The aetiology and outcome of 170 ulnar nerve lesions confirmed with electrophysiological testing

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The authors have studied 148 consecutive patients with 170 electrophysiologically confirmed ulnar nerve lesions, who were followed up for one to six years (median 3.8 years) to determine clinical progress and outcome. Injury and intra-operative pressure accounted for 12.9% and 7.1% of lesions respectively ; 58.2% were idiopathic with no identified clinical aetiological factor. Eighty-three percent received non-operative treatment initially ; 21% of these required operative intervention following further clinical/electrophysiological assessment. Partial or complete recovery occurred in 92% of intra-operative, 64% of idiopathic and 50% of injury cases respectively.

Ulnar nerve lesions predominate in males and can be treated non-operatively providing clinical and electrophysiological monitoring is possible. Bilaterality is common and should be excluded. Lesions due to injury have a worse prognosis than those caused by direct continuous or repeated pressure, inflammation or where no aetiological factor exists.

INTRODUCTION

Ulnar neuropathy is the second most frequent entrapment neuropathy occurring in adults (14) and the majority of these occur at the elbow in the cubital tunnel. Many causes of neuropathy at the elbow have been cited including anatomical anomalies (17), trauma (18), habitual leaning on the elbow (19), flexion in sleep (2), rheumatoid arthritis (11) diabetes mellitus (15) and iatrogenic. The last of these can be secondary to injudicious positioning while under anaesthetic (20) direct trauma during upper limb surgery (6) or infarction following transposition (21). The second most common site of entrapment is at the wrist in the canal of Guyon, but damage to the nerve at any stage along its course can result in the development of a neuropathy.

The cause and site of the lesion should be elucidated by careful history and examination with electrophysiological testing as an important adjunct. Whether subsequent treatment should be operative or non-operative is a controversial issue and depends on many factors. While some cases require early surgical intervention, others do not and this is a decision for the attending physician. Indeed, various classifications of ulnar nerve lesions exist that attempt to guide treatment (5, 7, 12). These classification systems each have their advantages and are based upon patient symptoms irrespective of underlying aetiology.

The published results of operative and non-operative treatment of ulnar neuropathy are wide-ranging reflecting variable patient selection and variable techniques. In an analysis of published data on treatment of ulnar nerve entrapment at the elbow, Dellon (5) describes 58% excellent results

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for non-operative treatment in patients with minimal symptoms. Surgery in these 'minimal' cases resulted in 94% excellent results. With more severe lesions the success of operative treatment decreases to 30% excellent results for simple decompression, although it is stated this may be improved with transposition and neurolysis. When surgery is indicated the preferred method of intervention remains controversial in the surgical literature i.e. simple decompression, transposition or medial epicondylectomy (1, 8, 13) although simple decompression would seem to be adequate in the majority of cases (1).

The aim of this study is to identify aetiological factors in ulnar neuropathy and whether they determine patient outcome. The anatomical locations of these lesions are identified and the roles of non-operative and operative treatment are assessed.

PATIENTS AND METHODS

One hundred and forty eight consecutive patients with a diagnosis of ulnar neuropathy between 1994 and 1999 were prospectively assessed. The patient details, symptoms, known aetiology and treatment profile were recorded. A full sensory and motor examination of both upper limbs was performed including elbow flexion test and Tinel's test. Where no clear aetiology of lesion could be obtained from history or examination e.g. injury (fracture, crush, direct blow), osteoarthritis, recent surgery etc. then these were termed 'idiopathic'.

All patients had nerve conduction studies (NCS) and/or electromyography (EMG) to confirm the diagnosis. Patients were referred for electrodiagnosis from both general practitioners (48%) and in-hospital clinicians (52%). This was performed by a single rheumatologist (IMM), with a special interest in this field, who would then instigate treatment depending on the severity, chronicity and progression of the lesion based on clinical presentation.

All studies were undertaken in warmed limbs, by prior immersion in warm water.

The standard tests used included an orthodromic ulnar nerve sensory study stimulating with ring electrodes from the little finger to recording surface electrodes at the wrist ; these results were compared with the ipsilateral median nerve sensory studies from the index finger to the wrist, and the contralateral ulnar nerve sensory study. Velocities for sensory conduction were calculated and a velocity of more than 50 metres per second was considered normal. Motor studies were undertaken in the ulnar nerve with stimulation from the wrist and then more proximal points below and above the elbow as well as in the upper arm to the adductor digiti minimi (adm) using surface or concentric needle electrodes to record. Distal latency as well as conduction velocity in the forearm, around the elbow and in the upper arm were measured. These studies were compared with the ipsilateral median motor nerve from the antecubital fossa and the wrist to the abductor pollicis brevis, as well as the contralateral ulnar nerve. A normal motor nerve velocity was considered to be more than 50 metres per second in the forearm and upper arm, whilst more than 40 metres per second around the elbow in the ulnar nerve was considered normal too. If appropriate distal motor latency in the ulnar nerve from the wrist to the first dorsal interosseous was undertaken and a difference in latency compared with that to the adm of less than one millisecond was considered normal. EMG studies using concentric needle electrodes were performed where indicated, e ither when nerve conduction abnormalities were found or when there was clinical wasting and, or, weakness Abnormalities looked for were activity at rest (fibrillation and positive sharp waves) and reduced interference patterns on maximal voluntary effort, both of these indicating denervation.

All lesions with sensory changes alone were managed initially non-operatively. This was also the treatment of choice in those patients where motor changes were present but in whom there was no pain and the symptoms remained static with no clinical suggestion of progression or deterioration. Non-operative treatment involved advice regarding protecting the site of nerve compression by modified activity and the provision of a tubipad bandage when the elbow was involved. These patients were then all followed up clinically and by further NCS/EMG if indicated. If non-operative treatment failed then the patient was referred to an orthopaedic surgeon for operative decompression.

Operative treatment was only used as first line treatment in those patients with motor changes which were progressive or troublesome and all patients were advised that the aim of surgery was to prevent further deterioration although some recovery of nerve function might occur.

In this prospective longitudinal cohort study patients were contacted by telephone and/or questionnaire one to six years (median 3.8 years) following electrophysiological diagnosis to determine clinical progress and outcome.

Table I. — The aetiology of lesions of the ulnar nerve

Aetiology	Number of Patients	Bilateral expected	Bilateral unexpected
Idiopathic	89	10	6
Injury	21	1	2
contusion elbow	8	1	2
fracture elbow	5	0	0
crush forearm	3	0	0
fracture metacarpal	2	0	0
fracture forearm	1	0	0
Colles' fracture	1	0	0
fracture scaphoid	1	0	0
Iatrogenic	12	3	2
Osteoarthritis (elbow)	9	4	5
Repeated pressure	5	2	1
Rheumatoid (elbow)	3	1	1
Medial epicondylitis	3	1	0
Other	6	0	0
Total	148	22	17

RESULTS

Of the 148 patients investigated, 22 had bilateral symptoms of an ulnar nerve lesion (bilateral expected). A further 17 patients had bilateral changes on NCS/EMG but remained only unilaterally symptomatic (bilateral unexpected). Therefore 170 symptomatic lesions were available for review and in 98.8% of patients follow up by questionnaire/ telephone was achieved.

In our study the average age of patient was 56.3 years (range 9-92). Sixty-eight percent of patients were male (average age 54.5 years) and 32% female (average age 60.2 years). The aetiology and distribution of bilateral changes are shown in table I. The six cases tabulated as 'other' were due to inflammatory arthritis in three cases (juvenile chronic arthritis, psoriatic arthritis, ankylosing spondylitis) and a space occupying lesion in three (two lipomata and a ganglion).

Three of the 12 iatrogenic cases involved incisions anatomically related to the course of the nerve resulting in a non-continuous lesion (olecranon bursa excision, removal of haemangioma from canal of Guyon, removal of metalwork from elbow) whereas in the remaining nine patients it was due to prolonged pressure on the nerve in the cubital tunnel during 'distant' surgery (total knee replacement, total hip replacement, excision of popliteal aneurysm, transurethral resection of prostate, splenectomy, cholecystectomy, nephrectomy, laporotomy, shoulder hemiarthroplasty). All three bilateral cases as a result of pressure while on the operating table were in cases with the patient lying supine, undergoing surgery on a distant site.

Table II. — The anatomical distribution of symptomatic lesions of the ulnar nerve

Aetiology	Elbow (cubital tunnel)	Wrist (canal of Guyon)	Other
Idiopathic	94	4	1
Injury	13	2	7
Iatrogenic	14	1	0
Osteoarthritis (elbow)	13	0	0
Repeated pressure	6	1	0
Rheumatoid (elbow)	4	0	0
Medial epicondylitis	4	0	0
Other	4	0	2
Total	152 (89.4%)	8 (4.7%)	10 (5.9%)

The anatomical location of the 170 ulnar nerve lesions and the relation to aetiology is shown in table II. Lesions distant from the elbow or canal of Guyon were in the deep branch in the hand (five), the forearm (four) and the sensory branch on the dorsum of the hand (one). Seven of these cases were due to injury, two from mass lesions in the hand and a single deep branch lesion of no known aetiology.

Primary non-operative treatment was instigated in 83% of lesions, with 21% of these progressing to surgery. Overall, 65% of lesions did not have operative intervention and the outcome of these lesions is detailed in table III. Sixty surgical interventions were required - 54 simple decompressions, three delayed primary repairs and three 'lump' removals. The outcome of these cases is detailed in table IV. The 29 patients receiving surgical decompression following 'failed' non-operative treatment included all the patients with a clinical and/or electrophysiological deterioration and 17 in whom non-operative

Table III. — The results of non-operative treatment

Aetiology (number)	No symptoms	Partial recovery	No change	Worse
Idiopathic (64)*	28	14	20	0
Injury (12)	3	3	6	0
Iatrogenic (9)	5	4	0	0
Osteoarthritis (9)	3	6	0	0
Repeat pressure (7)	6	1	0	0
Epicondylitis (4)	2	2	0	0
Rheumatoid (2)	1	1	0	0
Other (3)	2	1	0	0
Overall (110)*	50	32	26	0

*2 lost to follow up.

Table IV. — Results of surgery

Aetiology (number)	No symptoms	Partial recovery	No change	Worse
Idiopathic (35)	11 (5)	9 (5)	15 (10)	0
Injury (10)	0	5 (2)	5 (2)	0
Iatrogenic (6)	0	2 (1)	4* (1)	0
Osteoarthritis (4)	0	2 (1)	2 (1)	0
Rheumatoid (2)	0	2 (1)	0	0
Other (3)	3**	0	0	0
Overall (60)	14	20	26	0

* including 3 primary repair, ** 2 lipoma, 1 ganglion excised.

Table V. — Comparison of lesions undergoing surgery

	Primary operative treatment (n = 29)	Operation following non-operative treatment (n=31)	Statistical significance (test)
Months to surgery from electrodiagnosis (range)	5.0 (2-8)	6.1 (3-10)	$p > 0.05$ (student t-test)
No symptoms (%)	17.2	29.0	$p > 0.05$ (Mann-Whitney U)
Partial recovery (%)	34.5	32.3	$p > 0.05$ (Mann-Whitney U)
No change (%)	48.3	38.7	$p > 0.05$ (Mann-Whitney U)
Worse	0	0	n/a

treatment had had no effect. Table V compares the patients undergoing primary operative treatment and those having surgery following an initial trial of non-operative treatment. There were no complications of non-operative treatment whereas surgical complications included one painful scar neuroma, one wound dehiscence, one haematoma, two superficial infections and seven cases of persistent numbness adjacent to the wound.

Table VI shows the percentage of patients with full or partial recovery from their symptoms with respect to their aetiology at follow up.

Ninety five patients required only one EMG, whereas 53 had two or more. The average time from first EMG to surgery (if indicated) was 5.6 months (range 1-12). Five diabetic patients had evidence of peripheral neuropathy at the time of assessment. No double-crush lesions were identified.

DISCUSSION

This study identifies many aetiological factors leading to ulnar neuropathy. We have described the

Table VI. — Percentage of patients with full or partial recovery at follow-up

Aetiology	Operative	Non-operative	All lesions
Idiopathic	57	70	64
Injury	50	50	50
Iatrogenic	67	100	92
Osteoarthritis	50	100	85
Inflammatory arthritis	100	100	100
Repeated pressure	0	100	100
Medial epicondylitis	0	100	100

anatomical location of these lesions and assessed their outcomes following non-operative and operative treatments or a combination of both. The age distribution and predominance in males is similar to that previously reported (3). Symptomatic lesions occurred at the elbow in 89.4% of cases, identifying this as the most common site of neuropathy. If a lesion is not identified here, the wrist, forearm and hand should be evaluated. This is particularly the case in injury when a careful history should indicate the site of the lesion.

The aetiological factors described in this study are similar to those reported previously : joint deformity (9), rheumatoid arthritis (11), pressure during surgery (20), trauma (18), space-occupying lesions (16), diabetes (15), medial epicondylitis (10). The majority of the patients in our study had no clearly definable aetiology and we termed them ‘idiopathic’. Ninety-five percent of these ‘idiopathic’ cases had ulnar neuropathy at the elbow, presumably due to susceptibility to compression of the nerve in the cubital tunnel. Our ‘idiopathic’ group may include patients that habitually lean on the elbow without noticing, those who flex their elbows at night or those who have congenital anomalies or bands around the elbow. We did not define these as a separate aetiology as there is no method of demonstrating this clinically without open exploration or constant 24 hour observation. Bilaterality of lesions of the ulnar nerve occurred in 23% of patients and in all cases this was at the elbow with 56% of them being symptomatic. Bilaterality is uncommon in injury, but relatively frequent when the cause is as a result of direct pressure (as in a general anaesthetic), osteoarthritis or when no cause could be identified. It therefore follows that some patients are clearly more susceptible than others to pressure on the nerve as it courses posterior to the medial epicondyle within the cubital tunnel. The contralateral upper limb should therefore always be assessed at presentation to identify a possible bilateral lesion.

Our study shows that non-operative treatment can be beneficial to the majority of patients with ulnar neuropathy and in particular when arthritis, direct pressure or epicondylitis is an aetiological factor. Of the 12 patients (8%) who had deteriorated following instigation of non-operative treatment, all had partial or complete recovery following surgery. From our results there was no statistical significance between patients treated primarily operatively to those treated operatively as a result of failed non-operative treatment. It is possible that the patients in the latter group may have had better outcomes if treated sooner, but we would disagree with this as the average time to surgery from the time of the first nerve conduction study for the primary operative and failed non-operative groups was 5.0 and 6.1 months respectively. No patients in our study had deteriorated as a direct result of our treatment protocol although 20% of surgical patients had complaints relating to the scar from the surgery. Eight of these complications related to damage to the medial antebrachial cutaneous nerve as previously reported by others (4).

Overall, patients in whom injury was an aetiological factor had poor outcomes, as did the three patients who had primary repair of the nerve following accidental damage during surgery : all three showed no improvement in their symptoms. This is in contrast to the excellent prognosis of an ulnar nerve lesion that has an aetiology such as direct pressure, injudicious positioning with pressure on the nerve under a general anaesthetic, an adjacent mass lesion (e.g. lipoma) or inflammation/arthritis. When no aetiological factor was identified then results achieved were similar to those previously reported for both non-operative and operative treatment (1, 5). In this study we do not consider the chronicity of the lesion, but appreciate that this may be a factor in subsequent recovery. Patient age and site of lesion may also be factors in the

recovery of an ulnar nerve lesion, although it is not possible to comment on this from our data.

We do not wish it to be misconceived that non-operative treatment is in any way better than operative treatment from this study. We use non-operative treatment for the 'less severe' painless cases with sensory changes alone or in those with non-progressive longstanding motor lesions. The fact that 21% of cases refractory to non-operative treatment are referred for surgery is due to our close monitoring of lesions enabling us to change our treatment plan accordingly. Outcomes for non-operative treatment alone are better than operative as noted in table VI, but this does not equate to non-operative treatment being *better* per se, as operative treatment is used for the painful, progressive and refractory cases in our unit. Indeed, it is possible that if operative treatment was used for all lesions (including 'sensory' only) then it would be more successful than non-operative measures. This could only be determined by a prospective randomized study to compare the two treatment modalities for ulnar nerve lesions at the same site, with similar aetiology and chronicity. Ethical approval and adequate numbers of cases would be very difficult to achieve.

In summary, our study shows that ulnar neuropathy is a relatively common condition occurring predominantly in males at the elbow. Bilaterality is not uncommon and should always be excluded. Many aetiological factors have been identified although this is often not possible and we term these cases 'idiopathic'. The most common defined causes include injury, arthritis, repeated pressure and those as a result of injudicious patient positioning on the operating table during surgery. Ulnar neuropathy as a result of acute injury has a poor prognosis whereas those as a result of repeated pressure and inflammation respond well to treatment.

We feel strongly that non-operative treatment has an important role to play in the management of ulnar neuropathy. Unlike non-operative treatment, surgery is not without its complications and should be reserved for those patients who have pain, exhibit motor symptoms that are progressive or in those patients who have failed to respond to non-operative treatment. We believe that patients undergoing surgery should be counseled that the main aim is to prevent further deterioration and that the degree of recovery depends to some extent on the chronicity and aetiology of the lesion. This protocol necessarily requires diligent and careful continual assessment of the patient at regular intervals by both clinical and electrophysiological investigation.

REFERENCES

1. **Bartels RH, Menovsky TH, Van Overbeeke JJ, Verhagen WI.** Surgical management of ulnar nerve compression at the elbow : an analysis of the literature. *J Neurosurg* 1998 ; 89 : 722-727.
2. **Bradshaw DY, Shefner JM.** Ulnar neuropathy at the elbow. *Neurol.Clin* 1999 ; 17 : 447-460.
3. **Contreras MG, Warner MA, Charboneau WJ, Cahill DR.** Anatomy of the ulnar nerve at the elbow : potential relationship of acute ulnar neuropathy to gender differences. *Clin Anat* 1998 ; 11 : 372-378.
4. **Dellon AL, MacKinnon SE.** Injury to the medial antebrachial cutaneous nerve during cubital tunnel surgery. *J Hand Surg* 1985 ; 10-B : 33-36.
5. **Dellon AL.** Review of treatment results for ulnar nerve entrapment at the elbow. *J Hand Surg* 1989 ; 14-A : 688-699.
6. **Dellon AL, Hament W, Gittelshon A.** Non-operative management of cubital tunnel syndrome : an eight year prospective study. *Neurology* 1993 ; 43 : 1673-1677.
7. **Gabel GT, Amadio PC.** Reoperation for failed decompression of the ulnar nerve in the region of the elbow. *J Bone Joint Surg* 1990 ; 72-A : 213-219.
8. **Geutjens GG, Langstaff RJ, Smith NJ, Jefferson D, Howell CJ, Barton NJ.** Medial epicondylectomy or ulnar nerve transposition for ulnar neuropathy at the elbow? *J Bone Joint Surg* 1996 ; 78-B : 777-779.
9. **Khoo D, Carmichael SW, Spinner RJ.** Ulnar nerve anatomy and compression. *Orth Clin North Am* 1996 ; 27 : 317-338.
10. **Kurvers H, Verhaar J.** The results of operative treatment of medial epicondylitis. J *Bone Joint Surg* 1995 ; 77-A : 1374-1379.
11. **Leffert RD, Dorfman HD.** Antebrachial cyst in rheumatoid arthritis –surgical findings. *J Bone Joint Surg* 1972 ; 54-A : 1555-1557.
12. **McGowan AJ.** The results of transposition of the ulnar nerve for traumatic ulnar neuritis. *J Bone Joint Surg* 1950 ; 32-B : 293-301.
13. **McKee MD, Jupiter JB, Bosse G, Goodman L.** Outcome of ulnar neurolysis during post-traumatic reconstruction of the elbow. *J Bone Joint Surg* 1998 ; 80-B : 100-105.
14. **Miller RG.** Ulnar nerve lesions. In : Brown WF, Bolton CF, eds. *Clinical Electromyography*. Stoneham, MA : Butterworths, 1987 : 99-116.

15. **Mulder DW, Lambert EH, Batron JA.** The neuropathies associated with diabetes mellitus. *Neurology* 1961 ; 11 : 275-284.
16. **Nakamichi k, Tachibana S, Kitajima I.** Ultrasonography in the diagnosis of ulnar tunnel syndrome caused by an occult ganglion. *J Hand Surg* 2000 ; 25-B : 503-504.
17. **O'Driscoll SW, Horii E, Carmichael SW,** *et al*. The cubital tunnel and ulnar neuropathy. *J Bone Joint Surg* 1991 ; 73-B : 613-617.
18. **Platt H.** The pathogenesis and treatment of traumatic neuritis of the ulnar nerve in the post-condylar groove. *Br J Surg* 1926 ; 13 : 409-431.
19. **Stewart JD.** The variable clinical manifestations of ulnar neuropathies at the elbow. *J Neurol Neurosurg Psychiatry* 1987 ; 50 : 252-258.
20. **Warner MA, Warner DO, Matsumoto JY, Harper CM, Schroeder DR, Maxsom PM.** Ulnar neuropathy in surgical patients. *Anaesthesiology* 1999 ; 90 : 54-59.
21. **Wilbourn AJ.** Iatrogenic nerve injuries. *Neurol Clin* 1998 ; 16 : 55-82.