwardly directed humeral head makes contact with a shallower glenoid surface one fourth its size. Its narrow superior and broad inferior comma-like shape, inclined to the scapula's horizontal axis, contributes to shoulder joint incongruity and its greater dependence on surrounding soft tissues for stability compared with the hip and knee (Fig. 3). The labrum adds limited stability, but multiple capsular folds and synovial pouches with twice the humeral head volume lead to laxity. Stability is thereby sacrificed for mobility so that the shoulder has the greatest resultant range of motion of any joint in the body.

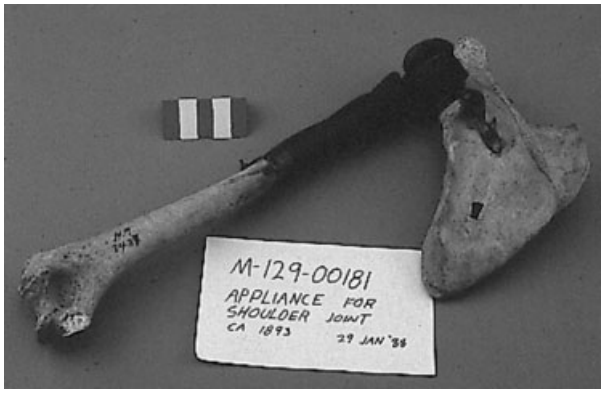


Figure 2 Cadaver specimen of Péan's first (1894) human shoulder arthroplasty. A hard rubber ball ''head'' joined to a platinum stem and glenoid by a metal plate and screws replaced a tuberculous joint.

Dynamic function relies on the rotator cuff. It arises from the glenoid rim and labrum, covers the shoulder joint, and blends with an inferior, synovium-lined fibrous capsule to insert on the anatomic neck and proximal humerus. Four muscles—the subscapularis, supraspinatus, infraspinatus, and teres minor—aid stability by depressing the head and fixing the fulcrum against which the deltoid contracts, abducts, and elevates the humerus (Fig. 4). The supraspinatus depresses the greater tuberosity and compresses the humeral head against the glenoid to prevent its downward subluxation or dislocation. The biceps long head prevents superior humeral head migration. The posterior deltoid, pectoralis major, and latissimus dorsi are active adductors. The anterior deltoid, clavicular portion of the pectoralis major, and biceps brachii are active flexors. A bony optimizer of function, the acromion, overhangs the posterosuperior humeral head, attaches to the coracoid with a ligament, and forms the coracoacromial arch, which guards the head from direct trauma but limits

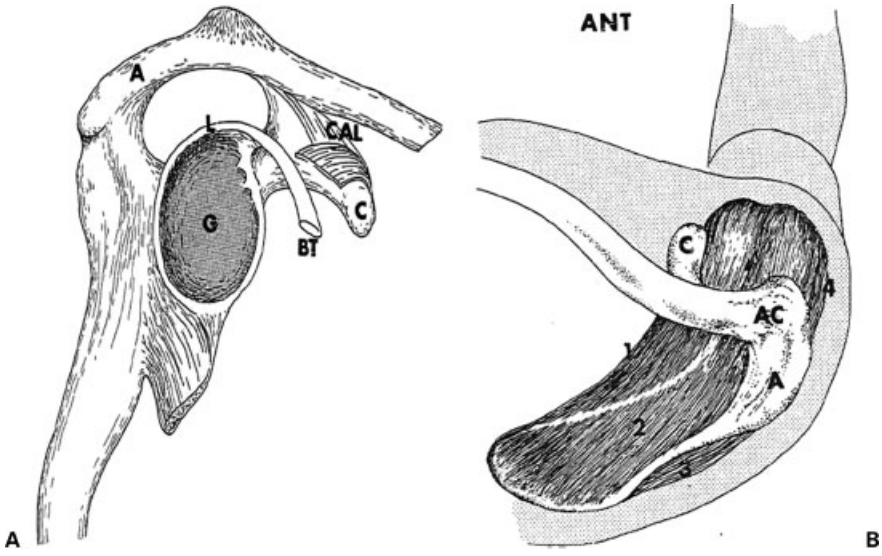


Figure 3 (A) Lateral scapular view. Shallow glenoid fossa (G), broad inferiorly, narrow superiorly, is rimmed and deepened by fibrous labrum (L), whose posterior lip blends with long biceps tendon head (BT). Coracoacromial arch composed of coracoid process (C), coracoacromial ligament (CAL), and acromion (A) protects underlying rotator cuff and humeral head. (B) Coracoacromial arch, axial view. Coracoid (C) and acromio (A)-clavicular junction (AC) form a roof overlying four rotator cuff muscles: subscapularis (1), supraspinatus (2), infraspinatus (3), and teres minor (4).

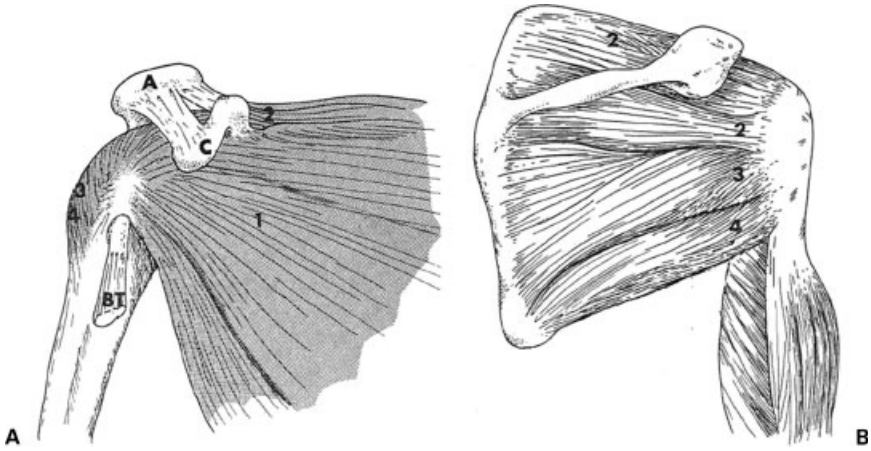


Figure 4 (A) Rotator cuff muscles and tendons. Anterior view shows subscapularis (1), supraspinatus (2), infraspinatus (3), teres minor (4), long biceps tendon (BT), coracoacromial arch and ligaments (AC). (B) Posterior view shows supraspinatus (2), infraspinatus (3), and teres minor (4).

abduction (Figs. 3, 4A).The subacromial bursa partially cushions the cuff from acromial impingement on a relatively hypovascular "critical zone" 1 to 1.5 cm proximal to the supraspinatus insertion that is equally vulnerable to mechanical "wear and tear" (Fig. 5). Ischemia, worsened by cuff angulation over the humeral head, compressive forces of supraspinatus contraction, and trauma may also play a role. Alternative causes include connective tissue degeneration related to aging and intrinsic cellular cuff alterations with decreased mucopolysaccharide or increased weaker type 3 collagen fibers noted in both young and old and, therefore, not age dependent. Immunohistochemical and molecular studies revealed increased expression of inflammatory mediator cytokine genes (i.e., tumor necrosis factor TNF; interleukin-1a,b,6; metalloproteases-MMP 1,9; and cyclooxygenases COX 1,2) by subacromial bursal cells, even without evidence of leukocytes, monocytes, or macrophages, suggesting the involvement of subacromial bursitis in the pathophysiology of rotator cuff disease. Accordingly, bursectomy has been advocated as an important component of treatment.[12–14]

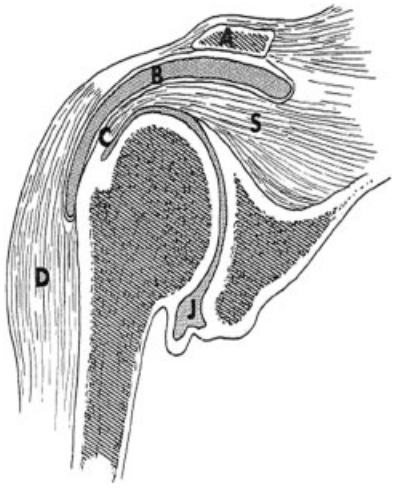


Figure 5 Shoulder joint, coronal section. Deltoid muscle (D) attaches to acromial process (A) above the communicating subacromial-subdeltoid bursa (B). Supraspinatus (S) overlies joint space (J), attaches to greater tuberosity, and blends with capsule. Note ''critical zone'' proximal to supraspinatus insertion (C).

Types of Surgical Reconstruction Types of surgical reconstruction have included hemiarthroplasty (HA) (Fig. 6) with humeral head replacement alone, total shoulder replacement (TSR) (Fig. 7A–C) of humeral and glenoid articulations, humeral head resurfacing with prosthetic surface replacement (Fig. 8A, B), glenoid resurfacing with biologic replacement, and glenohumeral arthrodesis.

Hemiarthroplasty Indications include proximal humeral articular deficiencies with intact glenoid surfaces and periarticular soft tissues, comminuted humeral head fractures, avascular necrosis unrelated to radiation, neoplasms, and old injuries in the young.

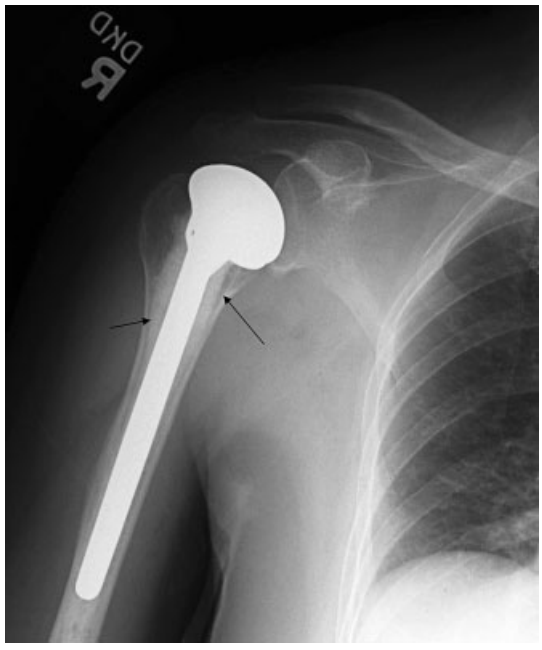


Figure 6 Hemiarthroplasty (HA). Modular prosthesis (Bigliani/Flatow) replaces humeral head. Note clinically insignificant thin, regular methylmethacrylate-bone interface lucency (arrows).

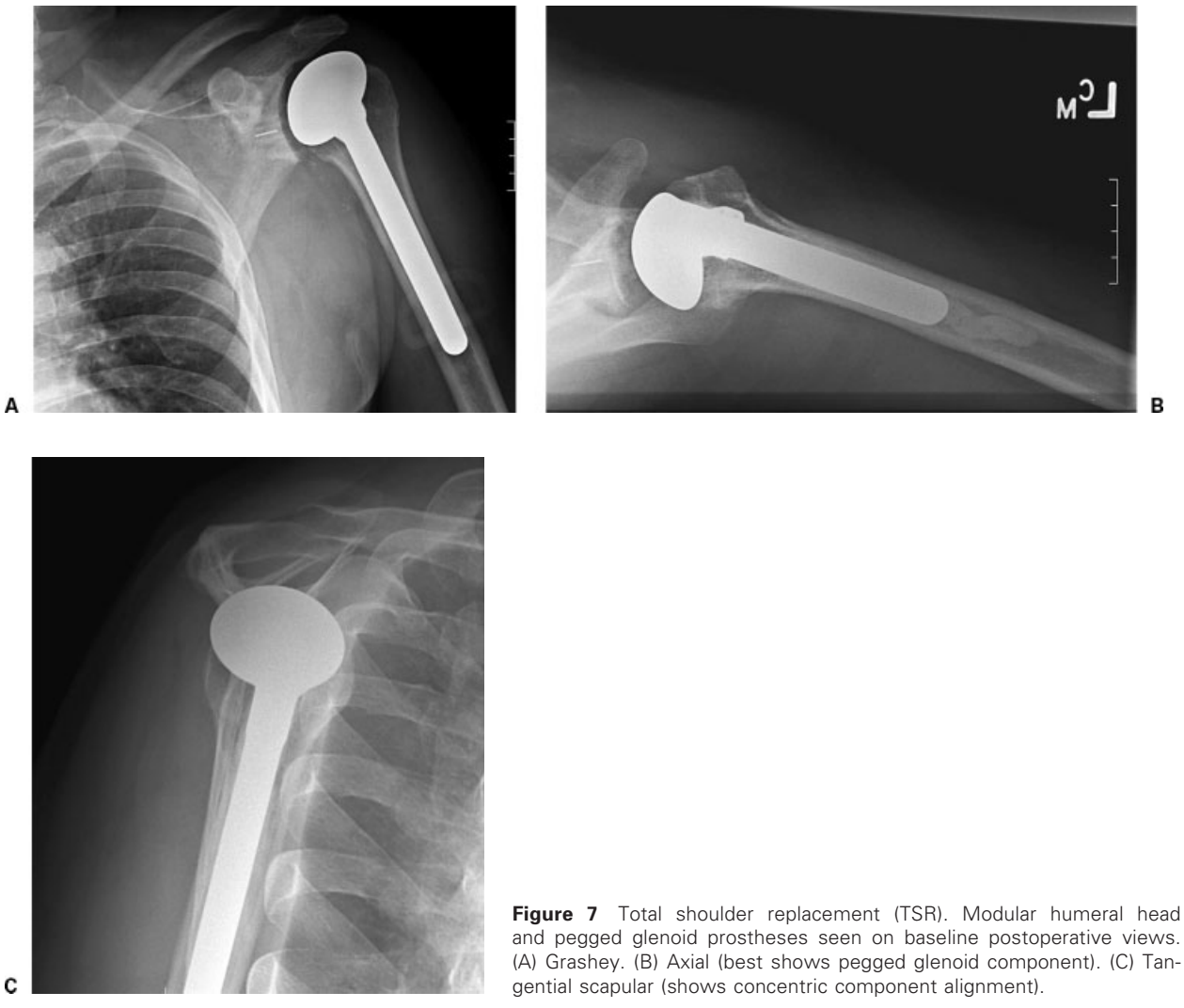


Figure 7 Total shoulder replacement (TSR). Modular humeral head and pegged glenoid prostheses seen on baseline postoperative views. (A) Grashey. (B) Axial (best shows pegged glenoid component). (C) Tangential scapular (shows concentric component alignment).

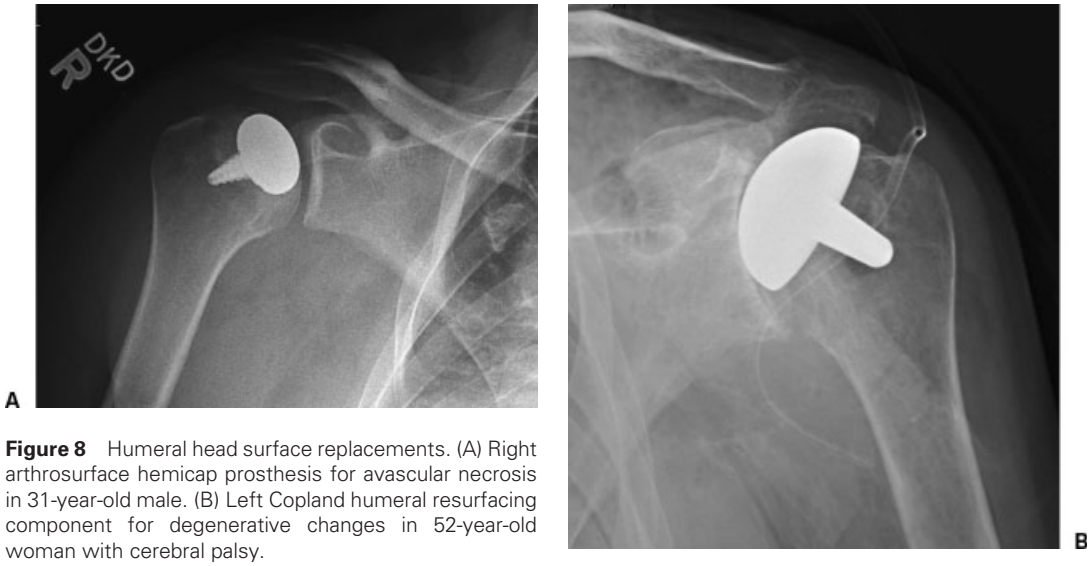


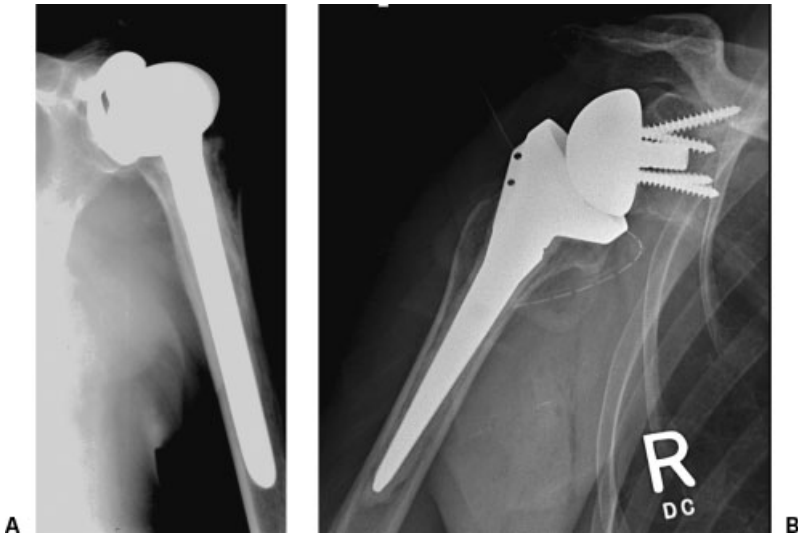
Figure 8 Humeral head surface replacements. (A) Right arthrosurface hemicap prosthesis for avascular necrosis in 31-year-old male. (B) Left Copland humeral resurfacing component for degenerative changes in 52-year-old woman with cerebral palsy.

HA component stems may be smooth and supported by "cement" or "press fit" with irregular ceramic, polymer, or metal beaded surfaces. The latter do not bond chemically, but provide greater surface area to interdigitate with newly reamed intramedullary trabecula in those with good bone stock. Contraindicated in osteopenia because of old age, medication, prior arthroplasty, or metabolic bone disease, they afford less immediate support than cement fixation due to time needed for ingrowth. Surface beads may also shed. Conversely, they are easier to remove, conserve bone stock for revision, and are not subject to cement fracture or "debris osteolysis."

Total Shoulder Replacements These include three basic designs: (1) unconstrained, unattached components for freely gliding motion (Fig. 7A–C); (2) semiconstrained with only partial play with stability achieved by larger glenoid socket designs, superior hoods or extensions preventing superior humeral component migration, or reverse ball and socket configurations (Fig. 9A–C); and (3) constrained interlocking components with a fixed fulcrum compensating for deficient periarticular soft tissues, now rarely used.

Indications for TSR Indications are primarily related to pain failing to respond to nonsurgical measures, such as rest, physiotherapy, anti-inflammatory agents, or potentially harmful medications. It is usually worse at night, and sleeping on the ipsilateral side, described as "unbearable," may lead to voluntary immobility or adhesive capsulitis. Pain of cervical spine, muscle, or acromioclavicular origin may be clinically distinguished by provocative anesthetic injection. Relief of restricted motion or function is a secondary objective with painless joints often not operated.

Glenohumeral dysfunction related to osteoarthritis accounts for over 60% of TSRs, followed by trauma, systemic or metabolic disorders such as crystal deposition disease, diabetes, sickle cell anemia, hemochromatosis, ochronosis, radiation necrosis, prior arthroplasty with excessive anterior capsular plication, posterior glenoid stress, and cuff tear arthropathy.[15] There is no specific age limit provided that patients are reasonable surgical risks, can cooperate in postoperative rehabilitation, and are well motivated with caution advised for those younger than 50 years.[11,16] TSR performs best with intact periarticular musculature that maintains unrestricted motion as in normal shoulders.



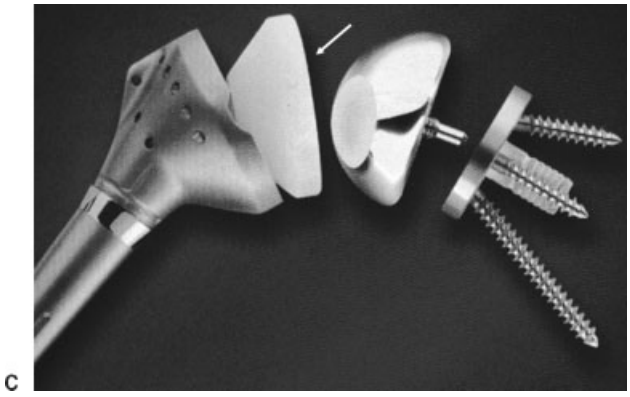


Figure 9 Semiconstrained TSR. (A) Only partial play occurs between humeral and metal-backed polyethylene articular surfaced glenoid component. Its deeper socket (200% greater humeral head coverage) increased stability after three failed rotator cuff repairs. (B) Depuy reverse ball and socket prosthesis replaced previously subluxing Biomet hemiarthroplasty because of deficient rotator cuff. (C) Ball and socket prosthesis—reversed humeral-glenoid articulations. Concave high-density polyethylene humeral surface (arrow) articulates with convex metallic glenoid.

Relative Contraindications for TSR Relative contraindications include severe osteoporosis, inadequate bone stock, and rotator cuff or glenoid deficiency. Inflammatory arthritides, Marie-Strümpell disease with shoulder ankylosis without pain, and hemophiliac, dialysis, or cuff tear arthropathy (CTA) account for 30% of TSRs. CTA may initiate a chain of mechanical and nutritional sequelae because closed joint space and synovial fluid loss reduce articular pressure and nutrient perfusion with resultant cartilage atrophy, subchondral bone collapse, and disuse osteoporosis.[15,17] TSR may be beneficial if periarticular tissues can be augmented. Neer noted that two thirds of such cases with repairable rotator cuffs benefitted from limited goals, such as self-feeding, hair combing, and perineal care, but recovered function slowly. Full-thickness tears were noted in 5 to 10% of this group, as well as poor quality muscles that could not hold sutures or function post repair.[15,17] HA or semiconstrained components may be alternatives in the severely affected.

Absolute Contraindications for TSR These include severe osteoporosis, suprascapular nerve paralysis, neurologic or Charcot arthropathies, alcoholism, psychiatric disorders, loss of deltoid or rotator cuff function, advanced osteoarthritis with superior humeral head migration, inferior acromial erosion and greater tuberosity blunting, and prior infection. Shoulder infections, without prior arthroplasty, presumably eradicated by antibiotics or open débridement, are being implanted. Revision arthroplasties of previously infected prostheses are evaluated on an individual basis, after prosthetic removal, open surgical débridement, antibiotic polymethylmethacrylate (PMM) mold placement, and negative cultures.

Semiconstrained TSR Indications include deficient periarticular soft tissues that can be augmented. As with nonconstrained designs, they have been modified with superior glenoid hoods or extensions to prevent proximal humeral component migration. More recent designs include reverse ball and socket configurations (Fig. 9A–C).

Constrained Prostheses Constrained prostheses, now rarely used, provided a fixed fulcrum to compensate for rotator cuff or muscle deficiency. Size constraints may preclude cuff reattachment, thereby limiting motion because external rotators supply more than 90% of external rotation.[17] They are also at risk for mechanical and material failure related to extreme constrained motion stresses across glenoid components and to trauma since the "safety valve" subluxation or dislocation of normal shoulders and unconstrained prostheses is absent. Constrained designs were proposed as salvage procedures for restoring limited function in those with severe musculotendinous compromise.

PROSTHETIC MATERIALS AND TECHNOLOGY

Prosthetic designs may be distinguished on radiographs, but their individual compositions may not. Stainless steel, tantalum, titanium, vanadium, cobalt, nickel, chromium, tungsten ceramics, and molybdenum have all been used as alloys, repeatedly tested, and redesigned to improve resistance to fatigue and wear. Early metal-to-metal articulations were likely to shed, with "metallosis" evidenced by dark-staining synovium and joint fluid.[18–20] Silastic and polyethylene components may similarly shed. Polyethylene is also used in powder form (PMM) as a support or cement. It does not form a biochemical bond with bone or prosthesis but promotes gradual load distribution between bone and prosthetic interfaces. PMM contains radiopaque barium sulfate, is mixed with liquid monomer at surgery, sets

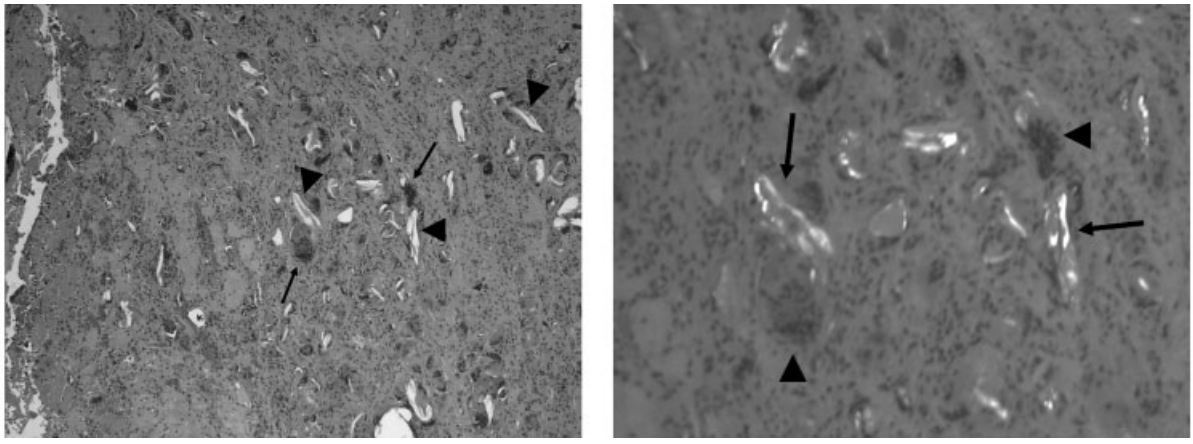


Figure 10 Debris disease. (A) Routine light microscopic H&E-stained section (× 120) reveals fibrotic tissue and giant cells (arrows) rimming linear vacant clefts (arrowheads) related to polyethelene fragments dissolved by tissue preparation. (B) Polarized light microscopy shows remaining refractile PMM particles (arrows), dense fibrous tissue, and cytokine-releasing macrophages (arrowheads) causing sterile osteolysis.

within 10 minutes, hardens in several hours, contracts on curing, and is exothermic with up to 100°C reached at bone-cement interfaces.[21]

Particles from PMM or high-density polyethylene surfaces are most often responsible for debris disease, noninflammatory tissue response, or sterile discharge.[22,23] Polarized light microscopy reveals polyethylene particles in dense fibrous tissue and macrophages, with cytokine release leading to progressive osteolysis (Fig. 10A, B). Mechanical attrition or regional exothermia may also cause diffuse or focal osteolysis at prosthetic-bone, prosthetic-cement, or bone-cement interfaces with endosteal cortical thinning, erosion, and weakened bone leading to fracture or prosthetic loosening. Raman vibrational spectroscopy of tissue from TSRs revised for osteolysis and aseptic loosening showed high-molecular-weight polyethylene particles that were larger, more fibrillar, and less globular than those about failed total hips and knees. Joint type may, therefore, influence wear and tear mechanisms as well as the nature of debris.[24] Nevertheless, ultrahigh-molecular-weight polyethylene-metal interfaces have provided long wear, least friction, and continue to be used in unconstrained TSR and weight-bearing joints.[11]

RADIOGRAPHIC EVALUATION—GENERAL GUIDELINES

Routine radiographs, an inexpensive means for documenting immediate postoperative changes, may initially be taken in two planes. A more complete baseline study, prior to discharge, should include erect anteroposterior humeral neutral, internal, external rotation, axillary, lateral scapular "Y," and 40-degree posterior oblique (Grashey) views (Fig. 7).

With proximal humeral bone loss related to fracture, tumor resection, or prosthetic revision, the prosthetic head (HA) is cemented "proud," neither too high nor low, about 5 mm above the greater tuberosity, to avoid postoperative impingement (Fig. 7).[11] HA replacements are supported by PMM except in younger patients with good press-fit potential or in those with concern for infection. The prosthetic head base should parallel the humeral neck's cut surface (Fig. 7) with 35- to 45-degree articular retroversion with the arm in neutral at the side or with 90-degree elbow flexion.[7] Ideally, the entire space between humeral prosthesis and adjacent bone is occupied by evenly distributed PMM with no evident discontinuity, gap, or trapped air, extending about 2 cm distal to the stem with or without a cement restrictor.[25] Gaps between humeral head replacement (HHR) and bone shafts may also be bone grafted to preserve humeral length and proper rotator cuff and tuberosity attachment. A sagging, relaxed deltoid cannot raise the arm. Slack muscles promote humeral component subluxation and joint instability.[7] Unsatisfactory postoperative performance is often due to muscle deficit rather than pain or implant breakage.[7,26] Stem holes for muscle and tuberosity reattachment sutures may be embedded in cancellous bone (Fig. 7). Ideal glenohumeral concentricity is difficult to achieve because of varied anatomy or preoperative insults, with minor HHR migration relative to the glenoid often due to positioning or motion.

Humeral implant instability with anterior, posterior, inferior, or most frequently superior migration (Fig. 11A–D) results from rotator cuff dysfunction, inadequate repair, or developmental deformity (Fig. 12).[27–29] Inferior instability or subsidence may be due to improper humeral length restoration or poor cement technique; anterior instability to excessive anteversion of either component, lost anterior deltoid function, or disrupted capsule or subscapularis; and posterior instability to excessive retroversion of either component.[11,28] Acute or late subluxation is most often due to reinjury (Fig. 11C).

Glenoid structural deformities may be due to developmentally altered inclinations (Fig. 12), to coracoid or glenoid surface attrition or erosion in inflammatory or severe osteoarthritis, or to recurrent dislocations. Marked joint space loss can lead to an "acetabularized glenoid" with medial joint line migration to the coracoid base and acromion, which substitute for anterosuperior glenoid deficiency.[30]

Surgical corrective techniques for optimal prosthetic function often relate to four problems:

1. Defective rotator cuff or deltoid muscle
2. Chronic humeral subluxation and dislocation
3. Deficient humeral or glenoid bone
4. Subacromial impingement related to cuff tear or contracture and acromioclavicular arthritis

Familiarity with corrective procedures such as acromial or clavicular resection, bone grafts for cuff or muscle reattachment, and augmentation or realignment of glenoid or humeral bones or components is essential for valid radiographic interpretation.[13,31]

The range of postoperative component orientations and their spatial relationships are also influenced by radiographic techniques. Patient positioning, tube angulation, centering, and slings or casts all affect roentgenographic criteria for optimum alignment. The significance of anatomic deviations should be judged in conjunction with orthopedic consultation and clinical findings. Multislice computed tomography (CT) or magnetic resonance imaging (MRI) with metal artifact reduction sequences, discussed elsewhere in this issue, may pre- and postoperatively aid documentation of abnormal alignment, version alteration, articular surface or bone stock deficiency, or other pathology potentially compromising fixation and function.

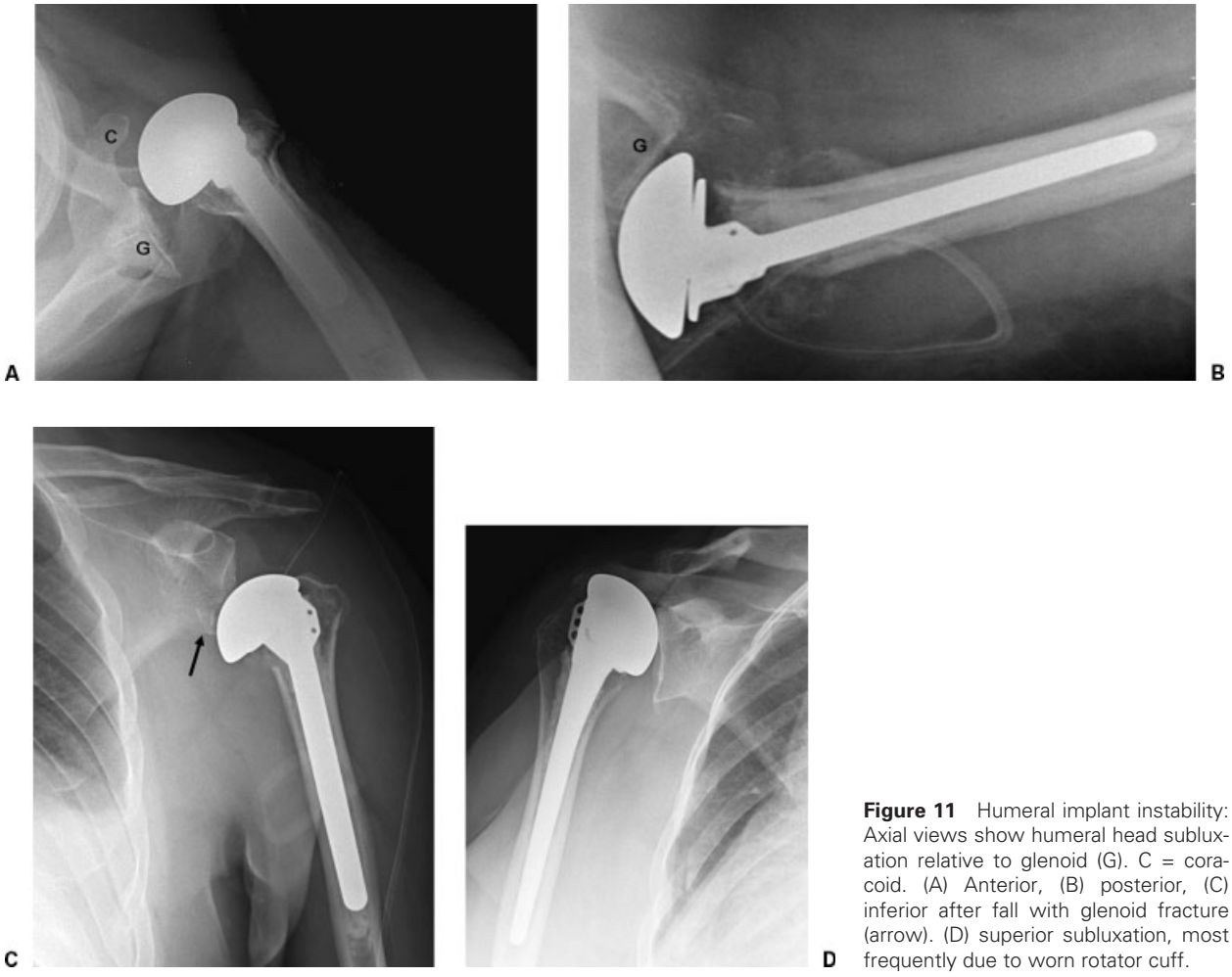


Figure 11 Humeral implant instability: Axial views show humeral head subluxation relative to glenoid (G). C = coracoid. (A) Anterior, (B) posterior, (C) inferior after fall with glenoid fracture (arrow). (D) superior subluxation, most frequently due to worn rotator cuff.

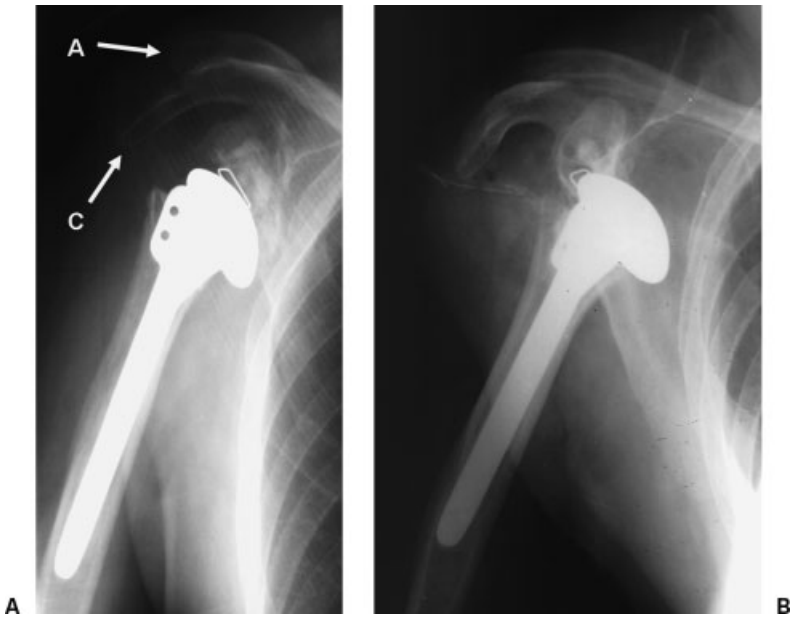


Figure 12 TSR of 61-year-old man with Erb's palsy, repeated humeral dislocations, secondary osteoarthrosis, and increasing pain related to muscular imbalance with partial deltoid paralysis but preserved external rotator function. At surgery a deformed mound replaced the glenoid articulation. (A) Postoperative anteroposterior (AP) view—a developmentally long coracoid (arrow c) partially supporting the glenoid prosthesis and a drooping acromial overhang (arrow a) are characteristic of Erb's palsy. The small humerus, fitted with a short prosthetic head and a small polyethylene keel-backed glenoid rather than a standard-sized component, was cemented into the coracoid base and scapular body and reinforced with bone graft. (B) Anterior humeral displacement 1 month after surgery. Developmental anatomic deformity rendered prosthesis susceptible to dislocation. After reduction, gradual removal of a supporting cast aided bone graft healing. Graduated exercise improved functional position, mobility, and pain.

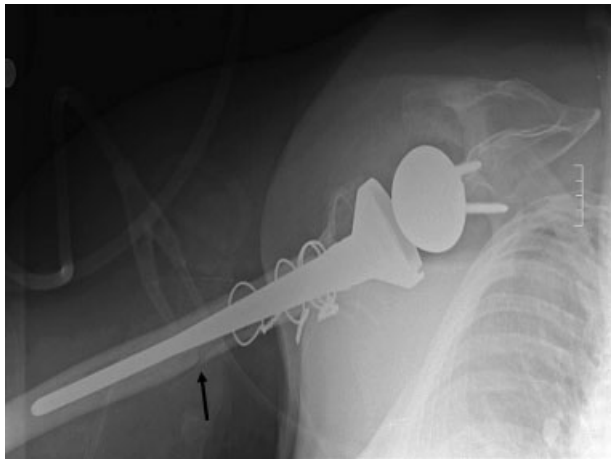


Figure 13 Fractured humeral shaft (arrow) during revision of failed TSR replaced by reverse ball and socket semiconstrained component. Fracture secured with cable.

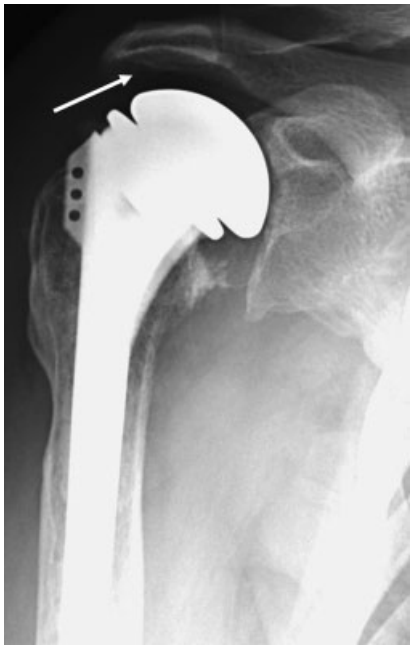


Figure 14 Hemiarthroplasty R shoulder with increasing superior humeral head migration due to massive cuff degeneration overpowered by deltoid. Inferior acromial concavity (arrow) due to chronic mechanical attrition by humeral head.

Complications Complications may be due to selection of patients, individual pathology, prosthetic design, or rehabilitation stress. Surgical or material causes include poor cementing technique, component failure, malalignment or migration, bone fracture, tuberosity avulsion or nonunion, heterotopic ossification, nerve injury, periprosthetic aseptic loosening, or infection.

Humeral Component Bending or Breaking This is uncommon with newer prosthetic materials. Native humeral shaft fractures occurred in 1.6% of 453 cases.[31,32]

Periprosthetic Fractures These are relatively rare but increasing because of the large numbers of arthroplasties inserted and revised, in part related to longer life expectancy. Humeral shaft or scapular stress fractures may occur during revision but may be spontaneous or posttraumatic (Fig. 13). Incidences vary with age of the patient, medication, osteoporosis, rheumatoid arthritis, or tumor; ranged from 1.6% in 436 cases to 2.4% in 252 cases; and have been categorized by site and anatomy.[32–35] One series[33] separated tuberosity fractures (A) from those about the stem (B); another[34] into three patterns with the periprosthetic tip as a focus. Fractures about the tip in A extended proximally; those in B had minimal proximal or distal extension. C, with shaft fractures distal to the tip, were best treated nonoperatively. In A or B the need for surgery varied, particularly with loose prostheses.[35]

Humeral Component Subluxations or Dislocations In TSRs these ranged from 1 to 7% (average $< 1\%$) in one series, 1 to 18% in another, with an overall incidence of 1 to 24%.[11,31,36] They frequently occur in the first few postoperative weeks. The commonest cause of superior migration is a deficient or injured rotator cuff, noted in 22 to 53% of cases.[14,27,31,37–42] Rheumatoid arthritis was causative in 79% of 188 TSRs and 10% with osteoarthritis.[8,27,43–46] Cuff degeneration may progress for years after arthroplasty with increasing superior HHR migration as the deltoid overpowers a dysfunctional cuff[11,28] (Fig. 14). Because large ball heads impinged on the acromion at extremes of motion, Neer replaced "end-stage bump" with joint dislocation using smaller heads to disassociate from glenoid components if specific torque forces were exceeded (Fig. 15A). However, each dislocation, as in normal shoulders, increases the likelihood of redislocation and debris accumulation.[7,8] Modular TSR designs allowing flexible selection of glenoid and head and neck dimensions decrease soft tissue tension and facilitate greater tuberosity repair with component selection facilitated by variously sized provisional templates[12,37–42] (Figs. 15B–D, 16)

Humeral Component Loosening Humeral component loosening that is symptomatic or clinically significant is relatively rare, ranging from 1 to 7% in unconstrained and a little higher in constrained TSRs.[31,36,46] Instability ranges from 1 to 2%[11] to 0 to 22%[28] and is less frequent in shoulder (Fig. 17) than knee or hip arthroplasties because shoulders are not weight bearing, the humerus is not impact loaded or rotationally strained, and forces across shoulder joints exert minimal bone-cement interface stress.[40–42,47,48]

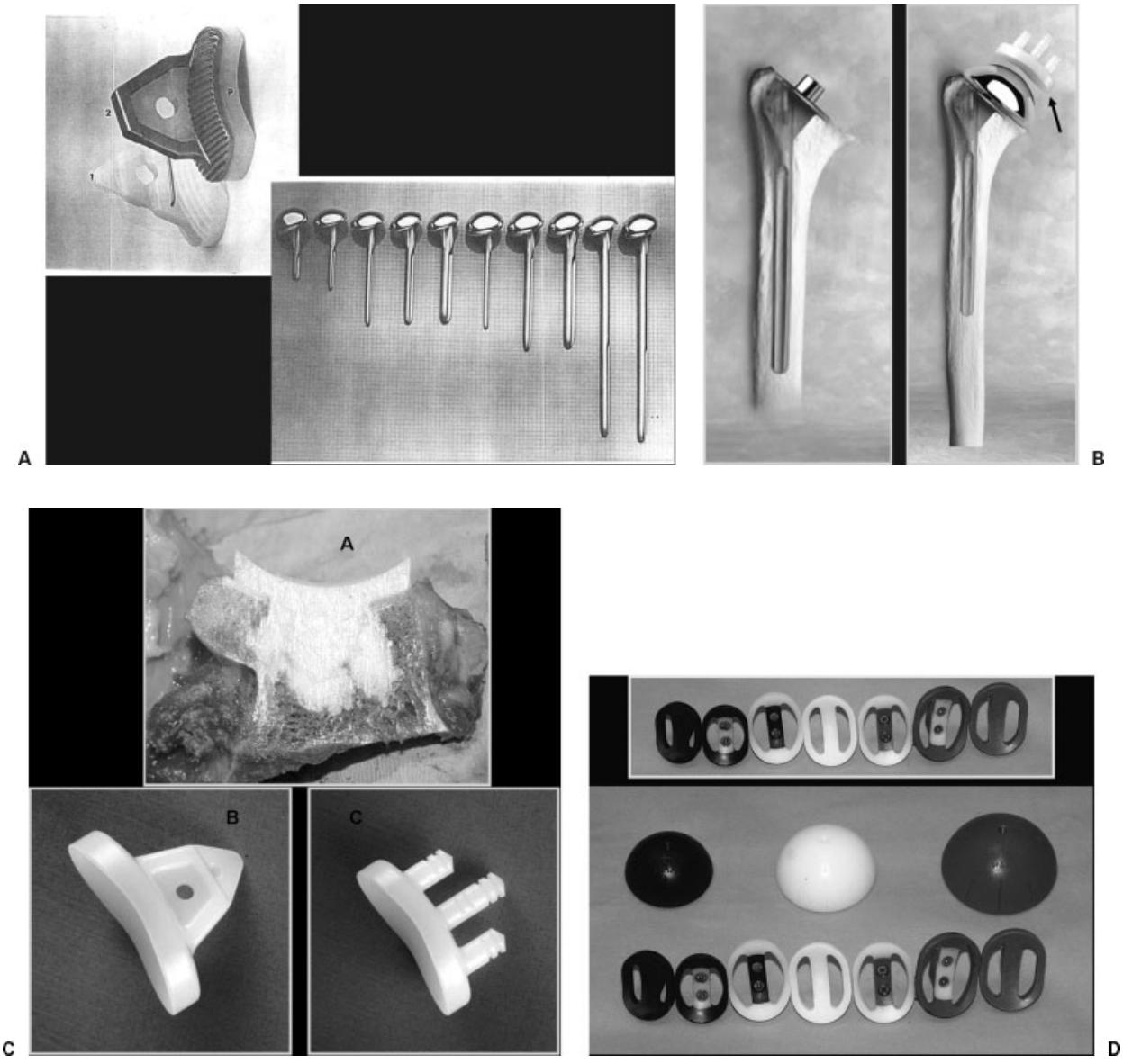


Figure 15 (A) Neer unconstrained TSR components with varying stem lengths, head diameters, and fixed head/stem angles: short heads used for small patients and repair of large cuff defects and shorter stems for humeral deformities (i.e., juvenile rheumatoid arthritis). Glenoid components had polyethelene keel bases with metal marker (1) or metal keels (2) with metal-backed polyethelene surfaces (P). (B) Bigliani/Flatow modular humeral components allow flexible selection of head, neck, and glenoid components. Note polyethylene pegged-based glenoid (arrow). (C) Glenoid components: a, pegged-cemented in cadaver section; b, keeled; c, pegged high-density polyethylene. (D) Variously sized, color-coded humeral head and glenoid templates facilitate intraoperative tailoring of components to individual anatomy.

Glenoid Component Loosening Glenoid component loosening is more common. The scapula's small volume, poor bone stock in those most in need of TSR, and operative excavation contribute to glenoid component vulnerability, particularly in constrained prostheses that directly transmit joint forces to bone-cement interfaces.[49–53] A review of 1459 TSRs from 29 series found 2% of glenoid components clinically loose but 30 to 90% considered "radiographically loose" at 3 years.[36] Longer follow-up found glenoid component lucencies in 30 to 90% of 89 TSRs increased 5 to 17 years after surgery.[54] Bone-cement interface lucencies noted in 75 (84%) glenoid components and 39 (44%) reported with "definite radiographic loosening" had a subsequent fourfold increase of osteolyses at more than one site, with pain relief decreasing in 67 of 73 (91%) to 72 of 89 (81%) cases.

Glenoid Component Instability This may be detected radiographically by its medial migration into the scapula, altered vertical tilt, medial displacement of its superior segment, and superiorly displaced articular surfaces. Metal glenoid backs enhance detection of their migration, settling, and articular surface inclination.

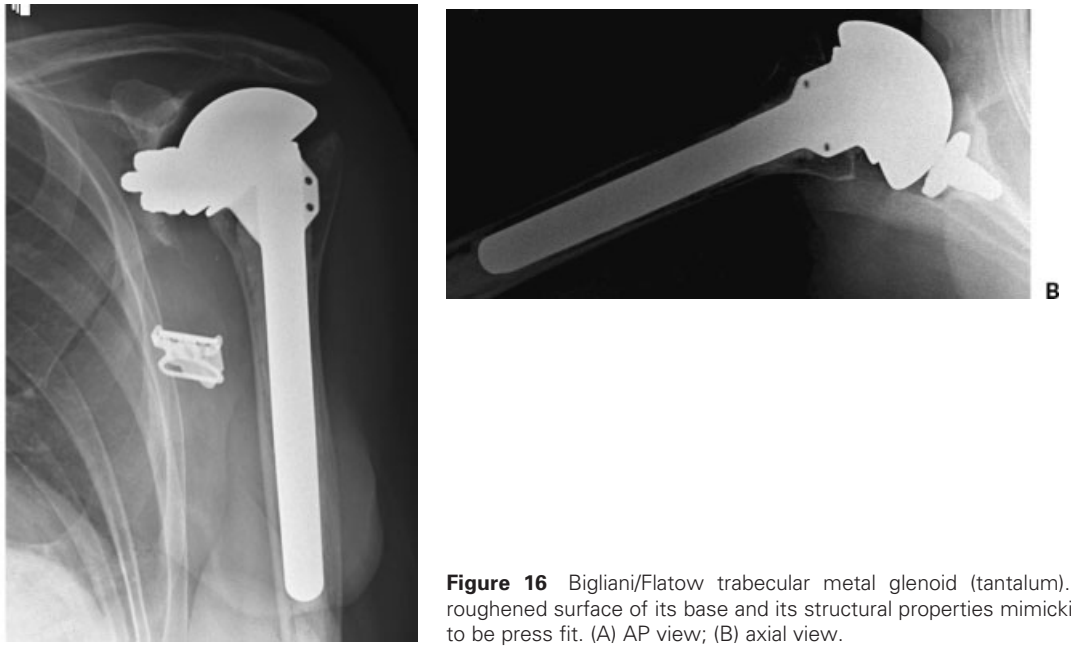


Figure 16 Bigliani/Flatow trabecular metal glenoid (tantalum). The irregularly roughened surface of its base and its structural properties mimicking bone allow it to be press fit. (A) AP view; (B) axial view.

Relatively radiolucent polyethylene components may contain subarticular metal markers, but barium-impregnated PMM bases are more easily seen (Fig. 18A–D). Displaced polyethylene cups are also difficult to detect on radiographs or CT without arthrography. Asymmetric joint narrowing related to worn metal backs or polyethylene glenoid surfaces also occurs (Fig. 18E).

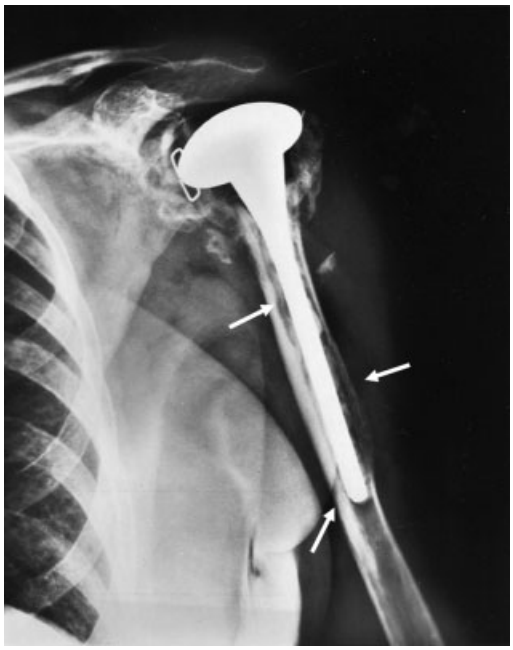
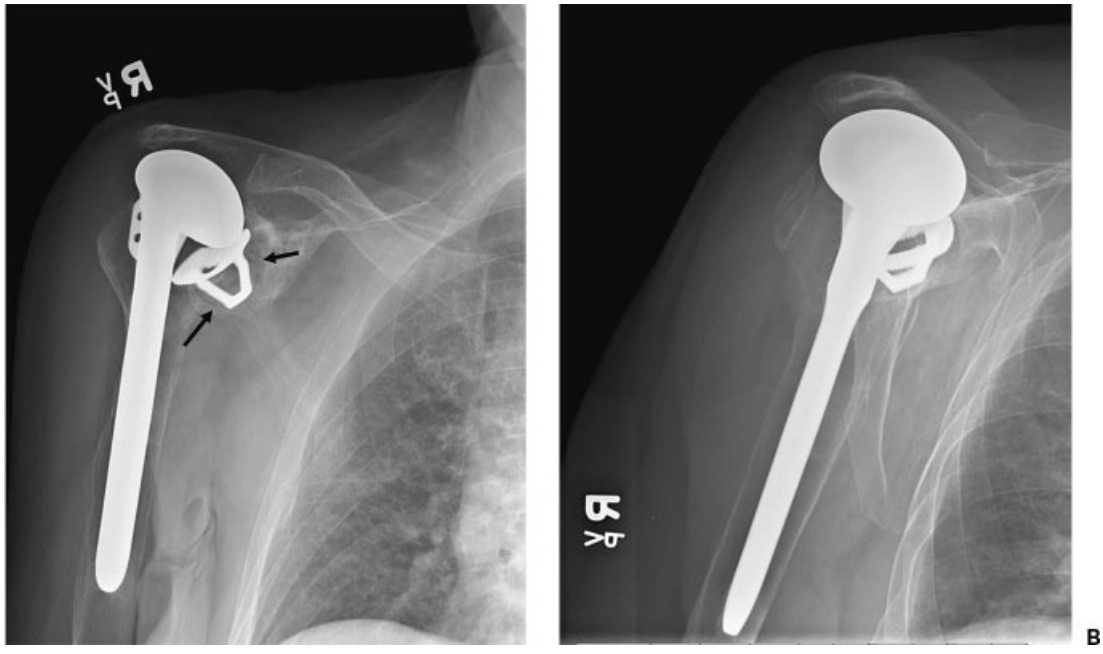


Figure 17 Loose humeral hemiarthroplasty. AP view—linear and focally irregular methylmethacrylate bone/-interface lucencies about humeral stem (white arrows) increased 2 ½ years after surgery. Cemented glenoid was unchanged (black arrow). Note ectopic bone.

Interface radiolucencies are commoner about glenoid components but not as easily seen as those about humeral components. They occur in 59% younger than 50 years[15] compared with 44% in older age groups[54] and do not always imply clinical loosening, particularly immediately after surgery.[53] Glenoid component failure may also be due to faulty cementing or cement fracture as evidenced by intra- and periarticular fragments. Despite metal backs and more ample keel and peg fixation, they remain problematic, with recent improvement credited to refined surgical preparation, better cementing techniques, and bone fixation rather than design alterations.[3,54–57]

Other complications involve greater tuberosity osteotomy healing. Complete bone union may occur at 6 months. Persistent osteotomy lucency may be due to solid fibrous union with the tuberosity unchanged in position on comparable poststress films. Early mineral deposition, usually not seen on radiographs, may nonspecifically concentrate radionuclide on bone scans. Radiographically evident, nonprogressive mild ectopic ossification, noted within 6 to 12 months in 24 to 50% of shoulder arthroplasties, usually corresponds to no clinical disability (Fig. 17), but extensive deposits may limit motion in 2 to 5% of TSRs. In 1 to 2%, nerve injury is most often due to scarring from prior surgery or trauma.[31]

Radiolucencies about TSRs may stabilize with time, but their ultimate behavior is unpredictable. They may be asymptomatic with no clinical implications or either asymptomatic or symptomatic when actually loose on longer follow-up. Symptomatic component loosening has accounted for one third of TSR complications, with most followed for relatively short periods.[11,53]



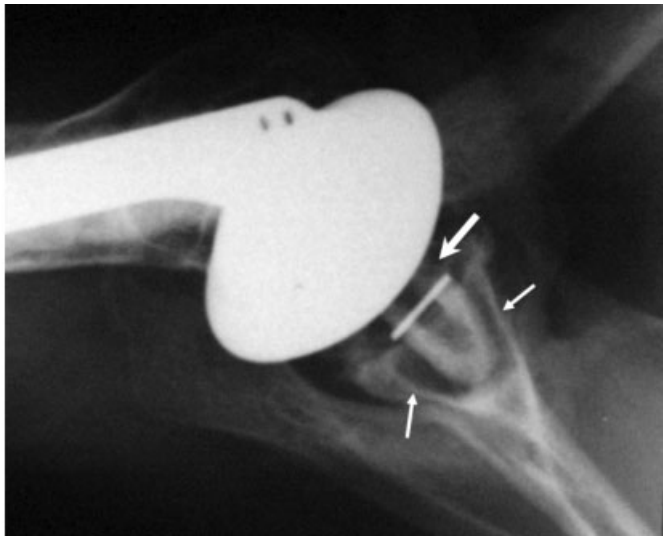
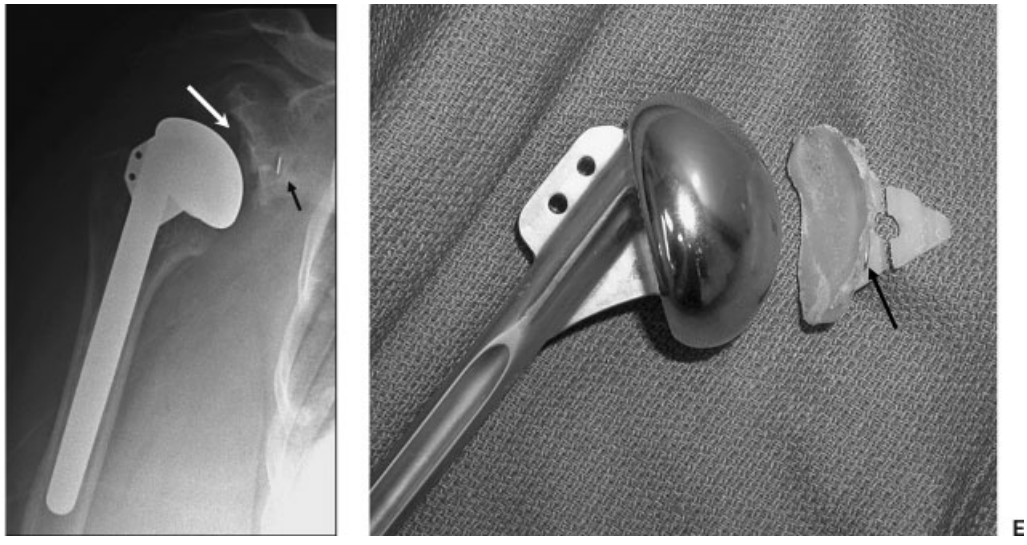


Figure 18 Glenoid component loosening and migration. (A) Anterior and (B) tangential scapular views. Inferior migration of metal-backed glenoid. Its radiolucent borders (arrows) and superiorly tilted articular surface are compatible with loosening. (C) Axial view of cemented polyethylene glenoid component with subarticular metal marker has a large, circumferential lucency (small arrows) with partial intra-articular displacement (large arrow). (D) Abnormal orientation of metal marker in polyethylene glenoid component base, intra-articular displacement, and lucencies bordering barium-impregnated PMM cement aid in establishing loosening. (E) Surgical specimen—failed worn polyethelene component had radiographic evidence of neighboring osteolysis, surgical evidence of loosening, and histopathology of debris disease. Note embedded metal marker (arrow) and fractured keel.



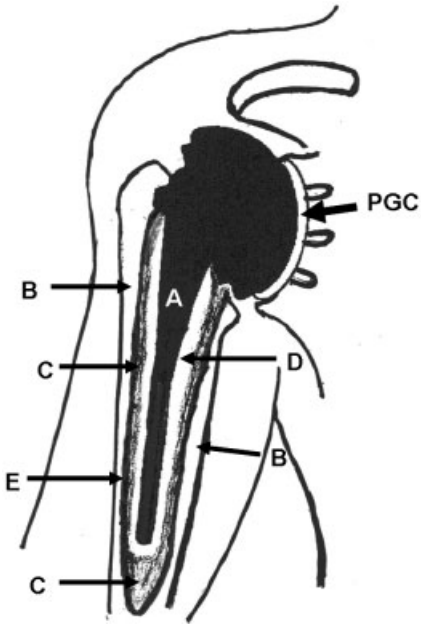


Figure 19 Interface lucencies. Stable—regular, thin < 2 mm ''membrane'' due to heat necrosis or tumor necrosis factor seen immediately or within 6 months after surgery. Unstable— irregular, focal or diffuse lucency, increasing > 2 mm due to infection, trauma, debris related to mechanical friction, metal or PMM sensitivity. (A) Metal implant stem; (B) bone; (C) gray zone, PMM) cement; (D) white zone, metal-PMM interface; (E) black line, bone-PMM interface.

Lucencies may be focal or completely marginate prosthetic or bone-cement interfaces. Smooth, narrow lucencies less than 2 mm, uniformly paralleling prosthetic contours, have appeared immediately or within 6 months after surgery in asymptomatic patients (Fig. 6). Their time of appearance, contour, and size in terms of length and width serve as radiographic criteria on which diagnoses of loosening or infection may be made provided that baseline radiographs are available for judging sequential development (Fig. 19). Etiologic factors include (1) surgical trauma with incomplete removal of interposed blood, cartilage, or soft tissue; (2) forces transmitted from prosthesis to bone creating stresses leading to bone resorption; (3) faulty cementing; (4) prosthetic motion during cement setting; (5) chemical damage from methacrylate and free radicals in cement; (6) fibrocartilaginous repair of tissue necrosis related to exothermic cement polymerization; and (7) micromotion related to thermal expansion and contraction of metal and cement.[47,58] Judging etiology is complicated because lucencies develop about uncemented and press-fit shoulder prostheses and immediately after surgery (Fig. 6). Generally, loosening of either component is suspected if lucencies extend, widen, or develop contour irregularities on serial films (Fig. 17), but similar changes may occur about infected prostheses (Fig. 20). A sclerotic line bordering a lucency may be an indirect late sign. Unless progressive, the latter is difficult to distinguish from cement condensation or fibro-osseous repair and has been noted earliest and most commonly along inferior glenoid keels.

Apparent altered alignments in clinically and surgically proven stable components may be due to differences in x-ray beam centering and patient positioning. True subluxation, angulation, or loosening may be accentuated by stress maneuvers and documented by radiographs, fluoroscopic spot, or video recordings. Baseline and sequential films are indispensable for distinguishing normal from loose or infected components (Figs. 17, 20). Infection, one of the gravest complications, decreased in incidence to less than 1% of HA and TSRs in 2 to 3% with constrained[31,36] and most recently in 0.7 to 0.9% of TSRs.[59,60] Among 49 infected shoulder prostheses, 1.9% occurred in primary and 4% after revision.[60] Those with acute infection were operated late despite diagnoses before the second month with reoperation further delayed for 9 months. Subacute infection was diagnosed within 2.9 to 11.8 months and more common chronic infection after 12 to 43 months, again with additional delays to reoperation.[60]

Predisposing factors continue to be primary or revision shoulder surgery (Fig. 20); rheumatoid arthritis; steroid therapy; obesity; hematogenous spread from bladder, respiratory, or dental infections; or any transient bacteremia seeding joints. Treatment may be difficult to evaluate. Neither antibiotics nor débridement was effective alone or in combination. Although revision after antibiotics has been advised, neither the antibiotic chosen nor the treatment length was optional.[60] Temporary intraoperatively prepared antibiotic-impregnated PMM molds have been used for local antibiotic dispersal, maintaining limb length, decreasing dead space, and preserving soft tissue planes, which facilitate revision (Fig. 20C). However, in 50% of the previously noted cases, the antibiotics chosen had no relation to their bone penetration properties or bacteria cultured.[60,61]

Signs and symptoms may be absent, minimal, or indistinguishable from those of loosening alone. In one series with infected prostheses, pain occurred in 100%, stiffness and limited motion in 40%, fever in 23%, and chills and night sweats in 10%.[59] Draining sinus tracts, local erythema, and effusion are important signs of infection (Fig. 20). Criteria used for prosthetic hip infections included sinuses or open wounds communicating with joints, hip pain with a systemic infection and purulent joint fluid, or positive results in at least three tests: erythrocyte sedimentation rate greater than 30 mm/ hour, positive preoperative aspiration, frozen section with more than 5 white blood cells per high-power field, and C-reactive protein levels greater than 10 mg/L.[62]

Routine radiographs often fail to distinguish infected from loose prostheses despite progressive interface

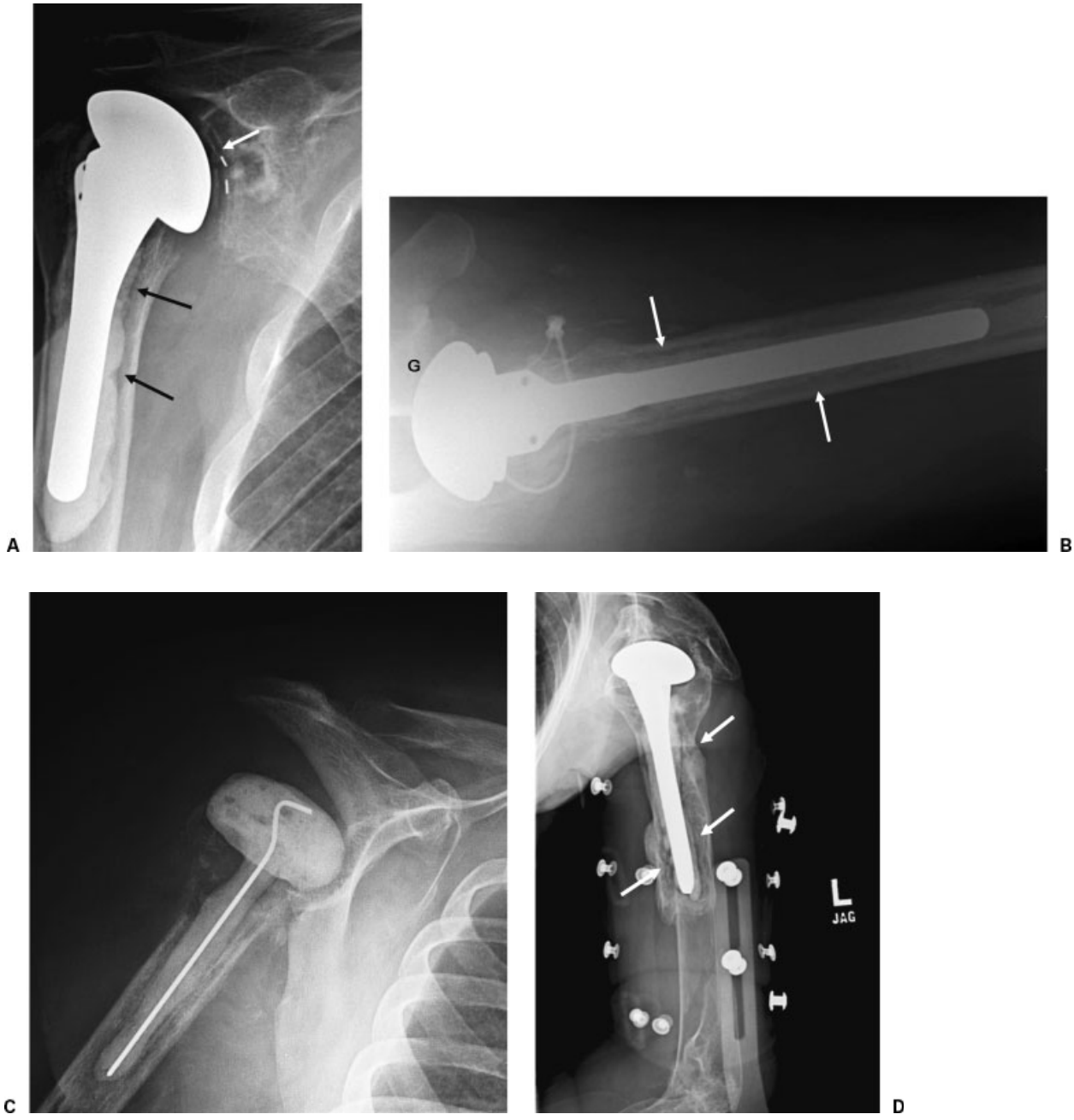


Figure 20 Infected components. (A) Patient 1, AP view R TSA. Irregular lucent interfaces about both components and in joint space prior to removal in patient with superficial focal swelling and erythema. (B) Patient 2, axial view. Irregularly contoured, progressively widened PMM-bone interface lucencies with posteriorly subluxed prosthetic head relative to glenoid (G). (C) Barium- and antibiotic-impregnated PMM mold also acts as ''spacer'' to preserve length for proper rotator cuff fixation during subsequent revision. Note eroded native glenoid surface. (D) Widened, irregular PMM-bone interface lucencies (inferior arrows), fractured cortices (superolateral and inferomedial arrows), with exuded cement, compatible with infection as well as loosening and fracture.

widening (Figs. 17–20). Antibiotics may also modify or obviate classic roentgenographic stigmata such as bone destruction, periosteal reaction, and sequestra.

OTHER IMAGING MODALITIES

Although effusions are nonspecific, ultrasound may guide joint aspiration. A positive culture is definitive evidence of infection. Negative culture, although not excluding infection, is associated with a greater probability of an aseptic prosthetic complication. CT air or nonbacteriocidal contrast arthrography can help detect defective or reinjured surgical cuff repairs or sinus tracts (Fig. 21), which may also be documented by MRI. Subtraction techniques were initially used in the hip to enhance detection of contrast material penetration adjacent to opaque cement. However, failure of interface contrast seepage does not exclude loosening because

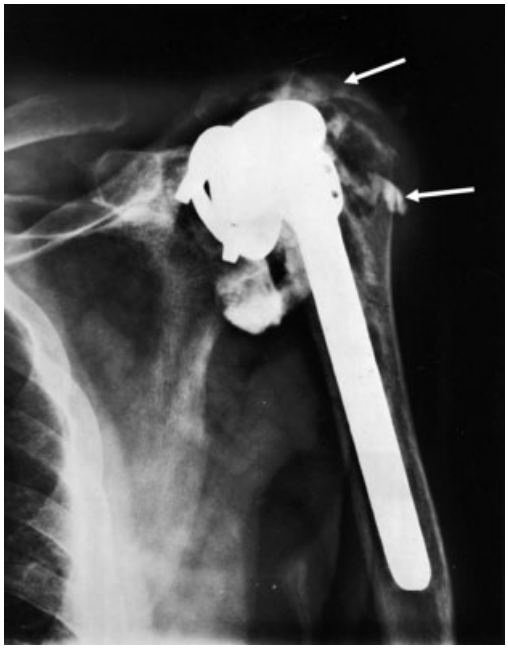


Figure 21 A 63-year-old male with painful shoulder 15 years after acromioclavicular acromioplasty and rotator cuff repair and 22 years after Neer semiconstrained TSR. Arthrogram with contrast material extravasated (arrows) through new posttraumatic cuff tear.

granulation or fibrous tissue may obstruct potential spaces. Conversely, contrast material that does penetrate an interface or pool in a localized area may be filling an innocuous fibrous-lined pocket or newly formed bursa. In addition to sinus tracts, rapid bone destruction generally suggests infection because bone resorption related to debris disease proceeds at a slower pace.

Technetium 99m–, gallium 67–, and indium 111–labeled white blood cell scans have been used to document infection.[63–65] Focal ^{99m}Tc uptake about distal femoral prostheses has been associated with loosening, with diffuse periprosthetic uptake favoring infection. Although highly sensitive, it is nonspecific. Radioactivity usually decreases gradually 6 to 9 months postoperatively, but increased uptake about distal femoral prostheses persisted in 11% in one series for 36 months.[63] The distribution of ^{67}Ga may parallel that of ^{99m}Tc in sterile loosening, but its hallmark in infection is patchy or diffuse intense concentration with spatial distribution incongruent with that of ^{99m}Tc. Computer correction of lesion-to-normal bone ratios for ^{67}Ga and ^{99m}Tc help establish gallium excess.[63] Specificity for osteomyelitis is enhanced by ^{99m}Tc triple-phase perfusion blood pool scans.[65]

Indium 111 is less sensitive to bone infection than Ga; it may be positive because of granulocytes in regional bone marrow and is more a time-consuming procedure. In Europe, ^{99m}Tc-labeled monoclonal anti-NCA-90 Fab fragments with an accuracy of 81% for detecting arthroplasty-associated infection have replaced it.[63–67] Other drawbacks include increased ^{67}Ga uptake up to a year after surgery, diminished uptake after antibiotics, simulating sterile reactions, and an affinity for bursas formed near orthopedic devices, which, if aseptically inflamed, may mimic soft tissue sepsis or abscess. Misinterpretation may be avoided by concurrent radiographs, which may explain increased but irrelevant nuclide uptake by identifying types and orientation of orthopedic devices, their radionuclide-deficient zones, fractures, osteotomies, or heterotopic bone, which may also be monitored if slated for excision. CT or CT air arthrography can further detect extraneous or interposed cement, bone, cartilage, or soft tissues.

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