Journal of Orthopaedic & Sports Physical Therapy
1999;29(1):31–38

Role of Scapular Stabilizers in Etiology and Treatment of Impingement Syndrome

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BACKGROUND

Shoulder pain and dysfunction with overhead activities resulting from subacromial impingement syndrome is common.[2,16] Subacromial impingement syndrome has generally been classified as primary or secondary. A thorough history and physical examination are essential to identifying the etiology of the subacromial impingement syndrome and to direct treatment.[4]

Primary subacromial impingement syndrome, resulting from mechanical encroachment into the subacromial space usually by an acromial hook or spurs, occurs in middle age. Individuals with primary subacromial impingement syndrome have symptoms of shoulder pain and weakness with overhead activities.[2] Impingement tests (eg, Neer,[19] Hawkins[11]) are positive. Typically, external rotation, flexion, and abduction of the shoulder are weak and painful. Night pain, usually an inability to sleep on the painful shoulder, is a common symptom of the full-thickness rotator cuff tears that can also occur in this age group.[3,15] Trauma is usually the mechanism of injury.

Persons with secondary subacromial impingement syndrome also have symptoms of pain and weakness with overhead activities. These individuals are usually young and often participate in sports that require repetitive overhead motion (eg, baseball, swimming, volleyball).[12] Symptoms with secondary impingement are attributed to rotator cuff tendinitis. These symptoms are thought to result from overuse of the rotator cuff tendons to compensate for subtle anterior or multidirectional glenohumeral instability.[26] More recently, scapulothoracic muscle weakness has been identified as a cause of secondary subacromial impingement syndrome. Here, the lack of scapular stability is thought to contribute to secondary subacromial impingement syndrome.[1,12,13]

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HISTORY

A 33-year-old, right-handed, male paramedic complained of a sudden onset of right shoulder pain with no apparent precipitating trauma that began approximately 1 month prior to evaluation. The pain occurred with movements of his arm above the horizontal. The pain was located in the posterior shoulder and axilla and was aggravated by lifting, especially overhead. The pain became more constant and he consulted his primary care physician. He was prescribed a nonsteroidal anti-inflammatory drug, but his symptoms worsened. He soon noticed that his right shoulder felt weak and he began having difficulty lifting things at work. He was referred to an orthopaedic surgeon and to physical therapy.

The patient had had no previous history of shoulder problems. He had no history of systemic illness. He had no pain at night. He was active and otherwise healthy with no change in appetite or energy level over the past few months. He had no complaints of any flulike symptoms or immunizations prior to the onset of his symptoms.[23,32]

He worked with the county paramedics on 10-hour shifts 3 days in a row followed by 2 days off. There were no light-duty options and he was unwilling to take time off from work for fear he might lose his job. He was, however, concerned that his shoulder pain and weakness would affect his job performance and the care of his patients. His goal was to return to the safe, pain-free performance of his work activities. We proceeded directly to the physical examination.

At this stage, we did not feel that laboratory tests and imaging were necessary. The patient had no signs of systemic illness. A careful musculoskeletal examination should allow for imaging studies to be better fo-

cused (eg, directed toward the appropriate anatomic location, ordering the correct view or type of imaging test). The normal shoulder series of plain radiographs does not include the definitive view (outlet) used to type acromial morphology.[14,17] Additionally, recent studies have demonstrated that many asymptomatic individuals have abnormal rotator cuff tendons as visualized by magnetic resonance imaging (MRI) studies.[18]

What diagnosis will guide physical therapy treatment?

Impression #1

Our working hypothesis about why the patient had shoulder pain and weakness was that he had a primary subacromial impingement syndrome with rotator cuff tendinopathy (Figure 1). Alternative hypotheses included rotator cuff tear, glenohumeral instability causing a secondary subacromial impingement syndrome, and muscular weakness causing a secondary impingement.

The patient had clear signs of subacromial impingement syndrome. His age, type of work, and symptoms suggest primary subacromial impingement syndrome with rotator cuff tendinopathy. This hypothesis accounts for his symptoms of pain with overhead activity and shoulder weakness. At this point, a rotator cuff tear is also possible, but the absence of night pain and his continued ability to perform overhead functions makes this option less likely. He had no history of trauma and had no symptoms of instability. He had never had any previous shoulder complaints. Glenohumeral instability leading to secondary subacromial impingement syndrome is therefore unlikely. Additionally, his history shows nothing strongly suggestive of secondary subacromial impingement syndrome from muscle weakness. The only odd finding is the axillary pain, which is a most uncommon presentation. Physical examination should therefore focus on testing for subacromial impingement syndrome and rotator cuff pathology.

PHYSICAL EXAMINATION

The patient's right scapula was very prominent even through his T-shirt. As he removed his shirt, pronounced unilateral scapular winging was noted. The patient reported that he too had noticed the winging and that he had, in fact, only noticed it for the past few weeks. Right scapular winging began at the onset of shoulder elevation (Figures 2 and 3). As expected, shoulder elevation brought on his symptoms of posterior right shoulder and axilla pain.

The finding of scapular winging influenced the course of the physical examination. Scapular muscle weakness and scapulothoracic instability has been incriminated in a plethora of shoulder dysfunction syndromes.[13] Frank serratus anterior muscle weakness or paralysis represents an extreme of scapulothoracic instability. Examination was indicated to determine the effect of scapular stability on the subacromial impingement syndrome signs and to identify the pattern of muscle weakness. A neurologic examination (associated myotomes, dermatomes, and reflexes [cervical C5, C6, C7]) was added and the effect of scapular position on symptoms was assessed. Other alternatives (postponing the examination until electromyography [EMG] or nerve conduction velocity tests were done or the MRI of the cervical spine was completed) were rejected.

Impression #2

The patient clearly had some recent injury affecting the serratus anterior muscle. Most likely sites of involvement were the cervical nerve roots and the upper brachial plexus. Anatomically, the long thoracic nerve arises from C5 and C6, which come together and pierce the scalenus medius muscle. The 2 branches come together with C7 after exiting the scalenus medius muscle. The nerve then travels vertically under the clavicle and angulates over the second rib.[30] The long thoracic nerve is purely motor and only innervates the serratus anterior muscle.

Given the patient's job requirements of lifting and carrying heavy paramedic equipment (eg, 40-lb cardiac monitors) and patients, he could have sustained an isolated nerve compression injury or a traction injury to the roots or trunks. Gregg et al have proposed a traction injury to be more likely the mechanism of a long thoracic nerve injury based on anatomic studies and clinical observations.[9] The examination should be expanded from the usual upper quarter neurologic screening examination to a more targeted and complete neurologic examination of the upper extremity to define the scope of the involvement. There was no danger in proceeding with the examination and no reason to postpone it until radiographic or electrophysiologic testing was performed.[29]

CONTINUED PHYSICAL EXAMINATION

Cervical range of motion was normal. Left cervical sidebending produced a burning sensation over the acromion, but did not reproduce his symptoms. No C5, 6, 7 weakness (other than serratus anterior) or sensory loss was evident. Reflexes were symmetric. Passive glenohumeral range of motion was within normal limits except mild internal rotation tightness was noted bilaterally. Manual muscle testing revealed

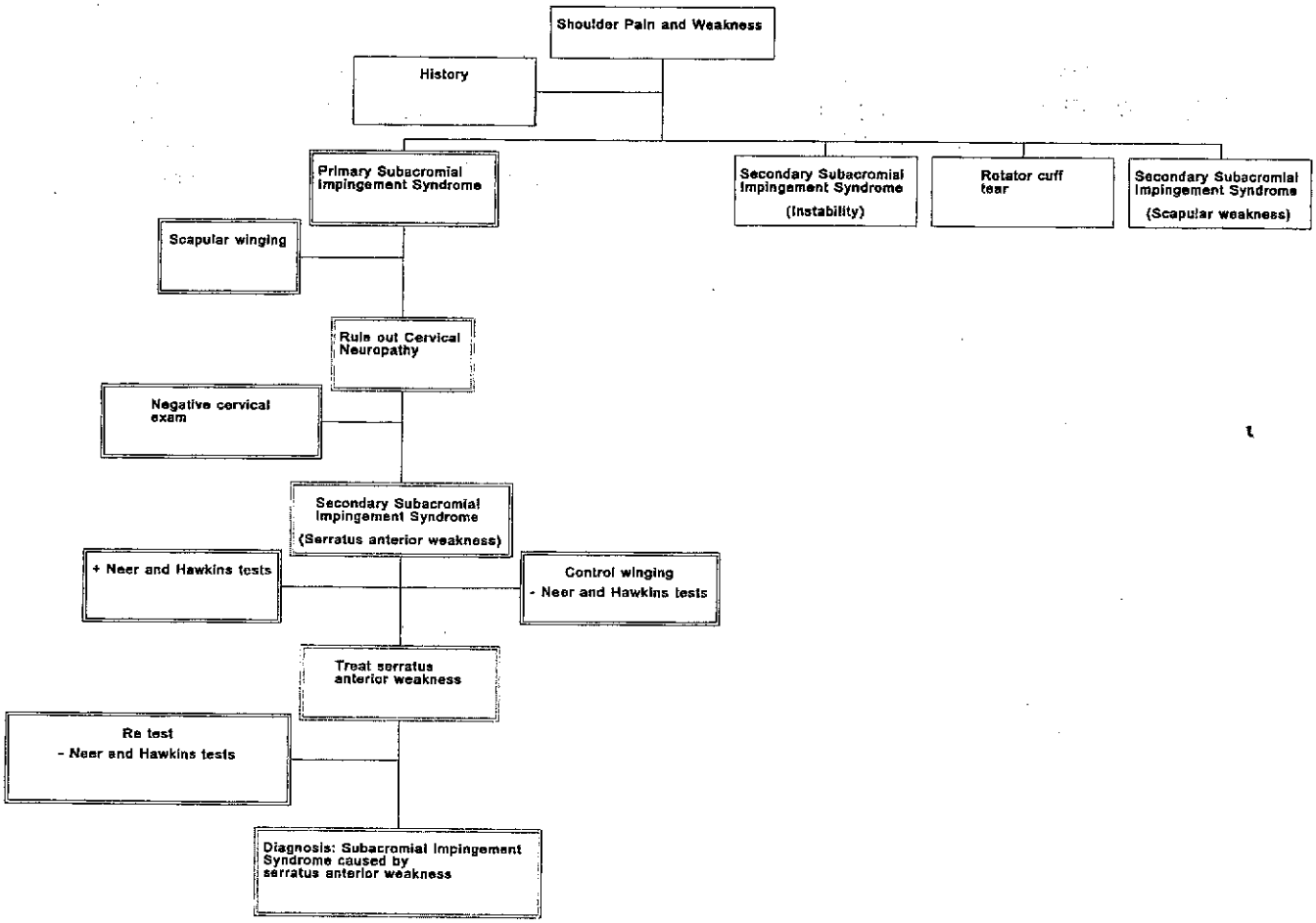

FIGURE 1. Diagnostic decision tree.

significant weakness of the right serratus anterior (2+/5) and moderate weakness of the right middle trapezius (4−/5) and lower trapezius (3+/5). Because shoulder flexion and abduction through the range of motion reproduced his pain, strength could not be accurately assessed. Both Neer and Hawkins subacromial impingement syndrome tests were positive for provoking the posterior shoulder pain.[11,19] Additionally, the scapula began to wing almost immediately with forward flexion. When the scapula was manually stabilized by the examiner, the axillary pain during elevation was eliminated and the posterior shoulder pain was somewhat reduced. Both Neer and Hawkins subacromial impingement syndrome tests were less painful when the scapula was manually stabilized. Shoulder internal and external rotation strengths were 5/5 and did not reproduce his symptoms. Resisted isometric testing of shoulder abduction and flexion were weak and painful.[21]

Impression #3

Our working hypothesis about why the patient's goals could not be met was modified as a result of the physical examination findings. We hypothesized that scapular instability resulting from serratus anterior muscle weakness was causing a secondary subacromial impingement syndrome. The weakness of his shoulder abductors and flexors could not accurately be assessed because the scapula could not be sufficiently stabilized.

There were no findings of painless weakness of other muscles that would have suggested a cervical or brachial plexus injury. The burning over the acromion with cervical side-bending was thought to be incidental. He had not felt this sensation before; he did not relate it to his complaints in any way; and there was no anatomic relation to the cervical segment for the long thoracic nerve. The anatomic location of the long thoracic nerve and its superficial nature make it susceptible to injury, especially stretching or compression.[15,25]

Although the patient appeared to have an isolated right long thoracic nerve palsy, it was not the nerve palsy, but the resultant scapular instability that was contributing to the development of the subacromial impingement syndrome symptoms. It is not possible to directly treat the long thoracic nerve palsy. We were charged with constructing a plausible hypothe-

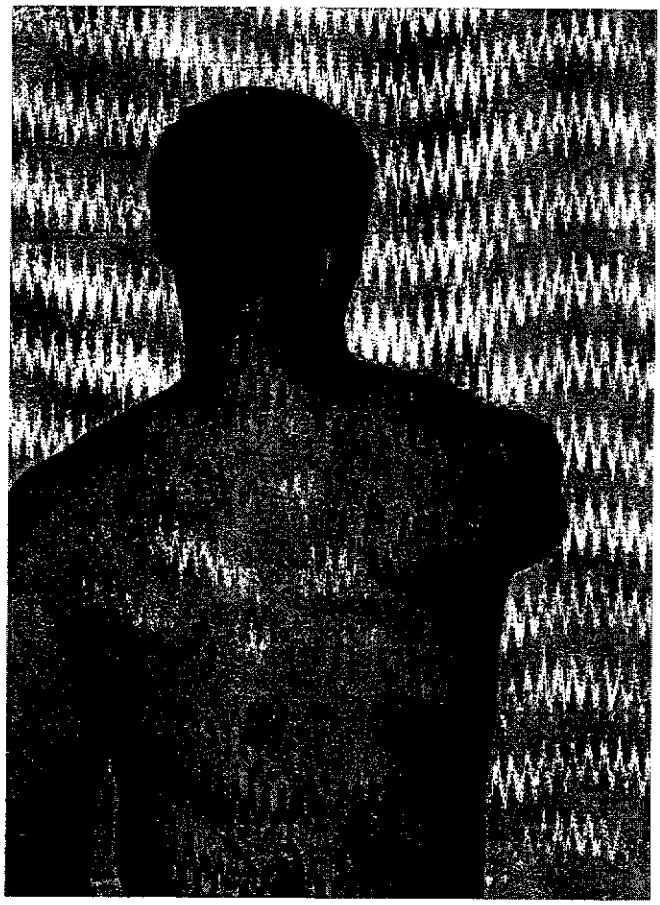

FIGURE 2. Right scapular winging at the onset of shoulder elevation.

sis that directs physical therapy treatment (ie, a physical therapy diagnosis).[22] Appropriate timing and function of the force couples between the serratus anterior and trapezius need to occur to avoid impingement of the rotator cuff and bursa in the subacromial space.[13] Together, the serratus and trapezius cause upward rotation of the scapula to maintain the subacromial space above 90 degrees of shoulder elevation. During the first 30 degrees of scapular rotation (0 to 90 degrees of shoulder elevation), the upper trapezius and the upper fibers of the serratus work together to upwardly rotate the scapula.[2] Upward rotation occurs because the axis of rotation is near the vertebral border of the scapula.[2] During the second 30 degrees of scapular rotation (90 to 180 degrees of shoulder elevation), the axis of rotation moves laterally to the acromio-clavicular joint. At this point, the lower trapezius plays a more significant role with the serratus anterior to provide upward rotation of the scapula as the upper trapezius reaches active insufficiency.[6]

In the case of this patient, the serratus weakness was great enough to impair its primary function of stability against the thoracic wall. As the serratus strength improved, scapulohumeral rhythm was more greatly disrupted above 90 degrees. The force couple of serratus anterior and upper trapezius was strong

FIGURE 3. Increased right scapular winging with addition of weight.

enough below 90 degrees to elevate the arm without scapular dysfunction. With elevation above 90 degrees, however, the lower trapezius weakness coupled with the serratus weakness contributed to the inability to control the scapula in this range of movement.

The amount of shoulder flexion achieved prior to scapular winging was used as a functional measure of progress. Although the scapula winging occurred almost immediately at the time of the initial evaluation, after 1 week of treatment the patient was able to flex the shoulder to 56 degrees before scapular winging began.

The strategies emphasized in designing a plan of care for this patient therefore included scapular stabilization, beginning with strengthening in a gravity-minimized position, and scapular strapping/bracing. If this intervention failed, referral or consultation with other medical professionals would have been warranted.[7] The results of the physical examination suggested that the symptoms could be reduced by stabilizing the scapula. The subacromial impingement syndrome may have continued long enough to be a problem independent of the scapular instability. The working hypothesis that scapular instability resulting from serratus anterior muscle weakness was causing a secondary subacromial impingement syndrome covered all the bases; it was clearly in the realm of physical therapy practice and set the direction for treatment.

Treatment of subacromial impingement syndrome alone may have alleviated some of the patient's symptoms but likely would not have resolved them completely. Without addressing the scapular instability, the patient's rotator cuff function would have continued to be compromised.[1,12] Gravity-resisted rotator cuff strengthening in the face of an unresolved mechanical subacromial impingement syndrome resulting from weakness of the scapular stabilizers would also not be the best choice.

Scapular stabilization reduced his subacromial impingement syndrome symptoms quickly as demon-

FIGURE 4. Taping technique used to stabilize scapula.

strated with negative Neer[19] and Hawkins[11] tests. The solution to the problem was 2-fold: stabilize the scapula and treat the subacromial impingement syndrome primarily. He did not have the ability to stabilize his scapula sufficiently to perform his job. He had some serratus anterior function present. Unfortunately, his right middle and lower trapezius already were showing signs of overuse and stretch weakness. We only had a snapshot of his muscle function and did not know if his nerve function was worsening or

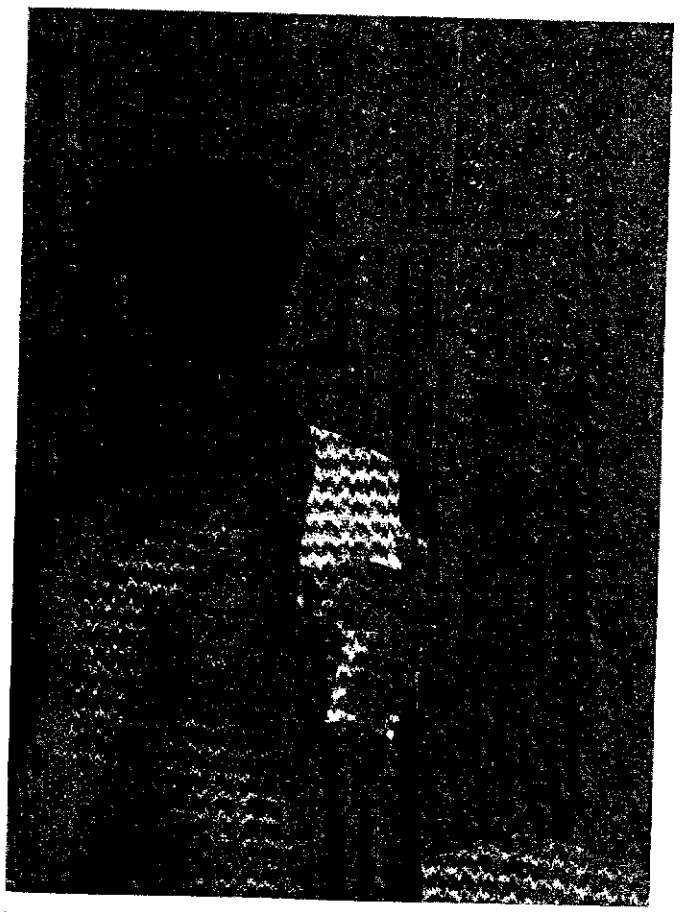

FIGURE 5. Improvement in winging with tape in place.

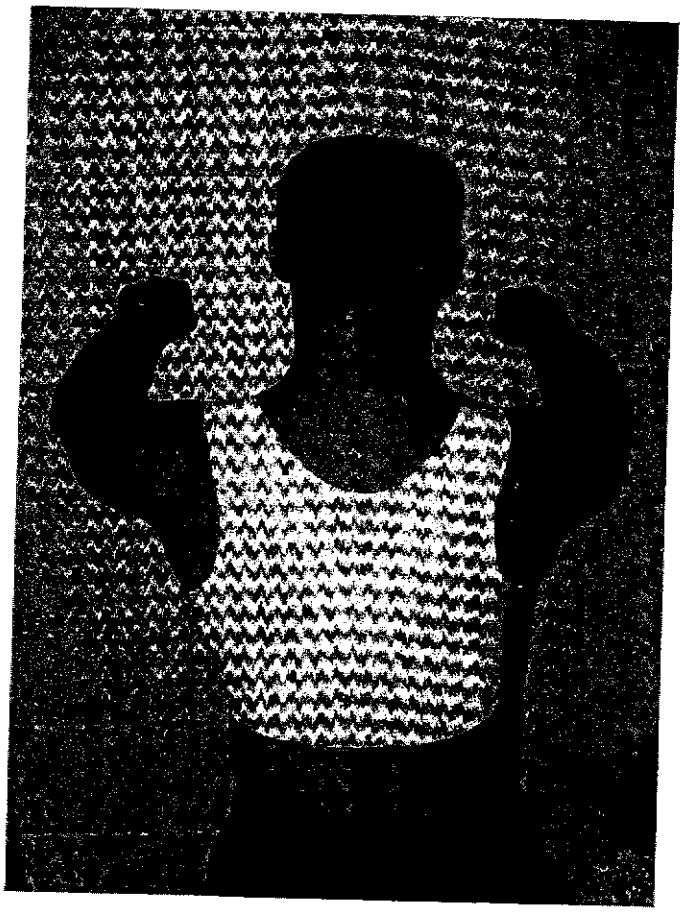

FIGURE 6. Posture support used to stabilize scapula.

improving. Treatment needed to focus on external stabilization of the scapula, while attempting to evoke intrinsic stabilization. This was done to prevent further weakness caused by lengthening the muscle beyond resting length of the middle and lower trapezius.[1,12,13] Additionally, the subacromial impingement syndrome symptoms of pain and inflammation could be directly addressed with treatments such as transverse friction massage, iontophoresis with corticosteroids, and ice massage.

COURSE OF TREATMENT

Scapular strapping/bracing

Initially, the patient's scapula was taped as shown in Figure 4. The taping improved scapular position during elevation (Figure 5) and reduced his posterior shoulder and axilla pain. It was impossible for the patient to apply the tape himself and he developed skin irritation from prolonged use. A posture support was applied that he could don and doff independently (Figure 6). Although his symptoms were better controlled with the tape, the posture support provided enough stability to give significant relief of his symptoms without the negative effects associated with the tape. He wore a T-shirt under the support

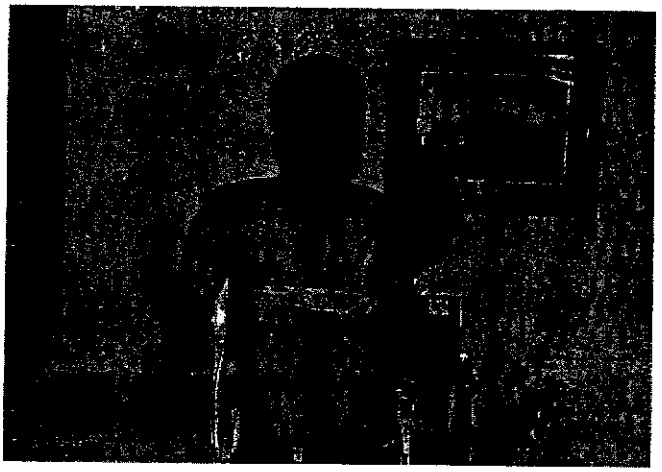

FIGURE 7. Scapular stabilization mechanically aided using mobilization belt.

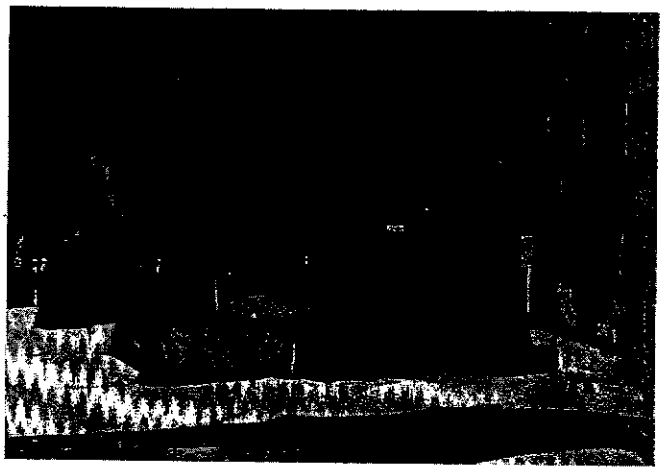

FIGURE 8. Supine position used to stabilize the scapula on the table.

to improve comfort and prevent friction during his 10-hour work days. The posture support and taping were used to assist the function of the scapular stabilizers. Likewise, this was used to prevent stretching of the serratus anterior and middle and lower trapezius.[25] This decreased the demand placed on these muscles as well as the glenohumeral musculature during daily activities. By providing support externally, the support enabled him to continue working. In addition to improving cosmesis and alleviating pain, supports of this nature may also improve muscle endurance.[13,28]

Scapular strengthening/stabilization

Initially, emphasis was placed on strengthening serratus anterior and maximizing middle and lower trapezius function. The demands on the middle and lower trapezius to stabilize the scapula were increased while the serratus anterior was functioning at less than optimal capacity.[30] Because of the increased demand, both serratus and trapezius were prone to fatigue and overuse.[20] Exercise sets and repetitions were initially low (sets of 3 to 5 repetitions) to help reduce this stress and provide optimal opportunity for strengthening to occur. The set was terminated as soon as the patient demonstrated improper technique or poor stabilization. We feel this helped allow the significantly weak muscles the opportunity to strengthen without substitution from stronger muscles. Nuber et al demonstrated that subacromial impingement syndrome can be aggravated by fatigue of the serratus anterior because of improper temporal sequencing of muscle firing.[20] Gradually, repetitions and sets were increased and weight was added to tolerance. Therapeutic exercises were progressed from gravity-minimized to gravity-resisted positions as muscle strength improved.

Subacromial impingement syndrome/tendinitis symptoms

External stabilization of the scapula improved the scapular stability and thereby decreased the stresses creating the subacromial impingement syndrome. The subacromial impingement syndrome symptoms resolved quickly and were never directly treated. Had the subacromial impingement syndrome symptoms continued, additional treatments, including electrical stimulation, ice massage, iontophoresis, and transverse friction massage, could have been used to control pain and inflammation.

Neuromuscular control/rotator cuff exercise

Initial exercises included glenohumeral neuromuscular control drills (eg, rhythmic stabilization with the shoulder in 90 degrees of flexion), isometric shoulder strengthening exercises, and gravity-assisted middle and lower trapezius strengthening. Resistive exercises were progressed quickly for internal and external rotation in neutral as he was able to stabilize the scapula in this plane well. Forward flexion of the shoulder measured at the point of scapular winging was used as a marker of functional improvement. As scapular stabilization improved, endurance became the limiting factor to the patient further achieving his goals. Scapular stabilization was initially mechanically aided using taping (Figure 5), posture support (Figure 6), mobilization belts (Figure 7), or supine positioning to stabilize the scapula on the table (Figure 8) to allow for appropriate strengthening of the rotator cuff while avoiding subacromial impingement syndrome. As coordination improved, exercises were progressed to include strengthening near and above shoulder height without external stabilization to challenge scapular control (Figure 9).

The patient was treated for a total of 10 visits in-

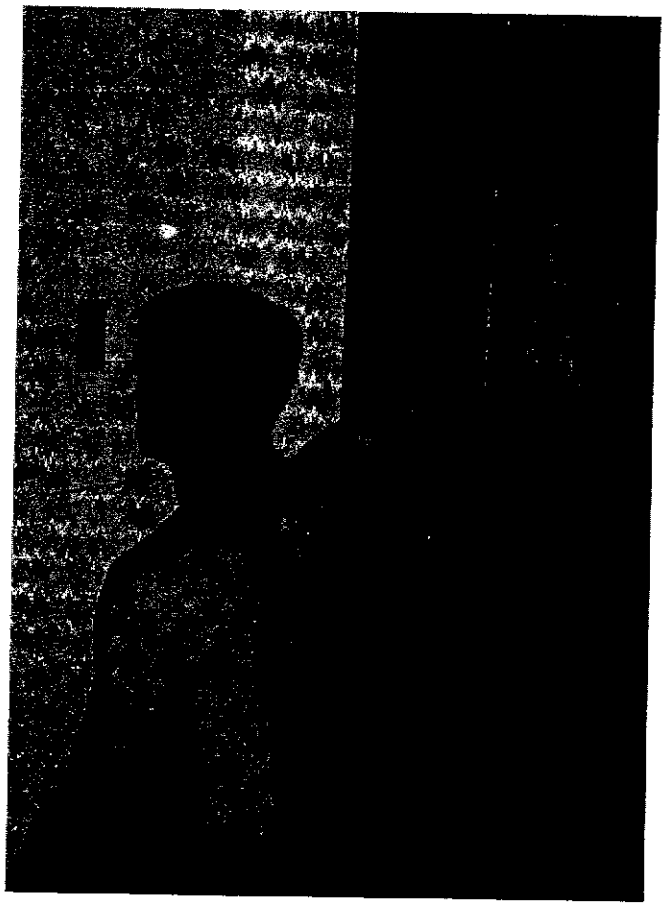

FIGURE 9. Strengthening exercise near and above shoulder height without external stabilization.

cluding the initial consultation in the University of Delaware Specialty Clinic. For the first 2 weeks, he was seen twice a week. When subacromial impingement syndrome symptoms were resolved and he demonstrated the ability to carry out his home exercise program, the frequency of his visits was decreased to once a week for 2 weeks. He continued to make slow, steady progress with strength and functional return and was followed once a month to monitor strength gains. His home exercise program was progressed to match his ability. As Schultz and Leonard mention, prognosis for a long thoracic nerve injury is good[23] but return of function after long thoracic nerve injury can be very slow and take up to 9 to 12 months to recover.[8,9,30] This was reflected in planning the patient's subsequent visits.

The patient's function and strength improved. He gained strength with overhead lifting, although fatigue continued to be a problem. He realized that he needed to pay careful attention to stabilizing his scapula with any upper extremity function. Forward flexion improved to 135 degrees prior to winging. Manual muscle testing revealed improved strength of the right upper extremity: serratus anterior to 4−/5; middle trapezius 5−/5; lower trapezius 4+/5; and shoulder strength 5/5, except abduction 4+/5 and flexion 5−/5, which continued to be influenced by the serratus anterior weakness. Fatigue was reflected by scapular winging after 3 to 4 repetitions of shoulder flexion and abduction exercise. Further strengthening exercises with emphasis on scapular stabilization continued to encourage return of serratus function. Subjectively, he reported his shoulder to be 100% but the stability of his scapula had improved to 85%. He did not feel 85% limitation with scapular stability impaired his function at work.

Impression #4

Serratus anterior strength improved. We never really identified the cause and also never determined the origin of the anatomic damage. We only knew that he had a very weak serratus anterior at the time of initial examination and that his function improved. Full functional return is expected following long thoracic nerve palsy; however, it is not uncommon for some scapular winging to persist.[23]

The patient never had radiologic, MRI, or electrophysiologic testing. Had his condition worsened, EMG would probably have been the most helpful test for defining a prognosis for recovery of his serratus anterior function. Had the function of his serratus anterior not recovered and training of the trapezius been insufficient to maintain scapular stability, scapular reconstructive surgery may have been necessary. Reconstructive surgery usually involves some form of muscle transfer and is typically technically challenging. Even when done by an expert shoulder surgeon, success is limited.[5,9] In a small sample, satisfactory results were fairly high (91%); however, it was recognized that strict adherence to excellent surgical technique and rehabilitation procedures plays an important role in success.[5] Gregg et al recommend surgical intervention only be considered after 2 years of unsuccessful rehabilitation.[9]

CONCLUSION

A definitive medical diagnosis, (ie, long thoracic nerve palsy) was not necessary to effectively treat this patient and in fact, would not have helped to direct *physical therapy* treatment. The clinical diagnosis of serratus anterior muscle weakness and resultant scapular instability causing subacromial impingement syndrome signs directed the care of this patient. Early recognition of scapular winging and rapid intervention have been shown to improve prognosis of recovery from long thoracic nerve palsy.[10] We believe that rapidly identifying the impairments that were limiting his function and specific targeting of the treatment to strengthen the weak muscles and not substitute with stronger muscles resulted in a rapid return of function.

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