

Heterotopic Ossification of the Deltoid Muscle After Arthroscopic Rotator Cuff Repair

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Abstract

Heterotopic ossification (HO), a well-known sequela of trauma, burns, head injury, and certain congenital or acquired metabolic conditions, has a predilection for the hip and the elbow. This disease has uncommonly been found after elective open shoulder surgery but extremely seldom after minimally invasive surgery. In our search of the peer-reviewed literature, we found no reports of HO after arthroscopic rotator cuff repair. The clinical importance of heterotopic bone after shoulder surgery remains unclear because of inconsistent definitions, varying correlations of symptom severity and radiographic findings, and lack of treatment efficacy data. Here we report a case of severely symptomatic HO after arthroscopic rotator cuff repair—successfully treated with excision of the heterotopic bone, interval release, and manipulation.

Heterotopic ossification (HO), a well-known sequela of trauma, burns, head injury, and certain congenital or acquired metabolic conditions, has a predilection for the hip and the elbow.¹ This disease may have a genetic, neurologic, or traumatic origin, with trauma including orthopedic surgery. Although the exact etiology of HO is unknown, several predisposing factors have been proposed: prostaglandin (specifically, prostaglandin E₂) activity, hypercalcemia, tissue hypoxia, alterations in sympathetic nerve activity, prolonged immobilization, and imbalances

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between parathyroid hormone activity and calcitonin. All these factors have been shown to contribute to HO.²

HO has uncommonly been found after elective open shoulder surgery but extremely seldom after minimally invasive surgery.³ In our search of the peer-reviewed literature, we found no reports of HO after arthroscopic rotator cuff (RC) repair. The clinical importance of heterotopic bone after shoulder surgery remains unclear because of inconsistent definitions, varying correlations of symptom severity and radiographic findings, and lack of treatment efficacy data.

Here we report a case of severely symptomatic HO after arthroscopic RC repair—successfully treated with excision of the heterotopic bone, interval release, and manipulation. The patient provided written informed consent for print and electronic publication of this case report.

CASE REPORT

The patient was a 65-year-old man with a 4-year history of left, nondominant shoulder pain. Over the preceding months, the pain had progressed to the point that it was markedly limiting his recreational activities. He reported experiencing loss of motion with weakness and managing this condition with physical therapy (PT), acupuncture, massage, and, over the past year, corticosteroid injections (3). There was no significant medical history, though the patient had undergone a successful arthroscopic right subacromial decompression (SAD). He did not smoke. Body mass index was 26.

The patient rated his pain on a verbal analog scale as 2/10 at rest and 7/10 with overhead activities. Cervical spine examination was unremarkable. There was focal



tenderness over the biceps tendon in the proximal bicipital groove. Other findings were active range of motion (ROM) of both shoulders with forward elevation (FE) to 160°, abduction to 160°, external

Figure 1. Initial anteroposterior radiograph shows no deltoid heterotopic ossification.

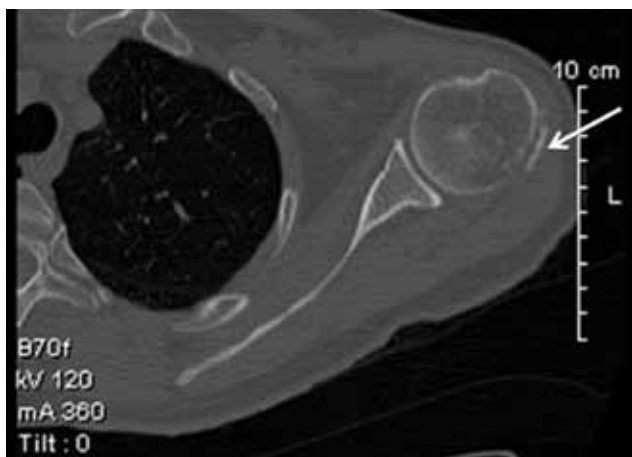


Figure 2. Axial view computed tomography of deltoid heterotopic ossification. Arrow identifies area of deltoid ossification.

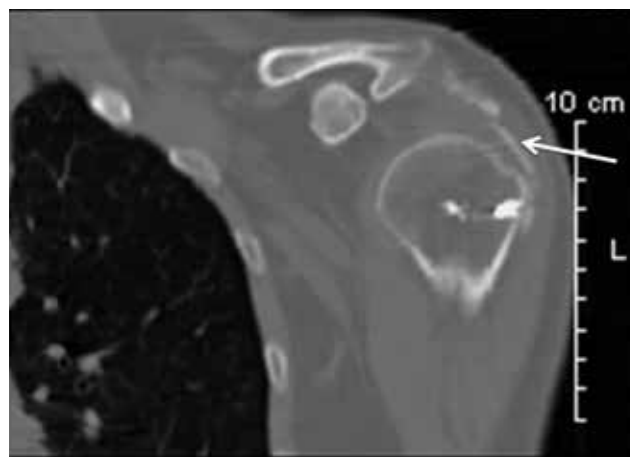


Figure 3. Anteroposterior computed tomography of heterotopic ossification. Arrow identifies area of deltoid ossification.

rotation (ER) of 90° at 0° of abduction, and internal rotation (IR) reach behind the back to the vertebral body of T8. Subscapularis function was normal with a negative lift-off test, while Speed's and O'Brien's tests for biceps dysfunction were positive. There was significant pain with resisted supination but no pain with cross-body adduction, negative anterior apprehension, load and shift, and sulcus signs. RC strength was 4/5 for all segments as assessed by standard manual muscle testing. Left shoulder radiographs showed significant acromioclavicular joint degenerative arthritis but were otherwise unremarkable (Figure 1). Magnetic resonance imaging (MRI) of the left shoulder was significant for RC tendinosis and degenerative changes of the biceps tendon. Given the chronic shoulder pain, physical examination findings, failure to progress with nonoperative intervention, and imaging findings, the surgical plan was set for a left shoulder arthroscopy with biceps tenodesis, acromioplasty, and SAD. Surgical evaluation of the integrity of the RC would dictate further surgical intervention.

Examination with the patient under interscalene anesthesia revealed ROM equivalent to that of the contralateral side and good stability. Arthroscopic evaluation with pump pressure maintained at 50 mm Hg demonstrated pristine cartilage on glenoid and humeral head, intact subscapularis, infraspinatus, and teres minor. The biceps tendon, however, was significantly degenerated. A full-thickness crescent-type supraspinatus tear was identified. The tear presented with fair tendon quality and had a footprint of approximately 20 mm. The subacromial space showed marked subacromial bursitis and a moderate-size anterior inferior osteophyte on the acromion. The supraspinatus tear had occurred during the delay between diagnostic MRI and arthroscopic evaluation, and the osteophyte had not been identifiable at the time of the MRI. The surgeon performed arthroscopic RC repair with 3 titanium anchors (Fastin RC; DePuy Mitek, Raynham, Mass), sutures passed in mattress fashion, and standard posterior and anterior portals.

For the acromioplasty and the SAD, a lateral portal was used approximately 3 cm below the acromion. The coracoacromial ligament was left intact, and a standard cutting block technique was used to resect the acromial osteophyte. During the arthroscopy, a type II superior labral anterior to posterior (SLAP) tear was found. This lesion was debrided back to the rim of the glenoid, and a subpectoral biceps tenodesis was performed as definitive management. The biceps tenodesis was completed through the lateral portal with a basket forceps and allowed to retract on the intertubercular groove. A separate incision was made through skin and subcutaneous tissue just under the pectoralis major tendon. The biceps was identified, and an anchor (Bioraptor Guide; Smith & Nephew, Andover, Mass) was inserted in the usual fashion. Sutures were passed into the biceps tendon with a single-pass technique at the musculotendinous juncture using a locking suture.

There was no significant intraoperative bleeding or postoperative hematoma associated with this procedure. A pain pump was placed, no drain was used, and the patient's arm was immobilized in a standard abduction sling (DonJoy, San Diego, Calif).

On postoperative day 3, the patient started PT with passive ROM as outlined by Millett and colleagues.⁴ He continued with passive ROM activities through the first 4 postoperative weeks. At 1-month follow-up, he had significant stiffness with FE at 90° and ER of 10° at 0° of abduction with capsular end feels. He was advised to be more aggressive with ROM. Over the next 8 postoperative weeks, he developed significant pain with ROM activities above 90° of FE or past 30° of ER. Three months after surgery, plain radiographs showed an ossification in the deltoid muscle. MRI showed an intact, well-healed RC. Computed tomography showed an ossification that seemed to be in the posterior surface of the middle deltoid with extension into the infraspinatus (Figures 2, 3). Concern was raised as to whether the patient had a history of diffuse idiopathic skeletal



Figure 4. One-year postoperative anteroposterior radiograph shows no heterotopic ossification. Four suture anchors are visible.

hyperostosis (DISH) or previous HO episodes. The patient denied previous issues. It was decided that he should undergo a course of radiation to minimize the likelihood of HO recurrence.^{5,6} The left shoulder was preoperatively treated with 750 cGy for HO prophylaxis. Data suggest that a single preoperative fraction dose of radiation is as beneficial as multiple postoperative fraction doses in minimizing HO recurrence.⁷⁻¹⁰

Approximately 3 months after the initial arthroscopic RC repair, the patient underwent open excision of heterotopic bone and an arthroscopic lysis of adhesions. The lysis of adhesions was performed to avoid traumatic manipulation after RC repair, as such manipulation may increase the chance of RC retearing.^{11,12} Examination with the patient under anesthesia revealed FE of only 80° and ER of 10° at 0° of abduction. When arthroscopic equipment was inserted into the subacromial space, thick scarring was noted between the RC and the acromion, and a marked amount of scar tissue was seen in the anterior, middle, and lateral gutters. The scar tissue was resected, divided, removed, and taken sharply off its intersection into the undersurface of the acromion. The heterotopic bone on the middle, deep surface of the deltoid did not involve any of the RC and was not associated with the scar tissue from the subacromial space. Given the size of the ossifications, open excision of the heterotopic bone was performed beginning with a saber-type incision in the shoulder. The deltoid was split in line with its previous lateral portal, and the calcifications were identified on the deep surface of the deltoid. These ossifications were removed with circumferential dissection, sent to pathology, and determined to be benign. The deltoid was meticulously reapproximated with specific attention given to repairing the posterior fascia without tension using Vicryl 0 sutures. The subcutaneous tissue, the more superficial tissues, and the saber incision were closed with Vicryl 0, Vicryl 3-0, and Monocryl 3-0 sutures, respectively. Portals were closed and sterile dressings applied.

Postoperative passive ROM demonstrated FE of 160° and ER of 90° at 0° of abduction. A Cryo/Cuff (Aircast, Austin, Tex) was applied, and the patient's arm was immobilized in a standard abduction sling (DonJoy). Four hours after surgery, the patient began postoperative rehabilitation with emphasis on passive ROM and active assisted ROM in all planes. He reported having none of the preoperative pain during the first therapy session. He did PT twice daily for the first 4 postoperative days, was discharged home on postoperative day 4, and began outpatient PT on postoperative day 5. His postoperative course was uneventful. By 5 months after surgery, his Shoulder Pain and Disability Index (SPADI)¹³ score was 17%, he was pain-free, and he had resumed full golf activity. Active ROM demonstrated FE of 150°, ER of 70° at 0° abduction, and IR reach behind the back to T12. Radiographs showed no HO recurrence. One and 2 years after surgery, SPADI score was 10%, and active ROM demonstrated FE of 150°, ER of 85° at 0° abduction, and IR reach behind the back to T10. Radiographs at 1 and 2 years demonstrated no recurrence of HO (Figure 4). Two years after surgery, left external RC strength as assessed by isometric handheld dynamometry was 6.4 kg compared with 6.7 kg on the right. Abduction strength in the plane of the scapula of the left shoulder was 4.77 kg compared with 5.7 kg on the right.

DISCUSSION

HO is abnormal formation of true bone in extraskeletal soft tissue. The etiology is incompletely understood but is thought to be multifactorial. Poorly understood systemic factors, usually relating to pathology of the central nervous system and genetic makeup, predispose to bone formation. Local factors, including decreased local oxygen tension,¹⁴ tissue trauma, and inappropriate expression of cellular signal proteins (eg, bone morphogenetic protein),^{15,16} synergize with the systemic factors to cause pluripotent mesenchymal stem cells to differentiate into active osteoblasts. Spindle cell proliferation and primitive osteoid formation initiate the histologic evolution of HO, typically over the first week of the inciting event. By week 2, cartilage and woven bone are present. Finally, mature trabecular bone forms 2 to 5 weeks after the inciting trauma. After approximately 6 weeks, a zonal phenomenon can be seen with immature, undifferentiated, central tissues and peripheral mature lamellar bone. Bone subsequently forms at triple the rate of normal bone, and it has twice the number of osteoclasts of age-matched bone.^{17,18}

Myositis ossificans classically was used to describe many diseases that result in abnormal bone formation. More recently, this general term fell into disfavor, as primary muscle inflammation is not a necessary condition for bone formation. In addition, ossification may occur in a variety of nonbone tissues, including fascia, tendons, and other mesenchymal soft tissues. HO should not be confused with another common shoulder

pathology, dystrophic calcification (calcific tendonitis or calciphylaxis). Mature bone trabeculae are formed in HO, whereas acellular amorphous calcium is deposited in dystrophic calcification.¹⁹ This physiologic difference has implications for methods and timing of treatment.

The most common acquired risk factors for HO are central nervous system injury, burns, and trauma with resultant hematoma formation. In the setting of nervous system injury with no surgical insult, HO in the shoulder occurs at a rate of 0.3% to 8%.^{18,20-22} Postoperative periarticular ossification about the shoulder has been clinically recognized as a complication since it was described by Smith²³ in 1835, but the definition of HO for the purposes of study is still heterogeneous. Multiple methods for grading HO have been used.²⁴ Apparently, the shoulder is predisposed to minor periosteal heterotopic bone formation after open surgery because of extensive soft-tissue coverage.²⁵ From the literature, the incidence may be estimated at 30% to 40%.^{26,27} Erggelet and colleagues²⁴ reported a 26.7% incidence after open acromioplasty and RC repair, with minor clinical impact. Risk factors for HO were prolonged surgical time and presence of osteoarthritis. Boehm and colleagues²⁸ found a 15% incidence of HO formation in patients who underwent total shoulder arthroplasty (TSA) or hemiarthroplasty—and no statistical difference between the procedures. Patients who underwent cuff-tear TSA, however, were at higher risk for HO formation (36%). Sperling and colleagues²⁹ found a 24% incidence of HO after TSA but did not note any preoperative risk factors or adverse effects on clinical outcomes. After conducting a systematic review, they found a range of 0% to 45% HO after TSA. With a critical review, it is evident that the trauma of open shoulder surgery may lead, relatively frequently, to clinically insignificant ossification about the shoulder.

Modern shoulder surgery has in large part evolved into arthroscopic techniques, which are mostly thought to be as efficacious as but less morbid than their open counterparts.³⁰⁻³⁴ It is believed that, for HO, constant irrigation with arthroscopic fluid dilutes osteoinductive marrow elements eluted from bone after SAD,^{35,36} lessening the risk for HO formation in muscle adjacent to the area of intramedullary penetration.^{37,38} Minimal soft-tissue trauma should theoretically limit hematoma formation and iatrogenic muscle damage as well, limiting another osteogenic stimulus. It could be argued that the initial RC repair in this case could have contributed to the patient's HO, but supporting data are minimal because of the rarity of the situation. The effect of accelerated rehabilitation after minimally invasive surgery remains unknown; at present, no determination can be made about the influence of rehabilitation on HO.²⁰

In multiple series,³⁹⁻⁴³ investigators have analyzed complications of arthroscopic shoulder surgery and found no evidence of postoperative HO. Berg and colleagues,⁴⁴ who first called attention to HO after

arthroscopic treatment, thought it was a source of residual impingement and surgical failure. They described their 10 patients as having ossicles that reformed in the area of previous bone resection, such as the coracoacromial ligament and the acromioclavicular joint. They believed this new periosteal bone formation may have been a result of a patient profile of hypertrophic pulmonary osteoarthropathy, obesity, diabetes mellitus, and smoking. In contrast to Berg and colleagues, who reported mild periosteal bone formation in the area of previous local resection, Boynton and Enders¹⁹ reported a severe case of HO after arthroscopic SAD that ultimately required fusion of the glenohumeral joint. This clinical presentation seems distinct from that described by Berg and colleagues in that there was immediate onset of extremely painful bone formation in a nonperiosteal location, the deltoid muscle belly. Our patient's case was similar—extreme pain presenting shortly after surgery, plus radiographic evidence that the deltoid muscle was immediately adjacent to the RC. These findings lead us to believe that this type of severe HO is a separate clinical entity related not to local bone resection but to preexisting systemic patient factors that may predispose to ossification after minor local trauma.

CONCLUSIONS

Our patient's case is significant in several ways. First, it provides additional evidence that HO may occur in the deltoid muscle even after the relatively mild trauma imparted by arthroscopic surgery. Second, a review of the literature suggests that 2 types of HO may arise after elective shoulder surgery—a clinically benign type, which occurs at muscle insertion and in periosteal locations after local surgical trauma, and a severe type in which bone forms in a diffuse periarticular pattern (the severe type is more painful, and its onset more rapid). Third, we demonstrate that the aggressive condition can be successfully treated with resection of the pathologic bone and surgical treatment of the ensuing soft-tissue contracture by conventional open interval release and manipulation, with adjuvant radiotherapy to the affected area. This treatment preserves patients' active function and mobility after elective surgery. Notably, passive ROM did not lead to further HO formation in our patient, who had been treated with radiation. External radiation therapy is a well-accepted treatment for prophylaxis of heterotopic bone. Given our patient's long-term intolerance to nonsteroidal anti-inflammatory drugs (NSAIDs) and the suboptimal results of using NSAIDs for HO prophylaxis, radiation therapy was selected. Although the data support administration up to 24 hours after surgery, immediate preoperative administration facilitates treatment and eliminates the need to transport a sedated patient for this treatment.^{5,45}

This report should heighten awareness of the postoperative factors of atypical limited ROM and pain, which hinder postoperative recovery and warrant radiologic investigation. When HO occurs, it may be treated with

resection and early motion after prophylaxis. Capsular ROM restrictions should also be addressed at the time of surgical intervention.

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

REFERENCES

- Hardy AG, Dickson JW. Pathological ossification in traumatic paraplegia. *J Bone Joint Surg Br.* 1963;45:76-87.
- Baird EO, Kang QK. Prophylaxis of heterotopic ossification—an updated review. *J Orthop Surg Res.* 2009;4:12.
- Kircher J, Martinek V, Mittelmeier W. Heterotopic ossification after minimally invasive rotator cuff repair. *Arthroscopy.* 2007;23(12):1359.e1-e3.
- Millett PJ, Wilcox RB 3rd, O'Holleran JD, Warner JJ. Rehabilitation of the rotator cuff: an evaluation-based approach. *J Am Acad Orthop Surg.* 2006;14(11):599-609.
- Blokhuis TJ, Frolke JP. Is radiation superior to indomethacin to prevent heterotopic ossification in acetabular fractures? A systematic review. *Clin Orthop.* 2009;(467):526-530.
- Vavken P, Castellani L, Sculco TP. Prophylaxis of heterotopic ossification of the hip: systematic review and meta-analysis. *Clin Orthop.* 2009;(467):3283-3289.
- Gregoritch SJ, Chadha M, Pelligrini VD, Rubin P, Kantorowitz DA. Randomized trial comparing preoperative versus postoperative irradiation for prevention of heterotopic ossification following prosthetic total hip replacement: preliminary results. *Int J Radiat Oncol Biol Phys.* 1994;30(1):55-62.
- Kantorowitz DA, Miller GJ, Ferrara JA, Ibbott GS, Fisher R, Ahrens CR. Preoperative versus postoperative irradiation in the prophylaxis of heterotopic bone formation in rats. *Int J Radiat Oncol Biol Phys.* 1990;19(6):1431-1438.
- Kantorowitz DA, Muff NS. Preoperative vs. postoperative radiation prophylaxis of heterotopic ossification: a rural community hospital's experience. *Int J Radiat Oncol Biol Phys.* 1998;40:171-176.
- Seegenschmiedt MH, Martus P, Goldmann AR, Wölfel R, Keilholz L, Sauer R. Preoperative versus postoperative radiotherapy for prevention of heterotopic ossification (HO): first results of a randomized trial in high-risk patients. *Int J Radiat Oncol Biol Phys.* 1994;30(1):63-73.
- Cho NS, Rhee YG. Functional outcome of arthroscopic repair with concomitant manipulation in rotator cuff tears with stiff shoulder. *Am J Sports Med.* 2008;36:1323-1329.
- Hsu SL, Ko JY, Chen SH, Wu RW, Chou WY, Wang CJ. Surgical results in rotator cuff tears with shoulder stiffness. *J Formos Med Assoc.* 2007;106(6):452-461.
- Williams JW Jr, Holleman DR Jr, Simel DL. Measuring shoulder function with the Shoulder Pain and Disability Index. *J Rheumatol.* 1995;22:727-732.
- Leung FW, Williams AJ, Fan P. Indomethacin therapy for hypertrophic pulmonary osteoarthropathy in patients with bronchogenic carcinoma. *West J Med.* 1985;142(3):345-347.
- Kawaguchi H, Kurokawa T, Hoshino Y, Kawahara H, Ogata E, Matsumoto T. Immunohistochemical demonstration of bone morphogenetic protein-2 and transforming growth factor-beta in the ossification of the posterior longitudinal ligament of the cervical spine. *Spine.* 1992;17(3 suppl):S33-S36.
- Soren A, Waugh TR. The morphologic and pathogenetic bases of heterotopic bone formation in the extremities. *Zentralbl Allg Pathol.* 1983;127(1-2):91-96.
- Sawyer JR, Myers MA, Rosier RN, Puzas JE. Heterotopic ossification: clinical and cellular aspects. *Calcif Tissue Int.* 1991;49(3):208-215.
- Wharton GW, Morgan TH. Ankylosis in the paralyzed patient. *J Bone Joint Surg Am.* 1970;52:105-112.
- Boynton MD, Enders TJ. Severe heterotopic ossification after arthroscopic acromioplasty: a case report. *J Shoulder Elbow Surg.* 1999;8:495-497.
- Damanski M. Heterotopic ossification in paraplegia: a clinical study. *J Bone Joint Surg Br.* 1961;43:286.
- Garland DE, Blum CE, Waters RL. Periarticular heterotopic ossification in head-injured adults. Incidence and location. *J Bone Joint Surg Am.* 1980;62:1143-1146.
- Hoffer MM, Garrett A, Brink J, Pery J, Hale W, Nickel VL. The orthopaedic management of brain-injured children. *J Bone Joint Surg Am.* 1971;53(3):567-577.
- Smith JS. Pathological appearances of seven cases of injury of the shoulder joint, with remarks. *Am J Med.* 1835;16:219-224.
- Erggelet C, Eggensperger G, Steinwachs M, Lahm A, Reichelt A. Postoperative ossifications of the shoulder. Incidence and clinical impact. *Arch Orthop Trauma Surg.* 1999;119(3-4):168-170.
- Holz U, Kraner F, Weller S. Periarticular ossification following replacement of the hip-joint. Clinical and radiologic examination of 697 hip-joints [in German]. *Z Orthop Ihre Grenzgeb.* 1977;115(2):146-158.
- Brooker AF, Bowerman JW, Robinson RA, Riley LH Jr. Ectopic ossification following total hip replacement. Incidence and a method of classification. *J Bone Joint Surg Am.* 1973;55(8):1629-1632.
- Hartwig CH, Sell S, Küsswetter W. Periarticular ossification following cement-free and cement fixed total endoprosthesis implantation in the hip joint [in German]. *Z Orthop Ihre Grenzgeb.* 1989;127(3):296-301.
- Boehm TD, Wallace WA, Neumann L. Heterotopic ossification after primary shoulder arthroplasty. *J Shoulder Elbow Surg.* 2005;14(1):6-10.
- Sperling JW, Cofield RH, Rowland CM. Heterotopic ossification after total shoulder arthroplasty. *J Arthroplasty.* 2000;15(2):179-182.
- Bishop J, Klepps S, Lo IK, Bird J, Gladstone JN, Flatow EL. Cuff integrity after arthroscopic versus open rotator cuff repair: a prospective study. *J Shoulder Elbow Surg.* 2006;15(3):290-299.
- Buess E, Steuber KU, Waibl B. Open versus arthroscopic rotator cuff repair: a comparative view of 96 cases. *Arthroscopy.* 2005;21:597-604.
- Sauerbrey AM, Getz CL, Piancastelli M, Iannotti JP, Ramsey ML, Williams GR Jr. Arthroscopic versus mini-open rotator cuff repair: a comparison of clinical outcome. *Arthroscopy.* 2005;21(12):1415-1420.
- Severud EL, Ruotolo C, Abbott DD, Nottage WM. All-arthroscopic versus mini-open rotator cuff repair: a long-term retrospective outcome comparison. *Arthroscopy.* 2003;19(3):234-238.
- Verma NN, Dunn W, Adler RS, et al. All-arthroscopic versus mini-open rotator cuff repair: a retrospective review with minimum 2-year follow-up. *Arthroscopy.* 2006;22(6):587-594.
- Ellman H. Arthroscopic subacromial decompression: analysis of one- to three-year results. *Arthroscopy.* 1987;3:173-181.
- Ellman H, Kay SP. Arthroscopic subacromial decompression for chronic impingement. Two- to five-year results. *J Bone Joint Surg Br.* 1991;73:395-398.
- Brumback RJ, Wells JD, Lakatos R, Poka A, Bathon GH, Burgess AR. Heterotopic ossification about the hip after intramedullary nailing for fractures of the femur. *J Bone Joint Surg Am.* 1990;72(7):1067-1073.
- Marks PH, Paley D, Kellam JF. Heterotopic ossification around the hip with intramedullary nailing of the femur. *J Trauma.* 1988;28:1207-1213.
- Burns TP, Turba JE. Arthroscopic treatment of shoulder impingement in athletes. *Am J Sports Med.* 1992;20:13-16.
- Hawkins RJ, Chris T, Bokor D, Kiefer G. Failed anterior acromioplasty. A review of 51 cases. *Clin Orthop.* 1989;(243):106-111.
- Imhoff A, Ledermann T. Arthroscopic subacromial decompression with and without the Holmium:YAG-laser. A prospective comparative study. *Arthroscopy.* 1995;11:549-556.
- Kay SP, Ellman H, Harris E. Arthroscopic distal clavicle excision. Technique and early results. *Clin Orthop.* 1994;(301):181-184.
- Roye RP, Grana WA, Yates CK. Arthroscopic subacromial decompression: two- to seven-year follow-up. *Arthroscopy.* 1995;11:301-306.
- Berg EE, Ciullo JV, Oglesby JW. Failure of arthroscopic decompression by subacromial heterotopic ossification causing recurrent impingement. *Arthroscopy.* 1994;10:158-161.
- Seegenschmiedt MH, Keilholz L, Martus P, et al. Prevention of heterotopic ossification about the hip: final results of two randomized trials in 410 patients using either preoperative or postoperative radiation therapy. *Int J Radiat Oncol Biol Phys.* 1997;39(1):161-171.