American Academy of Physical Medicine and Rehabilitation

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January 1999 EMG Case-of-the-Month

This case is no longer available for CME credit.

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HISTORY

A 30-year-old, right-handed male body builder presents with a four week history of right shoulder pain and weakness. He noted onset of the pain while performing an overhead lift, then completed his workout without further incident. Over the next several weeks, he experienced increasing discomfort and weakness in the shoulder, interfering with his weight training. There has also been prominence of the right scapula, causing discomfort when sitting in a chair. The pain is described as dull and aching in character without radiation. He denies sensory symptoms in the right hand and problems with other limbs.

- **Prior to continuing, please develop a differential diagnosis and list each possible diagnosis in order of likelihood.**
- **Is there any additional information regarding the clinical history that might be helpful in clarifying your differential list or changing its order of priority?**

COMMENTARY I

The differential diagnosis at this stage is quite broad. Neurogenic causes include an anterior horn cell lesion, cervical radiculopathy and upper trunk brachial plexopathy. A focal nerve injury about the shoulder girdle, such as axillary, suprascapular, long thoracic and spinal accessory neuropathy should also be considered. Neuralgic amyotrophy is a possibility, however the character of the pain and onset during activity make this less likely. Fascioscapulohumeral muscular dystrophy, limb girdle muscular dystrophy and a scapuloperoneal syndrome may cause shoulder weakness, however, the unilateral presentation is atypical for these disorders. Musculoskeletal causes include rotator cuff injury, biceps tendinopathy and muscle strain.

HISTORY, continued

There is no history of a recent viral illness, surgical procedure or immunization. He is otherwise healthy without history of diabetes mellitus, connective tissue disease or thyroid disorder. His only medication is ibuprofen, taken as needed for shoulder pain. Family history is negative for neuromuscular disease. Review of systems is non-contributory.

- **If necessary, revise your differential diagnosis based on the additional clinical history.**
- **On which details of the physical examination should you focus at this point?**

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COMMENTARY II

The negative family history makes muscular dystrophy and scapuloperoneal syndrome unlikely diagnoses. There is no history of precipitating factors often associated with neuralgic amyotrophy.

PHYSICAL EXAMINATION

The physical examination reveals winging of the right scapula at rest. Muscle bulk is otherwise normal and symmetric. Strength is normal in the upper and lower extremities. There is no facial weakness. Sensation is intact in the upper extremities. Muscle stretch reflexes are normal and symmetric. Cervical range of motion is normal and pain-free. Spurling maneuver is negative. There is no tenderness over the supraspinatus insertion or bicipital groove. Impingement sign is negative. Active abduction of the right shoulder is full, but scapulohumeral rhythm is grossly abnormal.

- **At this point, review your differential diagnosis and revise as appropriate.**
- **Are there additional observations on physical examination that might be helpful in narrowing your differential list?**

COMMENTARY III

The presence of unilateral scapular winging connotes a neurogenic process and is highly suggestive of a long thoracic or spinal accessory neuropathy. Additional provocative maneuvers should be performed to better characterize the winging. Cervical radiculopathy and upper trunk plexopathy are less likely, given the lack of other neurologic abnormalities.

PHYSICAL EXAMINATION, continued

Shoulder heights are symmetric. Forward flexion of the arms produces marked winging of the entire medial border of the right scapula. Winging is accentuated by performing a push up against a wall. Abduction does not increase the winging.

- **If necessary, revise your differential diagnosis based on the additional physical findings.**
- **Design your approach to the electrophysiologic examination based on the existing data.**

COMMENTARY IV

This type of scapular winging is consistent with serratus anterior weakness. The primary function of the serratus anterior muscle is to stabilize the medial scapula against the thorax when the upper limbs are thrust forward. This particular action is unique to the serratus and accounts for the characteristic winging seen with weakness of this muscle. The serratus anterior also functions as an upward rotator of the scapula, thus active abduction of the limb is typically limited. In this patient, shoulder abduction is full, indicating that the lower trapezius, another upward rotator, is compensating for the weak serratus.

Isolated trapezius weakness results in a different clinical picture. The lateral scapular angle is left unsupported and pulled downward by the weight of the arm, resulting in a drooped

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shoulder on the affected side. A compensatory increase in the activity of the rhomboids and levator scapulae produces an exaggerated downward rotation of the scapula. In contrast to serratus anterior weakness, trapezius weakness produces winging which is most pronounced with abduction rather than forward flexion of the limb.

ELECTROPHYSIOLOGIC DATA

ELECTROMYOGRAPHY

n = normal incr = increased decr = decreased 0 = absent
1+ = minimal 4+ = maximal
crd = complex repetive discharge fasc = fasciculation potential
myk = myokymic discharge myt = myotonic discharge
nmt = neuromyotonic discharge
p wave = positive sharp waves fib = fibrillation potentials
recr = recruitment
amp = amplitude dur = duration poly = polyphasic potential

R/L	MUSCLE	INSERTION		SPONTAN		VOLUNTARY				
		activ	p wave	fib	other	recrt	amp	dur	poly	effort
R	biceps	n	0	0	0	n	n	n	n	n
R	triceps	n	0	0	0	n	n	n	n	n
R	deltoid	n	0	0	0	n	n	n	n	n
R	pronator teres	n	0	0	0	n	n	n	n	n
R	FIDH	n	0	0	0	n	n	n	n	n
R	trapezius	n	0	0	0	n	n	n	n	n
R	serratus anterior	incr	1+	1+	2+	n	n	n	n	n
R	paraspinals mid cervical	n	0	0	0	-	-	-	-	-

SENSORY NERVE CONDUCTION

nr = no response

NERVE	LATENCY			AMPLITUDE (µV)			CONDUC VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
median	-	-	-	-	-	-	-	-	-

wrist to index	3.3	-	<3.7	26	-	>20	52	-	>52
ulnar	-	-	-	-	-	-	-	-	-
wrist to 5th digit	3.5	-	<3.5	22	-	>10	54	-	>52

MOTOR NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE(mV)			CONDUC VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
median	-	-	-	-	-	-	-	-	-
wrist to thenar	3.4	-	<4.4	5.4	-	>4.0	-	-	-
median	-	-	-	-	-	-	-	-	-
elbow to thenar	7.2	-	-	5.3	-	-	-	53.9	>49
ulnar	-	-	-	-	-	-	-	-	-
wrist to hypothenar	3.4	-	<3.6	-	12.8	-	>6.0	-	-
long thoracic	-	-	-	-	-	-	-	-	-
erbs to 6th rib	3.6	4.0	<4.0	1.6	6.5	>4.0	-	-	-

Needle electromyography demonstrates evidence of denervation and decreased voluntary motor unit recruitment in the serratus anterior. Nerve conduction studies are normal with the exception of decreased amplitude of the right serratus anterior CMAP. The long thoracic nerve is assessed by placing the recording electrode over the fifth or sixth rib in the midaxillary line, the reference electrode over the same rib in the anterior axillary line and stimulating at Erb's point. No additional electrophysiologic data are offered.

- **On the basis of both the clinical and electrophysiologic evaluations, formulate your diagnostic impression. List the most likely diagnosis first and follow in order with the other possibilities that are not excluded by the data. Eliminate those diagnoses not supported by the data.**
- **Are there additional electrophysiologic data that you feel would further delineate the diagnosis? (Remember, collecting data that are not needed for the diagnosis is costly and uncomfortable for the patient.)**

ELECTROPHYSIOLOGIC DATA, continued

No additional electrophysiologic data were collected.

- **Make the final revisions of your diagnostic impression(s).**

DIAGNOSTIC IMPRESSION

Right Long Thoracic Neuropathy

The clinical presentation and physical examination are typical of this disorder. The electrodiagnostic abnormalities are limited to the long thoracic nerve (LTN), confirming the diagnosis. The decreased LTN compound muscle action potential and abnormal spontaneous

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activity present in the serratus anterior muscle indicate an axon loss lesion. A significant number of motor units remain viable, suggesting a favorable prognosis. The motor unit action potential morphology in the serratus anterior is normal, consistent with his symptom duration of four weeks. The normal median sensory nerve action potential and normal needle examination of the biceps and deltoid argue against an upper trunk brachial plexopathy. The trapezius is also normal, thus a spinal accessory nerve injury is highly unlikely. Cervical radiculopathy is effectively excluded by the lack of abnormalities in the cervical paraspinals and other limb muscles.

The LTN is a purely motor nerve arising from branches of the fifth, sixth and seventh cervical nerve roots shortly after they exit the intervertebral foramina. The branch from the seventh root is absent in 8% of patients. The nerve occasionally receives a contribution from the eighth cervical root. The branches from the fifth and sixth roots unite immediately after piercing the scalenus medius muscle. The branch arising from the seventh root joins the nerve as it courses across the scalenus posterior muscle. It then travels along the outer surface of the serratus anterior just posterior to the midaxillary line, supplying each digitation of the muscle as it progresses. The lower portion of the serratus is innervated by the intercostal nerves in approximately 20% of patients.

Injury to the LTN is frequently attributed to trauma, including motor vehicle accidents, surgical, carrying a knapsack, lifting and electric shock. Athletic activity, especially tennis, is also associated with long thoracic neuropathy. Other reported activities include baseball, football, hockey, gymnastics, weight lifting, wrestling, golf, bowling and archery. The LTN is vulnerable to injury from direct, blunt trauma as well as traction forces. Raising the arm overhead and turning the head to the opposite side is reported to place a significant stretch upon a proximal segment of the LTN. In this patient, overhead lifting appears to have caused a traction-induced injury to the nerve.

Long thoracic neuropathy is also reported to occur following viral infection, immunization, cold exposure and childbirth. These cases likely represent a variant of neuralgic amyotrophy. Long thoracic neuropathy is frequently the only manifestation of this disorder.

Treatment includes relative rest, shoulder range of motion exercises and strengthening of the serratus anterior and other scapular stabilizer muscles. Activities that cause overstretching of the serratus should be avoided. Scapular bracing is helpful in some patients. The prognosis for recovery in long thoracic neuropathy is favorable. Most patients recover with minimal or no residual dysfunction, although scapular winging may persist in some. The average recovery time is nine months but may take as long as two years. If there is no electrophysiologic evidence of nerve function at two years, surgical stabilization of the scapula should be considered.

The patient responded well to a rehabilitation program. One year later, he had regained full functional use of his right arm with only mild residual winging of the scapula. A follow up EMG at that time demonstrated improved LTN function. He was able to resume his weight training program, however, the residual winging prevented a return to competitive bodybuilding.

- **What other diagnostic procedures (laboratory tests, etc.), if any, are needed?**
- **What treatment would you recommend?**

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