Accessory Nerve Palsy: A Review of 23 Cases

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ABSTRACT: In a series of 23 patients, the commonest cause of accessory nerve palsy was surgical trauma at the time of lymph node biopsy. The less common causes were penetrating or blunt trauma and a few were of spontaneous onset. There was involvement of adjacent motor sensory nerves in about half of the patients. The prognosis was better following blunt trauma, stretch injuries and after a spontaneous onset. The anatomical relationships of the accessory nerve and aspects of the clinical picture and management are discussed.

RÉSUMÉ: Paralysie du nerf spinal: revue de 23 cas. Dans une série de 23 patients, la cause la plus fréquente de paralysie du nerf spinal était le traumatisme chirurgical au moment d'une biopsie d'un ganglion lymphatique. Parmi les causes plus rares, on notait les traumatismes pénétrants ou contondants et quelques ces étaient spontanés. Il existait une atteinte des nerfs moteurs ou sensitifs adjacents chez à peu près la moitié des patients. Le pronostic était plus favorable à la suite des traumatismes contondants, des blessures par étirement et lorsque le début était spontané. Nous discutons des relations anatomiques du nerf spinal, de certains aspects du tableau clinique et de la conduite du traitement.

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Accessory nerve palsy commonly occurs as a result of a surgical trauma within the posterior triangle,[1,2,3,4,5] usually at the time of lymph node excision.[6,7,8,9] It has also been described following carotid endarterectomy,[10,11] accidental laceration,[7] irradiation[12] and as a spontaneous event.[13] This series is unusual as it contains a relatively large number of cases in which the trauma was of a non-surgical type.

Anatomical considerations

The normal anatomical relationships of the accessory nerve and the contents of the posterior triangle are shown in Figure 1. The spinal portion of the accessory nerve descends from the jugular foramen into the neck where it lies along with other nerves between the internal carotid artery and internal jugular vein.[14] It then passes obliquely downwards and laterally, superficial or deep to the vein, under cover of the posterior belly of the digastric and it pierces the deep surface of the sternomastoid muscle and supplies it. It appears at the posterior border of the sternomastoid at or below the junction of the upper and middle third and runs obliquely downward and backward in the fascial roof of the posterior triangle to reach the anterior border of the trapezius muscle. It passes under the muscle and helps to supply it along with the branches of the cervical plexus. The accessory nerve communicates with the second, third and fourth cervical nerves as part of the cervical plexus, in the region of the sternomastoid, posterior triangle and the trapezius. In its course beneath the sternomastoid and in the posterior triangle, the accessory nerve is intimately associated with lymph glands.

The posterior triangle, as formed by the middle third of the clavicle, the posterior border of the sternomastoid and the anterior

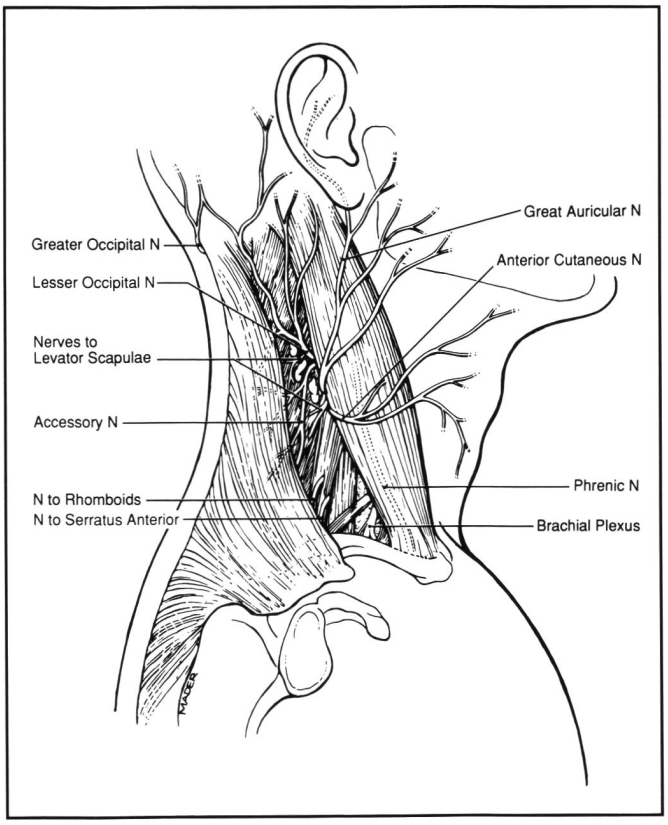

Figure 1. — The normal anatomical relationships of the accessory nerve and contents of the posterior fossa. Lymph nodes are shown adjacent to the accessory nerve.

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border of the trapezius, has a muscle floor formed by the scalenus capitus, the levator scapuli and the scalenus medius and posterior muscles, all of which run obliquely downwards and backwards. The muscular floor is covered with a layer of fascia and with the motor nerves to four muscles: levator scapulae (C3, 4), rhomboids (C5), serratus anterior (C5, 6, 7) and diaphragm (C3, 4 and 5). The nerves to the first three are found between the accessory nerve above and the upper border of the brachial plexus below and are therefore vulnerable to injury along with the accessory nerve. The suprascapular nerve, as the uppermost and most lateral branch of the brachial plexus, can also be involved in conditions effecting the posterior triangle. Several sensory nerves, the lesser occipital (C2, 3), the greater auricular (C2, 3), the anterior cutaneous colli (C2, 3) and the supraclavicular nerves (C3, 4) are also found in the posterior triangle and can be involved along with the accessory nerve.

Methods

This series consists of 23 patients referred to the Electrophysiology Laboratory of a large urban teaching hospital for the assessment of accessory nerve palsy. The cause, electromyographic finds, findings at operation, treatment and eventual outcome are summarized in Table 1. The mechanism of injury was assessed in each patient. The trapezius muscle was examined by inspection and by palpation of its upper fibres. Scapular movements were observed; the upper portion of the muscle is most active during scapular elevation and the mid and lower parts during retraction of the scapula and abduction and flexion of the limb. When the trapezius is paralyzed the glenohumeral joint becomes displaced laterally and inferiorly and there is considerable limitation of scapulothoracic rotation such that abduction of the arm is limited to about 90 degrees.

Sternomastoid bulk is assessed by inspection and palpation with the head and neck rotated in the opposite direction against resistance and also by forward flexion of the neck and head against resistance. Evidence for injury to adjacent nerves was also sought. Cutaneous sensory loss around the pinna and angle of the mandible was attributed to a lesion of the greater auricular nerve. Sensory loss between the back of the pinna and the occiput was attributed to a lesion of the lesser occipital nerve. Sensory loss between the clavicle and the upper and posterior portions of the shoulder was attributed to lesions of the supraclavicular nerves and sensory loss below the angle of the jaw and anterior to the sternomastoid was seen in lesions of the anterior cutaneous colli nerve. Weakness of the levator scapuli was assessed by observing the muscle action during elevation of the shoulder and by palpating its bulk. Rhomboid bulk and strength were assessed by an inspection of the muscle during scapular abduction with the hands on the hips while attempting to push the elbows backward against resistance. Infraspinatus and supraspinatus bulk was assessed by inspection and by their strength during lateral rotation and abduction during the first 30 degrees of the latter movement.

Electromyographic examination was done by the use of a DISA 1500 Digital EMG System. Evidence of denervation, motor units under voluntary control, completeness of interference pattern, the presence of polyphasic motor units during serial examinations, were noted. The number of motor units under voluntary control were graded on a scale of 0 to 4+.

Follow-up information was obtained when possible by clinical and electromyographic examination and through the records of the referring physicians.

Results

The series consists of 23 patients and details of causation, electromyographic changes, findings at operation, treatment and outcome are recorded in Table 1. The causation and mechanism of paralysis is listed in Table 2. The single commonest cause of this condition was that of injury to the nerve at the time of surgical removal of an enlarged lymph node or similar mass in the posterior triangle as a form of surgical trauma. Other forms of trauma were encountered in 10 patients. Two patients had suffered blunt injuries in automobile accidents. In one patient, the palsy had followed a fall which resulted in a head blow and another followed a beating with a baseball bat. Accidental laceration as a form of penetrating injury of the posterior triangle in industrial and other accidents was the cause in 3 patients. Stretch injuries were encountered in 3 patients when the upper limb was caught in machinery, following sudden angulation and after apparent traction during carotid endarterectomy. The condition occurred spontaneously, without any definable cause, in 2 patients.

The accessory nerve alone was involved in 11 patients (Table 3). The trapezius was the only muscle involved in about half of these patients and this indicates the lesion to be below the level of supply to the sternomastoid muscle. Five patients had trapezius as well as sternomastoid involvement. We did not always assess skin sensation over the neck, adjacent jaw and ear region and therefore involvement of sensory branches may have been missed in a number of patients. Blunt trauma is more likely to produce an isolated palsy and the 2 cases of spontaneous paralysis were also limited to the accessory nerve. Approximately half of the patients (12) had additional nerve and muscle involvement, namely greater auricular and lesser occipital, rhomboid, serratus anterior, surpacapular, levator scapuli, supraclavicular and recurrent laryngeal nerve. Stretch injuries produce either isolated accessory nerve or multiple nerve involvement. Multiple nerves were more commonly involved following node excision and penetrating injuries. Four of the patients developed a frozen shoulder as a complication of the paralysis and immobility.

Thirteen patients underwent surgical exploration and the findings are noted in Table 4. Scarring around an intact nerve was encountered in 2 of the patients who had suffered stretch injuries and also after accidently laceration and node excision. The finding of a severed nerve was most often attributable to surgical excision of a mass or node in the posterior fossa.

The relationship of outcome to causation is noted in Table 5. As could be anticipated, the outcome is poorest when the palsy has followed lymph node excision or accidental penetrating injury and is best when caused by blunt trauma. The rare cases of spontaneous onset had a good outcome.

The relationship between treatment and outcome is described in Table 6. Patients with palsy following blunt injury, of spontaneous onset and in whom surviving muscle action can be demonstrated clinically or electromyographically are usually treated in a conservative manner and the outcome, as would be expected, is better in such patients with lesions in continuity.

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Patients with complete paralysis or in whom nerve severance is suspected are best managed by surgical exploration and grafting or anastomosis as required. The outcome is less favourable although it should be noted that partial recovery occurred in 4 patients following a sural nerve graft procedure.

Discussion

The diagnosis of accessory nerve palsy can usually be made without difficulty. The patient describes weakness of the shoulder following surgical biopsy or excision of a lymph node in the posterior triangle, or after a blunt, penetrating or more rarely a stretch injury or the condition can be of spontaneous onset. Pain may be present and examination reveals drooping and slight outward displacement of the shoulder with weakness on shrugging. Thinning of the upper fibres of the trapezius muscle can be readily seen and palpated. Wasting of the mid and lower fibres of the trapezius muscle results in increased prominence of the scapula and the appearance can be mistaken for the winged scapula of serratus anterior palsy.

Involvement of adjacent nerves, as after a surgical procedure or penetrating injury, can result in paralysis of the levator scapu-

Table 1

Patient	Cause	E.M.G. Motor Units (0-4+) pp = polyphasic potentials	Nerve at Operation	Procedure	Recovery (m, yr) P.S. = post surgery
1. E.T.	lumpectomy post. triangle	0	intact.	neurolysis	6 1/2 m P.S. - complete
2. G.N.	cyst. post. triangle	1+	severed*	end to end anastomosis	3 yr P.S. - none
3. F.N.	node biopsy post. tri.	3+PP	severed*	sural nerve graft	15 m P.S. - partial
4. H.S.	lipoma post. tri	1+PP	severed*	sural nerve graft	11 m P.S. - none
5. K.D.	node biopsy post. tri.	3+PP	no surgery		18 m - complete
6. E.P.	node biopsy post. tri.	1+PP	no surgery		1 yr - partial
7. D.P.	node biopsy post. tri.	3+	no surgery		8 m - partial
8. C.D.	node biopsy tri.	0	no injury		28 yr - none
9. E.M.	node biopsy post. tri.	1+	no surgery		5 m - partial
10. B.G.	stretch inj.	2+PP	scarring	neurolysis	4 m. P.S. - partial
11. W.P.	stretch inj.	1+	scarring	neurolysis	6 m. P.S. - none
12. J.P.	laceration. post. tri.	1+PP	scarring	neurolysis	11 m. P.S. - none
13. E.B.	laceration post. tri.	1+PP	severed at muscle*		15 m.P.S. - none
14. C.G.	blunt injury	3+PP	no surgery		1 yr - partial
15. M.A.	cerv. lymph node biopsy	3+PP	intact, neuroma and scarring	neurolysis	5 m. P.S. - partial
16. V.C.	blunt trauma	4+	no surgery		3 m. -partial
17. K.D.	blunt trauma	4+	neuroma in sternomastoid	post. auricular nerve graft	8 m. P.S. - partial
18. D.S.	blunt trauma	1+PP	intact	neurolysis	6 m. P.S. - incomplete
19. B.S.	spontaneous onset		no surgery		19 m P.I. - complete
20. V.M.	removal tuberculous granuloma	0	severed	sural nerve graft	9 m. P.S. - partial
21. J.G.	penetrating injury	0	no surgery		6 m P.I. - partial
22. B.B.	penetrating injury	3PP	severed,** scarring	sural nerve graft	19 m.P.S. - partial
23. N.S.	spontaneous onset	2+	no surgery		2 yr - partial

* surviving motor units in trapezeus muscle can be on the basis of direct cervical innervation
** proximal neuroma, distal transection

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Table 2

Accessory nerve palsy: Causes		23 patients
Lymph node biopsy		11
Trauma		10
penetrating	3	
blunt	4	
stretch	3	
Spontaneous		2

Table 3

Accessory Nerve palsy: Clinical Picture		23 patients
Isolated palsy		11
trapezius alone	6	
trapezius and sternomastoid	5	
Additional nerve involvement		12
one	8	
multiple	4	

Table 4

Findings at Operation	13 patients
in continuity, scarring	5
normal	1
severed	6
neuroma	1

Table 5

Accessory Nerve Palsy: **Outcome vs Cause** **23 patients**

		Trauma			
Recovery	**Biopsy**	**- Ptg***	**Blunt**	**Stretch -**	**Spontaneous**
Complete (5)	2		2		1
Partial (12)	6	1	3	1	1
None (6)	3	2		1	

*penetrating

Table 6

Accessory Nerve Palsy: **Outcome vs Treatment** 23 patients

Recovery	Neurolysis	Graft	Anastomosis	Conservative
Complete (5)	2			3
Partial (12)	2	4		6
None* (6)	2	1	1	1

*1 - nerve severed at entry to muscle

lae, rhomboid or serratus anterior muscle. Electromyographic examination after about 14 days of onset, will detect denervation in addition to any surviving motor units and allows an assessment of the completeness or incompleteness of the lesion. The degree of involvement of the trapezius muscle is difficult to assess by clinical examination alone and electromyographic examination should be done whenever muscle contraction cannot be demonstrated and in all cases of post-operative palsy and which follow penetrating injury. The upper fibres of the trapezius are the easiest to examine as they can be palpated between the thumb and forefinger and the needle electrode can be accurately inserted into the muscle. The middle and lower fibres consist of a thin sheet of muscle overlying the rhomboids and latissimus dorsi. These fibres are more difficult to examine and we do not routinely sample them. Follow up examinations about six months after onset, in patients with a complete lesion will reveal evidence of re-inervation in the form of nascent motor units and this is about the time beyond which nerve anastomosis or grafting is no longer very successful.

Management

Management of the individual patient is influenced by the severity of the lesion and the cause of the palsy. If the palsy is incomplete as confirmed by the presence of surviving motor units on EMG examination, then conservative treatment with appropriate exercise or physiotherapy in order to prevent the complication of frozen shoulder is all that is required. If the palsy which follows blunt or penetrating trauma is complete by clinical and electrical criteria, then surgical exploration is indicated in order to establish the nature of the lesion. A severed nerve can be anastomosed or a graft may be interposed. Neurolysis is sometimes done when there is continuity but the efficacy of this procedure is not established. Although nerve grafting or anastomosis are not uniformly successful, they offer the only hope of reinervation and of a return of function in the patient with a severed nerve; in this series, partial recovery occurred in 4 out of 5 such patients. This procedure should be done in the early months after injury and before 6 months have elapsed.

A persistent, complete trapezius muscle palsy does result in significant disability. As the scapula is not anchored, it rotates and is displaced when the shoulder is flexed or abducted. The elevators of the shoulder cannot exert power or act through a full range and the limb is considerably weakened. The altered movements of the scapula also result in aching about the shoulder. This can be overcome to some extent by surgical transposition of the sternal head of the pectoralis muscle to the undersurface of the scapula and by other transposition procedures.[15]

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