Nerve lesions in proximal humeral fractures

Cornelis P. J. Visser, MD,[a] L. Napoleon J. E. M. Coene, MD,[b] Ronald Brand, PhD,[c] and Denes L. J. Tavy, MD,[b] *Leiderdorp, The Hague, and Leiden, The Netherlands*

In the literature nerve injury is not frequently considered a problem in proximal humeral fractures. Only a few studies exist concerning traction injury of nerves in fractures of the proximal humerus after low-velocity trauma. Almost all of them are retrospective and did not use electromyography. Patients with identical fractures can show quite different outcomes, which vary between complete recovery and severely limited shoulder function. On the assumption that nerve lesions can play a role in the recovery of conservatively and operatively treated proximal humeral fractures, we started a prospective follow-up study with electromyographic investigation. For this study, 143 consecutive proximal humeral fractures due to low-velocity trauma were included. According to the Neer classification, 93 were nondisplaced and 50 were displaced fractures. Denervation on the electromyogram was found in 96 patients (67%). The nerves most frequently involved were the axillary nerve (83 [58%]) and the suprascapular nerve (69 [48%]). Frequently a combination of nerve lesions was seen. Nerve lesions were much more frequent in displaced fractures (82% [41/50]) than in nondisplaced fractures (59% [55/93]). Complicating nerve lesions in patients older than 20 years of age were seen in about the same percentage of patients per decade. Nerve injury and the corresponding loss of muscle strength recovered well in all patients; however, the duration of the recovery was prolonged in cases with nerve lesions. Restoration of the function of the shoulder was less favorable. It is important to realize that, in both conservative and operative treatment of proximal humeral fractures, a paresis due to nerve injury can affect the restoration of shoulder motions. An electromyogram can be useful in the investigation of nerve lesions, because detection only by clinical examination proved to be very difficult. Because of the favorable electrophysiological recovery, no indication for exploration of nerve lesions was present in this series of fractures due to low-velocity trauma.
(J Shoulder Elbow Surg 2001;10:421-7.)

From Rijnland Hospital, Leiderdorp,[a] Leyenburg Hospital, The Hague,[b] and the Department of Statistics, Leiden University Medical Centre, The Netherlands.[c]

Reprint requests: Cornelis P. J. Visser, MD, Rijnland Hospital, Department of Orthopaedic Surgery, Simon Smitweg 1, 2353 GA Leiderdorp, The Netherlands.

1058-2746/2001/$35.00 + 0 **32/1/118002**
doi:10.1067/mse.2001.118002

INTRODUCTION

Proximal humeral fractures are osteoporosis-related fractures and are much more common in women than in men. Frequently the cause of injury is a fall on the outstretched hand from standing height.[14] Patients with identical fractures can show quite different levels of recovery, which vary between complete restoration and severely limited shoulder function. Furthermore, in the literature, no satisfactory explanation exists to explain why acute prosthetic replacement after displaced humeral head fractures has a variable prognosis regarding shoulder movement.

Nerve lesions are not frequently considered a problem in proximal humeral fractures. Only a few studies exist concerning nerve lesions in fractures of the proximal humerus. Almost all of them are retrospective and did not use electromyography (EMG). Three prospective studies with EMG exist,[2,7,12] but all are incomplete. Nerve lesions are produced by the same mechanism as that of anterior dislocations of the shoulder and fractures of the proximal humerus and are caused by extreme movements of the arm beyond physiological limits, leading to traction injury.[5,24]

Because of the suspicion that nerve injury can play a more important role than is recognized, we began a prospective follow-up study. The aim of the study was to determine the severity, the pattern, and the recovery of nerve lesions by electrophysiological investigation.

MATERIALS AND METHODS

In a prospective follow-up study 142 patients with proximal humeral fractures were evaluated. Patients with previous shoulder trauma or shoulder complaints or with poor cooperation (psychogeriatric) were excluded.

The radiographic evaluation included conventional radiographic views in 2 directions (anteroposterior and lateral views) and a 45° craniocaudal view[3,22] for determination of associated fractures of the tuberosities or glenoid. The fractures were classified according to the Neer classification.[17,18] In cases of pain persisting after 3 months, magnetic resonance imaging (MRI) was added to rule out rotator cuff ruptures. Radiographic evaluation was repeated after 4 weeks, in unstable fractures weekly, until consolidation. The position of healing of the frac-

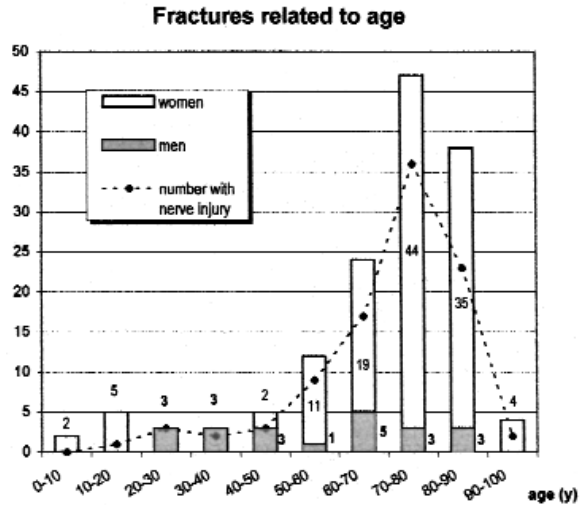

Figure 1 The number of fractures in men and women and the number of patients with nerve injury related to age. Note the large increase in the number of fractures in women older than 50 years of age.

ture was recorded and scored as anatomic, fair (bony impingement probable), or poor (fragments of bone blocking the joint).

Clinical evaluation was performed by measurement of active and passive motion of the shoulder and by testing muscle strength. Shoulder function was recorded from neutral position as described by Cave and Roberts[4] because measurement of motions in the scapular plane are not reliable and not reproducible in paretic shoulders. Internal rotation and external rotation were measured with the elbow at the side. Examination of abduction of the shoulder was also performed with the scapula fixed to eliminate compensatory movements of the scapulothoracic joint in evaluation of motions of the glenohumeral joint.

Muscle strength of the deltoid, supraspinatus, infraspinatus, biceps, triceps, flexor carpi radialis, and abductor digiti minimi was graded according to the Medical Research Council scale.[16] Strengths between the grades of this scale were scored as –0.5. In cases of severe paresis, investigation of other muscles of the corresponding nerve was added. No scoring system was used because we wanted to evaluate the effects of nerve lesions on separate motions and strength.

Follow-up ended when the recovery was complete or when the recovery of nerve injury or shoulder motions was not complete, but muscle strength and the function of the shoulder did not improve between 2 consecutive clinical control sessions (interval of 6 to 8 weeks).

Electrophysiological investigation

For diagnosis of the severity of nerve lesions, the number of nerves involved, and the recovery of the nerve injury, EMG was performed with a concentric needle electrode and was repeated every 8 weeks until recovery. The following muscles were tested[12]: deltoid—anterior, middle, and posterior part (axillary), infraspinatus (suprascapular), biceps (musculocutaneous), triceps (radial), flexor digitorum sublimis (median), and adductor pollicis (ulnar nerve).

For classification of the severity of active denervation, motor unit action potentials morphology and the pattern of voluntary contraction were scored. Denervation was scored according to a scale based on the quantity of fibrillation potentials and the pattern of recruitment, modified after Daube[6] and Wilbourn.[27] This resulted in a quantitative scale, which has been described previously.[12] Disturbances of nerve conduction were not studied because nerve conduction velocity studies in the shoulder region are difficult to perform and are not reliable.[19,28] Recovery was also evaluated by needle examination on the basis of reinnervation and the maximal voluntary contraction pattern and was scored according to a quantitative scale ranging from 0 to 4.[26]

Table I Details of 142 patients with 143 fractures of proximal humerus

Characteristics	Data
Absolute No.	
Fractures	143
Sex	
Men	21
Women	122
Side	
Left	75
Right	68
Nerve injury	96
Men	15
Women	81
Type of fracture (Neer class)	
I	93
II	1
IIIA	12
IIIB	9
IIIC	7
IV2	5
IV3	4
IV4	3
V2	1
VI2A	2
VI4A	2
Caput	4
Means	
All fractures (n = 143)	
Age (y) (95% CI)	68.8 (65.7, 71.9)
Range	5-92
Men	56.5
Women	71
With nerve injury (n = 96)	
Age (y) (95% CI)	70.1 (68.0, 73.2)
Range	13-91
Men	53.9
Women	73.1
Nerves involved*	
Mean No. (95% CI)	2.8 (2.5, 3.1)
Mean maximum severity (95% CI)	1.8 (1.6, 2.0)
Mean severity/nerve (95% CI)	1.6 (1.5, 1.7)

*Calculated per patient in 96 patients with nerve lesions.

Table II Combinations of nerves involved (total number of nerves affected, 273)

	Combination of nerves								
No. of nerves	**SS**	**AX**	**RA**	**MC**	**ME**	**UL**	**All fractures**	**Neer I**	**Displaced**
1	+						7	5	2
		+					14	10	4
2	+	+					14	7	7
	+		+				1	—	1
	+			+			1	1	—
		+	+				3	1	2
		+		+			3	3	—
		+			+		2	1	1
			+		+		1	1	—
3	+	+	+				5	3	2
	+	+		+			4	2	2
	+	+			+		2	2	—
		+	+		+		1	—	1
		+	+			+	1	1	—
		+		+	+		1	1	—
			+	+	+		1	—	1
4	+	+	+	+			16	9	7
	+	+	+		+		2	—	2
	+	+	+			+	1	1	—
	+	+		+	+		1	1	—
	+	+		+		+	1	—	1
	+		+	+	+		1	—	1
	+		+	+		+	1	1	—
5	+	+	+	+	+		7	2	5
6	+	+	+	+	+	+	5	3	2
Total	69	83	46	42	24	9	96	55	41

Note that both the axillary (AX) and suprascapular (SS) nerves are frequently involved in cases with a combination of nerve lesions. Note also that in displaced fractures a combination was more frequent (86%) than in nondisplaced (Neer I) fractures (72%).
SS, Suprascapular nerve; *AX,* axillary nerve; *RA,* radial nerve; *MC,* musculocutaneous nerve; *ME,* median nerve; *UL,* ulnar nerve.

Statistical methods

Several statistical analysis techniques were used in this study. Logistic regression models were used to quantify the probability of an event, in particular that of a nerve injury. The independent variables were treated either as covariates (age) or as factors (presence of displacement of fractures, presence of rotator cuff lesion). Estimates are presented as odds ratios and associated 95% CIs. The severity of nerve injury (when present) is described by 3 parameters on an ordinal scale (ranging from 1 to 4): number of nerves involved, the maximum severity among all nerves injured, and the mean severity score for all nerves injured. In the case of a simple group comparison, we used the Mann-Whitney nonparametric test. To quantify the difference between the healthy shoulder and the shoulder affected in terms of range of motion, we applied a multivariate analysis of variance.

Treatment

The most important factor in the choice of treatment was the Neer classification.[17,18] Other important factors were the age and general condition of the patient. Duration of immobilization in conservative treatment depended on the patient's age. Patients younger than 50 years of age and all patients with a Neer III fracture were immobilized for 4 weeks to avoid any risk of secondary dislocation. After this 4-week period, exercises with a physiotherapist were begun to restore muscle strength and shoulder function.

Physiotherapy followed a standard protocol given by a group of selected therapists. The frequency was 3 times or more per week. It included passive and active exercises and translation techniques to prevent capsular stiffness of the glenohumeral joint.[9,13,15] All patients received the same kind of therapy; in cases of nerve injury the therapy was more frequent and lasted longer. All but 8 patients were treated conservatively. Two patients were treated with an abduction cast. The type of operation performed was reduction (Neer IV2A [2 patients]), osteosynthesis (unstable Neer III [2 patients], Neer IV2 [2 patients]), and prosthetic replacement (Neer IV4A [2 patients]).

RESULTS

Most of the 142 patients were women (85%). One woman had a fracture on both sides at different times; thus 143 fractures were included in the study. Women were older (71 years) than men (56.5 years) (Table I). The ages of our patients are comparable to the epidemiology of these fractures as reported in the literature[1,8,11,21] and correspond to a J-shaped curve (Figure 1).

Of the 143 fractures, 93 (65%) were nondisplaced (Neer I) and 50 (35%) were displaced. In patients

Table III Frequency of axonal lesions and mean severity per nerve

Nerve	No. (%)	Neer I (%)	Displaced (%)	Severity [mean ± SEM (range)]
Suprascapular	69 (48)	37 (40)	32 (64)	1.8 ± 0.08 (0.5-3.5)
Axillary	83 (58)	47 (51)	36 (72)	1.7 ± 0.08 (0.5-4.0)
Radial	46 (32)	23 (25)	23 (46)	1.5 ± 0.08 (0.5-2.0)
Musculocutaneous	42 (29)	21 (23)	21 (42)	1.6 ± 0.10 (1.0-3.5)
Median	24 (17)	12 (13)	12 (24)	1.5 ± 0.11 (1.0-3.5)
Ulnar	9 (6)	6 (5)	3 (6)	1.3 ± 0.23 (1.0-2.5)

Subdivision is made in nondisplaced (Neer I, n = 93) and displaced fractures (n = 50). The percentage of nerve lesions was greater in displaced than in nondisplaced fractures. The axillary and suprascapular nerves are the most frequently and most severely injured.
*Severity is the mean for all patients with lesions of that particular nerve (denervation score is graded 0-4; see text).

Table IV Observed and adjusted differences between nondisplaced (Neer I) and displaced fractures

	Neer I*	Displaced*	Difference†	95% CI†	P value†
Maximal severity (mean)	1.7	2.0	0.7	0.4, 1.0	<.001
Nerves involved (mean)	2.7	3.1	1.0	0.5, 1.6	<.001
Mean severity/nerve	1.4	1.7	0.3	0.1, 0.5	.004

The number of patients with nerve lesions is 96 (Neer I, 55; displaced, 41). Denervation was scored according to a scale from 0 to 4 (see text). On average, nerve lesions are more severe in displaced fractures and more nerves are involved. The differences are significant for all parameters of nerve injury.
*Observed (nonadjusted) estimates.
†Estimates adjusted for age and duration between trauma and EMG.

younger than 50 years of age (n = 18), 14 Neer I and 4 Neer III fractures were seen. The other types were seen in those aged between 50 and 90 years (Table I).

EMG findings

Needle examination performed at 5.6 weeks (range, 2.0-10.1 weeks; SD, 2.0 weeks) showed axonal loss (denervation) in 67% (96/143). Solitary nerve injury was only seen in 21 cases (7 suprascapular and 14 axillary nerve lesions) (Table II). The mean number of nerves involved for all patients with nerve injury was 2.8 nerves (Table I). This implies that frequently a combination of nerve lesions was seen. The axillary nerve was most frequently involved (83 [58%]) (Table III).

The most important factor in the incidence of nerve lesions is the type of fracture. Nerve lesions were seen more frequently in displaced fractures (82% [41/50]) than in nondisplaced (Neer I) fractures (59% [55/93]). In displaced fractures the risk of additional nerve injury was 4 times as high as that in nondisplaced fractures (odds ratio, 4.0; 95% CI, 1.64-9.98; $P = .002$). In addition, the percentage of individual nerve lesions was greater in displaced fractures (Table III) and axonal lesions were more severe, according to the denervation score (Table IV).

In patients older than 20 years of age, the incidence of nerve injury was more or less equally divided over the decades. There was a significant, but only slight, increase of the incidence with increasing age. The odds ratio per 10 years of age was 1.1 (95% CI, 1.02-1.30; $P = .007$).

Clinical results

One hundred twenty-three patients fulfilled the complete clinical follow-up. The mean duration of clinical follow-up was 26.5 weeks (range, 4.5-94 weeks; SD, 13.5 weeks). Clinical testing proved to be of low value in detection of axonal nerve lesions in these fractures. Nearly all patients showed muscle weakness at testing in the first few weeks after the trauma. It was difficult to distinguish between paresis due to a nerve lesion and muscle weakness due to pain or the fracture. Testing of shoulder muscles innervated by the axillary and suprascapular nerve was possible in only 55% (78) of the patients during the first 4 weeks. Of this number, muscle weakness was present in the deltoid or the rotator cuff muscles in 73% (57) due to axillary or suprascapular nerve lesions. Of all muscles tested, the most reliable predictor was testing of the deltoid muscle in the case of an axillary nerve lesion; however, the sensitivity was low (77%). The correlation of the clinical and EMG results was described in detail in another study.[26]

Clinical testing of sensibility proved to be of low value because motor abnormalities of the axillary and musculocutaneous nerves seldom were accompanied by disturbances of sensibility. Clinical sensory loss was present in only 7% of the patients with a lesion of the axillary nerve as shown by EMG and in 4% of those

with a lesion of the musculocutaneous nerve as shown by EMG.[26]

Muscle weakness due to nerve injury recovered well in all patients. Shoulder movements did not recover completely in all patients, even in young patients with fractures healed in anatomic position. We could not find a satisfactory explanation for this.

The influence of temporary nerve injury of the axillary nerve or the suprascapular nerve on shoulder motions was studied in Neer I fractures. The recovery of shoulder function in patients with lesions of the axillary nerve took longer, but this did not influence the ultimate recovery of shoulder movements in conservative treatment. However, this contradicts the results in shoulder dislocations found in another study.[25] In nondisplaced fractures without nerve injury, the recovery took 18 weeks (range, 4-43 weeks; SD, 9.9 weeks); in the case of lesions of this nerve, it took 26 weeks (range, 12-36 weeks; SD, 3.5 weeks).

Temporary injury of the suprascapular nerve (leading to decreased cuff function due to a paresis of the supraspinatus and infraspinatus muscles) led to significantly diminished active glenohumeral abduction (4.8°, $P = .04$) and active flexion (18°, $P = .02$) at the end in comparison with the healthy side.

At the end of follow-up, no or only slight limitations remained in general function and activities of daily living in 73% (90/123) of all patients with a complete follow-up (eg, lifting weight overhead, putting on brassiere). Nineteen percent (23/123) had limitations in ability to perform housekeeping or sports activities, and in 6% (7/123) there was disability in activities of daily living (eg, hair combing, washing the opposite axilla, perineal care).

With regard to the type of fracture, loss of shoulder motion was minimal in fractures healed in anatomic position. In general, the worse the position of the fracture, the worse the recovery of shoulder function ($P \leq .005$).

With regard to age, the final loss of range of motion of the shoulder was small in patients younger than 50 years of age. With advancing years, the loss of motion increased significantly. An evident deterioration of shoulder motion occurred, especially in those over the age of 50 years.

It is important to realize that these results were obtained with an assiduous therapy protocol focused on preserving passive movements of the glenohumeral joint. The consequences of nerve lesions may have been more serious if patients were treated without or with another type of physiotherapy because of loss of passive mobility of the glenohumeral joint, especially in cases with muscle weakness.

Radiologic findings

The position of Neer I fractures did not change during the radiographic follow-up despite the early start of physiotherapy. Secondary displacement was seen only 3 times. Severe inferior subluxation of the humeral head (top of the head depressed for more than half of the height of the glenoid) was found in 9 fractures (Neer I or III category) in the first few weeks after the injury. All of these patients (aged 35-89 years) had a large hematoma (bruising) of the arm and a severe paresis of the deltoid muscle. In 8 of 9 patients, axonal lesions were found with EMG (axillary nerve, 8; suprascapular nerve, 6). In all cases the subluxation disappeared during follow-up.

MRI was done in 17% (24/143) of the fractures. In 11% (16/143) a rotator cuff tear was found (9 by MRI and in 2 perioperatively). Because the MRI was performed after 3 months, it remained unclear whether these tears were caused by the trauma or were preexisting. A delayed union was present 3 times.

DISCUSSION

This study shows that axonal nerve lesions in proximal humeral fractures are much more common than has been demonstrated in the literature and are even more frequent than in humeral dislocations.[25] A possible explanation is that fractures occur as a result of a greater force than a dislocation. A greater force during the trauma can result in a more severe bony and more severe nerve injury. In older patients with a decreased ability of reaction and poor muscle strength, the trauma can be more forceful. The differences in nerve injury in nondisplaced and displaced fractures can also be explained by this greater force.

A second explanation of why more nerves are involved in fractures than in dislocations can be the rotational position of the arm. For a subcoracoid dislocation (the most common type of shoulder dislocation) to be produced, the arm must be in external rotation during the trauma. If the arm does not rotate externally, a subglenoid dislocation or a fracture will occur. In external rotation of the abducted arm, the neurovascular bundle slips off the humeral head, whereas in internal rotation, the bundle (especially the axillary nerve) is stretched over the humeral head. A fall on the outstretched arm in internal rotation not only may produce fractures but can cause major tension in all nerves[5,10,24] (Figure 2).

A remarkable finding of this study was the great number of suprascapular nerve lesions. We could find no other reason than a more forceful trauma in fractures to explain the great number of axonal lesions of this nerve (48%) when compared with the number in shoulder dislocations (14%).[25]

The clinical consequences of nerve lesions for the conservative treatment include the increased risk of capsular stiffness. A comparison of the results in the literature is difficult because information about nerve injury or a proper description of the physiotherapy provided is generally not present. Although this study showed that the position in which the fracture heals and the patient's age are the most important factors in

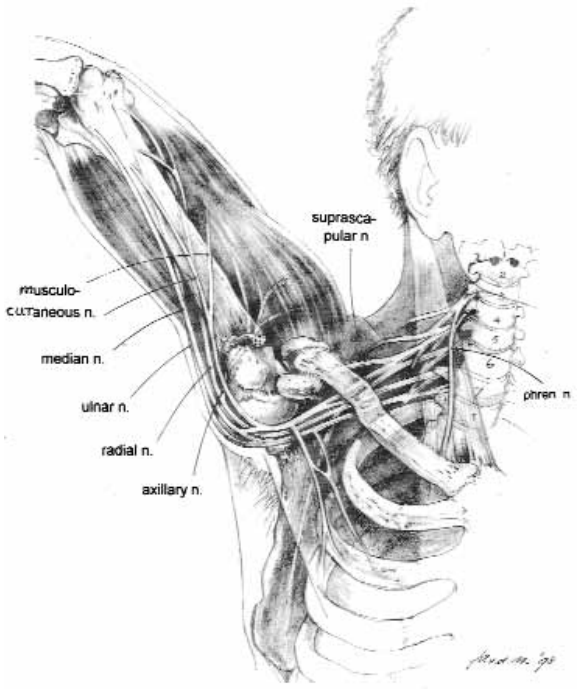

Figure 2 Trauma mechanism of proximal humeral fractures and associated nerve lesions. Traction at the neurovascular structures is greatest in abduction/hyperabduction, extension, and internal rotation of the arm.

the restoration of shoulder function, in our opinion, patients with proximal humerus fractures should be given a well-considered physiotherapy program. In cases of temporary nerve injury, exercises should not be left to patients alone. They may or will not be able to exercise actively because of muscular weakness. Physiotherapy must be initiated soon after the trauma to prevent shoulder stiffness and should be continued until muscle strength has recovered, and the maximal possible range of motion has been reached.

EMG investigations can be done (after 3 weeks) in patients with severe paresis or paralysis of muscles to determine the number of axonal nerve lesions and the severity of the axonal injury. After 3 months, an electromyogram can then be used to determine the recovery. In surgical patients with a displaced fracture this strategy can also be used. In case of doubt (for example, because of pain) it may also be advisable to perform EMG to detect nerve injury in order to get an impression about the patient's ability to exercise and about the duration of the recovery. Because traction injuries of the nerves in these shoulder traumas caused by low-velocity traumas recover well in general, it is not necessary to perform EMG routinely.

The consequences of our study findings for operative treatment are similar. From this study, we know that, in the majority of displaced fractures, nerve lesions are present. After osteosynthesis or prosthetic replacement, the patient may not be able to exercise properly because of paresis. This will increase the risk of capsular stiffness. This may well be one of the explanations for the poor results of prosthetic replacement in humeral head fractures.

No randomized study exists in which the results of acute and chronic prosthetic replacement in relation to transient nerve lesions are compared regarding the recovery of shoulder function. A prospective, randomized trial with EMG will be necessary to evaluate the benefit of operative treatment in post-retirement patients with displaced fractures.

Many possible reasons for inferior subluxation of the humeral head after proximal humeral fractures exist, such as humeral shortening, disappearance of negative intra-articular atmospheric pressure in the presence of a rupture of capsular ligaments, insufficient muscle strength due to a cuff rupture, or a paresis of the deltoid or the rotator cuff muscles due to nerve injury. Inferior subluxation after proximal humeral fractures has only been described twice before in the literature.[20,23] Thompson and Winant[23] described 40 displaced fractures with subluxation. Pritchett[20] found inferior subluxation in 60% (15/24) of fractures with prosthetic replacement and in 42% (11/26) of conservatively treated fractures. He explained the subluxation by "atonia of the deltoid and the rotator cuff muscles." He did not relate the atonia to nerve injury. In both studies the subluxation disappeared spontaneously in all patients.

In our study axonal nerve lesions were present in 8 of the 9 patients with severe inferior subluxation. All 9 patients were initially treated with a collar and cuff without any support of the elbow, leading to caudal traction at the shoulder in the presence of a very large hematoma. The electromyogram did not show axonotmesis in 1 of the 9 patients. This finding does not, however, exclude nerve injury in this patient. Because the nerve conduction velocity study to determine neurapraxia is not reliable in the shoulder girdle[19,28] and the prognosis of neurapraxia is excellent within 1 month, we did not study its presence. As expected, the subluxation recovered spontaneously in all patients. In our opinion, inferior subluxation of the humeral head in proximal humeral fractures points to loss of muscle strength of the deltoid or cuff muscles, which can point to nerve lesions.

REFERENCES

1. Bengnér U, Johnell O, Redlund-Johnell I. Changes in the incidence of fracture of the upper end of the humerus during a 30-year period. A study of 2125 fractures. Clin Orthop 1988; 231:179-83.
2. Blom S, Dahlbäck LO. Nerve injuries in dislocations of the shoulder joint and fractures of the neck of the humerus. Acta Chir Scand 1970;136:461-6.
3. Bloom MH, Obata W. Diagnosis of posterior dislocation of the shoulder with use of a Velpeau axillary and angle-up roentgenographic views. J Bone Joint Surg Am 1967;49:943-9.

4. Cave EF, Roberts SM. A method for measuring and recording joint function. J Bone Joint Surg 1936;18:455-65.
5. Coene LNJEM. Mechanisms of brachial plexus lesions. Clin Neurol Neurosurg 1993;95(Suppl):S24-9.
6. Daube JR. AAEM minimonograph No. 11: needle examination in clinical electromyography. Muscle Nerve 1991;8:685-700.
7. Ebel R. The cause of axillary nerve paresis in shoulder luxations [in German]. Monatsschr Unfallheilkd Versicher Versorg Verkehrsmed 1973;76:445-9.
8. Horak J, Nilsson BE. Epidemiology of the fracture of the upper end of the humerus. Clin Orthop 1975;112:250-3.
9. Kabat H, Knott M. Proprioceptive facilitation technics in treatment of paralysis. Phys Ther Rev 1953;33:53-64.
10. Kleinrensink GJ. Influence of posture and motion on peripheral nerve tension [thesis]. Rotterdam, The Netherlands: The Erasmus University Rotterdam; 1997.
11. Kristiansen B, Barfod G, Bredesen J, Erin-Madsen J, Grum B, Horsnaes MW, et al. Epidemiology of proximal humerus fractures. Acta Orthop Scand 1987;58:75-7.
12. Laat de EAT, Visser CPJ, Coene LNJEM, Pahlplatz PVM, Tavy DLJ. Nerve lesions in primary shoulder dislocations and humeral neck fractures. J Bone Joint Surg Br 1994;76:381-3.
13. Lewit K. Manuelle medizin. Leipzig, Germany: Johan Ambrosius Barth; 1977.
14. Lind T, Krøner K, Jensen J. The epidemiology of fractures of the proximal humerus. Arch Orthop Trauma Surg 1989;108:285-7.
15. Maitland GD. Treatment of the glenohumeral joint by passive movement. Physiotherapy 1977;69:3-7.
16. Medical Research Council. Memorandum No. 45. Aids to the examination of the peripheral nervous system. London: Her Majesty's Stationery Office; 1967.
17. Neer CS II. Displaced proximal humeral fractures. I. Classification and evaluation. J Bone Joint Surg Am 1970;52:1077-89.
18. Neer CS II. Displaced proximal humeral fractures. II. Treatment of three-part and four-part displacement. J Bone Joint Surg Am 1970;52:1090-103.
19. Peterson GW, Will AD. Newer electrodiagnostic techniques in peripheral nerve injuries. Orthop Clin North Am 1988; 19:13-25.
20. Pritchett JW. Inferior subluxation of the humeral head after trauma or surgery. J Shoulder Elbow Surg 1997;6:356-9.
21. Rose SH, Melton LJ, Morrey BF, Ilstrup DM, Riggs BL. Epidemiological features of humeral fractures. Clin Orthop 1982;168: 24-30.
22. Rozing PM, de Bakker HM, Obermann WR. Radiographic views in recurrent anterior shoulder dislocation. Comparison of six methods for identification of typical lesions. Acta Orthop Scand 1986;57:328-30.
23. Thompson FR, Winant EM. Comminuted fracture of the humeral head with subluxation. Clin Orthop 1961;20:94-7.
24. Visser CPJ. Nerve injury in shoulder dislocations and fractures of the proximal humerus [thesis]. Leiden, The Netherlands: The University of Leiden; 1998.
25. Visser CPJ, Coene LNJEM, Brand R, Tavy DLJ. The incidence of nerve injury in anterior dislocation of the shoulder and its influence on functional recovery. A prospective clinical and EMG study. J Bone Joint Surg Br 1999;81:679-85.
26. Visser CPJ, Tavy DLJ, Coene LNJEM, Brand R. Electromyographic findings in shoulder dislocations and fractures of the proximal humerus: comparison with clinical neurological examination. Clin Neurol Neurosurg 1999;101:86-91.
27. Wilbourn AJ. Electrodiagnosis of plexopathies. Neurol Clin 1985;3:511-29.
28. Wilbourn AJ. Electrodiagnostic testing of neurologic injuries in athletes. Clin Sports Med 1990;2:229-45.