NERVE LESIONS IN PRIMARY SHOULDER DISLOCATIONS AND HUMERAL NECK FRACTURES

A PROSPECTIVE CLINICAL AND EMG STUDY

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The incidence of nerve injuries in primary shoulder dislocation and humeral neck fracture is uncertain. We made a prospective study of 101 patients, using clinical examination and extensive electrophysiological assessment when there was suspicion of nerve damage.

We found electrophysiological evidence of nerve injury in 45%, most involving the axillary, supra-scapular, radial and musculocutaneous nerves. There were significantly more nerve injuries in older patients and those with a haematoma. Most patients recovered partially or completely in less than four months, and only eight had persistent motor loss. Early diagnosis and physiotherapy are recommended.

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The signs of nerve injury in patients with shoulder dislocations or fractures of the humeral neck are easily overlooked and subsequent loss of shoulder function in elderly patients is often thought to be due to immobilisation and stiffness. Only the early detection of nerve lesions allows adequate treatment. The only three prospective studies which we could find were those by Blom and Dahlbäck (1970), Ebel (1973), and Pasila et al (1978) who reported incidences of about 21% to 36%.

We have studied the frequency and distribution of such nerve lesions in 101 patients with primary shoulder dislocation or fracture of the humeral neck.

PATIENTS AND METHODS

During 1992 all patients who attended with a primary shoulder dislocation or a fracture of the humeral neck were specially examined within one week, recording the cause and the presence of haematoma, sensory loss and muscle atrophy. The power of deltoid, supraspinatus, infraspinatus, biceps and triceps was tested and recorded on the MRC scale (MRC 1954) using the methods described by Kendall, Kendall and Wadsworth (1971). The presence of atrophy, sensory impairment and any loss of active and passive ranges of motion was also assessed. This examination was repeated at three weeks, two months, three months and then every month until recovery. Electrophysiological investigation was done after three weeks in all patients with clinical suspicion of nerve injury such as paresis of one or more muscles or sensory loss. EMGs were also performed on patients with severe pain when adequate clinical testing was not possible.

Fifteen patients were excluded from the study. The reasons were non-co-operation because of dementia (7), refusal to attend (3), paresis after cerebrovascular accident (2), death (1), severe rheumatoid arthritis (1) and mental retardation (1). A total of 101 patients (71 women) were included: 57 with a humeral neck fracture and 44 with a primary shoulder dislocation. Their mean age was 63.5 years (8 to 89), but for the women this was 72 years, and for the men 41 years. The mean age at dislocation was 53 years and at humeral neck fracture 69 years. Of the dislocation group, 7 of the 44 patients also had a fracture of the greater tuberosity. Of the humeral neck fractures, 49 were unifocal at the anatomical or surgical neck. In three the greater tuberosity was also involved and five patients had a more comminuted fracture. The ratio of left to right was 54 to 47.

The cause of injury was a simple fall in most cases, but 14 patients had sports injuries; in all cases this was relatively minor. Three of the women with a shoulder dislocation also had rotator-cuff ruptures, confirmed by ultrasound. Four months later they all had power of MRC grade 4 or 5 in all tested muscles.

Treatment. Dislocations were reduced by the Hippocratic, Stimson or Kocher methods with or without

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intravenous sedation. Radiographs were taken before and after reduction. Immobilisation, in a sling or preferably a Polysling (Seton Healthcare Group, Oldham, UK), was for one week for those over 40 years of age and for four weeks in younger patients. Humeral neck fractures were treated in either collar and cuff (54) or a hanging cast (3), closed reduction being used for three displaced fractures. Patients were asked to start exercise as soon as possible, and those with nerve palsy had an active programme of physiotherapy.

Electrophysiology. Where indicated needle (EMG) and nerve-conduction studies (NCS) were performed at 3 to 6 weeks after injury, repeated after 2 to 3 months in some. We used a Medelec MS-60 8-channel electromyograph (Medelec Ltd, Woking, UK) and a concentric needle electrode for EMG. The deltoid (anterior, middle, posterior; axillary nerve), the infraspinatus (suprascapular nerve), the biceps (musculocutaneous nerve), the triceps (radial nerve) and the adductor pollicis longus (ulnar nerve) muscles were examined. In patients with abnormalities in the adductor pollicis longus, other muscles supplied by the median nerve were also examined.

Fibrillation potentials (FP) at rest, consisting of positive sharp waves or fibrillations, were assessed. Motor unit action potentials (MUAP) were studied during slight voluntary contraction and scored as normal or polyphasic (short and long duration). The recruitment of MUAP during maximal voluntary contraction was recorded as normal, mildly reduced, moderately reduced, severely reduced or absent. NCS were performed for the axillary and suprascapular nerves with registration of the compound motor action potential (CMAP) over the deltoid and suprascapular muscle after supramaximal percutaneous electrical stimulation at Erb's point (Gassel 1964).

The severity and extent of nerve injury were recorded on the basis of EMG and NCS. A nerve injury was diagnosed only when FP were found, and its severity on the extent of FP because the density of the latter correlates with the number of denervated muscle fibres (Daube 1991). The absence of MUAP with no CMAP after electrical stimulation was recorded as complete denervation. We scored the severity of nerve injury on a quantitative EMG scale as follows:

- 0 = no denervation;
- 1 = mild partial denervation;
- 2 = moderate partial denervation;
- 3 = severe partial denervation; and
- 4 = complete denervation.

RESULTS

Three weeks after injury, 37 patients had full muscle power and did not have EMG. The other 64 patients were suspected clinically of having a nerve injury and all had EMG at 22 to 79 days (mean 38) after the injury. Forty-five of these (14 dislocations and 31 fractures) showed EMG abnormalities consistent with axonal degeneration.

All these 45 had fibrillation potentials and some had nerve-conduction abnormalities. No patient had abnormal nerve conduction with normal EMG.

Thirty-six (54%) of the patients aged 65 years and older had a neuropathy (54%), as compared with nine of the 64 younger patients (26%). The axillary nerve was most frequently involved, with an isolated lesion in eight cases. The most frequent of 13 different combinations of injuries were: axillary/suprascapular (9) and axillary/suprascapular/musculocutaneous/radial nerve (9). The nerves involved and the EMG severity of the nerve lesions are shown in Table I. All except eight of the patients

Table I. Involvement of the nerves (some multiple) and EMG severity of the lesions of 101 patients with shoulder dislocations or fracture of the humeral neck

	Number of patients
Nerve	
Axillary	37
Suprascapular	29
Musculocutaneous	19
Radial	22
Ulnar	8
EMG severity	
0	19
1	14
2	19
3	11
4	1

recovered well within four months; these eight had an MRC power of 3 or less in one or more of the tested muscles. Thirteen patients had a second electrophysiological investigation after a mean interval of 104 days. There was no recovery in two patients, partial recovery in three and full nerve function on EMG in eight.

In the 26 patients with a haematoma, electromyographic nerve injury was found in 73% compared with 35% of 77 without a haematoma. The difference was significant ($p < 0.001$; with Yates' correction $p < 0.01$). There were only three haematomas in the dislocation group, and all three had severe denervation (EMG 3.3 and 4).

Initial sensory loss was detected in 11 patients, eight of whom had a proven axillary-nerve injury. At the three-month follow-up nine of these 11 patients had complete sensory recovery. A total of 17 still had muscle atrophy, ten of them with EMG abnormalities.

DISCUSSION

There are few prospective studies of the incidence of nerve lesions in shoulder trauma (Table II) and these show divergent results. Pasila et al (1978) used clinical examination only. Blom and Dahlbäck (1970) and Ebel (1973) used EMG but examined only the deltoid.

We referred patients with only minimal suspicion of

Table II. Prospective studies of the incidence of nerve lesions in shoulder injuries

	Number	Mean age (yr)	Diagnosis	Incidence (per cent)	EMG	Follow-up
Blom and Dahlbäck (1970)	73	54	Dislocations and humeral neck fractures	36	+	+
Ebel (1973)	197	58	Dislocations and humeral neck fractures	30	+	-
Pasila et al (1978)	238	58	Primary dislocations	21	-	-
de Laat et al (1994)	101	63	Primary dislocations and humeral neck fractures	45	+	+

nerve injury for electrodiagnosis, and it is unlikely that we have missed any clinically relevant cases. We also tested nerves other than the axillary, and found suprascapular nerve involvement in 29 of 64 EMGs in 101 patients. This high incidence of suprascapular nerve injury (29%) compares with 37% for the axillary and 22% for the radial nerve and has not previously been reported in any prospective study. On EMG, all lesions were due to axonal loss.

All the injuries that we studied had low-energy lesions. Patients either fell from their own height or had minor sports trauma. The pattern of the nerve lesions which we found, however, is similar to that reported in severe infraclavicular brachial plexus injuries (Burge, Rushworth and Watson 1985; Coene and Narakas 1992), and it is likely that the mechanism was the same.

Our older patients had a much higher incidence of nerve palsy, as reported by Blom and Dahlbäck (1970) and the higher general incidence which we found can therefore be partly explained by the higher mean age in our series (Table I). Another explanation may be our more extensive electrophysiological examination.

A haematoma may indicate the violence of the trauma or damage to the whole of a neurovascular bundle, thus accounting for the significantly higher incidence of nerve lesions in such patients. The clinical testing of sensation seemed to have no diagnostic significance: sensory loss was not associated with a higher incidence of motor lesions. Three out of 11 patients with sensory loss had no EMG evidence of abnormalities of motor nerve function, but many patients with normal sensation had motor lesions.

Nerve trunks are normally protected by routing across the flexor aspects of joints, their slackness and elasticity, and the cushioning effect of the epineurium (Sunderland 1978a). Sunderland (1978b) considered that most cases of axillary nerve palsy recover spontaneously, although there may be a delay of up to 12 months. Regrowth after axonotmesis at humeral level has been reported to take three to four months (Kline 1990; Coene 1993). Most of our patients recovered to MRC power 4 in less than four months which strongly suggests that the lesions were distally placed in our patients. The axillary, radial and musculocutaneous nerves are closely related to the head of the humerus and are easily injured during

hyperextension-abduction trauma. It seems unlikely that any of our patients had lesions of the more proximal brachial plexus.

Conclusions. Primary shoulder dislocation and humeral neck fractures cause nerve injury in at least 45% of patients, most often involving the axillary, suprascapular and musculocutaneous nerves. Elderly patients and those with a haematoma have a higher incidence of nerve palsy. Spontaneous recovery of EMG-proven lesions was good. Clinical diagnosis may be difficult because of pain, and electrophysiological assessment in suspected cases is valuable for diagnosis and in directing early active physiotherapy. Functional loss due to muscle paresis may result in shoulder stiffness, and early diagnosis and treatment may prevent lasting impairment of function.

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