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The incidence of nerve injury in anterior dislocation of the shoulder and its influence on functional recovery

A PROSPECTIVE CLINICAL AND EMG STUDY

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Opinion varies as to the incidence of nerve lesions in anterior dislocation of the shoulder after low-velocity trauma. Most studies are retrospective or do not use EMG. We have investigated the incidence and the clinical consequences of nerve lesions in a prospective study by clinical and electrophysiological examination.

Axonal loss was seen in 48% of 77 patients. The axillary nerve was most frequently involved (42%). Although recovery as judged by EMG and muscle strength was almost complete, function of the shoulder was significantly impaired in patients with lesions of the axillary and suprascapular nerves. Unfavourable prognostic factors are increasing age and the presence of a haematoma.

It is not necessary to carry out EMG routinely; an adequate programme of physiotherapy is important. In patients with a severe paresis, EMG is essential after three weeks.

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There are only six prospective studies of nerve lesions in anterior dislocation of the shoulder after low-velocity trauma, of which five include electromyography (EMG). The incidence of injury to a nerve in these five studies varies between 19% and 55%.[1-5] All of them, in differing ways, are incomplete and may not represent the real incidence.

In the study of de Laat et al,[5] EMG was not carried out in every patient, and only in a few with nerve injury was a second EMG available. There is a notable difference in the duration and quality of recovery between patients who have suffered an anterior dislocation of the shoulder. Recovery may be full or may leave the patient with severely limited function. Often there is no satisfactory explanation for this difference.

Nerve injury can cause paresis and inability to move the arm. It is a major factor influencing recovery. The role of physiotherapy is unclear. We therefore undertook a prospective study which included standardised electrophysiological examination and physical treatment in all patients.

The mechanism of anterior dislocation of the shoulder is well known and can be explained by extreme movements beyond physiological limits. In most cases the injury is due to low-velocity trauma, frequently a simple fall on the outstretched hand. In young patients sport injuries are more common.[6]

The mechanism of associated nerve damage is similar to that of the shoulder injury and results in a traction lesion of nerves stretched over the head of the humerus during the dislocation of the head. After the injury with the head in the dislocated position and the elbow against the body there is no traction on the nerves according to Milton[7] and Gariepy, Derome and Laurin[8]. The localisation of a lesion depends on the position and displacement of the arm during trauma. A fall with the arm in full abduction and internal rotation causes major tension on all nerves and cords. Extension of the elbow and wrist produces distraction of the median nerve and the medial cord; if the elbow is flexed the tension is mostly on the ulnar and radial nerves and the medial and posterior cords.[9,10]

The axillary nerve is easily damaged because of its close association with the glenohumeral joint and its course around the surgical neck of the humerus. The relationship of the musculocutaneous and the radial nerves to the joint is less close, but the distance between the anchorage points in the upper arm is short which makes the nerves vulnerable to traction. The median and ulnar nerves seem to be less at risk possibly because of this unencumbered pathway through the upper arm, providing the opportunity to stretch.

No lesion of nerves from the upper part of the brachial plexus, apart from the suprascapular nerve, has been described in dislocation of the shoulder due to minor trauma. Traction on the suprascapular nerve can cause damage in the short distance between its origin from the supraclavicular plexus and its anchorage point at the suprascapular

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notch. Lesions of this nerve are caused by abduction beyond the physiological limit of movement.[11,12]

According to Milton's cadaver study[7] nerve injury can occur during manipulative reduction of a dislocation in which traction together with rotation or abduction are applied at the same time. Methods of reduction with traction in abduction are particularly risky. Longitudinal traction parallel to the axis of the body imposes some distraction on the neurovascular bundle. Simultaneous lateral traction on the upper arm with the nerves anchored to the humerus creates an extra distraction which should be avoided. No traction lesion occurred in the dislocated position with the shoulder at 90° of flexion.[7,13]

Therefore safe methods of reduction, with reference to nerve injury, seem to be the elevation method with traction at 90° of flexion (Stimson, Cooper) or the method according to Kocher (which imposes no traction).[14] If the method of Hippocrates is used, the arm should be externally rotated before traction is applied to give the axillary and radial nerve the opportunity to slip over the humeral head.[7]

Patients and Methods

During a period of 31 months, 93 patients with anterior dislocation of the glenohumeral joint were seen at Leyenburg Hospital; 16 were excluded from the study for various reasons, such as lack of an image of the primary dislocation, very advanced age or multiple injuries. Details of the patients are shown in Table I.

We used EMG to determine the numbers, the severity, the pattern and the recovery of nerve lesions. Conventional radiological views were taken in two planes (AP and lateral) together with a 45° craniocaudal view. Other injuries such as ruptures of the rotator cuff, fractures of the greater or lesser tuberosity or dislocation of the tendon of the long head of the biceps muscle, which could influence muscle strength and shoulder mobility, were carefully assessed. If pain persisted beyond three months, MRI was undertaken.

Clinical examination included movements of the shoulder and arm with tests for muscle strength and sensation at one, four and six weeks, and then at six-weekly intervals. We measured the muscle power of deltoid, supraspinatus, infraspinatus, biceps, triceps, flexor carpi radialis and abductor digiti minimi graded according to the MRC scale (1 to 5).[15] In cases of severe paresis, other muscles supplied by the corresponding nerve were investigated.

Movements of the glenohumeral joint were recorded from the neutral position as described by Cave and Roberts.[16] Internal and external rotation were measured with the elbow positioned beside the body. Abduction of the shoulder was also achieved with the scapula fixed to eliminate compensatory movements of the scapulothoracic joint.

The chosen treatment depended on the patient's age. Those under 50 years were immobilised for four weeks, after which active and passive exercises commenced guided by a physiotherapist. Patients over 50 years of age started mobilising after one or two weeks, depending on pain. Physiotherapy followed a standard protocol supervised by trained therapists, and included passive and active movement with isometric exercises of pectoralis major and minor, and subscapularis. Translation techniques prevented capsular stiffness of the glenohumeral joint.

Electrophysiological investigation. We carried out EMG studies with needle electrodes on all patients after three weeks. Denervation, as an expression of axonal degeneration, can be found three weeks after injury; demyelination (neurapraxia) may be responsible for post-traumatic paresis. EMG does not show active denervation in these cases, whereas nerve-conduction studies show reduction of the amplitude of the potential and a reduced conduction velocity. Nerve-conduction studies are difficult and unreliable in the shoulder region.[16-18] Since neurapraxia has a favourable prognosis, with recovery within weeks,[19] it is not essential to diagnose it early by carrying out nerve-conduction studies.

The severity of nerve injury in our study was assessed according to the finding of axonal loss by needle EMG. We tested the anterior, middle and posterior parts of the deltoid (axillary nerve), the infraspinatus (suprascapular nerve), the biceps (musculocutaneous nerve), the triceps (radial nerve), the flexor digitorum sublimis (median nerve), and the adductor pollicis (ulnar nerve) routinely at three locations in order to optimise the chance of finding abnormalities.

The severity of nerve injury was scored by assessing active denervation, motor unit action potentials (MUAPs) and the pattern of voluntary contraction. In the subacute stage, denervation was scored according to a quantitative scale (0 to 4) because voluntary contraction is made unreli-

Table I. Details of the 77 patients with anterior dislocation of the shoulder

Mean age in years (range)	52.3 (16 to 94)
Gender	
Male	38
Female	39
Side affected	
Left	35
Right	42
Type of dislocation	
Subcoracoid	60
Subglenoid	8
Inferior	2
Unknown	7
Associated fracture	
None	63
Of greater tuberosity	12
Of glenoid rim	2
Haematoma	
No	20
Yes	55
Unknown	2
Nerve injury	
Male	15
Female	22

able by pain.[20-22] No denervation (0) was defined as no fibrillation potentials (FP) or positive sharp waves (PSW) and normal recruitment, slight partial denervation (1) as short or persistent series of one FP or one PSW and normal recruitment, moderate partial (2) as a moderate number of FP or PSW in three or more areas and decreased or normal recruitment, severe partial (3) as many FP or PSW in all areas with decreased or normal recruitment, and complete denervation (4) as many FP or PSW in all areas with recruitment absent. When there were differences of measurement for denervation in one muscle at different places, the average score was taken.

Needle examination was again used to assess recovery. It was not thought reliable only to measure denervation since this can disappear following fibrosis of the muscle. Recovery was measured on the basis of reinnervation and the pattern of maximal voluntary contraction. Needle examination was repeated until the EMG was normal or only minor disorders remained, coupled with a good reinnervation and a reasonable recruitment pattern. The recovery was scored as: 0, interference pattern, no FP or PSW; 1, mild reduced pattern, slight loss of MUAPs, FP or PSW possible; 2, moderate reduced pattern, mixed pattern with high frequent units, FP or PSW possible; 3, severe reduced pattern with one or two existing units, FP or PSW possible; and 4, no motor units, FP and PSW present.

Statistical methods. Several techniques for statistical analysis were used in this study. To quantify the probability of an event, in particular that of a nerve injury, logistic regression models were used. Independent variables were either treated as covariates (age) or as additional adverse factors as, for example, associated fractures or lesions of the rotator cuff. Estimates are presented as odds ratios and associated 95% confidence intervals. The severity of nerve injury, when present, is covered by three parameters on an ordinal scale, namely the number of nerves involved, the maximum severity among all nerves injured and the average severity score. The scale (1 to 4) is not a continuous one, but we feel justified in using the average value as an indication of severity of a nerve injury. It should be noted that the distribution is slightly skewed due to the fact that we only computed severity in nerves actually injured, and therefore the value of zero is excluded.

For simple group comparison we used the Mann-Whitney non-parametric test. To quantify the difference in mobility between the healthy shoulder and the affected side, we applied a MANOVA (multivariate analysis of variance) with the following structure. The dependent variable is one of several possible measurements of movement. The indicator of the shoulder, affected *v* healthy, is entered as a within-subject effect. The presence of a lesion of the rotator cuff, the presence of nerve injury and age (above or below 50 years) are entered as between-subjects effects. The difference between the two shoulders is denoted by 'loss' (see Table V). The estimates are adjusted of the difference (loss) between the shoulders where each effect is adjusted for all other factors involved. The p values are all based on one model containing all effects.

It should be noted that the outcome variable of primary interest is the 'loss', calculated as the difference between two within-patient measurements. There is a problem with non-normality of the within-patient differences between the two shoulders due to an over-representation of the value 'zero' where the measurements of the shoulders coincide; all other values follow a normal distribution. This problem cannot be solved by any transformation. In the absence of a reasonably interpretable non-parametric regression technique, we present averages based on these MANOVA results since they provide clinically useful and interpretable estimates; the data did not contain any outliers at all. Since the MANOVA is rather robust against skewness in the absence of outliers and a sufficient number of observations, we also provide confidence intervals and p values, although these should be interpreted with some caution.

Results

The number of dislocations in men and women and the number of patients with nerve injury are shown in Figure 1. Denervation (axonal loss) was seen in 37 patients (48%). The mean number of nerves involved was 1.8, the mean maximum severity was 1.8 and the mean severity per nerve was 1.6. The axillary nerve was the most frequently involved (42%). The numbers of the other nerve lesions were: suprascapular, seen in 14%, radial, 7%, musculocutaneous, 12%, median, 4% and ulnar, 8%.

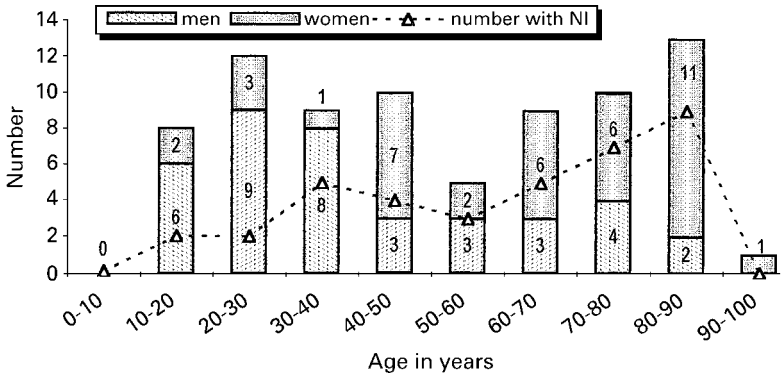

Fig. 1

The number of dislocations in men and women and the number of patients with nerve injury (NI) related to age. According to the literature there are two peaks in the incidence of dislocations.

Table II. Combinations of nerves involved in the 37 patients with denervation (total number of nerves affected 66). The axillary nerve is almost always involved in cases with a combination of nerve lesions

Number of nerves	Combination of nerves*						Number of patients
	SS	AX	RA	MC	ME	UL	
1	+						2
		+					15
				+			1
						+	1
2	+	+					6
		+	+				1
		+		+			3
				+		+	1
3	+	+	+				1
	+	+		+			1
		+	+	+			1
		+	+			+	1
		+		+	+	+	1
4		+		+	+	+	1
5							0
6	+	+	+	+	+	+	1

* SS, suprascapular; AX, axillary; RA, radial; MC, musculocut; ME, median; UL, ulnar

Table III. The frequency of nerve lesions and mean severity (± SEM; range) per nerve in 37 patients. The axillary nerve is the most frequently and most severely injured

Nerve	Count	Mean severity
Suprascapular	11	1.3 ± 0.17 (0.5 to 2.5)
Axillary	32	1.9 ± 0.17 (0.5 to 4.0)
Radial	5	1.5 ± 0.50 (1 to 3.5)
Musculocut	9	1.2 ± 0.15 (1.0 to 2.0)
Median	3	1.8 ± 0.44 (1.0 to 2.5)
Ulnar	6	1.4 ± 0.27 (1.0 to 2.5)

Frequently, combinations of nerve lesions were seen. A solitary nerve lesion was present in 51%. The average number of nerves involved in all patients with nerve injury was 1.8. The number of combinations, the frequency and the severity per nerve are given in Tables II and III.

Needle examination was used to investigate the recovery of nerve injury in 30 of the patients with nerve lesions (81%) at a mean of 15.7 weeks (9 to 26) after the trauma. Only two patients did not attend the follow-up. We discontinued the electrophysiological follow-up after the first EMG in five patients and after the second EMG in 19, because they had made a complete clinical recovery. A third EMG was made in 11 patients and two were followed for an extended period.

The mean nerve injury score at the last follow-up and EMG was 1. Four patients out of 32 with axillary nerve injury (12.5%) had persistent abnormalities of the EMG (grade 1 to 1.5) with associated paresis (M3 or M4). The initial denervation score in these patients was graded between 2.5 and 3.5.

In a patient with an almost complete denervation of the radial nerve (grade 3.5), with wrist drop and metacarpophalangeal paresis, recovery of triceps and brachioradialis was complete and extension of wrist and fingers graded M4. The final EMG score was 1.5. In the other patients suffering nerve injury muscle strength recovered well up to M4.5 or M5.

Attempts to relate sensation to lesions of a motor nerve were of low value. Motor abnormalities of the axillary and musculocutaneous nerve were only accompanied by disturbances of sensation in four out of eight cases. Abnormalities of sensation in the areas of the nerves of the lower arm occurred only twice, both in cases with severe nerve injury.

In most patients, recovery of shoulder function was incomplete (Table IV). Stiffness from capsular adhesion was not seen. Many of the patients showed a variable degree of restriction of motion in comparison with the healthy side, even after correction for adverse factors (Table V). We could not find a satisfactory explanation for this.

The duration of recovery from axillary nerve injury was prolonged, lasting as long as 35 weeks. Tables IV and V summarise the function of the shoulder and the influence of injury to the axillary nerve. Restricted movement of the shoulder was closely related to a lesion of the axillary nerve. The loss of movement, in comparison with the healthy side, is also shown in Tables IV and V. The reduction was most marked during active function and varied from 8° in external rotation, to 19.7° in flexion, compared with patients without a lesion of the axillary nerve.

Injury to the suprascapular nerve was found to be a relevant factor only in active glenohumeral abduction and flexion. The difference from patients without a lesion of this nerve was deterioration of abduction of 4.8° and of flexion 18°.

Age was a relevant factor in the recovery of function in the shoulder, with motion being most restricted in patients over 50 years of age (Table V).

In 19 patients persistent pain was the reason for MRI. A rupture of the rotator cuff was found in nine patients

Table IV. Observed range of shoulder movement[16] (degrees, confidence interval (CI)). Comparison of the mean function of the healthy and the involved shoulders (n = 72)

	Healthy side		Affected side		
Type of movement	**Mean**	**CI**	**Mean**	**CI**	**Mean difference**
Glenhumeral abduction*					
Active	89.9	89.6 to 90.2	84.2	81.4 to 87.0	-5.7
Passive	89.9	89.6 to 90.2	87.1	85.4 to 88.8	-2.8
Total abduction	174.5	170.1 to 178.9	158.8	151.0 to 66.6	-15.7
Flexion active	173.8	170.8 to 176.8	156.9	149.0 to 163.9	-16.9
Flexion passive	174.2	171.8 to 176.6	162.1	158.7 to 167.5	-12.1
External rotation active	52.1	49.3 to 55.9	42.1	38.0 to 46.2	-10.0
External rotation passive	52.8	50.3 to 55.3	46.1	42.8 to 49.4	-6.7
Internal rotation active†	6.0	5.6 to 6.4	5.0	4.6 to 5.4	-1 step

* the active and passive glenohumeral abduction comparable; loss of range of motion was due to capsular stiffness, recovery of muscle strength was nearly complete
† Internal rotation score; 1 hand able to reach lateral thigh; 2, buttock; 3, lumbosacral junction; 4, L3; 5, T12; 6, interscapular[27]

Table V. Estimates of mean losses (with confidence intervals, CI) of shoulder movement adjusted for lesions of the rotator cuff between the healthy and the affected side related to lesions of the axillary nerve and age

	Overall [(n,a)]		Axillary nerve injury [(a)]					Age (yr) [(n)]				
Type of movement	**Estim**	**CI**	**No**	**CI**	**Yes**	**CI**	**p value***	**<50**	**CI**	**>50**	**CI**	**p value***
Glenohumeral abduction												
Active	-10.0	-6.8 to 12.9	-6.0	-3.4 to 8.7	-14	-11.3 to 16.6	<0.001	-5.9	-2.7 to 10.1	-14.1	-9.9 to 18.3	0.01
Passive	-5.5	-1.7 to 9.3	-4.1	0.7 to 7.3	-6.9	-3.7 to 10.3	NS	-5.5	-0.3 to 10.7	-5.5	-0.3 to 10.7	NS
Total abduction	-22.1	-15.6 to -28.6	-14.9	-9.4 to 20.4	-29.3	-23.8 to 34.8	<0.001	-9.7	-1.2 to 15.9	-34.5	-26.3 to 43.0	0.03
Flexion												
Active	-25.0	-18.7 to 31.3	-15.4	-10.0 to 20.8	-35.1	-29.7 to 40.0	<0.001	-13.8	-5.3 to 22.7	-36.2	-27.7 to 43.7	0.001
Passive	-18.2	-12.7 to 13.6	-10.8	-6.2 to 15.5	-25.6	-20.9 to 30.2	0.002	-10.0	-2.6 to 17.2	-26.4	-19.7 to 33.8	0.02
External rotation												
Active	-14.0	-10.2 to 18.1	-10.0	-6.6 to 13.5	-18	-14.5 to 21.4	0.02	-6.1	-0.7 to 11.5	-21.9	-16.5 to 27.3	0.02
Passive	-8.6	-5.7 to 11.6	-5.9	-3.4 to 8.6	-11.3	-8.6 to 13.8	NS	-3.2	0.9 to 7.5	-11.8	-9.7 to 16.1	NS
Internal rotation active	-0.5	-0.3 to 0.8	-0.4	-0.2 to -0.6	-0.5	-0.3 to 0.8	NS	0.5	-0.2 to 0.8	0.5	-0.2 to 0.9	NS

[(n)] adjusted for axillary nerve lesions
[(a)] adjusted for age
* MANOVA model: between-subjects-effect
Even after correction of lesions of the axillary nerve, lesions of the cuff and age, restriction of movement between the two sides is significant for all tested directions ($p < 0.001$). Over the age of 50 years recovery of shoulder function becomes significantly worse.

(average age 59.6 years). It is probable that ruptures of the cuff were more frequent than were detected clinically, but since the MRI was carried out after three months it is uncertain whether these tears were caused by the initial trauma or were present before it. In all these patients, even in the presence of a tear, the pain eventually abated. Three patients with a cuff rupture were given a subacromial injection of corticosteroid. In all patients with cuff rupture, the mean loss of active abduction was 7.6° in comparison with patients without a clinically detectable or MRI-proven rupture of the cuff. In most of the tested directions the loss of function, attributed to cuff rupture, was not significant, although the number of patients who had demonstrable tears was small.

Risk factors of nerve injury. We searched for risk factors associated with nerve injury using logistic regression analysis and can identify the following:

Age. As shown in Figure 2, the probability (expressed as odds) increases with a factor of 1.3 for every ten-year period ($p = 0.007$). The severity of the lesion also increases with age, but was too small to be of relevance.

Haematoma. Bruising about the shoulder is associated with a 4.4 times (1.5 to 13.2; $p < 0.01$) greater probability of nerve injury. In patients with a haematoma the number of nerves involved and the severity of the lesion were significantly increased. The average increase in the number of affected nerves was 1.3 (0.6 to 2.0; $p < 0.001$), the average increase in the maximal severity 0.9 (0.4 to 1.4; $p < 0.001$) and of the average severity per nerve 0.4 (0.03 to 0.8; $p = 0.04$).

Associated fractures. These were present in 14 of our patients; the tuberosity was affected in 12 and the rim of the glenoid in two. In ten (71%) nerve lesions were present. The probability of nerve injury was doubled by the presence of an associated fracture. Although these figures do not reach significance, the associated confidence interval is quite wide (odds ratio 2; 0.5 to 7.8; $p = 0.3$). The severity of nerve damage and the number of nerves involved were significantly increased ($p = 0.02$; Mann-Whitney non-parametric test) by the coincidence of a fracture.

Method of reduction. The techniques of reduction used were as follows: Stimson in 6 patients, Milch in 4, Hippoc-

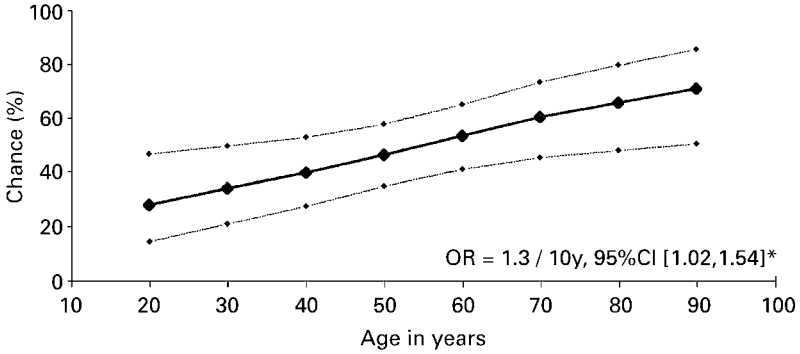

Fig. 2

The risk that a dislocation is complicated by nerve injury increases with age.

rates in 31, Kocher in 25, and in 11 it was not recorded. No relationship to nerve injury was observed in the method of reduction, the number of attempts or the difficulty encountered. There was no evidence to link the incidence of nerve lesions to technique. Gender, the cause of trauma, the position of the arm during the fall, the side, dominance, diabetes mellitus or the use of an anticoagulant did not influence the frequency of nerve damage.

Discussion

Our study has shown that nerve injury is common in dislocations of the shoulder. An incidence of 48% was present across all age groups. These lesions recovered within a period of 12 to 45 weeks. The recovery of the EMG was complete or almost complete and, in all but four patients, normal muscle strength returned. Shoulder function, however, showed significant loss of movement in injury to the axillary nerve, even after the nerve had recovered.

The functional outcome depends on the type of physiotherapy used. A less than assiduous protocol of physiotherapy may leave the patients with considerable limitation.

The most probable clue to the presence of a nerve lesion is paresis. Although there was a clear relationship between paresis and nerve injury we did not identify a precise point in testing of muscle power that can be used to determine the presence or absence of nerve injury. We found that the best indicator of nerve injury after one week was paresis of the deltoid muscle.

In order to detect nerve injury in the early stages, we tested the sensation in the distribution of the axillary and musculocutaneous nerves. Our results confirm the opinion expressed in the prospective studies mentioned above, that examination of sensation of the axillary nerve does not give a reliable indication as to the presence of lesions of the motor nerves. Abnormalities of sensation of the lower arm point to severe nerve injury.

Differentiating between nerve lesions and rupture of the rotator cuff, Neviaser, Neviaser and Neviaser[23,24] stated that, in patients over 40 years of age who are unable to abduct the arm after reduction, rupture of the rotator cuff is the probable cause. If this injury is found, repair of the lesion alone will correct the problem. We think that when paresis is present, nerve injury should be suspected. We found ruptures of the cuff in only 13% and repair was not required.

A combination of nerve injury and an MRI-proven cuff tear was found in seven of our series of 77 patients. In the acute stage we do not recommend exploration of the rotator cuff in a patient with apparent paresis who is beyond the age of retirement. If loss of abduction is due to a nerve lesion, muscle strength will recover spontaneously. If there is, in addition, a ruptured cuff, it is possible that the latter is of long standing. Furthermore, the symptoms of an acute rupture often resolve spontaneously. In young patients, however, with an extensive acute tear of the rotator cuff, restoration of the normal anatomy is important. In these cases, needle electrodiagnosis is essential in order to identify additonal nerve lesions.

Since the prognosis of nerve lesions after dislocations of the shoulder is favourable, it is not necessary to carry out EMG routinely. An assiduous programme of physiotherapy is important to prevent stiffness of the shoulder, especially in patients with poor muscle strength due to nerve injury. If, in the case of paresis, passive function is preserved by the therapist, active function will recover with the nerve palsy. Physiotherapy should be continued until muscle strength is regained and the function of the shoulder restored. In the treatment of patients with a rupture of the cuff, exercises are added which train other stabilising muscles of the shoulder.[25,26]

In cases of paralysis or severe paresis it is essential to perform an EMG at three weeks. If, after three months, no electrophysiological and clinical improvement occurs, exploration should be undertaken.[9]

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