Injuries to the spinal accessory nerve

A LESSON TO SURGEONS

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The integrity of the spinal accessory nerve is fundamental to thoracoscapular function and essential for scapulohumeral rhythm. This nerve is vulnerable along its superficial course. This study assessed the delay in diagnosis and referral for management of damage to this nerve, clarified its anatomical course and function, and documented the results of repair. From examination of our records, 111 patients with lesions of the spinal accessory nerve were treated between 1984 and 2007. In 89 patients (80.2%) the damage was iatrogenic. Recognition and referral were seldom made by the surgeon responsible for the injury, leading to a marked delay in instituting treatment. Most referrals were made for painful loss of shoulder function. The clinical diagnosis is straightforward. There is a characteristic downward and lateral displacement of the scapula, with narrowing of the inferior scapulohumeral angle and loss of function, with pain commonly present. In all, 80 nerves were explored and 65 were repaired. The course of the spinal accessory nerve in relation to the sternocleidomastoid muscle was constant, with branches from the cervical plexus rarely conveying motor fibres. Damage to the nerve was predominantly posterior to this muscle.

Despite the delay, the results of repair were surprising, with early relief of pain, implying a neuropathic source, which preceded generally good recovery of muscle function.

Owing to the nature of its anatomy, the spinal accessory nerve is susceptible to injury. Damage to the nerve anterior to the sternocleidomastoid muscle causes paralysis of the muscle and also of the trapezius. The trapezius alone is paralysed when the level of the lesion is deep, or posterior to the sternocleidomastoid. The sternocleidomastoid draws the head towards the ipsilateral shoulder and rotates it. When the left and right muscles act together, the head is flexed on the neck. The trapezius is the major suspensory muscle of the shoulder girdle,¹ and maintains scapulohumeral rhythm.² The upper fibres elevate the scapula and rotate the lateral angle upward. The intermediate portion adducts and retracts the scapula, and the inferior region depresses and rotates the scapula downward. Paralysis of trapezius leads to depression and lateral displacement of the scapula, classically described as winging, where the scapula drops down and away from the spine and posterior chest wall. The scapula is destabilised by the weight of the limb, and is pulled forward by the unopposed serratus anterior. This leads to loss of glenohumeral abduction, as the head of the humerus cannot move against the glenoid. The inferior scapulohumeral angle is

narrowed in active and passive movement.³ The result is severe disability, usually accompanied by pain.

The extent of innervation of the trapezius by branches of the cervical plexus and the thoracic spinal nerves, and the function of all the nerves supplying this muscle, remain controversial. Typically, the spinal accessory nerve has been described as a somatic efferent nerve, with upper cervical nerves conveying some motor fibres and all the muscle afferents.⁴ However, large numbers of small (< 2 µm) non-myelinated fibres have been found by Bremner-Smith, Unwin and Williams.⁵ Many of these are C-fibre polymodal nociceptors; others may be post-ganglionic sympathetic efferent fibres.

It was stated by Williams et al⁶ that "Injury to the accessory nerve results in a characteristic group of symptoms and signs - reduced shoulder abduction, drooped shoulder, and pain. Repair of the nerve improves symptoms in most cases. A sound grip of surgical anatomy, together with use of a nerve stimulator, ought to prevent this serious complication of surgery on the neck." Despite these comments, damage to the spinal accessory nerve still occurs, typically during surgery to the neck, especially

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lymph node biopsy,^{6,7} but there appears to be an unacceptable delay in diagnosing this injury and starting treatment.⁶

The elements evaluated in this study included the cause of injury, the interval between injury and diagnosis, the interval between injury and treatment, pain and shoulder function before and after operation upon the damaged nerve and the course and function of the nerve.

Patients and Methods

We reviewed the medical records of all 111 patients referred to our institution with lesions of the spinal accessory nerve between June 1984 and October 2007. Of these, ten patients had been previously included in the study by Williams et al.⁶ The group included 56 males and 55 females whose mean age was 37.1 years (2 to 75); 80 were right-handed. Data were collected regarding the cause of the lesion, the discipline and grade of surgeon initially involved, the interval to diagnosis of nerve injury and to referral for ongoing management, the results of neurophysiological investigations, the degree of active and passive movements of the shoulder and position of the scapula; the level of pain and medication used, details of the definitive treatment of the nerve injury with the date and findings at operation and the post-operative level of function, movements, pain and analgesia. For most patients data were collected prospectively, with the details entered in the records at the patient's first attendance. The patients were then followed prospectively with data entry as appropriate. Senior surgeons at the unit, together with the authors, were responsible for the final review. Where appropriate, the data were analysed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, Illinois).

Two systems were used to measure pain: the peripheral nerve injury pain score,⁸ which classes pain as 1 = none; 2 = controllable (daily activities possible, sleep undisturbed); 3 = severe (work interrupted, sleep difficult requiring medication); 4 = ungovernable (sleep disturbed regularly, work impossible); and a visual analogue scale (VAS), which measures pain on a numerical scale ranging from 0 to 10 (where 0 = no pain and 10 = worst possible pain). Both systems show strong concordance with each other.⁸ Shoulder function was assessed by measuring scapular depression and lateral displacement both at rest and in elevation. The active and passive inferior scapulohumeral angle between the longitudinal axis of the humerus and the lateral border of the scapula was also noted.³ As an outcome measure a categorical scale for function was applied.

The spinal accessory nerve was explored as follows. The nerve was exposed by extending the original incision or wound to display the anterior face of sternocleidomastoid. The transverse cervical and greater auricular nerves were then defined. Where the nerve had retracted into or was deep to the muscles, a nerve stimulator was employed to identify the branches to the sternocleidomastoid. The distal stump of the spinal accessory nerve often proved more difficult to identify. It was typically stuck to the inner face of

Table I. Cause of nerve lesion

Assault or accidental injury	22
Bottle injury/knife wound/missile	18
Road traffic accident	4
Iatrogenic injury	89
Lymph node biopsy or excision	59
Lymph node clearance for infection or neoplasm	3
Excision of brachial cyst	6
Other operation or procedure in the neck	21

the trapezius, running obliquely and vertically downwards, and connecting to branches from the cervical plexus just above the clavicle. If difficulty with identification was encountered, the inferior limb of the incision was extended distally into unscarred tissue planes. The spinal accessory nerve was then located adherent to the trapezius, marked by the adjacent longitudinal vessels. A nerve stimulator was used in all cases to assess conduction across the lesion. It was placed proximal to the lesion, and muscle contraction was observed. Conduction distal to the lesion was similarly evaluated. Further intra-operative neurophysiological studies, such as somatosensory evoked potentials, were not routinely recorded. The preferred graft, when required, was the medial cutaneous nerve of the forearm, rather than a supraclavicular nerve. Nerve transfer, where indicated, was undertaken with a standard technique, using the lateral pectoral nerve.⁹

Results

Characteristics of injury. Lesions in the 111 patients were divided equally between the two sides. Most (n = 89) were iatrogenic injuries in the course of medical treatment, of which 61 (55%) occurred during lymph node biopsy or excision for benign conditions (Table I). In 38 of the iatrogenic cases the nerve was damaged by general surgeons, and in a further 29 by otorhinolaryngological surgeons. Other disciplines accounted for damage to eight cases, two of these in general practice. The grade of the operating surgeon was known in 56 cases (consultant 38; registrar 14; staff grade 4).

The diagnosis of injury to the spinal accessory nerve was made by the operating surgeon in only 14 of 111 cases, in 59 by orthopaedic surgeons, and in 25 by neurologists, to whom the patient had been referred. In the remaining patients the diagnosis and referral were made by other disciplines, usually general practitioners, in ten cases at the prompting of the patient's solicitor. Time to diagnosis varied widely, but was generally longer in the iatrogenic group. The mean time from damage to diagnosis of the nerve injury was 384.9 days (0 to 9163) in the iatrogenic group, compared to 167.5 days (0 to 881) in the accident/assault patients.

Operation on the nerve was not recommended in 31 cases, either because the nerve was recovering (n = 18), because of

Fig. 1a

Fig. 1b

The posture a) and range of abduction b) of the scapula in transection of the right spinal accessory nerve. Note the depression and lateral displacement of the right scapula at rest, and the marked decrease in the inferior scapulohumeral angle (25° right; 170° left).

Fig. 2a

Fig. 2b

The posture of the scapula at rest a) and on forward flexion b) in a patient of athletic physique, with a lesion of the left spinal accessory nerve. Note the well-developed levator scapulae, and residual minor innervation of the uppermost fibres of trapezius, from the uppermost branch demonstrated during forward flexion.

the general medical condition of the patient or severe scarring in the neck from radiotherapy or sepsis ($n = 13$).

Pain. Pain at the first attendance at our unit was severe in 65 patients (63%) and significant in a further 26 (25%). In most the onset of pain had been immediate, following the initial procedure or accidental damage to the nerve. Pain was mitigated by non-steroidal anti-inflammatory medications (NSAIDs), opiates, anticonvulsants or calcium-ion channel blockers in 89 patients (80%), but no one agent was particularly effective. The specific constituents of each patient's pain, namely the neuropathic – *versus* mechanical elements, were not evaluated, nor did they correlate with the time to referral.

Function. On presentation to our unit, patients showed the characteristic features of injury to the spinal accessory nerve (Fig. 1) with wasting of the trapezius, downward and lateral displacement of the scapula and narrowing of the inferior scapulohumeral angle (ISHA). In most cases, active and passive abduction and the ISHA were diminished (mean ISHA: active 46.4° (20° to 100°), passive 53.6° (20° to 170°); mean abduction: active 63.5° (20° to 130°), passive 135.1° (30° to 180°)). Patients reviewed promptly following either traumatic or iatrogenic injury demonstrated the typical posture of the scapula. Reduction of the ISHA also seemed to occur early, although correlation of this with time to referral and definitive management was not formally evaluated. Winging with

Fig. 3

Intra-operative dissection of the posterior triangle showing the relationship of the spinal accessory nerve to other structures. Note particularly the proximal (3) and distal (4) stumps of the nerve, and the proximity (5 mm cephalad) of the spinal accessory nerve to the greater auricular nerve (2). The other nerves labelled are (1) the transverse cervical nerve and (5) a supraclavicular nerve (SCM, sternocleidomastoid muscle).

Table II. Findings at operation in 80 patients undergoing exploration

Level of lesion in relation to sternocleidomastoid	
Anterior	12
Deep	9
Posterior	59
Formation of nerve trunk*	
One trunk	69
Two or more	2
Treatment of nerve	
Neurolysis	10
Suture	1
Graft	64
Nerve transfer (lateral pectoral nerve)	5

* data only available for 71 patients

protrusion of the medial border of the scapula and the scapula pulled laterally by the action of the unaffected serratus anterior were common. In three patients of athletic physique the scapula was elevated. In these patients, the levator scapulae was particularly well developed (Fig. 2). Although typically reported by the patient to be present from the outset, weakness of the shoulder girdle, and resultant loss of shoulder function, was often not addressed until the patient was examined by an orthopaedic surgeon or neurologist.

Neurophysiological investigations. The results of neurophysiological investigations were often misleading. Pre-operative studies suggesting conduction along the nerve ($n = 16$) and electromyographical findings of polyphasia ($n = 55$) were reported in patients where the nerve was transected and the stumps wholly separated.

Findings at operation. Where the nerve was explored, it was seen consistently as one trunk, emerging from the sternocleidomastoid about 5 mm cephalad to the greater auricular nerve (Fig. 3, Table II). In two patients a slender trunk was seen running adjacent to the main nerve. A fine branch was usually found leaving the main trunk of the spinal accessory nerve within 1 cm of the sternocleidomastoid and passing to the uppermost part of the trapezius. When this was intact, loss of function was less severe. A branch from the cervical plexus was a consistent relation, passing to join the spinal accessory nerve just above the clavicle. In 26 patients this branch was stimulated. Weak muscle contraction was evoked in only two with no response in 24, illustrating that motor fibres were rare in the cervical contribution.

Most divided nerves were grafted (Table II), with the mean gap being 2.9 cm (1 to 10). External neurolysis alone was performed for ten patients in whom the nerve had been caught by a suture, or where intra-operative conduction across the lesion was identified using the nerve stimulator. Nerve transfer using the lateral pectoral nerve was carried out in five patients because the proximal stump could not be displayed safely and therefore nerve repair or grafting was not possible.

Pain response. Early relief from pain was usual, long before recovery of muscle function (Fig. 4), but in three patients relief of pain was not followed by useful recovery of the muscle. Pain relief was not as satisfactory after nerve transfer as it was after nerve repair or grafting. More than half of the operated patients discontinued their medications.

Recovery of function following suturing or grafting of the nerve, and nerve transfer. In 49 patients (70%) (Table III) functional recovery was excellent or good. Recovery was not related to delay before repair (Figs 5 and 6). In ten patients the gap was 5 cm or greater, and in seven of these the results were good. Five of the good results were in patients who underwent nerve repair within one year. All of the operated patients who had a poor outcome had been grafted.

Discussion

Bonney¹⁰ commented: "If there is an incision over the line of a main nerve and if, after operation, there is complete paralysis (including vasomotor and sudomotor paralysis) in the distribution of that nerve, speculation is unnecessary: the nerve has been cut, and there will be no recovery unless it is explored and repaired."

Iatrogenic injury to the spinal accessory nerve continues to be a problem.^{6,7} Indeed, the delay in diagnosis and referral for ongoing management appears to be increasing. In the report by Williams et al,⁶ the mean delay was 11.3 months, which has increased to 12.6 months in this study. Few of the initial operating surgeons, predominantly general or otorhinolaryngological surgeons, recognised the resulting spinal accessory nerve lesion. We found that an orthopaedic surgeon often made the diagnosis after the patient had been referred with a shoulder complaint. The

Fig. 4a

Fig. 4b

Figure 4a – visual analogue scale for pain for operated patients. Figure 4b – peripheral nerve injury scores for pain for operated patients. All available data included at each timepoint.

Table III. Frequency of the various outcomes in spinal accessory nerve surgical exploration/repair for the 71 patients with available outcome data

Outcome*	Number of patients (%)
Excellent	4 (5.6)
Good	45 (63.4)
Fair	16 (22.5)
Poor	6 (8.5)

* outcome was graded as follows: excellent = normal function, with no pain; good = good function (abduction $\geq 150^\circ$), with pain no more than a nuisance; fair = improvement over pre-operative state, but limited function (abduction $< 150^\circ$), with pain requiring analgesia; poor = no improvement

Fig. 5

Function at 16 months following nerve grafting, after an interval of 30 months from injury to repair. The patient had no pain, and considered function normal.

decline in anatomical teaching in medical schools is one factor,¹¹ and changes in surgical training are another. In 1996, Raftery¹² commented that “surgical skills are built upon anatomical knowledge, the study and examination of which must not be reduced to a level where it is detrimental to the care of the patients.” This advice is pertinent to operations in the neck.

It seems that discontinuity of care was also a contributory factor. Most patients were not seen by the operating surgeon after their procedure. Whether their injuries appeared in local audit remains unknown. A similar problem has recently been highlighted by Cannon¹³ regarding the quality of elective surgery in treatment centres, and the absence of comparative audit. Failure to recognise the complication of spinal accessory nerve injury and implement urgent appropriate treatment prolongs the duration of the patient’s pain and exacerbates upper limb dysfunction.

The clinical diagnosis of a spinal accessory nerve lesion is straightforward, with obvious physical signs and a temporal relationship between surgery and injury in the region of the nerve.

In contrast, paralysis of serratus anterior results in the scapula being translated medially and upwards. The medial border of the scapula becomes prominent on forward flexion of the shoulder. The diagnosis of immune brachial plexus neuropathy (neuralgic amyotrophy) should be considered only when there is a delay between surgery and the onset of symptoms, and when other muscles are affected. If a patient complains of the typical symptoms of a lesion of the spinal accessory nerve in the immediate post-operative period, then it is likely that the nerve has been cut.

In our study, the spinal accessory nerve had a constant course, which facilitates recognition and should prevent injury. The steps necessary for definite identification of the

Fig. 6

A plot of the relationship between time to surgical exploration/repair and functional outcome in 66 patients with available data.

nerve are to define the posterior border of the sternocleidomastoid at the apex of the posterior triangle and to identify the transverse cervical and greater auricular nerves, where they wind around the posterior margin of the sternocleidomastoid. The spinal accessory nerve lies 5 mm to 10 mm cephalad to the greater auricular nerve. A nerve stimulator is essential for identification. Conduction to muscle fibres in trapezius through the constant branch from C3 and C4 was rare.

Neurophysiological investigations were sometimes unreliable, which may have been due to the difficulties of electromyography in the thin atrophic muscle, where motor potentials from adjacent normal muscles may be detected.

The pain associated with a lesion of the spinal accessory nerve is swift in onset and often severe, suggesting a neuropathic origin. Mechanical pain in such a setting tends to be gradual in onset, and relieved by support of the limb and morphine-based analgesia. The early relief of pain after nerve repair or grafting long before muscle recovery occurs lends support to a neuropathic origin. In neglected cases, pain might also be caused by secondary entrapment of the suprascapular nerve, glenohumeral capsulitis, and/or from traction on the cervical spine and brachial plexus. These mechanisms could be contributing to a number of the poor and fair results.

The capacity of the nerve to regenerate after delayed repair is surprising, with good results following repair as long as three and a half years after injury. The critical time limit for delay prior to repair of the injured spinal accessory nerve is unknown, but repair, by either grafting or nerve transfer, offers definitive management of pain¹⁴ and the opportunity for a good or excellent functional recovery. Pain relief was less effective in the small number of patients who required nerve transfer rather than repair or grafting.

It has been recognised that nerve transfer should be undertaken only where repair or grafting cannot be performed.⁹ Patients typically found early relief of pain gratifying, and the later return of even limited function beneficial. Muscle transfers in response to spinal accessory nerve injury have been shown to improve function.^{15,16} However, because good results with respect to relief of pain and function of the shoulder girdle may be achieved with nerve grafting or transfer, even three and a half years after insult to the nerve, this unit no longer contemplates muscle transfers.^{17,18}

In summary, the anatomy of the spinal accessory nerve in the posterior triangle of the neck is constant, and identification of the nerve using a stimulator remains essential when undertaking any surgery in this region, however minor. The typical interval between injury and the diagnosis and treatment of injury to this nerve is many months. Injury should be suspected if a patient presents post-operatively with neuropathic pain, characteristic scapular displacement and limited shoulder function. Exploration, with or without nerve repair, despite a prolonged delay, maximises the chance of relief of pain and recovery of function.

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