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Electromyographic findings in shoulder dislocations and fractures of the proximal humerus: comparison with clinical neurological examination

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Abstract

There is no consensus of opinion about the frequency of associated nerve lesions in anterior shoulder dislocations and fractures of the proximal humerus. We undertook a prospective study to assess the incidence, the severity of the nerve injury and the diagnostic value of electromyographic examination; 215 patients were included. We performed neurological examination and needle electromyography (EMG). Nerve injury was graded according to a denervation score at the EMG. EMG disorders were seen in 133 patients (62%). Testing of sensibility and clinical reflexes proved not to be a reliable indicator for EMG abnormalities. Detection of axonal lesions by grading muscle strength based on the MRC score after these shoulder traumas is difficult. The findings of this study imply that by clinical examination alone a large number of axonal lesions remain undetected.

Keywords: Clinical examination; EMG; Nerve injury; Proximal humeral fractures; Shoulder dislocation

1. Introduction

There is no consensus of opinion about the frequency of associated nerve lesions in anterior shoulder dislocations and fractures of the proximal humerus. In particular, in fractures the frequency of this complication is not well known. In studies with electromyographic (EMG) investigation a greater number of nerve lesions is found than in studies with clinical examination alone. Only three prospective clinical studies with EMG investigation exist concerning dislocations and fractures of the shoulder, but all of them are incomplete. The percentages in these studies vary between 30 and 45 [1–3]. The study of de Laat et al. was performed by our group. EMG abnormalities were found in 45% of the patients. Electromyography was not performed in all (66%) and only in a few a second EMG was available.

We undertook a prospective clinical and EMG study to further assess the incidence, the severity of the EMG disorders and the value of EMG examination next to clinical examination. In this study the value of EMG is discussed and a comparison is made with clinical examination.

2. Patients and methods

In total, 215 patients (dislocations 74, fractures 141; men 58, women 157) were collected for a prospective follow-up study. All injuries were due to low-velocity traumas like a simple fall, in 21 patients in sports incidents. The average age was 64.2 years (range 12.6–94.3 years). Patients were seen for clinical examination at fixed times. This examination included sensory function of axillary, musculocutaneous, median and ulnar nerve, stretch reflexes of biceps and triceps, muscle strength of deltoid, supraspinatus, infraspinatus, biceps, triceps, flexor carpi radialis and abductor digiti minimi. Muscle strength was graded according to the MRC scale. In cases of severe paresis investigation of other muscles of the corresponding nerve was added. Note

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that testing of mustle strength was not possible in many patients within the first few weeks due to severe pain and fractures. In these patients no grading of muscle strength was performed. Grading was only performed in dislocations and clinically healed fractures in which pain was a minor problem (eventually after using adequate pain medication.)

Muscle atrophy by measuring the circumference of the arm was not scored because of the large swelling of the arm in many patients (68%) due to haematoma. A haematoma was scored as being absent or present. When present it was scored if the bruising involved the whole arm or only the upper arm. To detect cuff tears as the cause of persisting pain and persisting loss of muscle strength, magnetic resonance imaging (MRI) was added in these cases after 3 months. On MRI, cuff ruptures were scored as being absent or present, regardless of the size.

EMG needle examination was performed (after 3 weeks) with a concentric needle electrode (Medelec Sapphire Premiere) at three locations for each muscle to optimize the chance of finding abnormalities. These muscles were deltoid: anterior, middle, posterior part (axillary nerve), infraspinatus (suprascapular nerve), biceps (musculocutaneous nerve), triceps (radial nerve), flexor digitorum sublimis (median nerve), adductor pollicis (ulnar nerve). This set of muscles was chosen based on electrophysiological findings in an earlier study [3].

In almost all patients the shoulder injury was due to an indirect trauma; needle examination was not performed in direct traumatized muscles.

Active denervation, motor unit action potential (MU-
APs) morphology and the pattern of voluntary contraction was scored to classify the severity. In the acute stage the denervation was scored according to a scale based on the quantity of fibrillation potentials and the pattern of recruitment, modified after Daube [4] and Wilbourn [5]:

Severity	Properties
0	No fibrillation potentials (FP) or positive sharp waves (PSW), normal recruitment
1	Short or persistent series of 1 FP or PSW in two or more places, normal recruitment
2	Moderate number of FP/PSW in three or more places, normal or decreased recruitment
3	Many FP/PSW in all examined places, decreased recruitment
4	Screen filled with FP/PSW in all examined places, absent recruitment

In the case of a difference of severity of denervation
at different places in one muscle a score between both scores was used and expressed as (score a + score b)/2. This resulted in a 9-point scale (Table 1).

The average duration between trauma and EMG was 5.4 weeks. Not all EMGs were performed at the desired time (3–4 weeks). The range was 1.1–13.4 weeks. Due to the poor compliance of some patients two were seen within a 2-week period after the trauma and three after a 10-week period.

We used a simple linear regression model to detect if the number of nerves involved as well as of the severity of the injury, both adjusted for age and the diagnosis (dislocation/fracture), significantly decreased with time.

Recovery was evaluated by needle examination on basis of reinnervation and the maximal voluntary contraction pattern after 16.5 weeks (range 9–27 weeks). For recovery it is not reliable to score denervation only, because denervation can disappear by fibrosis of the muscle. The recovery was scored as follows:

Table 1
Number of electromyographic nerve lesions graded according to the denervation score ($n = 215$)[a]

	Normal[b]	0.5	1	1.5	2	2.5	3	3.5	4	Total NI+[c]
Axillary	100	4	27	37	13	18	9	5	2	115
Suprascapular	135	2	24	21	14	15	3	1	0	80
Radial	184	19	16	9	6	0	0	1	0	51
Musculocutaneous	164	0	21	12	14	3	0	1	0	51
Median	188	0	11	11	1	3	0	1	0	27
Ulnar	200	0	10	0	2	3	0	0	0	15

[a] Denervation score 0.5–4; see text.
[b] Normal: no EMG abnormalities.
[c] Total NI+: total number of axonal lesions for the specific nerve.

Score	Nerve injury at follow-up
0	Interference pattern, no fibrillation potentials or positive sharp waves
1	Mild reduced pattern, slight loss of MUAPs, FP/PSW possible
2	Moderate reduced pattern, mixed pattern with highfrequent units, FP/PSW possible
3	Severe reduced pattern with one or two existing units, FP/PSW possible
4	No motor units, FP/PSW

Again, when the EMG score was different in different places in the same muscle an average score was calculated. Needle examination was repeated (interval 2–3 months) until the EMG was normal or if there remained only minor disorders in presence of reinnervation and a reasonable recruitment pattern.

In the beginning of the study we tried to perform a nerve conduction velocity (NCV) study, because, when measuring denervation, cases of slight nerve injury with only demyelination and partial conduction block with sparing of axons could be missed. Because the site of the presumed nerve lesions in the shoulder is at the shoulder joint [3,6] and stimulation for NCV studies is only possible proximal from the site of lesions for the axillary, musculocutaneous and suprascapular nerves, the presence of a partial conduction block cannot be determined. Also, it is often impossible to achieve supramaximal stimulation of nerve fibers when supraclavicular stimulation is performed [7,8]. Performing NCV studies caused discomfort in a great number of the patients due to the movements of the injured shoulder. We therefore stopped this part of the investigation after 70 patients. We support the opinion in the literature that a nerve conduction study in the shoulder girdle region is difficult and not reliable [9,10].

2.1. Statistical analysis

To assess the value of clinical testing of muscle strength as an indicator of nerve injury, we calculated the sensitivity and the specificity of testing of the strength of one muscle of the corresponding nerve related to the EMG findings of this nerve (Table 2). Muscle strength was scored as normal (clinical 'not injured') if the strength was graded $>$ M4.

To quantify the ability of clinical evaluation of muscle strength testing to classify patients as 'nerve injured' or 'not injured', a discriminant analysis was used. Analyses were performed on a per-nerve basis, the grouping variable being the presence or absence of nerve injury in the EMG. Independent covariates (predictors) were: the corresponding muscle strength as measured, the duration of follow-up, the presence of pain and the presence of rotator cuff damage. Predictive ability was quantified as sensitivity and specificity. Moreover, the positive predictive value is mentioned. Basically two models are fitted: one relating muscle strength to nerve injury, adjusting for age, pain and cuff lesions; in the other, the presence of haematoma was added to the set of possible predictors.

In addition, a logistic regression model has been fitted to the data. Although this is approximately equivalent to a discriminant analysis (for normally distributed data) it has the added advantage of providing an odds ratio and corresponding confidence interval to quantify the relation between each independent predictor and the probability of nerve injury.

Table 2
Sensitivity and specificity of clinical testing of muscle strength (MRC) at 1 and 4 weeks related to EMG disorders of the corresponding nerve (unadjusted measurements)

Nerves	Sensitivity[a]		Specificity	
	At 1 week	At 4 weeks	At 1 week	At 4 weeks
Axillary	0.96	0.94	0.23	0.35
Supra-scapular	0.89	0.84	0.29	0.42
Musculocu-taneous	0.43	0.24	0.81	0.88
Radial	0.58	0.29	0.78	0.90
Ulnar	0.31	0.23	0.93	0.98
Median	0.31	0.17	0.93	0.96

[a] The sensitivity of clinical testing of muscle strength related to EMG abnormalities of the corresponding nerve is low except for testing of the deltoid in the case of axonal axillary nerve lesions.

3. Results

3.1. Electromyographic results

EMG indicated nerve injury was seen in 133 patients (62%) (dislocations 50%, fractures 68%). In patients with denervation the average number was 339 nerves involved in 133 patients. The most frequently involved nerve was the axillary nerve (115 times, 53%). The numbers of the other nerve lesions were: suprascapular nerve 80 (37%), radial nerve 51 (24%), musculocutaneous nerve 51 (24%), median nerve 37 (12%) and ulnar nerve 15 (7%). Frequently the lesions of these nerves were seen in combination.

The pattern of severity according to the denervation score of nerve injury per nerve is shown in Table 1. The variation of severity was largest in the axillary and the suprascapular nerve. The average severity per nerve was highest in the axillary nerve (grade 1.8, S.D. 0.8), suprascapular nerve grade 1.6 (S.D. 0.7) and equal for the other nerves tested (1.5, S.D. 0.6).

Regarding the duration between trauma and EMG we found a significant decrease of the number of nerves involved as well as of the severity of the injury with time, both adjusted for age and the diagnosis (dislocation/fracture). The number of nerves involved decreased with an average of one per 5 weeks (95% CI: -1.5, -0.5; $P < 0.001$). The average severity per nerve diminished with 0.3 on the scoring scale during the same period (95% CI: -0.6, 0.0; $P = 0.03$).

The probability to detect a nerve injury also decreased with time. The odds ratio was approx. 0.8 per week (95% CI: 0.68, 0.91; $P < 0.001$), adjusted for age and the diagnosis (dislocation/fracture) (logistic regression model). In Fig. 1 we estimate the actual probability of nerve injury as a function of time for the specific distribution of age and diagnosis (dislocation/fracture) in our study population.

3.2. Clinical results

Testing of muscle strength at 1 and at 4 weeks (average 4.4) showed considerable variation between patients. As mentioned in Section 2, it was not possible to test all muscles reliably in every patient due to pain and fractures. Grading of the strength of all muscles could be assessed in 169 patients.

Sensory disturbances of the axillary nerve were clinically seen in 12 patients and in three patients of the musculocutaneous nerve. In all of these patients the gnostic sensibility (cotton-wool) was diminished and in all but one the vital sensibility (disposable needle).

In two dislocations gnostic and vital sensory loss was found in region of the median and ulnar nerve (volar side of the second till the fifth finger).

Clinical reflexes of the biceps were reduced (2 patients) or absent (two patients) in four patients. Abnormal reflexes of the triceps were seen in two patients (one diminished and one absent).

In 150 patients (70%) a haematoma was present. In 27 the bruising was small and limited to the lateral side of the upperarm. In 123 there was a large haematoma involving a large part of the upper and (a part of) the lower arm.

3.3. Correlation between clinical and EMG results

In Table 2 an overview is given of the unadjusted measurements of the sensitivity and the specificity of testing muscle strength (according to the MRC scale) in relationship to the electromyographical outcome (regarding EMG disorders as 'nerve injured'). Testing of the deltoid muscle showed a high sensitivity (96% at 1 week, 94% at 4 weeks) for the presence of EMG abnormalities of the axillary nerve. For the other tested muscles per corresponding nerve the sensitivity was low. The specitivity was low for all the tested muscles, except for the muscles of the ulnar and median nerve; however, the number of patients with axonal lesions of these nerves was small.

Although the finding of clinical paresis was a significant predictor, this did not imply that clinical examination of muscle strength was reliably predicting in these shoulder traumas whether a nerve injury was actually present or not. To quantify this predictive ability, a discriminant analysis was used. Clinical muscle testing was significant for the detection of electromyographic nerve lesions (except for the median nerve at 4 weeks, and the suprascapular nerve at 1 week). Discriminant analysis could not find the optimal cut off point for the grade of muscle strength (based on the MRC score) per nerve to decide whether or not EMG disorders are present (taking duration of follow-up, diagnosis and cuff lesions into account).

Regarding the analysis it appeared that the sensitivity of clinical examination of muscle strength according to the MRC score for the detection of EMG disorders was low (even for the deltoid muscle: 81% at 1 week, 77% at 4 weeks). The specificity, however, of the musculocutaneous, radial, ulnar and median nerve at 4 weeks was over 96%.

Sensory disturbances of the axillary nerve were clinically seen in 12 patients; in eight of them there was a proven axonal injury by EMG. The grade of denervation varied between 1.0 and 3.5 (mean 2.2, S.D. 0.9). Thus in only eight of 115 cases with an EMG lesion of the axillary nerve (7%) clinical sensory loss was present.

In the three patients in which sensory disturbances of the musculocutaneous nerve were seen, two had axonal injury (grade 1.5 and 2). The incidence of sensory loss due to a lesion of the musculocutaneous nerve was 4% (2/51).

In the four cases with abnormal clinical reflexes of the biceps, three had severe injury of the musculocutaneous nerve (grade 1.5, 2, 3.5). In both patients with abnormal reflexes of the triceps there was a moderate injury of the radial nerve (both grade 2).

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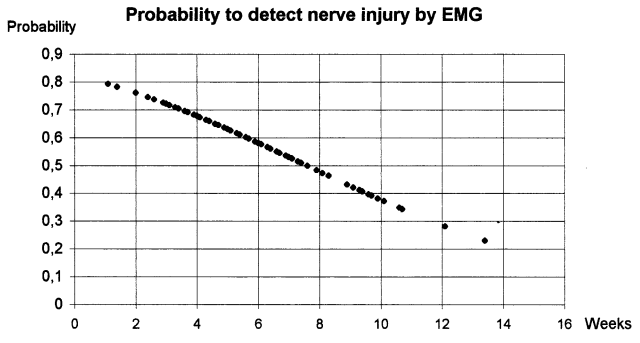

Fig. 1. Probability of detecting a nerve injury as a function of follow-up time (EMG). The probability decreases with time due to reinnervation.

presence of a haematoma demonstrated to be a sensitive predictor of suprascapular nerve EMG disorders. The association between (presence of) haematoma and (probability of) axonal injury of this nerve was highly significant (sensitivity 89%, $P < 0.001$) regardless of the size of the haematoma.

3.4. Follow-up

A second EMG was performed in 79% (105/133) of the patients with nerve lesions at 16.5 weeks (range 9–27 weeks) after the trauma; 12 patients dropped out of follow-up. In the other 16 patients the EMG examination was not repeated because there was only a slight grade of nerve injury without clinical deficit. A third EMG was done in 20 patients and two were followed for an extended period. The EMG investigation was stopped in patients who had a complete recovery of the strength of the corresponding muscles. This occurred after the first EMG in 18 patients and after the second EMG in 23 patients. The average grade of nerve injury at follow-up was 1 (S.D. 0.5).

Four patients out of 115 with axillary nerve injury (3%) had persistent EMG abnormalities (grade 1–1.5) with associated paresis (M3–M4). In a case with an almost complete denervation of the radial nerve (grade 3.5) with dropping hand and fingers the recovery of the triceps and brachioradialis was complete, of extensors of hand and fingers M4. The EMG score at the end was 1.5. The recovery of the muscles innervated by the other nerves was complete in all cases.

In all but two patients with sensory disturbances of the region of the axillary and musculocutaneous nerve, sensory recovery was complete within 18 weeks. In the other two cases (with severe axonotmesis of the axillary nerve) a slight clinical sensory loss was still present at the end of follow-up (16 and 27 weeks, respectively).

In the two patients with a shoulder dislocation and gnostic and vital sensory loss in region of the median and ulnar nerve, the sensory disturbances did clinically not recover completely.

In the three of four cases with a severe EMG injury of the musculocutaneous nerve and abnormal clinical reflexes of the biceps, the electrophysiological recovery was not complete and the reflex clinically stayed diminished. Muscle strength recovered completely in all.

In both patients with abnormal reflexes of the triceps and moderate EMG abnormalities of the radial nerve, the recovery was electrophysiologically complete and the paresis and the reflex recovered clinically well.

4. Discussion

Associated EMG evidence of axonotmesis in anterior shoulder dislocations and fractures of the proximal humerus due to a minor trauma was present in 62% of the patients. The infraclavicular nerves of the arm and the supraclavicular originated suprascapular nerve were involved frequently.

The findings of this study show that by clinical examination alone a lot of nerve lesions remain undetected. Of all 1290 nerves ($n = 215 \times 6$) assessed by clinical testing of the strength of one of the corresponding muscles (based on the MRC score), the number of axonal nerve lesions that could not be detected at all (possibly due to pain or fractures) was 149 at 1 week and 50 after 4 weeks. If $>$ M4 was regarded as being clinically 'not injured', 58 (clinically less important) axonal lesions of the nerves at risk were missed at 1 week and 114 at 4 weeks.

Due to reinnervation the chance to detect EMG abnormalities decreased with time. This means that to detect axonal lesions and to determine the severity of the injury properly the EMG examination should be performed as soon as possible (about 3 weeks). On the other hand, this finding does show that nerve injury in these shoulder traumas has a good prognosis for recovery.

The findings of this study show that in almost all cases with axonal lesions recovery occurres to a clinical acceptable function with MRC scores of $>$ M4 in 12–45 weeks. Only four patients had clinically significant impairment at follow-up.

The suprascapular nerve recovered fast and in all cases completely. 'Long' nerves with paresis of muscles of lower arm and hand did recover more slowly and electrophysiological recovery was not always complete.

Clinical function of muscle recovered well with conservative treatment. Persistent nerve injury was the reason of a lasting paresis only in four patients with axillary nerve lesions. A slight paresis of the extensors of the fingers (M4) remained after an almost complete denervation of the radial nerve (1/51). The other nerves recovered well in all cases.

In this study no clear answer could be given on the question if the neurological injury in these traumas is localized in the cords of the plexus or the peripheral nerves. Nerve injury is due to the same mechanism in shoulder dislocations and proximal humeral fractures in low-velocity traumas. During the trauma the nerves are stretched over the humeral head and traction injury can occur in the cords of the infraclavicular plexus or the terminal branches (nerves). Lesions of the suprascapular nerve in these traumas are due to forcefull abduction beyond physiological limits [1,11–13]. Due to the trauma mechanism it is likely that lesions of two individual nerves can occur at the same time both with electrophysiological disorders.

In 30% (40/133) only one nerve was involved and combinations of lesions of the same cord were not frequent. The most frequently found combination was a lesion of the axillary and the radial nerve (originated

from the posterior cord) in 38% (46) of 120 patients with axillary and/or radial nerve lesions. The number of the other cords were: median and musculocutaneous nerve 30% (18/60), median and ulnar nerve 24% (8/34). However, being that in the majority of all the nerves lesions no combination of two nerves from the same cord was seen and in addition lesions of two individual nerves of one cord also can occur at the same time, it is most likely that the lesions were in the peripheral nerves rather than in the plexus.

Clinical testing of sensibility to evaluate the presence of nerve injury in these shoulder traumas is not a useful clinical marker. Motor abnormalities of the axillary and musculocutaneous nerve were seldom accompanied by disturbances of sensibility. This means that the finding of a normal sensibility does not exclude the presence of significant axonal injury. Sensory disturbances of the median and ulnar nerve were rare (two instances). If present, however, this was always associated with axonal nerve injury.

Abnormalities of the reflexes of the triceps and biceps muscle were rare. They were only seen in severe cases of muscle palsy. On the other hand, in many cases with severe nerve injury reflexes were clinically intact. Therefore in the case of clinically normal reflexes axonal nerve injury can not be excluded.

Our findings show that detection of nerve lesions by clinical testing of muscle strength according to the ordinal scale of the MRC score after these shoulder traumas is difficult.

We conclude that clinical examination is not reliable in detection of axonal nerve injury after these shoulder traumas. Because of the favourable prognosis of these axonal lesions performing EMG routinely is not necessary, but one should keep in mind that a nerve injury can complicate the shoulder trauma leading to a prolonged recovery of muscle strength and function of the shoulder. In the case of sensory disturbances of the median and ulnar nerve or decreased clinical reflexes, the presence of severe nerve lesions is probable and EMG investigation is recommended. Also, because most patients recover clinically in 3 months, patients with clinical deficit at that time must be investigated with EMG for the presence of persistend nerve dysfunction.

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