JSES Reviews, Reports, and Techniques 4 (2024) 329–340

Contents lists available at ScienceDirect

JSES Reviews, Reports, and Techniques

journal homepage: www.jsesreviewsreportstech.org

Paralysis of the trapezius muscle: evaluation and surgical management

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A R T I C L E I N F O

Keywords:
Trapezius
Iatrogenic injury
Spinal accessory nerve
Levator scapulae
Rhomboids
Eden-Lange
Muscle paralysis
Trapezius palsy

Level of evidence: Level V; Review Article; Expert Opinion

Background: Paralysis of the trapezius muscle most commonly results from iatrogenic injury to the spinal accessory nerve.
Methods: The clinical presentation and physical examination findings of trapezius palsy have been well characterized, but unfortunately the diagnosis of this condition is oftentimes missed or delayed, sometimes leading to unnecessary surgery on the rotator cuff or tendon of the long head of the biceps.
Results: The diagnosis can be confirmed using electromyography with nerve conduction studies. Although nonoperative treatment may help some patients with temporary neurapraxia of the spinal accessory nerve, nerve repair with or without nerve grafting should be performed soon for patients suspected of a nerve transection. Nerve transfers can be considered within the first year after the injury when nerve repair and grafting cannot be completed. For chronic trapezius palsy, transfer of the levator scapulae and rhomboids has been refined and represents a very successful surgical procedure. Rarely, scapulothoracic arthrodesis is considered for individuals with failed tendon transfers or multiple nerve involvement.
Conclusion: Trapezius palsy is oftentimes missed. An accurate diagnosis allows consideration of various treatment modalities that have been reported to provide good outcomes for properly selected patients.

The trapezius muscle is one of the most important periscapular muscles. Loss of trapezius function can be associated with profound functional limitations and pain. Although loss of trapezius function can be due to (1) developmental abnormalities (trapezius agenesis or hypoplasia), (2) iatrogenic trapezius muscle damage during certain surgical exposures, (3) traumatic trapezius rupture, and (4) brachial plexus palsy, the most common condition leading to loss of trapezius function is dysfunction of the *spinal accessory nerve* (SAN), leading to trapezius paralysis. Certain reconstructive procedures currently considered for trapezius paralysis can also be beneficial for patients with the other conditions mentioned above, specifically tendon transfers. So called functional scapular dyskinesis, where patients develop loss of neuromuscular control of periscapular muscles, also presents with associated partial loss of trapezius function.

This review article summarizes current concepts regarding trapezius paralysis (or palsy) due to dysfunction of the SAN. Recognition of this condition is particularly important because, not uncommonly, it goes undiagnosed for quite some time, and may lead to unnecessary surgery, such as subacromial decompression, biceps tenodesis, or cuff débridement. In addition, when nerve reconstruction is not an option, transfer of the rhomboids and levator to the spine of the scapula can dramatically improve pain and function for patients with trapezius palsy.

Anatomic review

The trapezius muscle

Each trapezius muscle is flat and triangular, with the base of this triangle along the midline and the apex pointing to the scapula. Together, the trapezius muscles on both sides form the shape of a trapezium. Each trapezius originates from the superior nuchal line, external occipital protuberance, ligamentum nuchae, and the spinous processes of C7 to T12. The trapezius inserts into the clavicle, acromion and spine of the scapula (Fig. 1, *A* and *B*).[10]

Contraction of the trapezius fibers can result in movement of the scapula and/or the head, neck and spine. Regarding *the head, neck*

Institutional review board approval was not required for this review article. Investigation performed at Mayo Clinic Rochester.

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https://doi.org/10.1016/j.xrrt.2024.03.014
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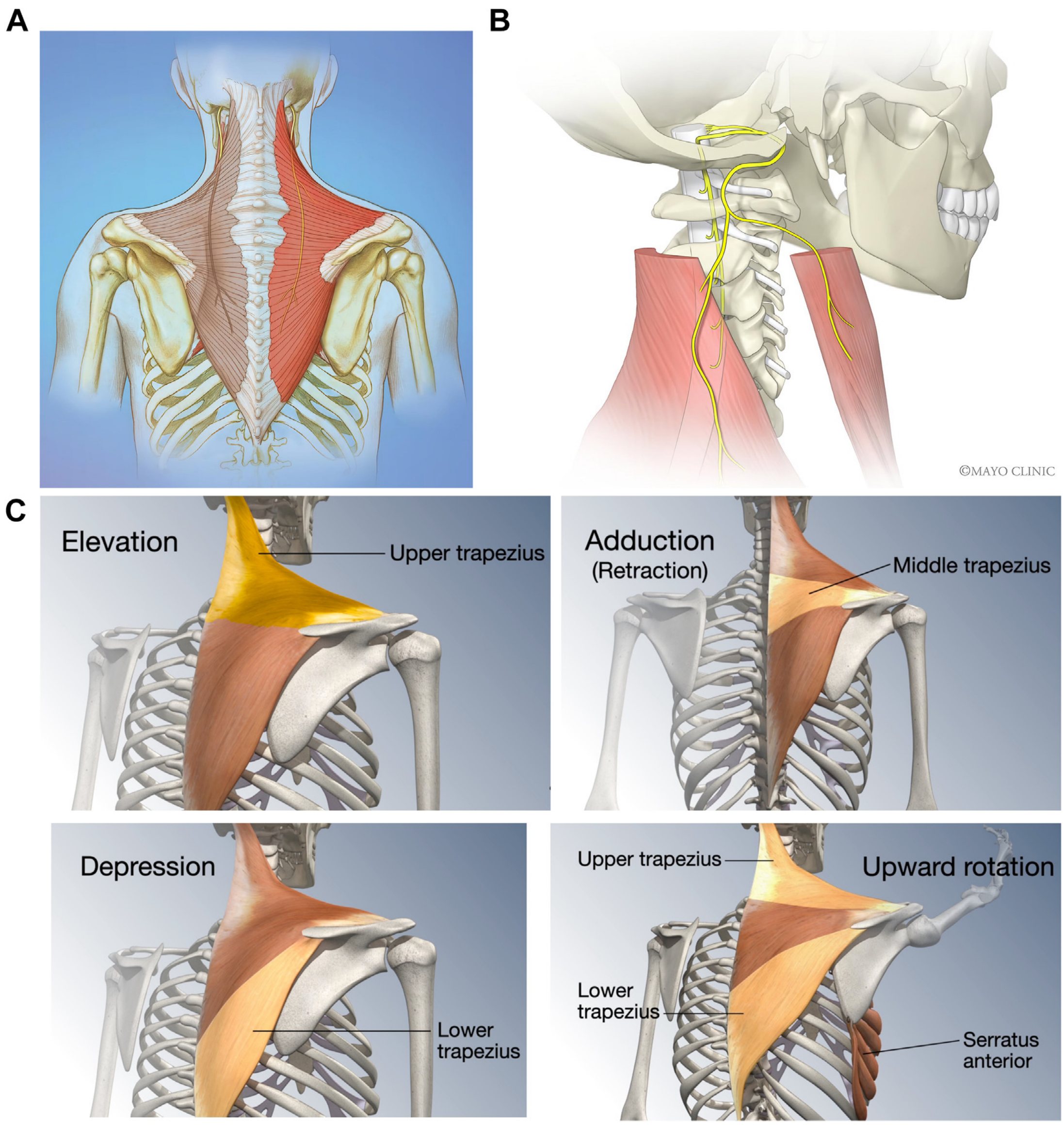

Figure 1 (**A**) Anatomy of the trapezius muscle (**B**) and spinal accessory nerve (cranial nerve XI). (**C**) Schematic representation of the mechanical function of the trapezius.

and spine, the trapezius extends and laterally flexes the head and neck to the side of the contraction and rotates the head and neck to the opposite side; it also contributes to extend the spinal column. Regarding the effect of trapezius contraction on the *scapula*, conceptually, the trapezius is considered to have 3 functional parts: upper, middle, and lower trapezius (Table I, Fig. 1, *C*).

In the resting position, the trapezius is the largest muscle responsible for *suspension of the scapula on the thorax*. The main functions of the trapezius include (1) maintaining the scapula against the chest wall and (2) *rotating the scapula upwards* with slight extension, so that the glenoid points superiorly and slightly posteriorly. Insertion of the trapezius fibers into the scapular spine, acromion and lateral clavicle is biomechanically advantageous for scapular upward rotation. Additionally, the upper trapezius elevates the scapula superiorly ("shoulder shrug"), whereas the middle trapezius retracts the scapula closer to the midline, and the lower trapezius contributes to lower or depress the scapula.[10]

The scapular contribution to shoulder elevation by upward rotation and extension while remaining close to the chest wall represents a beautiful example of human muscle coordination, with the trapezius controlling the scapula at the acromion and scapular spine posterosuperior while the serratus anterior controls the inferior pole of the scapula from anterior.

Trapezius innervation: the SAN

The SAN (cranial nerve XI) is responsible for the majority of the motor innervation of the trapezius; however, the trapezius is also partially innervated by the cervical plexus,[38] which explains why the lower trapezius may be spared in isolated SAN injuries. The ventral rami of C3 and C4 are responsible for its sensory innervation and proprioception.

The SAN arises from a column of ventral horn cells (accessory nucleus, levels C1 to C5) and its fibers coalesce in a single spinal root that first enters the cranium through the foramen magnum and then exits the cranium through the pars vascularis of the jugular foramen.

The SAN then descends between the internal carotid artery and the internal jugular vein, posterior to the stylohyoid and digastric

Table I
Anatomy and function of the trapezius muscle.

	Origin	Insertion	Action	Agonists
Whole trapezius			Scapular suspension Upward rotation	Serratus anterior
Upper trapezius	Superior nuchal line to C6	Clavicle and acromion	Elevation	Levator scapulae
Middle trapezius	C7 to T4	Acromion and superior scapular spine	Retraction	Rhomboids
Lower trapezius	T4 to T12	Medial third of the inferior scapular spine	Depression	Latissimus dorsi Pectoralis minor

Table II
Causes of trapezius palsy.

Condition	Cause
Central conditions	- Contralateral hemiplegia - Low spinal cord lesions - Tumors/injuries at the foramen magnum or jugular foramen
Brachial plexus conditions	- Traumatic brachial plexus injuries - Parsonage-Turner syndrome
SAN injury at the posterior cervical triangle	- Blunt or penetrating trauma - Iatrogenic (surgery and other procedures involving the posterior cervical triangle)
Neck radiation leading to SAN neuropathy	

SAN, spinal accessory nerve.

Table III
Physical examination findings in trapezius palsy.

Inspection	- Trapezius atrophy - Increased distance between spinous processes and the medial border of the scapular body
Active motion	- Abduction is limited or weak - Flexion may be reasonably well-maintained - Scapula does not rotate upwards with attempted elevation or abduction
Strength testing	- Weak shoulder shrug - Weak scapular retraction against resistance - Weak horizontal arm extension, patient prone, shoulder in external rotation and 90° (middle) or 120°-135° (lower) of abduction
Special tests	- Scapular flip sign - Active elevation lag sign - Triangle sign - Scapular assistance test (+/−) - Assessment of sternocleidomastoid strength

muscles, to enter and innervate the sternocleidomastoid muscle. It then emerges lateral to the sternocleidomastoid muscle (in close proximity to the great auricular nerve) to cross the posterior cervical triangle (anterosuperior trapezius border, posterior sternocleidomastoid border, and clavicle) on the surface of the levator scapulae to innervate the trapezius muscle.

Etiology

Trapezius palsy may be present in patients with contralateral hemiplegia, low spinal cord lesions (infarction, tumor or syringomyelia), injuries or tumors involving the foramen magnum or the jugular foramen, and traumatic brachial plexus injuries (Table II).

However, the most common reason for trapezius palsy is an injury to the SAN at the posterior neck triangle. These injuries can be the result of blunt or penetrating trauma or radiation but most commonly occur as iatrogenic lesions complicating procedures performed at the posterior cervical triangle (lymph node biopsy, surgical neck dissection for management of malignancy or vascular conditions, carotid endarterectomy, canulation of the inferior jugular vein, and other).[36] Many variations of the surface anatomy of the spinal accessory neck at the posterior cervical triangle have been described,[39] which explains why injury to the SAN is so difficult to prevent during procedures in the posterior cervical triangle; the SAN is the most commonly iatrogenically injured nerve to the shoulder.[25]

Additionally, complete or partial dysfunction of the SAN may be the result of inflammatory brachial plexopathy (Parsonage-Turner syndrome (PTS)). Patients with PTS may experience partial or incomplete recovery, with may lead to permanent trapezius paresis or paralysis. When no cause for trapezius palsy can be identified, it probably represents sequelae of PTS.[14]

Evaluation

Unfortunately, the diagnosis of trapezius palsy continues to be missed. In our practice, we have evaluated several patients with classic, isolated trapezius palsy who were wrongly diagnosed with biceps or cuff tendinopathy, were thus subjected to biceps tenodesis or rotator cuff débridement procedures with acromioplasty, and did not improve. This is partly due to the fact that loss of trapezius function will allow anterior scapular flexion or tilt, with symptoms consistent with secondary subacromial impingement. However, many trapezius palsies are missed because the cause of SAN injury is not identified in the history or in some cases because patients are examined with their shirts on and without a dedicated scapulothoracic exam.

History

When a patient presents with shoulder pain and dysfunction after a prior invasive procedure or radiation at the side of the neck, the index of suspicion for a possible trapezius palsy should prompt a dedicated evaluation of the trapezius. The same is true for patients referred with continuous shoulder issues after an otherwise resolved PTS. Pain is a common complaint of most patients with trapezius palsy; some attribute this pain to constant traction on the brachial plexus caused by shoulder drooping.

Physical examination

A few physical examination findings can be identified in patients with trapezius palsy (Table III). On inspection, atrophy may be noted, especially when comparing the bulk of the upper trapezius at the base of the neck with the opposite side. In the relaxed, resting position, the distance between the midline (vertebral spinous processes) and the medial border of the scapular body can be measured with a tape or ruler, and will be increased on the affected side (Fig. 2).

Some patients with trapezius paralysis are still able to reasonably elevate their shoulders, but abduction is typically limited or very weak. Careful comparison of scapular motion with attempted

Figure 2 Physical examination findings in patients with trapezius palsy (*Right* side). (**A**) Increased distance between midline and scapula in the resting position. (**B**) Limited active abduction. (**C**) Inability to properly shrug. (**D**) Weakness of scapular retraction against resistance. (**E**) Scapular flip sign.

active elevation will show compromised upward scapular rotation on the affected side.

We typically compare the strength of the trapezius with the contralateral sign by asking the patient to shrug the shoulders against resistance and to retract the shoulders against resistance; slight arm abduction during shrug can relax the rhomboids and help isolate the trapezius. Some prefer to test the strength of the middle and lower trapezius with the patient prone and the shoulder in abduction and external rotation; trapezius strength is tested by applying downward force on the arm in 90° of abduction (middle trapezius) and 120°-135° of abduction (lower trapezius). SAN injuries proximal to the posterior cervical triangle may also present with weakness of the sternocleidomastoid muscle, tested with resisted rotation of the chin to the opposite side.[29]

Some maneuvers have been described to unmask trapezius palsy. The so-called "*scapular flip sign*" (resisted active external rotation test) demonstrates posterior protrusion of the medial scapular border with resisted external rotation with the arm at the side.[7] The "*active elevation lag sign*" is assessed with the patient standing: on the affected side, the only way a patient with a trapezius palsy can fully elevate the arm is by spine hyperextension. With bilateral attempted elevation, if the examiner prevents spine hyperextension, elevation on the affected side lags behind the normal side. The "*triangle sign*" is similar to the active elevation lag

Figure 3 Plain anteroposterior (**A**) and axillary (**B**) radiographs show very lateral position of the scapula in reference to the chest wall, in addition to metallic anchors for rotator cuff surgery that retrospectively had been unnecessary. (**C**) Magnetic resonance may show atrophy and fatty infiltration of the *Right Upper* trapezius (→) compared to the *Left Upper* trapezius (→).

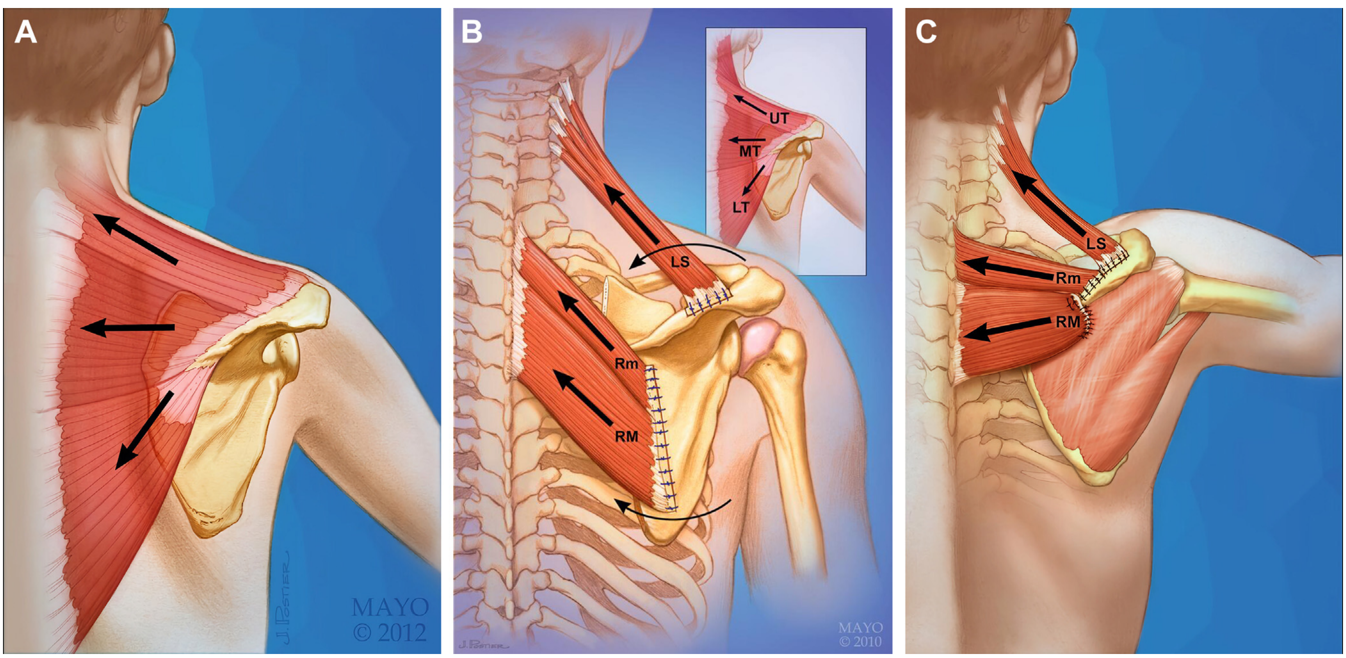

Figure 4 Schematic representation of (**A**) the 3 portions of the trapezius, (**B**) transfer of the levator and rhomboids according to Eden and Lange, and (**C**) the Elhassan modification of this procedure.

sign but performed prone: attempted forward flexion beyond 120° will not be possible; the patient attempts to compensate with hyperextension of the trunk, and the arm and trunk form a triangle with the examining table.[29] The "*scapular assistance test*" consists in supporting the scapula against the chest wall by the examiner to determine whether active elevation is easier; this test can dramatically change the ability to elevate the shoulder in patients with serratus anterior weakness, but not so much in patients with trapezius palsy.

Radiographs

Plain radiographs should be obtained in all patients evaluated for shoulder complaints. At first look, radiographs will appear normal in patients with a trapezius palsy. A very subtle finding may be observed secondary to the lateral drift of the scapula: radiographs will show much less overlap between the scapular body and the chest than in normal circumstances (Fig. 3, *A*). Radiographs may also show anchors or other implants in patients who have previously undergone rotator cuff surgery or a biceps tenodesis. One additional value of radiographs is to exclude bony deformities which could lead to a similar presentation, such as scapular malunion after trauma.

Magnetic resonance

To properly evaluate the trapezius with magnetic resonance imaging (MRI), the field of view needs to be the chest as opposed to the shoulder region. On the coronal views, atrophy and neurogenic fatty infiltration of the upper trapezius can easily be appreciated (Fig. 3, *B*). Axial cuts also allow comparative assessment of the trapezius on each side for changes in muscle bulk and increased signal change in the T1 and T2 sequences. As with x-rays, 1 additional value of the MRI is to exclude other causes of scapular abnormalities, such as osteochondromas or elastofibroma dorsi.[30]

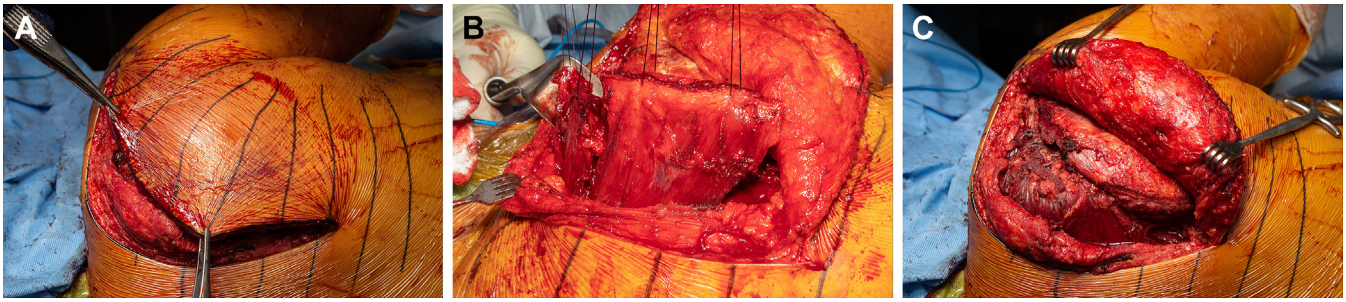

Figure 5 Levator scapulae and rhomboids transfer. (**A**) Curvilinear skin incision with elevation of a large subcutaneous flap. (**B**) Harvested levator scapulae and rhomboids. (**C**) Completed transfer.

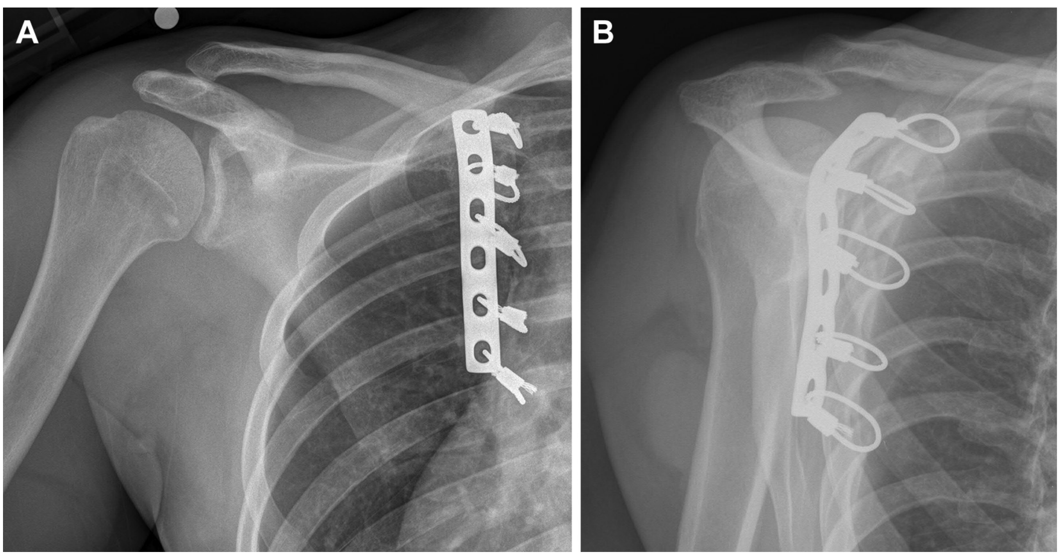

Figure 6 Postoperative radiographs after scapulothoracic arthrodesis. (**A**) Anteroposterior. (**B**) Lateral.

Electromyogram with nerve conduction studies

Well-conducted electromyograms with nerve conduction studies will confirm dysfunction of the SAN. However, since the trapezius is a relatively flat muscle, it can be so thin when atrophied that it may be difficult to know for sure whether the electromiogram (EMG) needles are inside the confines of the trapezius or too deep and into the rhomboids. Needle insertion into a denervated trapezius will show increases in insertional activity, fibrillation potentials, and positive sharp waves and will show reductions in muscle fiber recruitment and the number of rapidly firing motor unit action potentials (MUAPs). Classically, when reinnervation occurs, MUAPs become longer, polyphasic, and of increased amplitude.[31]

Management

Patients with isolated trapezius palsy may be best suited for conservative treatment, nerve procedures, tendon transfers, or scapulothoracic arthrodesis depending on the nature of the injury, time of presentation, and other factors. On one hand, nerve repair should be considered promptly when acute transection of the SAN has occurred secondary to penetrating trauma or iatrogenic surgery. On the other hand, mild stretch injuries may recover spontaneously, and patients presenting years after their index injury are not candidates to nerve procedures and best helped with tendon transfers or alternative salvage procedures.

Nonoperative treatment

Nonoperative treatment should only be considered for (1) individuals with a very high likelihood of spontaneous recovery or (2) tolerable subjective symptoms in patients able to cope. The challenge is to estimate the chances of spontaneous recovery: electromyographic findings consistent with loss of SAN continuity (complete absence of MUAPs) should prompt surgery, but there are reports of patients with MUAPs on EMG despite confirmation of complete transection of the SAN, possibly due to cervical plexus contributions to the innervation of the trapezius.[38] In questionable cases, MRI or ultrasound evaluation of the SAN may be considered. As such, in addition to EMG and imaging findings, serial physical examinations are very important, and conservative treatment should only continue when there is evidence of progressive recovery of strength.

Nonoperative treatment consists of pain control modalities, mechanical support of the shoulder girdle, and physical therapy. Pain may be managed with acetaminophen, nonsteroidal anti-inflammatory drugs, and gabapentin or pregabalin. Transcutaneous nerve stimulation and the occasional use of regional nerve blocks may be considered for patients with severe pain. Mechanical support of the shoulder girdle can provide some comfort as well. Patients should avoid carrying objects on the affected side. In addition, the shoulder girdle may be supported by placing the patient's hand in a pants pocket or use of a sling. Dedicated orthosis such as the Akman-Sari orthosis may be considered as

Table IV
Reported outcomes for various treatment modalities in patients with trapezius palsy.

Year	First author	Start of title	Treatment category	Procedure specific type	n	Follow-up (mo) mean (range)	General outcome (improvement)	Pain (VAS are for the averages)	Overhead function restored	Elevation/ Flexion/ Abduction	Reop %	Complications %	Satisfied %	Other metrics
2007	Shimada	Clinical Results of Rehabilitation	Nonoperative	Occupationnal therapy	35	2	35/35 improved ROM and function significantly	2.6-2.0	N/A	FF 106-125; Abd 57-65	N/A	N/A	N/A	N/A
2003	Romero	Levator Scapulae and Rhomboid	Nonoperative	Conservative treatment	4	180 (24-396)	0/4 regained satisfactory function	Disappeared in 2/4	0/4	N/A	N/A	N/A	N/A	N/A
2002	Chida	Occupational Therapy for ANP	Nonoperative	Occupational therapy	10	3	10/10 Improved function; 3/10 improved pain	N/A	N/A	FF 107-135; Abd 59-78	N/A	N/A	N/A	N/A
1991	Ogino	AN Injury	Nonoperative	Passive motion & shoulder exercise	10	28 (8-60)	Improved function (6/10), strength (4/10)	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1985	Bigliani	Treatment of Trap. Paralysis	Nonoperative	Conservative treatment	8	30 (12-54)	1/8 Improved ROM, stability, and pain	Some relief in 5/8	N/A	N/A	N/A	N/A	N/A	N/A
1984	Petrera	Conduction Studies along the AN	Nonoperative	Time (Self-healing)	10	N/A (11-96)	Incomplete Recovery	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1993	Donner	Extracranial SAN Injury	Nonoperative	Conservative treatment	34	14 (5-96)	29 of 34 improved	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2019	Mayer	Reconstruction of the SAN	Nerve	Nerve transfer	5	20 (12-26)	Good/ excellent: 5/5	7 to 1	5/5 = 100%	Abd 55-151	N/A	Winging persisted 2/5 = 40%	N/A	Medical Research Council Grade 4: 2, Grade 5: 3
2018	Cambon-Binder	SAN Repair Using a Direct Nerve	Nerve	Neurolysis or Transfer	11	25 (11-53)	Good/ excellent: 7/11	N/A	N/A	Abd 95-151	N/A	Unstable Scapula: 4/11	N/A	Medical Research Council Grade 3:1, Grade 4-5: 10
2016	Göransson	Patient Outcome after Surgical	Nerve	Neurolysis	24	131 (SD of 36)	Subjective: 1 excellent, 10 good, 11 fair, 2 poor	N/A	N/A	FF 136-137; Abd 103-152	N/A	Atrophy: 15 slight, 6 greater	N/A	Subjective pain: No: 7, Controllable: 8, Severe: 9
2016	Göransson	Patient Outcome after Surgical	Nerve	Nerve repair	9	101 (SD of 62)	Subjective: 2 excellent, 2 good, 2 fair, 2 poor	N/A	N/A	FF 129-143; Abd 83-159	N/A	Atrophy: 2 slight, 5 greater	N/A	Subjective pain: No: 3, Controllable: 2, Severe: 3, Ungovernable: 1
2016	Göransson	Patient Outcome after Surgical	Nerve	Nerve grafting	4	117 (SD of 65)	Subjective: 4 fair	N/A	N/A	FF 138-143; Abd 135-173	N/A	Atrophy: 2 slight, 2 greater	N/A	Subjective pain: Controllable: 2, Severe: 2
2011	Bertelli	Combined Injury of the AN	Nerve	Nerve grafting	2	N/A	Recovered shoulder elevation	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2011	Bertelli	Combined Injury of the AN	Nerve	utr	3	N/A	Recovered shoulder elevation	N/A	N/A	N/A	N/A	N/A	N/A	N/A

(*continued on next page*)

Table IV (*continued*)

Year	First author	Start of title	Treatment category	Procedure specific type	n	Follow-up (mo) mean (range)	General outcome (improvement)	Pain (VAS are for the averages)	Overhead function restored	Elevation/ Flexion/ Abduction	Reop %	Complications %	Satisfied %	Other metrics
2011	Bertelli	Combined Injury of the AN	Nerve	Neurolysis	1	N/A	Recovered shoulder elevation	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2008	Grossman	Iatrogenic SAN Injury in Children	Nerve	1 Neurolysis, 2 nerve grafting	3	20 (12-24)	Full elevation	N/A	N/A	N/A	N/A	N/A	N/A	Medical Research Council Grade 4: 3/3
2008	Flores	Suprascapular Nerve Release	Nerve	Nerve transfer	1	18	Recovered Abd capabilities	N/A	N/A	N/A	N/A	N/A	N/A	Medical Research Council Grade 3
2006	Bertelli	Refinements in Technique for Repair	Nerve	Nerve grafting	5	36 (24-84)	5/5 improved, full abduction	7 to 2 or 3	5/5 = 100%	N/A	N/A	Slight winging: 3/5 = 60%	4/5 = 80%	N/A
2004	Teboul	Surgical Management of TP	Nerve	Neurolysis, repair, or grafting	20	38 (12-140)	Good/ excellent: 16/ 20	N/A	N/A	Abd 126	N/A	N/A	N/A	N/A
2003	Chandawarkar	Management of Iatrogenic Injury	Nerve	Nerve repair	3	≥12	3/3 free of pain	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2003	Chandawarkar	Management of Iatrogenic Injury	Nerve	Nerve grafting	3	≥12	3/3 free of pain	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2003	Kim	Surgical Outcomes of 111 SAN	Nerve	Nerve repair or grafting	84	27 (12-44)	Function improved; 84/ 84 had pain reduced	N/A	N/A	N/A	N/A	N/A	N/A	Medical Research Council Grade 0-2: 19, Grade 3: 15, Grade 4-5: 50
2003	Kim	Surgical Outcomes of 111 SAN	Nerve	Neurolysis	19	24 (12-39)	19/19 improved greatly in function	N/A	N/A	N/A	N/A	N/A	N/A	Medical Research Council Grade 3: 1, Grade 4-5: 18
2003	Kim	Surgical Outcomes of 111 SAN	Nerve	Nerve transfer or burial into muscle	7	N/A	5/7 improved	N/A	N/A	N/A	N/A	N/A	N/A	Medical Research Council Grade 0-2: 5, Grade 3: 2
1999	Harpf	[Iatrogenic Lesion of the AN]	Nerve	Nerve repair	2	19 (8-30)	0/2 improved	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1999	Harpf	[Iatrogenic Lesion of the AN]	Nerve	Nerve grafting	3	27 (8-36)	3/3 Improved but not well	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1999	Harpf	[Iatrogenic Lesion of the AN]	Nerve	Burial into muscle	1	6	Did not improve	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1996	Williams	The Posterior Triangle	Nerve	Nerve grafting	24	N/A (3-48)	Good/ excellent: 14, Fair: 6, Poor: 4	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1996	Williams	The Posterior Triangle	Nerve	Neurolysis	12	N/A	Good/ excellent: 5, Fair: 4, Poor: 3	N/A	N/A	N/A	N/A	N/A	N/A	N/A

1993	Donner	Extracranial SAN Injury	Nerve	Neurolysis	7	18 (12-24)	7 of 7 improved greatly	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1993	Donner	Extracranial SAN Injury	Nerve	Nerve repair, grafting, or transfer	33	16 (5-22)	31/33 improved	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1993	Donner	Extracranial SAN Injury	Nerve	Burial into muscle	2	N/A	0/2 improved	N/A	N/A	N/A	N/A	N/A	N/A	Medical Research Council Grade 0: 2
1991	Ogino	AN Injury: Conservative or Surgical	Nerve	Nerve suture	2	31 (18-43)	2/2 regained full elevation, pain eliminated	N/A	N/A	Abd 62-180	N/A	N/A	2/2 = 100%	N/A
1991	Ogino	AN Injury: Conservative or Surgical	Nerve	Nerve grafting	5	15 (12-20)	4/5 regained full elevation, pain eliminated	N/A	N/A	Abd 83-180	N/A	N/A	5/5 = 100%	N/A
1991	Ogino	AN Injury: Conservative or Surgical	Nerve	Neurolysis	3	19 (8-36)	3/3 regained full elevation, pain eliminated	N/A	N/A	Abd 66-164	N/A	N/A	3/3 = 100%	N/A
1984	Vastamäki	AN Injury	Nerve	Neurolysis	5	13 (5-29)	Good: 2, fair: 2, poor: 1	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1984	Vastamäki	AN Injury	Nerve	Nerve Repair	2	22 (7-37)	Fair: 1, poor: 1	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1984	Vastamäki	AN Injury	Nerve	Nerve Grafting	2	39 (12-65)	Fair: 1, poor: 1	N/A	N/A	N/A	N/A	Atrophy: 1, Dysesthesia: 1	N/A	N/A
1952	Woodhall	Trap. Paralysis Following Minor	Nerve	Nerve Repair	3	>3	3/3 Regained Abd	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1952	Woodhall	Trap. Paralysis Following Minor	Nerve	Neurolysis	3	>3	3/3 Regained Abd and muscle mass	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2018	Amroodi	Single-Incision Eden-Lange	Tendon transfer	Eden-Lange	11	34 (24-48)	11/11 Improved in pain and ROM	VAS 8-2	N/A	FF 122-154; Abd 80-148	N/A	N/A	N/A	ASES 33-82
2015	Elhassan	Outcome of Triple-Tendon Transfer	Tendon transfer	Triple-Tendon Transfer (T3)	22	35 (13-61)	21/22 greatly improved in position and ROM	Diminished in 21/22	N/A	FF 102-150; Abd 71-118	N/A	1/22 ruptured rhomboid sutures	21/22	SSV 44%-67%, CSS 41-73, DASH 59-21
2008	Galano	Surgical Treatment of Winged	Tendon transfer	Bigliani-modified Eden-Lange	6	47 (16-89)	Improved, winging eliminated	VAS 7-2	N/A	FF 142-151	N/A	Infection: 2 = 33%	6/6 = 100%	ASES 33-65
2008	Flores	Suprascapular Nerve Release	Tendon transfer	Eden-Lange	2	14 (12-15)	2/2 recovered Abd above horizontal	N/A	2/2 = 100%	N/A	N/A	N/A	N/A	N/A
2004	Teboul	Surgical Management of TP	Tendon transfer	Eden-Lange	7	29 (14-54)	Good/ excellent: 4/7	N/A	N/A	Abd 120	N/A	N/A	N/A	N/A
2003	Romero	Levator Scapulae and Rhomboid	Tendon transfer	Eden-Lange	12	384 (156-456)	11/12 excellent/fair	Relieved in 11/12	9/12 = 75%	N/A	1/12 = 8%	N/A	N/A	Median Constant Score 74 vs. 83 contralateral
1996	Bigliani	Transfer of the Levator Scapulae	Tendon transfer	Bigliani-modified Eden-Lange	22	90 (24-168)	19/22 excellent/ satisfactory	Relieved in 19/22	19/22 = 86%	N/A	N/A	N/A	N/A	N/A
1985	Bigliani	Treatment of Trap. Paralysis	Tendon transfer	5 Eden-Lange, 2 Eden-Lange 2-stage	7	42 (24-56)	7/7 improved; 6/7 excellent or satisfactory	Relieved in 6/7	N/A	N/A	N/A	N/A	86%	N/A

(continued on next page)

Table IV (*continued*)

Year	First author	Start of title	Treatment category	Procedure specific type	n	Follow-up (mo) mean (range)	General outcome (improvement)	Pain (VAS are for the averages)	Overhead function restored	Elevation/ Flexion/ Abduction	Reop %	Complications %	Satisfied %	Other metrics
1973	Langenskiöld	Treatment of Paralysis of the Trap.	Tendon transfer	Eden-Lange	3	74 (18-180)	3/3 improved, increased stability	Diminished in 3/3	3/3 = 100%	N/A	0	Some difficulty in abduction: 3/3	3/3 = 100%	N/A
1959	Lange	[The Operative Treatment]	Tendon transfer	Eden-Lange	4	N/A	Strong elevation	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1951	Lange	[Treatment of Paralysis of the Trap.]	Tendon transfer	Eden-Lange	30	N/A	Drooping eliminated	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1924	Eden	[For the Treatment of Trap. Paralysis]	Tendon transfer	1 Eden-Lange, 1 Rhomboid transfer	2	24 (18-30)	Normal Elevation and Abd	Pain disappeared	2/2 = 100%	N/A	0	Infection: 1 = 50%	2/2 = 100%	N/A
2014	Goel	Scapulothoracic Fusion: Outcomes	Salvage procedures	Scapulothoracic Fusion	12	41 (8-72)	Improved	8-3	N/A	FF 90-117	2/12 = 17%	50%	7 patients	SSV 39%-63%
1983	King	Iatrogenic SANP	Salvage procedures	Scapulothoracic Fusion	1	N/A	ROM unimproved, pain improved	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1983	King	Iatrogenic SANP	Salvage procedures	Fascial Sling—Henry Procedure	3	N/A	Improved function, relieved symptoms	N/A	N/A	N/A	N/A	N/A	N/A	N/A

Abd, abduction; *AN*, accessory nerve; *ASES*, American Shoulder and Elbow Surgeons; *ANP*, accessory nerve palsy; *CSS*, constant shoulder score; *DASH*, disabilities of the arm, shoulder and hand; *FF*, forward flexion; *ROM*, range of motion; *SAN*, spinal accessory nerve; *SANP*, spinal accesory nerve palsy; *SD*, standard deviation; *SSV*, subjective shoulder value; *VAS*, visual analogue scale.

well. Physical therapy exercises are instituted to (1) prevent secondary glenohumeral joint contractures and (2) strengthen the remaining periscapular muscles (levator, rhomboids, serratus anterior).

Nerve procedures

As mentioned previously, SAN transections are best treated by nerve repair as soon as feasible. One study suggested that for each month of delay before primary repair of a peripheral nerve injury, the odds of recovery significantly decrease.[22] In addition, irreversible motor endplate degradation has been observed by 12 months after a nerve injury.[19] Thus, efforts should be taken to provide axons to motor endplates no later than 1 year after complete transections. As a general rule, neurolysis or nerve repair with or without grafting is recommended within the first 6 months after injury, and nerve transfers may be attempted between 6 months and 1 year after the injury.

Neurolysis, nerve repair and nerve grafting

Within the first 6 months after injury, the findings at the time of surgical exploration of the SAN will dictate the nature of the procedure. If intraoperative stimulation of the SAN leads to trapezius muscle contraction, an extrafascicular neurolysis is recommended. In the absence of electrically demonstrable transmission with direct nerve stimulation, a repair is performed by cleanly cutting the SAN on each side of the injury and suturing it using epineural sutures, provided the repair can be completed without undue tension. However, if the repair requires excessive tension, grafting of the defect with an autograft or a biosynthetic nerve guide is preferred. Common donor grafts for the SAN include the greater auricular nerve, sural nerve, anterior branch of the medial antebrachial cutaneous nerve, lateral antebrachial cutaneous nerve, and thoracodorsal nerve.

Nerve transfers

Nerve transfers are considered when nerve surgery is attempted late or very long grafts (over 5 cm) would be needed. Multiple options have been described, including the anterior C3 levator scapulae motor branch, medial pectoral nerve, lateral pectoral nerve, and posterior division of C7. Currently, our institution favors use of one fascicle from the posterior division of the upper brachial plexus trunk.[44] The benefits of this procedure include a single incision, short reinnervation time, and synergistic donor function to facilitate motor reeducation.

Tendon transfers

In patients with long-standing trapezius paralysis not amenable to nerve procedures, *transfer of the levator scapula, rhomboid major, and rhomboid minor* to alternative locations within the scapula was first reported 100 years ago. This procedure has been modified over time to optimize clinical outcomes.

Transfer of these 3 muscles was first suggested by Eden in 1924[21] and further refined by Lange in the 1950s into what is known in the literature as the Eden-Lange procedure.[12,27] In the original publication by Lange, all 3 muscles were harvested with their bony insertion; the levator was transferred to the lateral aspect of the scapular spine at the level of the acromioclavicular joint, whereas both rhomboids were transferred under the infraspinatus to the lateral aspect of the infraspinatus fossa (Fig. 4). Later on, Bigliani modified the procedure to transfer the rhomboid minor to the supraspinatus fossa.[4]

Since the trapezius muscle inserts into the spine of the scapula, transfer of the rhomboids to the infraspinatus or supraspinatus fossae may partially correct lateral drooping of the shoulder girdle, but does not replicate the upward rotational effect of the trapezius through the scapular spine. In 2015, Elhassan described a modification of the Eden-Lange procedure in which the levator scapulae was transferred to the spine just posterior to the acromion, the rhomboid minor to the dorsal aspect of the spine of the scapula just medial to the new location of the levator, and the rhomboid major to the dorsal and volar aspects of the medial third of the spine of the scapula.[13] The line of pull of the transferred rhomboids better replicates the line of pull of the normal trapezius. The Elhassan modification of the Eden-Lange procedure is considered by most today the transfer of choice.

Surgical technique

Our preference is to perform this procedure in the lateral decubitus position with the affected upper extremity in a sterile dynamic arm holder. A curvilinear incision is placed parallel to the body of the scapula and a few centimeters medial and curved laterally at the level of the superomedial border of the scapula to end at the level of the acromion (Fig. 5). A large skin flap is raised inferomedially to expose the location of the atrophic trapezius and scapular spine. The trapezius is then detached from the spine of the scapula and mobilized superomedially.

Next, anterior traction is applied to the arm to protract the scapula, which helps to identify the orientation of the muscle fibers of the levator and both rhomboids. The intervals between these 3 muscles are dissected. The arm is then released from the mechanical arm holder and placed behind the lumbar spine ("chicken-wing position"). The interval between the inferior end of the rhomboid major and the attachment site of the anterior serratus is easily identified in this position, so that inadvertent damage to the serratus anterior is avoided. The levator and rhomboid insertions are then detached with a 5-mm segment of its bone insertion using a microsagittal saw. The muscles are mobilized medially, and the dorsal scapular nerve is identified and protected during the remainder of the procedure. The bony attachment harvested with the rhomboid major is divided in the middle so that the rhomboid major can be folded over the os trigonum.

The dorsal aspect of the spine of the scapula and the undersurface of the medial spine are lightly decorticated with a high-speed bur. Fixation of the transferred muscles can be performed with buttons or transosseous sutures. We prefer transosseous sutures and use a 2-mm drill to perforate the spine of the scapula from superior to inferior, creating 2 tunnels for the levator, 2 for the rhomboid minor, and 4 for the rhomboid major.

The arm is then placed again into the mechanical arm holder and the scapula is brought as close to the midline as possible. Complete paralysis is requested from anesthesia. Looped sutures are placed around the bony attachments of the levator and rhomboid minor (2 per muscle) and sequentially passed through the 4 more lateral tunnels. Four additional looped sutures are placed sequentially through the superior half of the rhomboid major, bone tunnel and inferior half of the rhomboid major, using the 4 medial tunnels. All sutures are then tied using Nice knots. Once the transfer is finalized, the atrophic trapezius can be draped over the transferred muscles.

Postoperative management

Although the initial description of the Elhassan modification recommended use of a custom brace in 80° of abduction and 40° of external rotation, maintaining this position for several weeks is challenging for patients, and compliance is low. As such, we have migrated to use of an external rotation brace similar to what is used for posterior shoulder instability. The upper extremity is immobilized in this brace for 8 weeks. At that time, gentle motion exercises and isometric scapular stabilizing exercises are initiated. Strengthening exercises are advanced as tolerated starting at 3 months, and unrestricted activity is allowed at 6 months. In many patients, the levator and rhomboids hypertrophy over time.

Scapulothoracic arthrodesis

In our practice, the main indication for scapulothoracic arthrodesis in the setting of a trapezius palsy is a prior failed levator and rhomboid transfer. Other indications include patients with poor levator and rhomboid muscle bulk (typically secondary to aging or an associated injury to the dorsal scapular nerve) and patients with associated serratus anterior insufficiency (which can occur with incomplete recovery after a brachial plexus injury or PTS). Our preference is to use cable fixation of the body of the scapula to the ribs, passing the cables through a dorsal plate to avoid fracture of the body of the scapula, and augmentation with posterior iliac crest autograft (Fig. 6).

Reported outcomes

Multiple peer-reviewed publications have reported the outcome of the various management strategies summarized above.[1-3,5,6,8,9,11,13,15-18,20,21,23,24,26,28,32-34,35,37,40,41-43] Table IV summarizes data extracted from the most relevant publications organized by intervention (conservative treatment, nerve procedures, tendon transfers, and salvage procedures). In many of the older publications, granular information on restoration of function is not available. Some studies have reported pretty good outcomes across the board, whereas others have presented mixed results.

Conclusion

Paralysis of the trapezius muscle is a very debilitating condition. Unfortunately, a common cause for trapezius palsy continues to be iatrogenic injury to the SAN. Despite well-described physical examination findings classic for trapezius palsy, the diagnosis is oftentimes delayed, and unnecessary surgery on the biceps and rotator cuff is occasionally performed before the true nature of the problem is finally identified.

Nonoperative management may be considered for incomplete injuries with electromyographic findings suggestive of spontaneous recovery. However, evidence of SAN transection secondary to penetrating trauma or surgery should prompt surgical nerve repair with or without grafting; if nerve repair or grafting cannot be successfully achieved, nerve transfers can be considered. Nerve procedures are less likely to succeed once irreversible motor end-plate degradation occurs, and tendon transfers generally represent a more predictable procedure, especially when surgery is considered more than 1 year after the onset of the palsy. Transfer of the levator scapulae and rhomboids to the spine of the scapula according to the Elhassan modification of the Eden-Lange procedure is a very good procedure with the potential to provide pain relief and to improve function. Rarely, scapulothoracic arthrodesis is recommended; namely, if a prior tendon transfer failed or on patients with poor levator and rhomboids or with several combined nerve palsies.

Disclaimers:

Funding: No funding was disclosed by the authors.
Conflicts of interest: The authors, their immediate families, and any research foundations with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this article.

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