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SECTION O

Vascular Problems of the Shoulder

J. Michael Bennett

Vascular injuries of the shoulder are relatively uncommon and are often associated with direct trauma. Most acute sports-related vascular injuries occur with contact sports; however, repetitive motion and congenital malformation can lead to many chronic syndromes that can become painful and debilitating. Symptoms can be misinterpreted and attributed to one of the more common shoulder musculoskeletal abnormalities. Early recognition of vascular compromise is essential to avoid potentially catastrophic outcomes from misdiagnosis.

ANATOMY

A thorough understanding of the vascular anatomy of the shoulder is necessary to fully understand the complete spectrum of vascular injury (Fig. 170-1). Blood flow to the upper extremity begins with the heart. On the right, the subclavian artery branches from the innominate artery. On the left, the subclavian artery arises directly from the arch of the aorta. The subclavian artery then enters the thoracic outlet and extends to the lateral border of the first rib. The thoracic outlet is composed of the upper border of the first rib, inferior border of the clavicle, and anterior and middle scalene muscles. From the lateral border of the first rib to the inferior border of the latissimus dorsi, the subclavian becomes the axillary artery. The artery travels beneath the pectoralis minor and is divided into three sections, with the number of branches from each section corresponding with the number of the section. The first section is above the superior border of the pectoralis minor and gives off the superior thoracic artery inferiorly, which supplies vessels to the first, second, and third intercostal spaces. The second section travels deep to the pectoralis minor consisting of the lateral thoracic and the thoracoacromial arteries, which further arborize into clavicular, acromial, deltoid,

and pectoral branches. The third section is distal to the lateral border of the pectoralis minor and contributes the largest branch of the axillary artery, the subscapular artery. The subscapular artery further divides into the scapular circumflex and the thoracodorsal artery. The anterior and posterior humeral circumflex arteries are the last two remaining branches from the axillary artery before it becomes the brachial artery. The posterior humeral circumflex descends posteriorly into the quadrilateral space

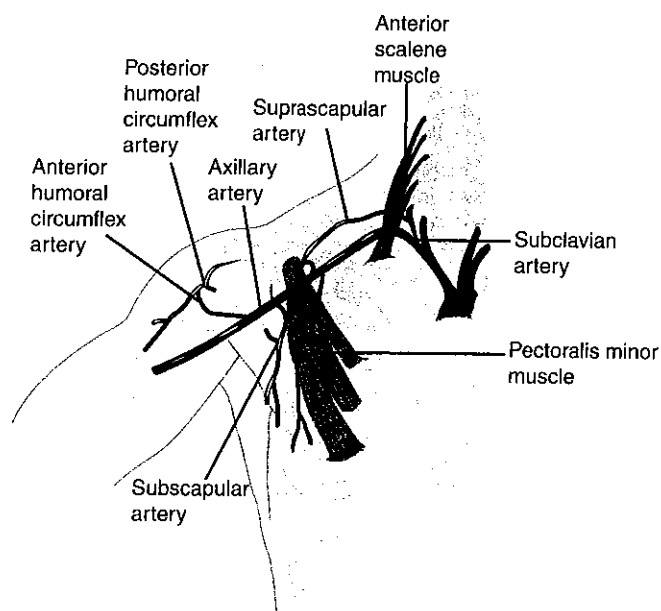


Figure 170-1 Arterial anatomy of the shoulder demonstrates arterial relationships to surrounding musculoskeletal structures and potential sites of compression.

with the axillary nerve. The anterior humeral circumflex artery is smaller than the posterior branch and travels laterally around the front of the surgical neck of the humerus, supplying most of the blood supply to the humeral head. The humeral head is perfused by the arcuate artery, which is an anterolateral ascending branch of the anterior circumflex artery that enters the bone in the area of the intertubercular groove and supplies branches to the lesser and greater tuberosities.

The subclavian vein begins as a branch from the brachiocephalic vein medially and becomes the axillary vein at the lateral border of the first rib. At the inferior border of the latissimus dorsi, the axillary vein becomes the basilic vein, and it continues distally. The cephalic vein is a superficial vein that pierces the clavipectoral fascia and empties into the axillary vein. The axillary and cephalic veins are responsible for most of the venous drainage in the shoulder. The lymphatics end in the thoracic and right lymphatic ducts.

CLINICAL PRESENTATION

The differential diagnosis of shoulder pain and swelling should include vascular injury. Vascular lesions have been described in baseball, volleyball, tennis, cycling, marksmanship, and kayaking athletes.¹ Initial symptoms are vague and nonspecific; however, complaints of easy fatigability, venous congestion, pallor, coolness of the hand, paresthesias, diminished pulses, and cold intolerance should increase suspicion of a vascular lesion. The throwing athlete is at particular risk for developing a vascular injury. The increased stresses across the shoulder can place vascular structures at risk. Other potential causes to consider include penetrating and blunt trauma, thrombosis, and compression by muscle, tendon, fascia, callus, or bone.



Figure 170-2 Adson's test indicates subclavian arterial compression between the scalene muscles when there is a diminished radial pulse with arm extension, external rotation, and the patient facing the involved extremity.

PHYSICAL EXAMINATION

A thorough history emphasizing timing of symptoms, activities, and precipitating causes is necessary. Physical examination of both upper extremities should include inspection of the hands and fingers, looking for ulcerations, cold intolerance, capillary refill, color differences, and nailbed abnormalities.² Standard range of motion, strength testing, blood pressure, shoulder swelling, auscultation of the axilla and brachial artery, Allen's test, and pulse palpation from the wrist to the shoulder should be evaluated. The position of the arm is important and should be tested at the side and in abduction and external rotation to identify any clinically significant differences that would need further neurovascular evaluation.

In addition to the extremities, the cervical spine and clavicle must be thoroughly examined. Vascular compression in the cervical region can be evaluated using Adson's test, the costoclavicular maneuver, and the hyperabduction maneuver. Adson's test (Fig. 170-2) indicates subclavian arterial compression between the scalene muscles when there is a diminished radial pulse with arm extension, external rotation, and the patient facing toward the involved extremity. The costoclavicular maneuver (Fig. 170-3) indicates compression among the structures between the clavicle and the first rib when there is a diminished pulse after thrusting the shoulders back in an erect posture. For the hyperabduction maneuver (Fig. 170-4), the arm is extended, abducted, and externally rotated with the patient facing away from

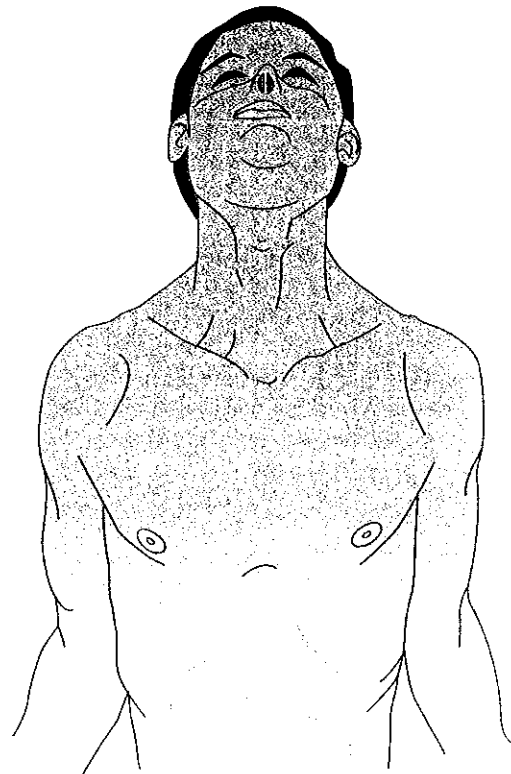


Figure 170-3 The costoclavicular maneuver indicates compression among the structures between the clavicle and the first rib when there is a diminished pulse after thrusting the shoulders back in an erect posture.

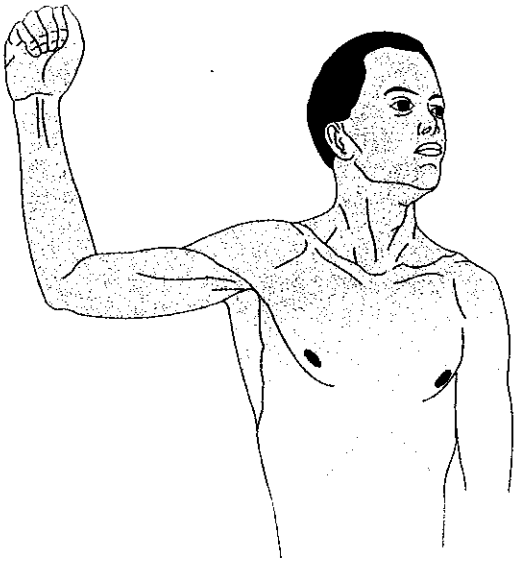


Figure 170-4 For the hyperabduction maneuver, the arm is extended, abducted, and externally rotated with the patient facing away from the affected extremity, which compresses the axillary artery as it passes underneath the insertion of pectoralis minor and the coracoid process.

the affected extremity, which theoretically compresses the axillary artery as it passes underneath the insertion of the pectoralis minor and coracoid process.³

IMAGING

Once a history and physical examination have been obtained, a systematic diagnostic work-up is indicated. Standard radiographs of the cervical spine and shoulder should be obtained to rule out bony abnormalities associated with vascular compromise such as a cervical rib, a mass occupying bone lesion, fracture, or dislocation.

If a vascular lesion is suspected, a Doppler ultrasound of the extremities is the best initial screening test^{1,4}; however, its use should be limited as a dynamic test. Arteriography remains the gold standard for the diagnosis of arterial injuries. Magnetic resonance angiography (MRA) has gained recent popularity for its detailed images of blood vessels and blood flow without having to insert a catheter into the area of interest, minimizing the risk for arterial damage. In all cases, a vascular consultation is recommended to aid in the initial evaluation and work-up.

VASCULAR TRAUMA

Vascular injury to the shoulder can occur secondary to blunt or penetrating trauma. Most traumatic vascular injuries to the shoulder occur from penetrating trauma such as bone fragments or a foreign object (Fig. 170-5). Axillary artery injury has been associated with scapular neck fractures, humeral neck fractures, and clavicle fractures. Vascular status and fracture pattern must be quickly assessed with plain films and a thorough physical examination followed by arteriography or MRA (if indicated). If surgery is indicated, the injury must be addressed within 6 hours to reduce risk for limb ischemia. Although blunt



Figure 170-5 Arteriogram demonstrating an axillary artery aneurysm after a gunshot injury to the right shoulder. (Courtesy of Drs. Raphael Espada and James B. Bennett.)

trauma-induced injury is in the minority, the consequences from delay in diagnosis can be just as devastating. Shoulder mobility can create injury from traction and avulsion of underlying neurovascular structures without creating a bony injury. Low-energy shoulder dislocations or high-energy traction injuries such as scapulothoracic dissociation can lead to similarly poor outcomes if a misdiagnosis or delay in diagnosis is made.

Scapulothoracic Dissociation

Scapulothoracic dissociation (SCD) describes a complete disruption of the scapulothoracic joint and its underlying neurovascular structures, which can be associated with acromioclavicular separation, displaced clavicle fracture, or sternoclavicular disruption. Vascular lesions have been reported in 88% of patients, and severe neurologic injuries occur in 94% of patients. One study reported a 10% mortality rate and nearly 100% of deaths were associated with vascular lesions.⁵ Overall, clinical outcome after SCD is uniformly poor. Outcomes such as flail extremity, early amputation, and death have been reported.⁶ The scapula, clavicle, acromioclavicular joint, and the surrounding ligamentous, tendinous, and capsular structures create a superior shoulder suspensory complex. Injuries to single components of the complex may be treated nonoperatively because the complex maintains a stable construct. However, if two or more components are compromised, the complex is unstable and requires at least partial repair to restore stability.⁶

Shoulder Dislocation

Anterior shoulder dislocation is a common injury with potential complications associated with the initial dislocation as well as the reduction. Fortunately, vascular injury associated with anterior dislocation is rare. Because of anatomic location and primary restraints, the axillary artery remains at risk with this type of injury. There are a number of mechanisms that have been proposed to describe arterial injury. Some authors have proposed that the artery is fixed by the circumflex scapular artery, which reduces the

artery's mobility on impact and can lead to arterial disruption.^{7,8} Others have suggested⁹ that the pectoralis minor acts as a fulcrum against which the artery is angulated, contused, and ruptures as the humeral head displaces the artery anteriorly (Fig. 17O-6). Vascular injuries secondary to shoulder dislocation occur primarily in older patients with stiffer, calcified, more delicate vessels. Other injuries to the axillary artery include axillary artery occlusion, which has been documented with luxatio erecta, and pseudoaneurysm (Figs. 17O-7) and can occur after recurrent anterior dislocations.⁹ In addition to arterial injuries, venous injuries such as venous thrombosis (Fig. 17O-8) can have a delayed presentation with unilateral extremity swelling and pain. Noninvasive Doppler imaging or venography can be used for diagnosis.

Sternoclavicular Dislocation

Posterior sternoclavicular joint dislocation is a rare entity, with only 120 cases documented in the medical literature since it was first described by Sir Astley Cooper in 1824.¹⁰ The sternoclavicular joint is the articulation between the medial clavicle and the manubrium of the sternum. The joint is considered to be gliding with an intra-articular

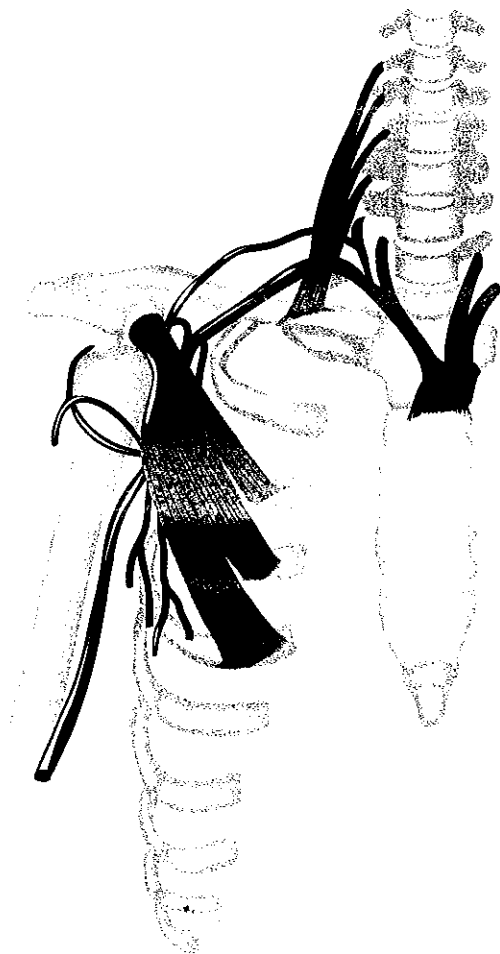


Figure 17O-6 During anterior dislocation, the pectoralis minor can act as a fulcrum against which the artery is angulated and contused, and it ruptures as the humeral head displaces the artery anteriorly.

disk, allowing up to 60 degrees of angulation in extreme of shoulder girdle movement.¹⁰ Younger patients have a higher rate of dislocation due to increased joint laxity. Stability of the joint is maintained with anterior ligaments and a thicker, stronger posterior ligament. The proximity of the brachiocephalic vein and innominate artery on the right and the common carotid artery and subclavian vein on the left can make posterior dislocation of the sternoclavicular joint a potentially life-threatening injury. Compressive or violent force is usually required to cause a dislocation, although a few cases of atraumatic dislocation have been reported.¹¹ Contact sports and motorcycle injuries are the most common causes worldwide.^{12,13} Symptoms include pain, inability to move the affected shoulder, and a palpable depression on the affected side. Rarely, patients may present with dyspnea or respiratory compromise and immediate reduction is indicated. Other life-threatening complications, which can occur in up to 25% of cases, include tracheal damage, hemopneumothorax, and damage to the larynx with vocal cord palsy.¹³ Standard radiographic views can be difficult to interpret; therefore, computed tomography (CT) evaluation in stable patients is the ideal method for confirming the suspected diagnosis. CT angiography can delineate related injuries, and intravenous contrast can be used to enhance computed tomographic interpretation of vascular injuries. Treatment of posterior dislocation is immediate reduction. Attempt at closed reduction is made and if unsuccessful or the joint is found to be unstable, open reduction is indicated. If open reduction is indicated, a cardiothoracic surgeon should be present or on standby. Arteriography can be used before and after reduction; however, because of its invasive nature, it is reserved for selected cases.¹⁰

VASCULAR INJURY IN THE ATHLETE

Subclavian and Axillary Artery Occlusion

The axillary artery is a continuation of the subclavian artery and is responsible for perfusing the entire upper extremity. There are a number of anatomic compression sites along this pathway that may lead to arterial occlusion. Compression can occur at the subclavian artery as it angulates over a cervical transverse process, cervical rib, or first rib or is compressed by the anterior scalene muscle. Symptoms may include intermittent blanching of the hand and fingers associated with cooler temperatures, fatigue, and exertional pain. Physical examination may reveal a diminished or absent radial pulse, supraclavicular bruits, and a positive Adson's test. The diagnosis is confirmed with arteriography. Treatment involves a first rib resection. An acute arterial occlusion is an emergency, and immediate surgery is indicated with first rib resection, removal of the thrombus, and embolectomy.¹⁴ In 1945, Wright first demonstrated occlusion of the axillary artery from direct compression of the pectoralis minor as the arm is brought into a position of hyperabduction.^{3,15} Tullos and colleagues further expanded on this description to include a position of abduction, extension, and external rotation, which is consistent with the cocking phase of the throwing cycle. They concluded

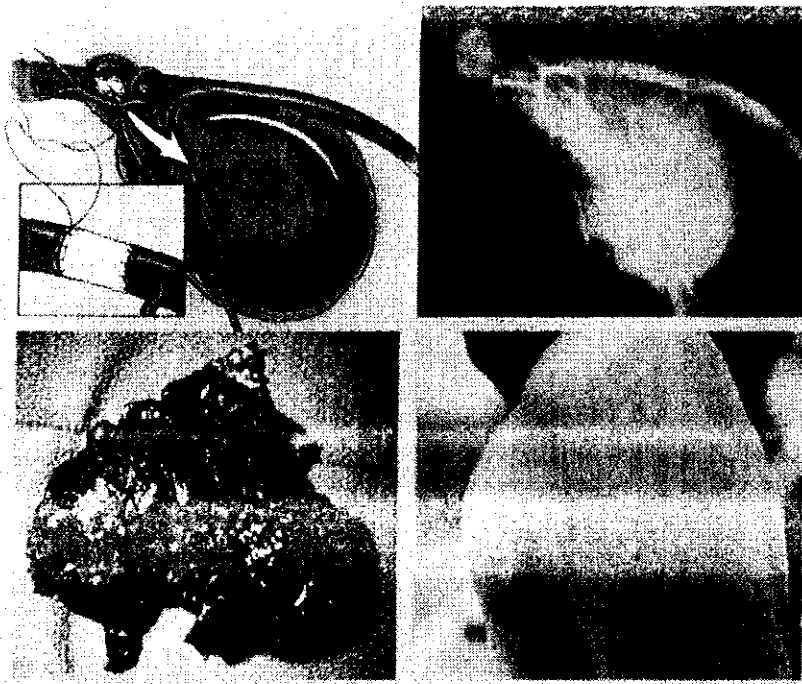


Figure 170-7 Diagnosis and treatment of a large pseudoaneurysm in a patient after recurrent shoulder dislocations. (Courtesy of Drs. Raphael Espada, Baylor College of Medicine, Houston, TX, and James B. Bennett, the London Orthopedic group, Houston, TX.)

that repetitive throwing can lead to repeated local trauma to the artery creating intimal damage and the development of subsequent thrombosis.³¹⁶ Symptoms included claudication pain, rapid fatigue, poor control of the pitch, diminished or absent distal pulses, cyanosis, and decreased skin temperatures particularly in the position of hyperabduction and external rotation. Noninvasive Doppler studies can be diagnostic; however, definitive diagnosis is made with arteriography (Fig. 170-9). Treatment of this condition is usually surgical, and options include thrombectomy,

sympathectomy, segmental excisions, bypass with vascular graft, anastomosis, and angioplasty.¹⁶⁻¹⁹

Effort Thrombosis

Thrombosis of the axillary venous system was first described by Sir James Paget in 1875 and by Von Schroetter in 1884. This condition has been termed *effort-induced thrombosis* because of its frequent association with repetitive vigorous activity or blunt trauma with direct or indirect injury to the



Figure 170-8 Venogram demonstrating venous thrombosis, which can be a complication after dislocation. (Courtesy of Drs. Raphael Espada and James B. Bennett.)



Figure 170-9 Arteriogram demonstrating axillary artery thrombosis. (Courtesy of Dr. Raphael Espada.)

axillary vein. The basilic vein becomes the axillary vein at the lower border of the teres major muscle, which becomes the subclavian vein at the lateral margin of the first rib. There are several points along its anatomic course where venous compromise may occur. Compression occurs with hyperextension of the neck or hyperabduction of the arms and can occur between the first rib and the clavicle, the subclavian muscles, or the costocoracoid ligament.^{20,21}

Risk factors for the development of a thrombus include a hypercoagulable state, dehydration, oral contraceptives, and vascular injury. The repetitive throwing motion involved with overhead athletes stretches the subclavian vein and can predispose to the development of tears within the intima of the vein.

Symptoms occur within 24 hours of the inciting trauma or activity and consist of activity-related fatigue, dull and aching extremity pain, numbness, and swelling. Often, patients describe a "heaviness" of the upper arm and shoulder following activities. Physical examination may reveal superficial venous dilation, extremity cyanosis, swelling, and a painful axilla or deltopectoral groove. Pulses and neurologic examination can be normal. The physical findings may become more prominent with exercising testing. Diagnosis is made with history and physical examination and can be confirmed using venography. The venogram will show complete occlusion of the axillary or subclavian vein with extensive collateral venous return.

Once the diagnosis is made, the first line of treatment for effort-induced thrombosis is conservative, emphasizing rest, heat, and elevation of the involved extremity. Pain and swelling will resolve within 3 to 4 days; however, many of these patients continue to suffer symptoms.^{17,22} In the acute phase, heparin, followed by warfarin, is often used to inhibit progression of the thrombus. Recently, thrombolytic agents such as streptokinase have been found to be effective in the lysis of acute clots less than 2 weeks old; however, these agents are ineffective with chronic clots. Early thrombectomy with simultaneous decompression of the thoracic outlet or first rib resection following fibrinolytic therapy has been associated with good long-term results.^{17,23}

Quadrilateral Space Syndrome

The quadrilateral space is defined as the area enclosed by the teres minor superiorly, the humeral shaft laterally, the teres major inferiorly, and the long head of the triceps medially. Within this space traverses the axillary nerve and posterior humeral circumflex artery (PHCA). In 1983, Cahill and Palmer first described the "quadrilateral space syndrome," which involves compression of the PHCA or the axillary nerve within the quadrilateral space.^{1,24} Compression can occur from fibrous bands within this space, creating tension across the neurovascular bundle with abduction and external rotation of the affected extremity. Typical patients are between 25 and 50 years of age. Symptoms are unilateral and often involve the dominant extremity. The symptoms described are nonspecific and include pain and paresthesias not associated with a traumatic event. These symptoms progressively worsen with abduction and external rotation. Clinical findings include tenderness to palpation over the quadrilateral space in addition to reproduction of symptoms with abduction and external rotation

of the extremity for longer than 1 minute. Dampening of the radial pulse may also occur with the arm in this cocked position. In chronic cases, there may be atrophy of the deltoid. Neurologic examination and electromyographic studies are usually normal. Diagnosis may be confirmed using bilateral dynamic subclavian arteriograms. Cahill and Palmer^{2,24} found that with abduction and external rotation, the PHCA remained patent in the asymptomatic shoulder and became obstructed in the symptomatic shoulder. Mochizuki and associates²⁵ found that asymptomatic volunteers demonstrated angiographic occlusion of the PHCA while in the cocked position. Angiography is nonspecific and should serve only as a supplement to a clinical diagnosis.

The standard treatment is nonoperative for patients with quadrilateral space syndrome. Rest, modification of activities, and the initiation of a formal therapy program is the first line of treatment. Therapy should emphasize stretching the posterior capsule and teres minor. If conservative measures fail, surgical decompression of the quadrilateral space through a posterior approach is indicated. The surrounding muscles, tendons, and fibrous bands that constrain the space are released, removing all pressure on the neurovascular bundle when the shoulder is brought into abduction and external rotation. Cahill and Palmer reported 16 of 18 patients with good or excellent results and 2 of 18 with no change in symptoms after decompression.²⁴ Further interpretation of the results associated with decompression is difficult owing to the small number of cases and the short follow-up.

Thoracic Outlet Syndrome

Thoracic outlet syndrome involves compression of the neurovascular structures supplying the upper limb as they course from the neck to the axilla. The boundaries of compression include the clavicle, the scapula, and the first thoracic rib or cervical rib. Etiology, symptoms, and treatment are further discussed in Chapter 17N.

CRITICAL POINTS

- Early recognition of vascular compromise is essential to avoid potentially catastrophic outcomes from misdiagnosis.
- Initial symptoms are vague and nonspecific; however, complaints of easy fatigability, venous congestion, pallor, coolness of the hand, paresthesias, diminished pulses, and cold intolerance should increase suspicion of a vascular lesion.
- The position of the arm is important and should be tested at the side and in abduction and external rotation to identify any clinically significant differences that would need further neurovascular evaluation.
- Standard radiographs of the cervical spine and shoulder should be obtained to rule out bony abnormalities associated with vascular compromise such as a cervical rib, a mass occupying bone lesion, fracture, or dislocation.

SUGGESTED READINGS

- Arteriography remains the gold standard for the diagnosis of arterial injuries.
- Vascular injuries secondary to shoulder dislocation occur primarily in older patients with stiffer, calcified, more delicate vessels. Other injuries to the axillary artery include axillary artery occlusion, which has been documented with luxatio erecta, and pseudoaneurysm and can occur after recurrent anterior dislocations.
- The proximity of the braciocephalic vein and innominate artery on the right and the common carotid artery and subclavian vein on the left can make posterior dislocation a potentially life-threatening injury.
- Risk factors for the development of a thrombus include a hypercoagulable state, dehydration, oral contraceptives, and vascular injury.
- The repetitive throwing motion involved with overhead athletes stretches the subclavian vein and can predispose to the development of tears within the intima of the vein.

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SECTION P

Parsonage-Turner Syndrome

Adam Nelson Whatley

Parsonage-Turner syndrome, also known as *brachial neuritis* or *neuralgic amyotrophy*, is a condition of unknown etiology. It affects the brachial plexus and causes pain followed by weakness of the shoulder and upper extremity. It has been described numerous times in the literature since it was first reported by Parsonage and Turner in 1948.¹

The classic presentation begins with an acute onset of sharp pain in the shoulder region. As the pain subsides, weakness arises in the shoulder musculature. Diagnosis of this condition is primarily clinical in nature and is exceedingly difficult in the acute stage.

There is a 3:2 male-to-female ratio in the idiopathic form of the disease, although a hereditary form of the disease has been reported widely in the literature.² Age of onset is usually in the second or third decade, but cases have been reported ranging in age from neonates to patients in their eighth decade. The exact cause is unknown, but the current hypothesis is one of an immune-mediated response to a patient's own peripheral nerves.² This theory is supported by the fact that about half of attacks are preceded

by some event that can trigger the immune system, including infection, surgery, pregnancy and puerperium, mental and strenuous physical stress, immunizations, and immunomodulating treatment regimens (interleukin-2 or interferon- α 2).²

RELEVANT ANATOMY AND BIOMECHANICS

The brachial plexus comprises the ventral rami of spinal nerve roots from C5 to T1. These rami, or roots, form subsequent trunks, divisions, cords, and terminal branches that innervate the shoulder and upper extremity. The brachial plexus resides in the upper shoulder region between the anterior and middle scalene muscles. It encircles the subclavian artery and enters the upper arm through the axilla. The brachial plexus is the site of many traumatic and atraumatic conditions, which must be considered in the differential diagnosis of Parsonage-Turner syndrome.