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290 Surgical Procedures for Ulnar Nerve Entrapment at the Elbow: Physiopathology, Clinical Experience and Results

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Summary

Ulnar nerve entrapment at the elbow is an important and relatively frequent pathological condition that may be related to diffent causes depending on individual or external factors. The cause of the nerve lesion is also idiopathic in about one-quarter to one-third of cases. This variable aetiopathogenetic presentation has often suggested different diagnostic and clinical approaches and, moreover, various surgical procedures. We present our 8-years surgical experience with 290 cases of ulnar nerve entrapment at the elbow analysing the salient clinical features and the results of the surgical treatment in the light of the relevant literature available on this topic.

Keywords: Ulnar nerve; elbow; entrapment neuropathy, epitrochlear groove; surgical management; electromyography; tardy ulnar palsy.

Introduction

The entrapment of the ulnar nerve at the elbow results from different pathological conditions such as degenerative or inflammatory diseases, congenital defects, iatrogenic or occupational diseases, metabolic or nutritional disorders [8, 11, 12, 16, 18, 37, 43, 46, 47, 54]. The increased vulnerability to compression of the nerve trunk at this level is conditioned by its anatomical relationships [3, 5, 7, 10, 13]: in fact, the nerve is running between the medial belly of the triceps muscle and humerus condyle. The aponeurosis, connecting to the intermuscular septum, and the muscle itself are closely following the nerve as far as it enters the trochlear groove, covered by a sheath of connective tissue extending to the two-folded belly of the flexor carpi ulnaris. Ulnar nerve compression may be related, besides external factors, to anatomical individual variations and it is a common cause of arm and hand pain, paraesthesias and weakness [7, 13, 14, 37]. A correct preoperative evaluation and careful planning of the surgical procedure are essential for a good therapeutic result. Our 8-years experience (1985–1992) with 290 surgically treated instances of ulnar nerve entrapment is reported and results compared with the relevant literature, focusing on the main clinical and therapeutic findings of this pathological condition.

Patients and Methods

236 patients with ulnar nerve entrapment at the elbow (140 males and 96 females) were observed from January 1985 and December 1992. In 54 patients bilateral ulnar nerve involvement was present (290 surgical procedures for ulnar nerve compression). Patients were between 17 and 69 years old with a mean age of 42.5 (median 45.5). History ranged from 1 to 7 years with an average lenght of 3.5 years. In 33% of cases the presence of local (fractures with deformities or dislocation, cubitus valgus, muscles anomalies, rheumatoid arthritis) or systemic (diabetes, alcohol abuse, polineuropathies) factors were diagnosed.

Subjective symptoms and neurological signs both contributed to the clinical picture. As far as these are concerned, we found paraesthesias in 93%, motor weakness in 66%, pain 44% of cases. Neurological examination revealed a sensory deficit in 80%, a motor deficit in 75%, and muscular loss in 66% of instances. A clinical grading of ulnar loss of function was devised, featuring three classes: 1) patients complaining of subjective disturbances but without neurological signs and showing only mild neurophysiological test (NPT) alterations (17%); 2) patients with neurological signs and showing significative NPT pathological changes (61%); 3) patients with neurological signs and serious NPT alterations (22%). NPT alterations were considered the detection of signs of denervation, alteration of intention patterns and reduction of conduction velocities across elbow to less than 48 m/sec..

The following therapeutic clinically-related criteria were used to select the patient for the proper surgical treatment: a) for the patients with acute neuropathy conservative treatment was advised; b) a decompressive surgical procedure was performed in patients of groups 1 and 2, with an history of less than 12 months, if clinical examina-

Table 1. *1985–1993: 290 Surgically Treated Cases (in 54 Cases Ulnar Nerve Entrapment was Bilateral)*

Three symptom classes (12 mths min.)	Subjective symptoms	Neurological examination	Surgical treatment	Follow up
I: Pat. with subj. dist. without neurol. sympt. & showing mild EMG alterations only (17%)	paraesthesia 93%	sensory deficit 80%	decompr. 75 c.	excellent 58%
	motor weakn. 66%	motor deficit 45%	superf. transp. 195 c.	good 27%
	pain 40%	muscular loss 66%	deep transp. 20 c.	unchanged 15%
II: Pat. with neurol. sympt. + signs showing a signi-ficative EMG alter. (61%)				deteriorate 0%
	sex	*age*		
III: Pat. with neurol. sympt. + signs showing a serious EMG alteration (22%)	140 males 96 females ..... 236 patients	17–69 yrs mean 42.5 median 45.5		

tion disclosed an ulnar nerve entrapment syndrome, in spite of a wide trochlear groove; c) in patients bearing anomalies or muscular hypertrophy a deep transposition was the procedure of choice; d) a superficial transposition (subcutaneous) was preferred in the remaining cases. Since acute neuropathy cases were not included in the present report (no surgery), the decompression procedure was performed in 13% of patients, the superficial transposition in 80% and the deep transposition in 7% of instances.

The follow-up of patients, although ranging from 1 to 8 years, was considered at 1 year after surgery, to permit a more homogeneous evaluation of results and to overcome the effects of poor co-operation of patients requested to attend for check-ups over longer periods.

Table 1 summarizes surgical results of the whole series of patients. We registered no major surgical complication and few minor complications such as a hypertrophic scar (5 cases) with a poor cosmetic result and one short lasting superficial infection (successfully treated with systemic antibiotics).

Results

Between 1985 and 1993 236 patients with ulnar nerve compression at the elbow were operated on at our Institution. Males prevailed in comparison to females (140 vs. 96) and age ranged between 17 and 69 years (mean 42.5; median 45.5). In 54 instances the entrapment syndrome was bilateral and surgical treatment was accomplished in two stages. Preoperative clinical evaluation of patients prompted us to devise a three classes classification according to subjective symptoms, objective signs and EMG findings, as above described. Distribution of patients in the three classes was as follows: 40 patients (17%) in Class I, 144 patients (61%) in Class II and 52 patients (22%) in Class III (Table 1).

The most common subjective symptom was paraesthesia (93%), followed by motor weakness (66%) and pain (40%). At neurological examination sensory deficit resulted in the most frequent finding (80%), while muscular loss and motor deficit were less common (respectively accounting for 66% and 45%). Surgical procedure consisted mainly of superficial (subcutaneous) transposition (195 nerves – 80%), followed by decompression (75 nerves – 13%) and deep (intramuscular) transposition (20 nerves – 7%). Follow-up ranged from 12 to 96 months, but our evaluation was confined to 1 year after surgery. After this interval 58% of cases showed marked improvement of the neurological status with subjective normalization (excellent); 27% of cases revealed a fair improvement of the neurological symptoms with mild sensory subjective disturbances (good); 15% of cases remained unchanged. We did not register any further deterioration in the surgical cases.

Discussion

Both systemic and local affection may induce the onset of an ulnar nerve distress at the elbow: congenital anomalies (cubitus valgus, epithrocleoanconeus, thickening either of the triceps, medial belly, or of the aponeurosis of the deep flexor pronator), trauma (elbow luxations, distal humeral lesions), arthritis, tumoural lesions (ganglion cysts, lipomas), iatrogenic injuries (post-anaesthetic, haemorrhagic, due to wrong positioning during operations or in bedridden patients), nutritional or metabolic disorders (diabetes, alcoholic addiction, toxics exposure), occupational diseases (manufacturers, truck drivers, computer or desk employees) and rare illnesses (lepromatosis, biceps rupture) [2, 8, 12, 16, 18, 36, 37, 38, 39, 43, 44, 45, 46, 56, 60]. Individual anatomical variations of the nerve course and situation as well as the peculiar pathologic effect of the different aetio-

logical factors, remarkably influence the degree of functional impairment [3, 7, 13, 27, 28, 29, 30, 54].

Though general principles seem to be lacking to enable the neurosurgeon to predict the degree of functional impairment induced by a compromised nerve situation, the factors involved in causing a theorical nerve trouble seem actually counterbalanced by some kind of neural (and/or biological) defense reaction [36]. The ability of the nerve structure to slide and the action of gliding surfaces also between the inner nerve fascicles must a role play [3, 10, 14, 36]. That is to say that a compression neuropathy is almost never a simple compression neuropathy.

The compression will result in a functional problem if surrounding oedema, microstretching injuries, extraneural scarring, epineural fibrosis are serious enough to affect the intraneural microcirculation, the axonal transport of nutrients, the cell bodies and so on. These alterations will finally lead to demyelination, conducting problems and fiber degeneration [11, 14, 17].

For the very high length/diameter ratio and the distance from the cell body, the peripheral nerve axons are strictly dependent on the neurovascular environment as far as metabolic and catabolic exigences are concerned.

The small epineural vessels divide in ascending and descending branches. This epineural network, after forming an anastomotic network in the subepineurium, further form a vascular plexus at the perineurium. The vascular plexus at the level of perineurium sends small end-arteries and capillaries within the fascicles to complete the deep vascular network [17, 52].

Endoneural microvessels feature big diameters, large intercapillary distances, and scarce perivascular smooth muscle. These anatomical grounds explain the poor autoregulation of endoneural vasculature, so that even small changes of nerve blood flow and perfusion pressure are not compensated [52].

Endoneural oedema, further enhancing vessel distance and determining vessel collapse, greatly affects the integrity of the blood-nerve interface [42, 51, 36]. In fact, since the perineurium is not permeable and endoneural capillaries are very selective in molecule filtration, the endoneural compartment results in being relatively isolated. Therefore, oedema increases endoneural hydrostatic pressure with endothelial hypoxia and consequent axonal damage [51, 58]. There is also experimental evidence that endoneural oedema is able to negatively influence axonal regeneration inducing fibroblastic invasion with intraneural scar formation [41].

The production of free radicals or of oxidated products of low density lipoproteins has been claimed to induce cytotoxicity and decrease of the antioxidative capacity [1, 9, 31]. Long peripheral axons, for their content of phospholipidic membranes are very sensitive to oxidative damage [23, 49].

Axoplasmic transport of molecules synthetised in the cell body, uses oxidative phosphorilation of the axonal mitochondria to produce high-energy phosphates. Segmental axonal ischaemia due to a mild decrease of blood flow implies a loss of energy for the transport as well as for the sodium-pump system. Cell membrane is also affected by this energetic default, with consequent loss of conduction and transmission by the axon. The segment of axon in which ischaemia is produced, for local anatomy and/or raised internal pressure, not only will give rise to vascular mechanisms, but will also change its own ionic content, with further changes in endoneural hydrostatic pressure [4, 50, 51].

A correlation between symptoms and histological findings was often attempted aiming to establish objective criteria for surgical indications, prognosis and therapeutic result evaluation.

Tingling and paraesthesias were related to microcirculation dysfunction leading to hypoxia [36].

Intermittent oedema was claimed to cause intermittent symptoms, however permanent sensory and motor deficits should be interpreted as initial demyelination. Muscle wasting and loss of sensibility should, on the other hand, imply fiber degeneration [36].

Moreover, there is evidence suggesting that other factors play a determining role in the so called compression neuropathy. Nerve fascicle arrangement, anatomically classified as ''simple'' and ''compound'', may influence the compression tolerance as well as the distance of the entrapped branch from the main trunk [30]. A vascular mechanism or a compromised axonal transport may respectively be invoked.

Anatomical studies have also suggested that the nerve fibers from the terminal digital sensory branch and to the small muscles of the hand, at the elbow lie deeply in the nerve, adjacent to the bone where they could be more easily exposed to injuries [50, 51].

Similar aetiopathogenetic hypotheses could explain the ''double crush'' syndrome in which a cervical root compression would make a minor elbow pathology

able to produce ulnar neuropathy [11, 13, 37]. The reported higher damage susceptibility of the three terminal digital sensory fascicles and fascicles to intrinsic hand muscles could be interpreted analogously.

For the correct diagnosis and treatment of ulnar nerve entrapment, a careful clinical and electrophysiological evaluation and a fair surgical planning are recommended. Options for the best treatment are conditioned by the ability to find a correspondence between the clinical picture and the anatomical situation, grouping the patients in homogeneous classes addressed to the surgical procedures. Preoperative grading of patients was introduced by McGowan in 1950 [38] and Osborne in 1966 [43], based respectively on ulnar motor impairment and and ulnar sensory-motor deficit progression. Our experience prompted us to rely on a syncretic three-class selection, based upon subjective, objective and electrophysiological criteria. Predisposing conditions such as alcoholism, diabetes, in addition to symptoms duration, are well known to compromise at different levels the vascular or the neurobiological condition of the compressed nerve [6, 17, 18, 24]. The interpretation of the symptomatic compression neuropathy as a result of a series of multifactorial local and non local dysfunction mechanisms, represents a possible explanation of the fact that these patients have usually more than one complaint. Besides neurological examination (''en griffe'' shaped hand, muscle hypotrophy, sensory disturbances and the presence of the sign of Froment,) EMG represents an invaluable diagnostic tool, supplying decisive informations about the conduction speed of the affected nerve and the site of compression, although the neurophysiological testing barely correlates with the clinical features [54]. One possible explanation is that conduction velocities are not sensitive to axonal loss as they are to demyelination [40]. However when clear dysfunction was detected, a pathological process was always identified at surgery and the role of EMG studies is still relevant in the differential diagnosis, e.g. with the thoracic outlet syndrome or Pancoast's tumour [17].

Tardy ulnar palsy (TUP) is the fourth most common entrapment neuropathy (after carpal tunnel, cervical rib compression and meralgia paraesthetica) [27].

The clinical features more commonly include paraesthesias and signs of sensory-motor involvement, while pain and relevant motor deficits are less represented [17]. We were able to detect systemic or local predisposing factors in 1/3 of the reported cases. Factors favourably influencing the outcome were: age less than 50 and a short clinical history, while a worse prognosis could be expected in patients with serious muscle hypotrophy, concurrent systemic diseases and a longer history. The typical patient is middle-aged, male (more frequent to trauma exposure), Tinel's sign positive and complaints of motor and sensory disturbances on the non-dominant side. These consist of paraesthesias of 4th and 5th fingers, pain on the ulnar side of the forearm, weakness in the ulnar muscles of the hand. TUP is mainly caused by trauma (15–47%), sometimes not mentioned in the history [27, 28]. A childhood trauma is reported in 20% of the cases [27]. This frequently follows fractures if these are associated with cubitus valgus [28, 49]. Other causes are arthritis (20–38%) [12] and recurrent trauma (4%) [44, 45, 47]. Although bilaterality of elbow neuropathy is sustained by some Authors [25], the EMG evidence without clinical symptoms does not justify any therapeutic procedure. In our series bilateral TUP was present in 18.6% of cases and no correlation with predisposing conditions or factors was found.

Plain X-ray films of the elbow may reveal degenerative bony changes, old fractures and increased valgus angulation [28, 53]. CT and MRI data are still anecdotal in the literature. In our experience, while MRI studies provide available information about aponeurotic and/or muscle abnormalities and lesions narrowing the cubital space, CT is more helpful in revealing bony changes (e.g. osteophytes) in the cubital tunnel.

Electromyography and conduction velocities studies often demonstrate denervation potentials in the flexor carpi ulnaris or in the abductor digiti minimi and slowing of motor (not frequently) and/or sensory nerve conduction [6, 20, 48, 54].

The differential diagnosis is based on clinical and neurophysiological criteria, expecially taking into account C8-T1 root involvement by cervical spondylosis or discopathy, thoracic outlet syndrome, Pancoast tumours [17].

Since widely accepted grading criteria for these patients are lacking, the therapeutic guidelines are still a matter of debate by various Authors. Moreover, controversies arise because the relevant literature consists of series of a single surgeon, invariably accustomed to a particular surgical technique (therefore surgical strategies are not the result of comparable criteria of patient selection).

In order to avoid these problems, we attempted to strictly define three supposedly homogeneous classes

of patients, tailoring for each the therapeutic strategy and an appropriate surgical option. The surgical results at the 1-year follow-up are satisfactory. In fact, 85% of patients improved after surgery (65% excellent and 20% good), whereas only 15% resulted in no change. Cases with particular features benefit from simple decompression (13%) and from deep transposition (7%), while the technique most frequently employed was the superficial transposition (80%). Surgical approach appears to deserve little consideration to the specific cause. This reflects the lack of prospective randomized studies, performed using more than one procedure in the screening of the clinical material. Moreover, follow-up studies are usually too short for a pathological condition that often shows long lasting recovery and spontaneous healing. Therefore, the published studies are contradictory concerning the efficacy of the different surgical procedures and the rate of satisfactory results. The only conclusion is that the postoperative results reflect the preoperative status regardless of the surgical procedure [5, 6, 19, 28, 32, 55]. General agreement is also registered concerning the conservative treatment of patients with intermittent symptoms and the worse prognosis of patients complaining of symptoms lasting more than one year or with muscle atrophy [5, 14, 15, 22, 34, 39].

The role of epicondylectomy, of transposition and of intramuscular relocation techniques is controversial [21, 26, 33, 35, 59]. Retro-epicondylar compression is claimed to be more frequent than cubital tunnel syndrome, but this opinion requires further confirmation [7, 57].

Transposition may be more effective than simple decompression in patients with a history longer than one year [14, 28, 34].

However, three months after surgery, conduction is still below normal in 30% of cases, often with residual symptoms and signs [20, 40]. Five months postoperatively action potentials may remain subnormal [20]. The widespread use of a rating system for the evaluation of results is greatly desirable and the Bishop's scale may represent a starting point [32]. Conduction studies are advisable in surgical patients, whether they have or have not improved after the procedure.

These remarks are essentially in agreement with those of the major series in the literature.

On the other hand, we must be able in the future to fruitfully compare data and conclusions drawn from very large series and different surgical experiences.

Eventually, the correlation between anatomo-clinical data and surgical strategy, in spite of the protean aetiopathogenesis of the syndrome, remains, in our opinion, the desirable solution to tailor the treatment to the requirements of the single case.

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