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Complications of treatment of acromioclavicular and sternoclavicular joint injuries

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Injuries to the acromioclavicular (AC) joint are among the most common injuries to the shoulder girdle. Injuries to the sternoclavicular (SC) joint are far less common. Nonetheless the sequelae of these injuries and their treatment is not without the potential for significant, and in some cases, fatal complications. This article will review both the common and uncommon complications of AC and SC joint injuries.

Anatomy of the shoulder girdle complex

Clavicle

Among the first bones in the body to ossify, the clavicle begins to form through intramembranous ossification in the fifth week of gestation at two separate diaphyseal centers that fuse by day 45 of fetal life [1–3]. The clavicle increases in diameter through intramembranous ossification, and in length by endochondral ossification at the physes. The medial clavicular physis provides approximately 80% of longitudinal growth and is the last long bone physis to appear as it ossifies between age 18 and age 20. Ossification and fusion of the lateral clavicular physis to the shaft occurs by approximately age 19, and that of the medial physis occurs between age 22 and age 26 [4–6].

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The clavicle provides the only bony articulation between the thorax and upper extremity. Derived from the Latin clavis (key), the s-shaped clavicle has an anterior convexity medially and a posterior convexity laterally that provide a bony roof over the contents of the thoracic outlet (subclavian vessels and brachial plexus). The clavicle serves as the attachment site for the trapezius and deltoid muscles laterally, and the pectoralis major, subclavius, and sternocleidomastoid muscles medially. Clavicular range of motion occurs in multiple planes, with the predominant motion occurring at the sternoclavicular joint which allows for approximately 50° of rotation, 30° of elevation, and 35° of protraction and retraction (translation) in the anteroposterior plane [7–9].

Scapula

The scapula first appears during the fifth week of gestation as a cartilaginous anlagen at the level of the fifth and sixth cervical vertebrae. It continues to enlarge and, during the seventh gestational week, descends to its position over the lateral thorax [10]. Failure of this descent results in Sprengel's deformity [11,12]. Intramembranous ossification of the scapula's primary ossific nucleus is completed by birth. The coracoid has 2 to 3 ossification centers that first appear at 1 year of age, and a common epiphysis for the base of the coracoid and upper glenoid that appears by age 10 [10]. A variable ossification center may be present at the tip of the coracoid, which may resemble an avulsion fracture. The ossific nuclei of the coracoid all fuse by age 15 to age 16. Acromial ossification centers (2 to 5) appear at puberty and fuse by age 22, with failure of fusion resulting in an "os acromiale"[11,13].

Acromioclavicular joint anatomy

The AC joint is a diarthrodial joint containing a perforated fibrocartilaginous disk. Approximately 20° of rotation occurs at the AC joint [14]. Its articular surfaces are covered by hyaline cartilage, and its disk is incomplete in over 90% of the population [15]. Stabilization of the AC joint is provided by the acromioclavicular and coracoclavicular (CC) ligaments, with the latter comprised of the conoid and trapezoid ligaments. The coracoclavicular ligament complex is the primary stabilizer of the AC joint, acting as a vertical stabilizer, whereas the acromioclavicular ligaments act as the secondary stabilizer providing anteroposterior stability [16].

The AC ligaments surround the capsule on its four surfaces and insert on the clavicle approximately 1.5 cm from the joint with the larger superior AC ligament reinforced by the deltoid and trapezial fascia [15]. A sequential ligament-cutting study by Fukuda et al used load displacement testing of the AC joint to demonstrate that the AC ligaments act to resist 90% of anteroposterior translation and joint distraction with a greater contribution to joint constraint at smaller degrees of displacement. At large displacements, the more lateral trapezoid ligament of the coracoclavicular ligament complex resists approximately 75%

of AC joint compression, and, together with the more medial conoid ligament, the coracoclavicular ligaments resist greater than 60% of superior clavicular motion [15,17]. The coracoclavicular ligamentous attachments to the undersurface of the distal clavicle are stronger than its periosteum, and, in children, this frequently leads to superior displacement of the distal clavicle at the physis from periosteal disruption rather than ligamentous detachment and displacement of the AC joint [18]. In addition, fusion of the lateral clavicular physis at age 19 makes children and adolescents far more likely to sustain this "pseudodislocation" than an AC separation [6,15,19,20].

Sternoclavicular joint anatomy

The SC joint is an incongruous, diarthrodial saddle joint composed of the medial clavicle, sternum, and first rib. It is the only true articulation between the axial skeleton and the upper extremity by way of the clavicle [21]. The surfaces of the SC joint are covered with fibrocartilage and are highly incongruent. Osseous stability of the SC joint is among the lowest of the major joints in the body because less than half of the medial clavicle articulates with the superior angle of the sternum [21,22]. As a result, this incongruity requires stability from its surrounding ligamentous supports. A fibrocartilaginous disk provides cushioning and limited stability as it separates the joint into two compartments. This disk is a ligamentous structure that originates from the synchondral junction of the sternum and first rib, passes through the SC joint, and attaches to the posterior and superior portions of the medial clavicle [21,23,24]. DePalma has shown that approximately 6% of intra-articular disks are incomplete [14]. The disk acts primarily as a checkrein against medial displacement of the proximal clavicle and blends with the fibers of the capsular ligament anteriorly and posteriorly [21]. Elevation and depression occur between the clavicle and SC disk, whereas anteroposterior and rotatory motion occur between the disk and the manubrium [25]. Fusion of the SC joint limits shoulder girdle abduction to approximately 90°.

The anterosuperior and posterior aspects of the capsular ligament provide the primary support for the SC joint, with greater strength provided by the posterior component. Bearn demonstrated that the capsular ligament is the most important structure preventing superior displacement of the medial clavicle and inferior descent of the distal clavicle [26,27]. The interclavicular ligaments connect the superomedial aspects of the clavicles to the capsular ligament and the manubrium.

The costoclavicular (rhomboid) ligament running from the first rib to the medial clavicle consists of anterior and posterior fasciculae. The anterior fasciculus fibers are directed laterally and cross the medially directed posterior fasciculus fibers. Together, they provide stability during elevation and rotation of the medial clavicle [26,28]. The constellation of ligaments that stabilize the SC joint are pivotal for its 50° of clavicular rotation, 35° of elevation (pivot), and 35° of antero-posterior glide (translation).

Classification of acromioclavicular joint injury

The most common mechanism of injury to the AC joint is a direct force produced by a fall on the point of the shoulder with the arm in adduction. The acromion is driven medially and downward and an abrasion or laceration of the overlying skin is not uncommon. Bearn [26] demonstrated that the SC ligaments are the primary restraints to downward displacement of the distal clavicle. In addition, contraction of the trapezius muscle provides a second mechanism by which inferior clavicular displacement is resisted. The direct downward force results in a sequence of events that may ultimately lead to failure of suspension of the upper extremity by the clavicle, and downward displacement of the shoulder girdle. Such an AC joint injury is commonly called a "shoulder separation."

AC joint injuries were initially classified into three different types [29,30]; however, Rockwood expanded the classification to include six different types [31]. A type I injury consists exclusively of an AC ligament complex sprain without ligamentous disruption. Type II injuries involve disruption of the AC ligament complex and joint capsule, with up to 50% of relative vertical subluxation of the distal clavicle. With type III injuries, both the AC and CC ligaments are disrupted, resulting in a complete dislocation of the AC joint with superior clavicular displacement. Type IV, V, and VI injuries include the components of a type III but are subdivided based on position of the displaced clavicle. In a type IV injury, the distal clavicle is displaced posteriorly, often into the belly of the trapezius muscle (confirmed with an axillary radiograph). Type V injuries are defined by extreme superior displacement of the clavicle (between 100% and 300% of the clavicular width) and complete disruption of the trapezius and deltoid fascia from the distal clavicle. Finally, type VI injuries are characterized by inferior displacement of the clavicle to a subcoracoid or subacromial position [31,32].

Complications of treatment of acromioclavicular joint injuries

Complications of AC joint injuries may develop in patients treated with and without surgical intervention. Consideration of potential complications is important when contemplating management of AC injuries to provide optimal patient counseling regarding potential outcomes. Discussion of complications of AC joint injury treatment is best divided into nonoperative and operative. The former is the mainstay of treatment for acute type I and type II AC injuries. Treatment of type III AC injuries is dependent on both the injury severity and activity level of the patient. Type IV, V, and VI injuries are typically treated by surgical means.

Complications of nonoperative management

Nonoperative management of AC injuries typically consists of shoulder immobilization with a sling or a harness (eg, Kenny-Howard sling) that attempts to hold the lateral clavicle "reduced" with downward clavicular pressure and upward support of the limb. The initial goal is limitation of motion to allow soft-tissue healing followed by a rehabilitation program that emphasizes re-establishment of shoulder motion. Restriction of heavy lifting and unprotected contact sports is recommended for the first several weeks, with return to full unlimited activity in approximately 3 to 6 weeks as comfort allows [33]. Full range of shoulder motion and normal shoulder girdle and rotator cuff strength should be achieved before return to full athletic participation.

Skin or wound complications

Skin or wound complications may develop in patients treated with or without operative intervention. The abrasion or laceration of the skin frequently present over, or adjacent to, the AC joint may limit compliance or success of harness immobilization caused by downward pressure placed on the distal clavicle. Allman [34] reported a 20% incidence of harness treatment failure resulting from adjacent skin compromise. For this reason, use of Kenny-Howard type devices is no longer recommended.

Post-traumatic arthritis

The literature regarding long-term outcomes of nonoperative treatment for AC joint injuries is controversial. AC joint injuries may lead to residual instability, degenerative changes, pain, and disability [32,35,36]. Symptomatic and radiographic evidence of AC arthritis has been reported by Berkefeld [37] as a late sequela of types I and II injuries. This finding is supported by Cook and Heiner [38], who presented a review of AC joint injuries in which degenerative changes were found in up to 24% of patients. It would not appear that the degree of degenerative changes are definitively related to the type of injury because Cox [35] reported radiographic changes in 70% of patients after a type I injury and 75% after a type II injury. In addition, 36% of these patients were symptomatic after the type I injuries, and 48% were symptomatic after type II injuries [35]. Taft et al [39] reported post-traumatic arthritis developed in 43% of patients treated nonsurgically and 25% of patients treated surgically after AC dislocation. The authors noted, however, that the development of symptoms did not correlate directly with radiographic changes.

Symptomatic degenerative arthritis after an AC joint injury is initially managed conservatively with activity modification, nonsteroidal anti-inflammatory medications, and the judicious use of intra-articular corticosteroid injection. Diagnosis of the condition is made by history, physical exam, and radiography, and may be facilitated by diagnostic intra-articular injection of local anesthetic. Patients who do not respond to conservative therapy may benefit from distal clavicle excision for pain relief through an arthroscopic or open approach [40–43]. With type II or III injuries in which horizontal or vertical instability is commonly present, distal clavicle excision alone will frequently prove inadequate because the instability remains and abutment of the posterior clavicle on the scapular spine may result in persistent pain and disability [33]. In these situations, resection of the distal clavicle should be accompanied by an appropriate stabilization procedure in either the horizontal (type II injury) or vertical plane (type III injury).

After AC injuries, patients may develop activity-related pain, weakness, and persistent deformity, as well as joint stiffness from prolonged immobilization after the initial injury. Calcification and ossification of the ligamentous supports of the AC joint is common and has been reported to occur in injured coracoclavicular ligaments up to 40% of the time [44]. This finding has not been shown to affect outcomes, however [15,45,46]. Interesting, persistent stiffness tends not to develop as a consequence of the injury when the glenohumeral joint is not involved. For this reason, prolonged immobilization is not recommended after AC joint disruption.

Distal clavicular osteolysis

Posttraumatic distal clavicular osteolysis (DCO) is a recognized complication of AC joint injuries [47,48]. This condition primarily manifests as pain (particularly with arm abduction and flexion) and is frequently self-limited. Diagnosis is facilitated by a Zanca view (15° cephalic tilt and 50% decrease in penetrance) or a technetium bone scan. Osteolysis, osteopenia, osteophyte formation, and tapering of the distal clavicle may be seen radiographically (Figs. 1, 2). The etiology of posttraumatic DCO has not been definitively identified. Proposed mechanisms extrapolated, however, from study of both post-traumatic DCO and atraumatic DCO in athletes include bone resorption related to subchondral stress fractures, followed by altered regional blood flow, and a stress failure syndrome with increased osteoblastic activity [47,49,50]. Activity modification is the initial mainstay of treatment, with or without a course of anti-inflammatory medication. Reconstitution of the distal clavicle has been reported [32]. Patients with persistent

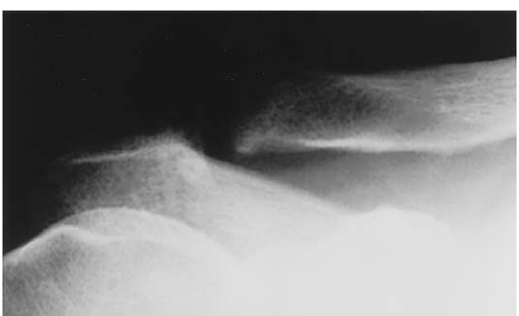

Fig. 1. Posttraumatic distal clavicle osteolysis occurring 6 months after type I AC separation. Note resorption of superior distal clavicle.

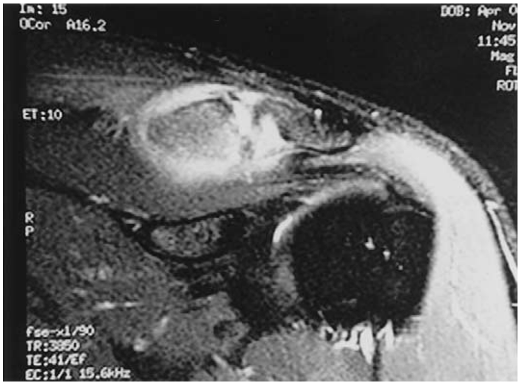

Fig. 2. MRI evidence of posttraumatic osteolysis of the distal clavicle 3 months after type I AC separation.

pain unresponsive to conservative management may be effectively treated with excision of the distal clavicle.

Neurovascular complications

Chronic AC joint instability may result in arm weakness, trapezial fatigue, and paresthesias consistent with a brachial plexopathy [33,44]. Shoulder girdle instability may result in a symptomatic traction neuropraxia of the brachial plexus. A case of brachial plexus neuropraxia presenting 8 years after a type III AC joint injury has been reported in the literature [51]. In addition, a review of 59 patients with brachial plexus injuries by Sturm and Perry included two cases of AC separation (though, in these two cases, the brachial plexus injury was probably caused by the initial injury, and this should therefore be considered simply an association) [52]. Vascular symptoms may be present, suggesting thoracic outlet syndrome; however, AC joint stabilization has been described as an effective treatment for these symptoms [32].

Complications of operative management

Surgical treatment of AC joint injuries has several potential complications. These complications are most effectively subdivided into those that develop in the preoperative and perioperative periods. Complications noted in the preoperative period are common to injuries treated both operatively and nonoperatively.

Preoperative complications

Accurate diagnosis of the injured AC joint requires a thorough assessment of possible associated injuries. Coracoid fractures may be easily missed in the

Fig. 3. Tenting of the skin with type V AC separation.

setting of an acute AC joint injury [44,53]. Barber reported an ipsilateral pulmonary contusion and contralateral pneumothorax in a patient with a type IV AC injury [54]. Finally, associated fractures of the distal or mid-clavicle and the acromion may be present and may influence the choice of treatment and risks for perioperative complications [33,55].

As with non-operative treatment, skin injury sustained in an AC joint injury may complicate surgical management. An abrasion or laceration at the site of a

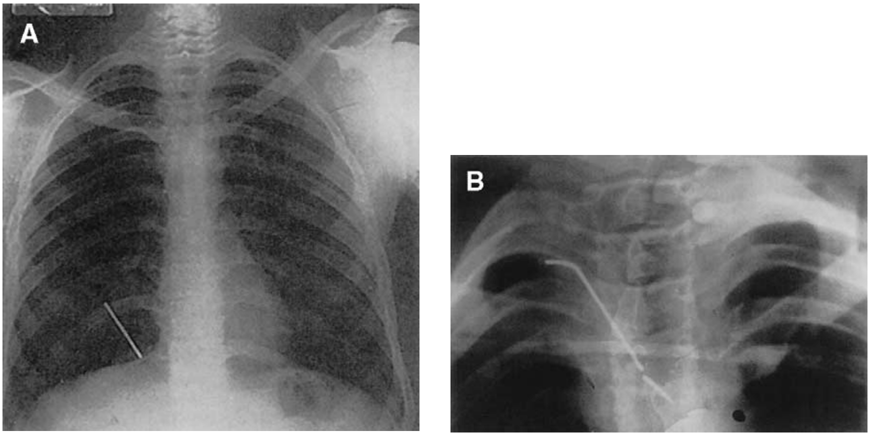

Fig. 4. Two examples of migration of smooth pins in the lung (*A*) and mediastinum (with broken tip in pericardial sac (*B*) used for AC joint fixation following AC separation. (*From* Galatz LM, Williams GR Jr. Acromioclavicular joint injuries. In: Bucholz RW, Heckman JD, editors. Rockwood and Green's fractures in adults. Philadelphia: JB Lippincott; 2001. p. 1209–44; with permission [Fig. 4A]; and Wirth MA, Rockwood Jr CA. Injuries to the sternoclavicular joint. In Heckman JD, Bucholz RW, editors. Rockwood and Green's fractures in adults. 5th edition. Philadelphia: Lippincott-Raven; 2001. p. 1245–92; with permission [Fig. 4B].)

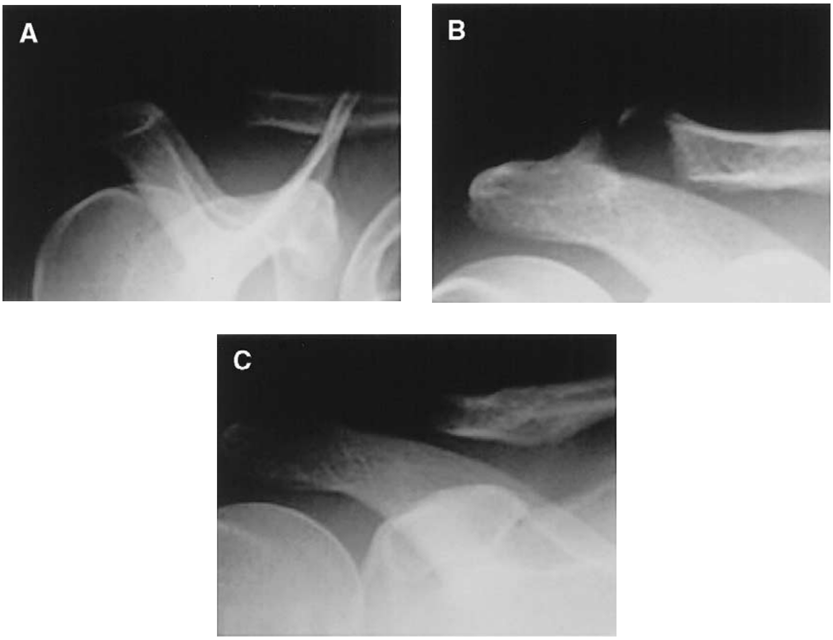

Fig. 5. (*A*) Apparent adequate arthroscopic distal clavicle resection for AC joint arthritis following AC separation. (*B*) At 6 months postop from index procedure, the patient presented with complaints of recurrent pain. Radiographs demonstrate calcification and ossification of the superior aspect of the distal clavicle resection site. (*C*) Radiographs s/p revision distal clavicle resection and resection of focus of ossification.

planned surgical incision must heal before surgery as skin breakdown and wound complication risk is significantly increased in these situations. Skin tenting is most common with type V injuries and may necessitate early intervention to limit soft tissue compromise [32] (Fig. 3).

Perioperative complications

Intraoperative and postoperative complications are related, in part, to the method of repair and reconstruction. Gentle handling of the soft tissues at surgery is critical to reduce the risk of skin infection and wound dehiscence. The most devastating complication following surgery of the shoulder, in general, is a potentially fatal great vessel injury related to pin migration. The migration of Steinman pins, Hagie pins, and Kirschner wires from the AC joint to the neck [56], spinal cord [57], and lung [58,59] has been reported in the literature. As a result, the current trend is to avoid transfixion pins around the shoulder whenever possible [32,44,60]. The use of hardware has also been associated with breakage and fixation failure [15,61] (Fig. 4) as well as erosion of the clavicle by non-absorbable suture or wire used for fixation [62–65]. Currently, the most common form of surgical treatment of a complete AC joint dislocation is a modified form of the Weaver-Dunn procedure which involves transfer of the acromial origin of the coracoacromial ligament to the lateral clavicle with supplemental fixation of the resected distal clavicle to the coracoid process [66]. Recalcification following apparent adequate AC joint resection can be a cause of pain requiring revision distal clavicle resection (Fig. 5).

Sternoclavicular joint injuries

Sternoclavicular joint injuries are rare and require an application of tremendous force to the shoulder. Cave [67] presented a series of 1603 shoulder girdle injuries of which only 3% were SC dislocations. The medial location of the SC joint protects it from low energy trauma to the shoulder. Indirect forces transmitted medially, however, from the shoulder and high energy direct forces may produce SC sprains, fractures, and potentially devastating retrosternal dislocations. The most common causes of SC joint injuries have been identified in a review of 150 traumatic SC dislocations in three series [68–70] conducted by Wirth and Rockwood [27]. These reviews found that 40% occurred as a result of vehicular accidents and approximately 21% resulted from sports-related activity. The remaining 39% were associated with miscellaneous trauma, falls, and industrial accidents.

The most common mechanism of injury to the SC joint is an indirect force [71,72]. This mechanism involves a lateral compressive force applied to the anterolateral or posterolateral shoulder producing anterior dislocation of the SC joint if the shoulder is rolled backward at the time of compression. In this situation, the underlying first rib acts as a fulcrum upon which the medial clavicle levers out anteriorly. Posterior dislocation of the SC joint occurs if the shoulder is rolled forward at the time of a lateral compressive force [27]. Posterior dislocation represents the most potentially devastating injury to the SC joint as the posteriorly displaced medial clavicle enters the mediastinum and threatens the great vessels, esophagus, and airway.

Classification of sternoclavicular joint injury

Sternoclavicular joint injuries may be classified by their etiology or more straightforwardly through an anatomic approach. This article will use the anatomic classification.

Anterior sternoclavicular joint dislocation

The vast majority of SC joint dislocations are anterior and consist of the medial clavicle being displaced anteriorly or anterosuperiorly relative to the anterior aspect of the manubrium. Approximately 98% of the SC dislocations in Cave's series [67] were anterior dislocations. This high percentage is consistent with data presented by Nettles and Linscheid [69] in which 57 of 60 (95%) patients with SC dislocation sustained anterior injuries. In fact, the world literature contains fewer than 125 cases of posterior SC dislocation [27,67,73,74]. Neurovascular injuries following an anterior SC injury are far less common than with posterior SC joint dislocation, but the clinical appearance and exam may be misleading, and a thorough evaluation is critical.

Treatment of acute and chronic anterior SC joint dislocation remains controversial. The rarity of SC dislocation has made it difficult to definitively formulate an optimal treatment plan based on validated outcome measures and an adequate length of follow-up. The majority of acute anterior dislocations are unstable following reduction. Though many operative procedures have been described to reconstruct or repair the joint, questions remain regarding the need for anatomic reduction and the reliability of outcomes. For example, one series of 10 patients treated nonoperatively for traumatic anterior SC dislocation with a mean follow-up of 5 years revealed good results in 70%, fair results in 20%, and poor results in 10% [75].

Some authors have advocated an attempt at closed reduction if the patient presents within 7 to 10 days of the injury [27,69,76]. As described, however, the instability of the injury frequently results in redisplacement and has led some authors to recommend definitive treatment with sling immobilization for approximately 6 weeks. When patients are older than 23 to 25 years of age, and fusion of the medial clavicular epiphysis has occurred, a persistent prominence at the anterior aspect of the SC joint may be expected; but this has been reported not to significantly interfere with activities of daily living, even in some cases involving heavy manual labor [69,73].

Open reduction and repair or reconstruction of an anterior SC dislocation is rarely indicated. Nevertheless, some authors have advocated operative treatment with a variety of techniques including soft-tissue reconstruction, medial clavicle resection, and hardware transfixion [69,77–86]. Eskola et al [77] advocated operative treatment of SC dislocations and presented good or fair results of SC joint reconstruction after failure of conservative management in eight patients treated with tendon or fascia lata grafts [78]. In addition, the authors argued against resection of the medial clavicle, as advocated by several authors [87,88], and presented four patients with poor results including pain and weakness of the operative extremity following medial clavicular resection [78]. It should be noted that these cases involved resection of approximately 2.5 centimeters of the medial clavicle without stabilization, which has subsequently been shown to adversely affect the results [89,90].

Wirth and Rockwood have advocated operative intervention for an anterior SC joint dislocation only when persistent symptoms of post-traumatic arthritis are present for 6 to 12 months, and if these symptoms can be completely relieved by injection of a local anesthetic into the SC joint. In these cases, they prefer a SC joint arthroplasty involving resection of 1.0 to 1.5 inches of the medial clavicle when the anterior capsule is incompetent, and ligamentous reconstruction with transfer of the intra-articular disk ligament into the osteotomized clavicle [91]. The operative treatment of anterior SC dislocation remains controversial. Operative management, like closed reduction, has a significant rate of redisplacement and potential complications. The clear consensus in the literature at present is to avoid metallic transfixion of the SC joint as discussed below under complications of operative management.

Posterior sternoclavicular joint dislocation

Though the vast majority of SC joint dislocations occur anteriorly, posterior SC dislocations can be truly devastating injuries [27,67]. Improvements in emergency medical care, technologic advances in trauma management and diagnostic imaging, and an increasing index of suspicion may be responsible for an increase in the number of posterior as opposed to anterior SC dislocation in two series. In 1988, Fery and Sommelet [79] reported on 49 SC dislocations, 8 (16%) of which were posterior. In 1996, Rockwood and Wirth [73] presented a series of 185 SC dislocations in which 50 patients (27%) sustained posterior dislocation.

The great vessels, trachea, esophagus, and brachial plexus immediately posterior to the SC joint make posterior dislocations life-threatening and require a thorough clinical and radiographic evaluation. Signs of airway compromise, venous congestion, arterial compromise, and symptomatic dyspnea or dysphagia are consistent with compression of vital structures [74,92–99] (Fig. 6). Of the 102 cases identified in the English-language literature by Ono et al [74] in 1998, approximately 30% included complications associated with the posteriorly displaced medial clavicle compressing the great vessels, trachea, esophagus, and brachial plexus. Prompt radiographic evaluation of the suspected posterior SC dislocation should include roentgenograms, CT scans of both medial clavicles to compare the injured and uninjured sides, and angiography if indicated [100]. Reports in the literature of great vessel laceration and compromise [97,101] support the attempt of closed reduction in a controlled environment coordinated with general trauma or thoracic surgical consultation.

The current recommendation for treatment of a posterior SC joint dislocation is closed reduction for all patients older than age 23 to 25 years presenting within 7 to 10 days of the injury [27,87,92]. After closed reduction of a posterior SC dislocation, the joint is usually stable [27], and postreduction care consists of

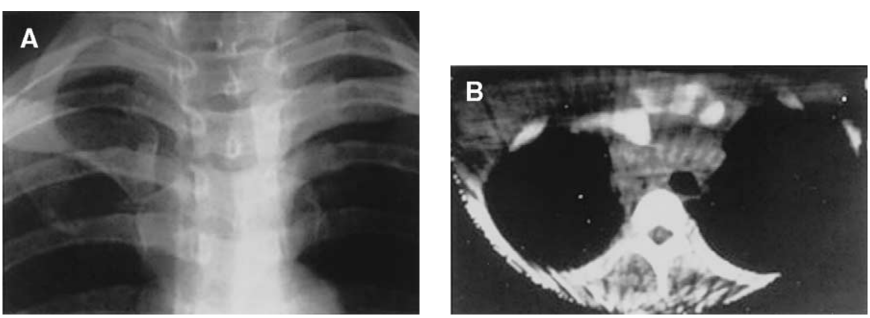

Fig. 6. (*A*,*B*) Plain radiographs and CT demonstrating posterior SC joint dislocation.

shoulder immobilization for 4 to 6 weeks with a figure-of-eight clavicle strap. Adhesion formation of the retrosternal structures supports the use of open reduction and stabilization for those patients who present after the initial 7- to 10-day period.

With unsuccessful closed reduction of an acute dislocation, chronic posterior dislocation, or instability, operative management is indicated. Several procedures have been described for open reduction and stabilization, including simple reduction and capsular repair, suture reconstruction of the capsule, use of subclavius or fascia lata tendons, osteotomy of the medial clavicle, and internal fixation across the joint [27,83,84,86,87,92,102–106]. Postoperatively, the shoulders are immobilized with a figure-of-eight clavicle strap for 4 to 6 weeks. Again, it must be emphasized that internal fixation with transfixion pins is no longer indicated because of the unacceptably high rate of potentially fatal complications from pin migration discussed below under complications of operative management.

Physeal injuries

Many presumed SC joint dislocations in patients under age 22 to 26 years are actually misdiagnosed medial clavicular physeal injuries [27,107]. The medial clavicular epiphysis begins to ossify approximately between age 18 and age 20, and it is the last physis of the body to close with fusion to the clavicular shaft at approximately age 22 to 26 years [5,108,109]. The SC ligaments primarily insert on the epiphysis and leave the physis unprotected outside the capsule. Concern for complications from compression of the vital structures posterior to the SC joint has led some authors to recommend operative management of these injuries [110]. Others feel the potential for remodeling and healing support-expectant management when there is no evidence of mediastinal structure compression, and when the patient presents more than 10 days beyond the period of injury [18,27,111]. Closed reduction of an acute posterior physeal injury is advocated when the patient presents within 10 days of injury [27]. Persistent deformity of the medial clavicle can occur as the result of medial physeal injury (Figs. 6 and 7).

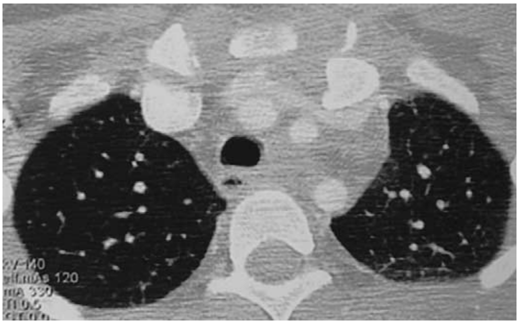

Fig. 7. Medial clavicular physeal injury with posterior displacement of clavicle. Note small crescent-shaped fragment of medial clavicular epiphysis.

Complications of nonoperative management

The primary complications of nonoperative management of anterior dislocations are cosmetic deformity, and late post-traumatic degenerative changes [89,112,113], as well as persistent pain, weakness, and instability with activity [75,78,102,105]. It is difficult to ascertain definitively whether these sequelae are exclusively caused by the nonoperative management of an SC dislocation, or to the injury itself. Recurrent dislocation may occur with the frequently unstable anterior SC joint dislocation after closed or open reduction. As stated previously, operative treatment of anterior SC dislocations is rarely indicated, and conservative, expectant management has been shown to be an effective treatment that may not interfere with activities of daily living [69,73,75,92,114]. Nonoperative management of unreduced or unstable posterior SC joint dislocations, however, may result in the aforementioned complications of great vessel compromise, myocardial conduction abnormalities, tracheal or esophageal injury, pulmonary injury, brachial plexus compression, dysphagia, dyspnea, and voice changes [27,68,73,74,89,113].

Complications of operative management

The most catastrophic complications of operative management of SC joint injuries, like that of AC joint injuries, are associated with the migration of transfixion pins. In 1990, Lyons and Rockwood [60] reported on the complications of pin migration in shoulder surgery. Of the 49 complications in 47 patients, 21 had transfixion pins placed for treatment of anterior SC joint dislocations. Seventeen pins (smooth pins, threaded Steinman pins, Kirschner wires bent at the skin, and Hagie pins) migrated to critical vascular structures including the heart, ascending aorta, pulmonary artery, and subclavian artery resulting in eight deaths. Six of these fatalities were sudden. The other 2 occurred within 3 months of surgery. An additional six patients suffered cardiac tamponade, requiring surgical intervention. Additional reports of Steinmann pin and Kirschner wire migration to the aorta, heart, innominate artery, pulmonary artery, lung, and spinal cord have been presented [27,88,115]. As a result of these devastating potential complications, the use of transfixion pins in SC joint stabilization is clearly contraindicated.

Infection is a recognized complication of any surgical procedure and may carry significant morbidity when it results after operative management of a SC injury. Infection of the SC joint may require multiple debridements to eradicate a need for resection of additional clavicle or the lateral aspect of the sternum, resulting in instability and perhaps leading to a disfiguring scar [89]. In addition, SC joint infection brings a risk for mediastinal spread and abscess formation [92].

Overzealous resection of the medial clavicle, or failure to stabilize it, with careful attention to the integrity of the capsular and costoclavicular ligaments, may result in recurrent pain and functional deficits related to limited shoulder mobility, fatigue, neurologic symptoms, and thoracic outlet syndrome [83,86,88,89]. Similarly, insufficient medial excision has been reported to cause pain from posterior spurring. Wirth and Rockwood [89] have offered methods to avoid this potential complication through resection of sufficient bone, removal of the proximal periosteum, use of bone wax at the osteotomy site, and release of the clavicular head of the sternocleidomastoid. Failure to recognize pathology involving degenerative changes at the inferior pole of the medial clavicle where it articulates with the upper angle of the sternum may also result in persistent symptoms.

Summary

Although common, AC joint injuries and their treatments are not benign. The injury itself and both nonsurgical and surgical treatments may result in complications yielding persistent pain, deformity, or dysfunction. Sternoclavicular joint injuries are far less common and are typically the result of higher energy trauma. As such, the associated complications may be more serious. Familiarity with the potential complications of these injuries can help the treating physician to develop strategies to minimize their incidence and sequelae.

References

[1] Abbott LC, Lucas DB. The function of the clavicle: its surgical significance. Ann Surg 1954; 140:583–99.
[2] Gardner E. The embryology of the clavicle. Clin Orth 1968;58:9–16.
[3] Ogata S, Uhthoff HK. The early development and ossification of the human clavicle: an embryologic study. Acta Orth Scan 1990;61:330–4.
[4] Webb PA, Suchey JM. Epiphyseal union of the anterior iliac crest and medial clavicle in a modern multiracial sample of American males and females. Am J Phys Anthropol 1985;68: 457–66.
[5] Ogden JA, Conlogue GJ, Bronson ML. Radiology of postnatal skeletal development. III. The clavicle. Skel Radiol 1979;4:196–203.
[6] Todd TW, D’Errico Jr J. The clavicular epiphyses. Am J Anat 1928;41:25–50.
[7] Abbott LC, Lucas DB. The function of the clavicle: its surgical significance. Ann Surg 1954; 140:583–99.
[8] Moseley HF. The clavicle: its anatomy and function. Clin Orth 1968;58:17–27.
[9] Inman VT, Saunders JB, Abbott LC. Observations on the function of the shoulder joint. JBJS 1944;26:1–30.
[10] Chung SMK, Nissenbaum MM. Congenital and developmental defects of the shoulder. Orth Clin North Am 1975;6:381–92.
[11] McClure JG, Raney RB. Anomalies of the scapula. Clin Orth 1975;110:22–31.
[12] Galpin RD, Birch JG. Congenital elevation of the scapula(Sprengel’s deformity). Orthopedics 1987;10:965–70.
[13] Liberson F. Os acromiale: a contested anomaly. JBJS 1937;19:683–9.
[14] DePalma AF. The role of the disks of the sternoclavicular and the acromioclavicular joints. Clin Orthop 1959;13:222–33.
[15] Nuber GW, Bowen MK. Disorders of the acromioclavicular joint: pathophysiology, diagnosis, and management. In: Ianotti JP, Williams GR, editors. Disorders of the shoulder: diagnosis and management. Philadelphia: Lippincott Williams & Wilkins; 1999. p. 739–62.

[16] Salter Jr. EG, Nasca RJ, Shelley BS. Anatomical observations on the acromioclavicular joint and supporting ligaments. Am J Sports Med 1987;15(3):199–206.
[17] Fukuda K, Craig EV, An KN, Cofield RH, Chao EY. Biomechanical study of the ligamentous system of the acromioclavicular joint. JBJS [Am] 1986;68(3):434–40.
[18] Kwon Y, Sarwark JF. Proximal humerus, scapula, and clavicle. In: Beaty JH, Kasser JR, editors. Rockwood and Wilkins' fractures in children. 5th edition. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 754–64.
[19] Falstie-Jensen S, Mikkelsen P. Pseudodislocation of the acromioclavicular joint. JBJS 1982;64: 368–9.
[20] Ogden J. Distal clavicular physeal injury. Clin Orthop 1984;188:68–73.
[21] Wirth MA, Rockwood Jr C. Disorders of the sternoclavicular joint: pathophysiology, diagnosis, and management. In: Ianotti JP, Williams GR, editors. Disorders of the shoulder: diagnosis and management. Philadelphia: Lippincott Williams & Wilkins; 1999. p. 765–813.
[22] Flatow EL. The biomechanics of the acromioclavicular, sternoclavicular, and scapulothoracic joints. In: Heckman JD, editor. Instructional course lectures 42. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1993. p. 237–45.
[23] Eskola A. Sternoclavicular dislocations: a plea for open treatment. Acta Orthop Scand 1986; 57:227–8.
[24] Eskola A, Vainionpaa S, Vastamki M, Slatis P, Rokkanen P. Operation of old sternoclavicular dislocation: results in 12 cases. J Bone Joint Surg 1989;71B:63–5.
[25] Zuckerman JD, Matsen III FA. Biomechanics of the shoulder. In: Nordin M, Frankel VH, editors. Basic biomechanics of the musculoskeletal system. 2nd edition. Philadelphia: Lea & Febiger; 1989. p. 225–47.
[26] Bearn JG. Direct observations on the function of the capsule of the sternoclavicular joint in the clavicular support. Anat 1967;101:159–70.
[27] Wirth MA, Rockwood Jr CA. Acute and chronic traumatic injuries of the sternoclavicular joint. JAAOS 1996;5:268–78.
[28] Cave AJE. The nature and morphology of the costoclavicular ligament. J Anat 1961;95:170–9.
[29] Tossy JD, Mead NC, Sigmond HM. Acromioclavicular separations: useful and practical classification for treatment. Clin Orthop 1963;28:111–9.
[30] Allman Jr FL. Fractures and ligamentous injuries of the clavicle and its articulation. JBJS 1967;49:774–84.
[31] Rockwood Jr CA, Young D. Disorders of the acromioclavicular joint. In: Rockwood Jr CA, Matsen FA, editors. The Shoulder. Philadelphia: WB Saunders; 1990. p. 413–76.
[32] Guttmann D, Paksima NE, Zuckerman JD. Complications of treatment of complete acromioclavicular joint dislocations. In: Price CT, editor. Instructional course lectures 49. Rosemont, IL: AAOS; 2000. p. 407–13.
[33] Galatz LM, Williams Jr GR. Acromioclavicular joint injuries. In: Bucholz RW, Heckman JD, editors. Rockwood and Green's fractures in adults. Philadelphia: JB Lippincott; 2001. p. 1209–44.
[34] Allman Jr FL. Fractures and ligamentous injuries of the clavicle and its articulation. JBJS 1967; 49:774–84.
[35] Cox JS. The fate of the acromioclavicular joint in athletic injuries. Am J Sports Med 1981;9: 50–3.
[36] Post M. Current concepts in the diagnosis and management of acromioclavicular dislocations. CORR 1985;200:234–47.
[37] Bergfeld JA, Andrish PT, Clancy WG. Evaluation of the acromioclavicular joint following first and second degree sprains. Am J Sports Med 1978;6:153–9.
[38] Cook DA, Heiner JP. Acromioclavicular joint injuries. Orthop Rev 1990;19:510–6.
[39] Taft TN, Wilson FC, Oglesby JW. Dislocation of the acromioclavicular joint. An end-result study. JBJS 1987;69A:1045–51.
[40] Mumford EB. Acromioclavicular dislocation: a new operative treatment. JBJS 1941;23: 799–802.

[41] Flatow EL, Duralde XA, Nicholson GP, Pollock RG, Bigliani LU. Arthroscopic resection of the distal clavicle with a superior approach. J Shoulder Elbow Surg 1995;4:41–50.
[42] Gartsmann GM. Arthroscopic resection of the acromioclavicular joint. Am J Sports Med 1993; 21:71–7.
[43] Snyder S, Banas M, Karzel R. The arthroscopic Mumford procedure: an analysis of results. Arthroscopy 1995;11:157–64.
[44] Nuber GW, Bowen MK. Acromioclavicular joint injuries and distal clavicle fractures. JAAOS 1997;5:11–8.
[45] Millbourn E. On injuries to the acromioclavicular joint: treatment and results. Acta Orthop Scand 1950;19:349–82.
[46] Hessman M, Gotzen L, Gehling H. Acromioclavicular reconstruction augmented with polydioxanonsulphate bands: surgical technique and results. Am J Sports Med 1995;23:552–6.
[47] Murphy OB, Bellamy R, Wheeler W, Brower TD. Post-traumatic osteolysis of the distal clavicle. Clin Orthop 1975;109:108–14.
[48] Madsen B. Osteolysis of the acromial end of the clavicle following trauma. Br J Radiol 1963; 36:822–8.
[49] Cahill B. Atraumatic osteolysis of the distal clavicle: a review. Sports Med 1992;13:214–22.
[50] Zawadsky M, Marra G, Wiater JM, Levine WN, Pollock RG, Flatow EL, et al. Osteolysis of the distal clavicle: long-term results of arthroscopic resection. Arthroscopy 2000;16:600–5.
[51] Meislin RJ, Zuckerman JD, Nainzadeh N. Type III acromioclavicular joint separation associated with late brachial plexus neuropraxia. J Orth Trauma 1992;6:370–2.
[52] Sturm JT, Perry JFJ. Brachial plexus injuries from blunt trauma: a harbinger of vascular and thoracic injuries. Ann Emerg Med 1987;16:404–6.
[53] Bernard TN, Brunet ME, Haddad RJ. Fractured coracoid process in acromioclavicular dislocations: report of four cases and review of the literature. Clin Orthop 1983;175:227–32.
[54] Barber FA. Complete posterior acromioclavicular dislocation: a case report. Orthopedics 1987; 10:493–6.
[55] Wurtz LD, Lyons FA, Rockwood Jr CA. Fractures of the middle third of the clavicle and dislocation of the acromioclavicular joint. JBJS 1992;74A:133–7.
[56] Lindsey RW, Gutowski WT. The migration of a broken pin following fixation of the acromioclavicular joint: a case report and review of the literature. Orthopaedics 1986;9:413–6.
[57] Norrell H, Llewellyn RC. Migration of a threaded Steinmann pin from an acromioclavicular joint into the spinal canal: a case report. JBJS 1965;47:1024–6.
[58] Eaton R, Serletti J. Computerized axial tomography—a method of localizing Steinmann pin migration: a case report. Orthopaedics 1981;4:1357–60.
[59] Mazet RJ. Migration of a Kirschner wire from the shoulder region into the lung: report of two cases. JBJS 1943;25:477–83.
[60] Lyons F, Rockwood Jr CA. Migration of pins used in operations on the shoulder. JBJS 1990; 72-A:1262–7.
[61] Cappello T, Nuber GW, Nolan KD, McCarthy WJ. Acute ischemia of the upper limb fifteen years after anterior dislocation of the glenohumeral joint and a modified Bristow procedure. JBJS 1996;78:1578–82.
[62] Nelson CL. Repair of acromioclavicular separations with knitted Dacron graft. Clin Orthop 1979;143:289.
[63] Kappakas GS, McMaster JH. Repair of acromioclavicular separation using a Dacron prosthesis graft. Clin Orthop 1978;131:247–51.
[64] Park JP, Arnold JA, Coker TP, et al. Treatment of acromioclavicular separations: a retrospective study. Am J Sports Med 1980;8:251–6.
[65] Colosimo AJ, Hummer III CD, Heidt Jr RS. Aseptic foreign body reaction to Dacron graft material used for coracoclavicular ligament reconstruction after type III acromioclavicular dislocation. Am J Sports Med 1996;24:561–3.
[66] Weaver JK, Dunn HK. Treatment of acromioclavicular injuries, especially complete acromioclavicular separation. JBJS 1972;54:1187–94.

[67] Cave EF. Fractures and other injuries. Chicago: Year Book Medical Publishers; 1958.
[68] Waskowitz WJ. Disruption of the sternoclavicular joint: an analysis and review. Am J Orthop 1961;3:176–9.
[69] Nettles JL, Linscheid R. Sternoclavicular dislocations. J Trauma 1968;8:158–64.
[70] Omer GE. Osteotomy of the clavicle in surgical reduction of anterior sternoclavicular dislocation. J Trauma 1967;7:584–90.
[71] Heinig CF. Retrosternal dislocation of the clavicle: early recognition, x-ray diagnosis, and management. J Bone Joint Surg 1968;50A:830.
[72] Mehta JC, Sachdev A, Collins JJ. Retrosternal dislocation of the clavicle. Injury 1973;5:79–83. 1996;5(1):79–83.
[73] Rockwood Jr CA, Wirth MA. Injuries to the strenoclavicular joint. In: Rockwood Jr CA, Green DP, Bucholz RW, et al, editors. Rockwood and Green's fractures in adults. 4th edition. Philadelphia: Lippincott-Raven; 1996. p. 1415–71.
[74] Ono K, Inagawa H, Kiyoto K, Terada T, Suzuki S, Maekawa K. Posterior dislocation of the sternoclavicular joint with obstruction of the innominate vein: case report. J Trauma 1998;44: 381–3.
[75] De Jong KP, Sukui DM. Anterior sternoclavicular dislocation: a long-term follow-up study. J Orthop Trauma 1990;4:420–3.
[76] Haas N, Blauth M. Injuries to the acromioclavicular and sternoclavicular joints—surgical or conservative treatment. Orthopade 1989;18:234–45.
[77] Eskola A. Sternoclavicular dislocations: a plea for open treatment. Acta Orthop Scand 1986; 57:227–8.
[78] Eskola A, Vainionpaa S, Vastamki M, Slatis P, Rokkanen P. Operation of old sternoclavicular dislocation: results in 12 cases. J Bone Joint Surg 1989;71B:63–5.
[79] Fery A, Sommelet J. Dislocation of the sternoclavicular joint: a review of 49 cases. Int Orthop 1988;12:187–95.
[80] Brown JE. Anterior sternoclavicular dislocation: a method of repair. Am J Orthop 1961;31: 184–9.
[81] Habernek H, Hertz H. Origin, diagnosis and treatment of traumatic dislocation of sternoclavicular joint. Aktuel Traumatol 1987;17:25–8.
[82] Kahle M, Filler RL, Forster R. Luxations in the sternoclavicular joint. Aktuel Traumatol 1990;20:83–6.
[83] Booth CM, Roper BA. Chronic dislocation of the sternoclavicular joint: an operative repair. Clin Orthop 1979;140:17–20.
[84] Burrows HJ. Tenodesis of subclavius in the treatment of recurrent dislocation of the sternoclavicular joint. J Bone Joint Surg 1951;33B:240–3.
[85] Barth E, Hagen R. Surgical treatment of dislocations of the sternoclavicular joint. Acta Orthop Scand 1983;54:746–7.
[86] Lunseth PA, Chapman KW, Frankel VH. Surgical treatment of chronic dislocation of the sternoclavicular joint. J Bone Joint Surg 1975;57B:193–6.
[87] Salvatore JE. Sternoclavicular joint dislocation. Clin Orthop 1968;58:51–4.
[88] Omer GE. Osteotomy of the clavicle in surgical reduction of anterior sternoclavicular dislocation. J Trauma 1967;7:584–90.
[89] Wirth MA, Rockwood Jr CA. Complications following repair of the sternoclavicular joint. In: Bigliani LU, editor. Complications of shoulder surgery. Baltimore: Williams & Wilkins; 1993. p. 139–53.
[90] Rockwood Jr CA, Groh GI, Wirth MA, Grassi FA. Resection arthroplasty of the sternoclavicular joint. J Bone Joint Surg 1997;79(3):387–93.
[91] Wirth MA, Rockwood Jr CA. Injuries to the sternoclavicular joint. In: Heckman JD, Bucholz RW, editors. Rockwood and Green's fractures in adults. 5th edition. Philadelphia: Lippincott-Raven; 2001. p. 1245–92.
[92] Medvecky MJ, Zuckerman JD. Sternoclavicular joint injuries and disorders. In: Price CT, editor. Instructional course lectures 49. Rosemont, IL: AAOS; 2000. p. 397–406.

[93] Gale DW, Dunn ID, McPherson S, Ini OOA. Retrosternal dislocation of the calvicle: the Stealth dislocation: a case report. Injury 1992;23:563–4.
[94] Jougon JB, Lepront DJ, Dromer CE. Posterior dislocation of the sternoclavicular joint leading to mediastinal compression. Ann Thorac Surg 1996;61:711–3.
[95] Wasylenk MJ, Busse EF. Posterior dislocation of the calvicle causing fatal tracheoescophageal fistula. Can J Surg 1981;24:626–7.
[96] Howard FM, Shafer SJ. Injuries to the clavicle with neurovascular complications: a study of fourteen cases. J Bone Joint Surg 1965;47A:1335–46.
[97] Worman LW, Leagus C. Intrathoracic injury following retrosternal dislocation of the clavicle. J Trauma 1967;7:416–23.
[98] Gangahar DM, Flogaites T. Retrosternal dislocation of the clavicle producing thoracic outlet syndrome. J Trauma 1978;18:369–72.
[99] Selesnick FH, Jablon M, Frank C, Post M. Retrosternal dislocation of the clavicle: report of four cases. J Bone Joint Surg 1984;66A:287–91.
[100] Cope R, Riddervold HO. Posterior dislocations of the sternoclavicular joint: report on two cases, with emphasis on radiologic management and early diagnosis. Skeletal Radiol 1988;17: 247–50.
[101] Cooper GJ, Stubbs D, Waller DA, Eilkinson GAL, Saleh M. Posterior sternoclavicular dislocation: a novel method of external fixation. Injury 1992;23:565–7.
[102] Lowman CL. Operative correction of old sternoclavicular dislocation. J Bone Joint Surg 1928; 10:740–1.
[103] Allen AW. Living with suture grafts in the repair of fractures and dislocations. Arch Surg 1928; 16:1007–20.
[104] Key JA, Conwell HE. The management of fractures, dislocations and sprains. 5th edition. St. Louis, MO: CV Mosby; 1951. p. 458–61.
[105] Bankart ASB. An operation for recurrent dislocation (subluxation) of the sternoclavicular joint. Br Surg 1938;26:320–3.
[106] Milch H. The Rhomboid ligament in surgery of the sternoclavicular joint. J Int Coll Surg 1952;17:41–51.
[107] Brooks AL, Henning CD. Injury to the proximal clavicular epiphysis. J Bone Joint Surg 1972; 54A:1347–8.
[108] Webb PA, Suchey JM. Epiphyseal union of the anterior iliac crest and medial clavicle in a modern multiracial sample of American males and females. Am J Phys Anthropol 1985; 68:457–66.
[109] Todd TW, D'Errico Jr J. The clavicular epiphyses. Am J Anat 1928;41:25–50.
[110] Goldfarb C, Bassett G, Sullivan S, Gordon J. Retrostemal displacement after physeal fracture of the medial clavicle in children: Treatment by open reduction and internal fixation. JBJS 2001; 83-B:1168–72.
[111] Zaslav KR, Ray S, Neer II CS. Conservative management of a displaced medial clavicular physeal injury in an adolescent athlete. Am J Sports Med 1989;17:833–6.
[112] Wirth MA, Rockwood Jr CA. Chronic conditions of the acromioclavicular and sternoclavicular joints. In: Chapman MW, editor. Operative orthopaedics, part XI. 2nd edition. Philadelphia: Lippincott; 1992. p. 1683–93.
[113] Wirth MA, Rockwood Jr CA. Complications of treatment of injuries to the shoulder. In: Epps CH, editor. Complications in orthopaedic surgery. 3rd edition. Philadelphia: Lippincott; 1994. p. 229–55.
[114] Barth E, Hagen R. Surgical treatment of dislocations of the sternoclavicular joint. Acta Orthop Scand 1983;54:746–7.
[115] Daus GP, Drez Jr D, Newton Jr BB, Kober R. Migration of a Kirschner wire from the sternum to the right ventricle: A case report. Am J Sports Med 1993;21:321–2.