Anterior transposition of the ulnar nerve: an electrophysiological study

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Of 13 patients with lesions of the ulnar nerve at the elbow, 11 were investigated before and after anterior transposition of the nerve and two before and after a period of conservative management. This has given us the opportunity of observing the way in which sensory and motor fibres recover when in continuity and no longer exposed to trauma, and of reconsidering the indications for surgery.

Patients

The clinical data are given in Table I. When first examined all patients had sensory impairment in the ulnar distribution and weakness of the interossei; using McGowan's (1950) grading of severity four patients (cases 6, 4, 12, 32) belonged to grade III (severe) and the rest to grade II (intermediate).

Method

Sensory and motor conduction were investigated in the ulnar nerve. The patient lay supine on an examination couch, the arm at 45° to the trunk, the elbow extended and the forearm supinated. The limb was warmed before and throughout the investigation and the temperature measured on the skin and near the nerve. At each recording site the temperature near the nerve was 34 to 36°C.

Sensory The electrodes were stainless steel needles 0.6 mm in diameter insulated except at the tip. They were placed at the wrist, about 5 cm distal to the medial epicondyle (‘below-sulcus’) and about 5 cm proximal to it (‘above-sulcus’). The exploring electrode (3 mm bared tip) was placed close to the nerve by adjusting its position until the lowest threshold for the muscle action potential was reached when the electrode was used to stimulate the nerve (<1 mA). The reference electrode (5 mm bared tip) was placed subcutaneously at a transverse distance of at least 4 cm from the exploring electrode. Sensory action potentials were recorded according to the procedure described by Buchthal and Rosenfalck (1966).

Motor The electrodes used for recording sensory potentials were then used to evoke a muscle action potential. The motor threshold (<1 mA) was checked to ensure that the needles were still close to the nerve. The stimulus, a rectangular current pulse 0.2 msec in duration, was then increased to be supramaximal—that is, at least 10 times the motor threshold. Muscle action potentials were led off by concentric needle electrodes 0.45 mm in diameter with a leading-off area of 0.07 mm² placed in the innervation zone of a hypothenar muscle.

The distal latency was measured as the time interval between the onset of the stimulus and the onset of the initial deflection of the muscle action potential. The distance between stimulating cathode and recording

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electrode was 6-9 cm S.D. 1.1. The latency was corrected to a distance of 7-0 cm according to:

\[
\text{lat}_{\text{corr}} = \text{lat}_{\text{meas}} - \frac{\text{distance}}{v_m (\text{wrist to below-sulcus})} - 7.0
\]

(Slomic, Rosenfalck, and Buchthal, 1968). Conduction velocity in the fastest fibres (\(v_m\)) was calculated for the segments above-sulcus to below-sulcus and below-sulcus to wrist. The amplitude was measured peak-to-peak and the degree of synchronization noted. The distal motor latency to m. flexor carpi ulnaris was measured when stimulating above-sulcus (distance 13-5 cm S.D. 1.9).

A change in conduction time or amplitude of sensory potentials or evoked muscle action potentials was considered significant when the percentage change corresponded to or was more than two standard deviations from the normal mean, or when the percentage change exceeded the 95% limit obtained from the cumulative distribution curve in normal subjects. These limits comprise inter- and intra-individual variation in normal subjects and may be stricter than is warranted; they have been used because repeated investigations in the same subjects were not available.

**RESULTS**

1. **Changes in sensory fibre conduction and in the sensory potential after transposition** After transposition sensory conduction velocity in the trans-sulcal segment was still below normal (30%) in eight cases, but three investigated before and after showed an increase of at least 55% (Table 2), the earliest increase being found five months after transposition. The potential recorded below- and above-sulcus was at most half the normal amplitude, and remained low after transposition (Fig. 1) whether or not the velocity had increased. \(v_s\) was slow from wrist to below-sulcus after transposition in seven cases, one of which had become normal across the sulcus (case 22, Table 2). Transposition affected \(v_s\) from digit V to wrist unsystematically; it was unchanged, slower, or tended to be even faster than before regardless of more proximal changes in velocity. There was an increase in amplitude of the potential recorded at the wrist of at least six times in all cases investigated seven months or longer after transposition (see Fig. 4) except one; no increase occurred earlier. Desynchronization present before transposition persisted, except in case 6 where the shape of the potential became almost normal.

2. **Changes in motor fibre conduction and in the muscle action potential after transposition** Conduction velocity in the trans-sulcal segment increased after transposition in 10 cases but became normal in only one, the others remaining slowed by about 30%; the earliest increase was seen at three months. Changes from below-sulcus to wrist were unsystematic. After transposition, the distal motor latency to hypothenar muscles was still prolonged in seven cases, but had shortened in two; the distal

**TABLE 1**

**CLINICAL AND ELECTROPHYSIOLOGICAL RESPONSE TO ANTERIOR TRANSPOSITION OF THE ULNAR NERVE AT THE ELBOW AND TO CONSERVATIVE MANAGEMENT**

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yr)</th>
<th>Aetiology</th>
<th>Duration of symptoms</th>
<th>Interval transp.- 2nd invest. (mo.)</th>
<th>Subjective change</th>
<th>Phys. signs</th>
<th>Elctrl. improv.</th>
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<tbody>
<tr>
<td>1</td>
<td>60</td>
<td>After prostatectomy</td>
<td>1 yr</td>
<td>34</td>
<td>+</td>
<td>+</td>
<td>SM</td>
</tr>
<tr>
<td>5</td>
<td>49</td>
<td>Acute trauma</td>
<td>2 yr</td>
<td>18</td>
<td>wo</td>
<td>0</td>
<td>SM</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>Unknown</td>
<td>10 yr</td>
<td>18</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>3</td>
<td>56</td>
<td>Unknown</td>
<td>2 yr</td>
<td>16</td>
<td>++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>Acute trauma</td>
<td>3 mo</td>
<td>16</td>
<td>N</td>
<td>N</td>
<td>SM</td>
</tr>
<tr>
<td>6</td>
<td>47</td>
<td>Unknown</td>
<td>7 mo</td>
<td>14</td>
<td>++</td>
<td>++</td>
<td>SM</td>
</tr>
<tr>
<td>25</td>
<td>46</td>
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<td>3 mo</td>
<td>8</td>
<td>++</td>
<td>++</td>
<td>SM</td>
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<tr>
<td>12</td>
<td>38</td>
<td>After lumbar laminectomy</td>
<td>4 mo</td>
<td>7</td>
<td>0</td>
<td>+</td>
<td>SM</td>
</tr>
<tr>
<td>18</td>
<td>57</td>
<td>After plaster cast</td>
<td>18 mo</td>
<td>6</td>
<td>+</td>
<td>0</td>
<td>SM</td>
</tr>
<tr>
<td>22</td>
<td>56</td>
<td>After colectomy</td>
<td>6 wk</td>
<td>5</td>
<td>0</td>
<td>+</td>
<td>SM</td>
</tr>
<tr>
<td>34</td>
<td>59</td>
<td>Unknown</td>
<td>2 yr</td>
<td>3</td>
<td>wo</td>
<td>0</td>
<td>M</td>
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Conservative management

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Duration of symptoms</th>
<th>Interval 1st-2nd invest. (mo.)</th>
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<tbody>
<tr>
<td>28</td>
<td>34 Chronic trauma (occupat.)</td>
<td>5 yr 7</td>
</tr>
<tr>
<td>32</td>
<td>50 After colectomy</td>
<td>2 mo 4</td>
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</table>

yr: years; mo: months; wk: weeks +: slight; ++: marked improvement; N: normal; wo: worse; S: in sensory fibres; M: in motor fibres.
motor latency to m. flexor carpi ulnaris was still prolonged in eight cases and had lengthened in one.

In brief, after transposition there was improvement in both motor and sensory fibres in 10 patients and in only motor fibres in one. The patients fell into two groups according to the interval between transposition and reinvestigation: with an interval of eight months or longer clinical improvement was marked except in one patient; with an interval of seven months or less clinical improvement was slight or absent, but even in these patients there was evidence of electrophysiological improvement, seen earliest in sensory fibres at five months and in motor fibres at three months.

Conservative management In two patients transposition was not performed: they were merely advised to avoid leaning on their elbows.

Case 28 A 35-year-old male university teacher had for several years experienced paraesthesia in the ulnar sensory distribution while leaning on his elbows, but for three months preceding the first investigation these symptoms had been constantly present on the left and he had noticed a tendency for the little finger to drift into abduction. On examination he described the touch of cotton wool as ‘different’ in the ulnar distribution, and the two sides of the ring finger did not feel the same. Two points were discriminated when 3 mm apart on the pulp of the little finger. There was minimal wasting of the hypothenar eminence and dorsal interossei and slight weakness of all ulnar-innervated hand muscles. When re-examined seven months later the patient claimed that a steady improvement had taken place, a ‘slight dead feeling’ at the tips of the ring and little fingers being the only symptom. Appreciation of cotton wool was normal except in those areas, and there was no wasting or weakness. The first electrophysiological investigation had shown a markedly slowed sensory conduction velocity across the sulcus, small and much-desynchronized potentials below and above the sulcus, and a small, normally shaped potential at the wrist. Conduction

### Table 2

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Before/after transposition</th>
<th>Potential amplitude (μV)</th>
<th>κv(m/sec)</th>
<th>Amplitude ev. pot. (mV)</th>
<th>Latcorr (msec)</th>
<th>vα(m/sec)</th>
<th>Lat a-f (msec)</th>
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<tr>
<td>1</td>
<td>before</td>
<td>5.0*</td>
<td>1.5*</td>
<td>1.0*</td>
<td>43</td>
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<td>40</td>
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</tr>
<tr>
<td>2</td>
<td>before</td>
<td>1.2*</td>
<td>0.7*</td>
<td>0.5*</td>
<td>45</td>
<td>65</td>
<td>47</td>
</tr>
<tr>
<td></td>
<td>after</td>
<td>1.2*</td>
<td>0.7*</td>
<td>0.5*</td>
<td>45</td>
<td>65</td>
<td>47</td>
</tr>
<tr>
<td>3</td>
<td>before</td>
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<td>1.0*</td>
<td>0.5*</td>
<td>44</td>
<td>38</td>
<td>37</td>
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<td>after</td>
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<td>1.0*</td>
<td>0.5*</td>
<td>44</td>
<td>38</td>
<td>37</td>
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<td>24</td>
<td>before</td>
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<td>0.3</td>
<td>0.4</td>
<td>36*</td>
<td>63*</td>
<td>51*</td>
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<td>0.4</td>
<td>36*</td>
<td>63*</td>
<td>51*</td>
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<td>before</td>
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<td>0.2*</td>
<td>0.2*</td>
<td>24</td>
<td>27</td>
<td>27</td>
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<tr>
<td></td>
<td>after</td>
<td>0.2*</td>
<td>0.2*</td>
<td>0.2*</td>
<td>24</td>
<td>27</td>
<td>27</td>
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<td>18</td>
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<td>0.4</td>
<td>0.5</td>
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<td>0.5</td>
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<td>62</td>
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<td>22</td>
<td>before</td>
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<td>0.4</td>
<td>0.5</td>
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<td>36</td>
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<td>0.5</td>
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<td>before</td>
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<td>1.2</td>
<td>44</td>
<td>62</td>
<td>56</td>
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<td></td>
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<td>1.0</td>
<td>1.2</td>
<td>44</td>
<td>62</td>
<td>56</td>
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Conservative management

<table>
<thead>
<tr>
<th>Case 28</th>
<th>Before/after transposition</th>
<th>Potential amplitude (μV)</th>
<th>κv(m/sec)</th>
<th>Amplitude ev. pot. (mV)</th>
<th>Latcorr (msec)</th>
<th>vα(m/sec)</th>
<th>Lat a-f (msec)</th>
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<td>3.0</td>
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<td>2.0</td>
<td>46</td>
<td>58</td>
<td>24</td>
</tr>
<tr>
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<td>after</td>
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<td>1.8*</td>
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<td>52</td>
<td>65*</td>
<td>53*</td>
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<td>before</td>
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<td>0.2*</td>
<td>0.2*</td>
<td>43</td>
<td>37</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>after</td>
<td>&lt;0.1*</td>
<td>&lt;0.1*</td>
<td>&lt;0.1*</td>
<td>43</td>
<td>37</td>
<td>39</td>
</tr>
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</table>

Normal

<table>
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<th>Mean</th>
<th>S.D.</th>
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<tr>
<td>13.9</td>
<td>7.7(8.2)</td>
</tr>
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</table>

w: wrist; b: below the sulcus (about 5 cm distal to the medial epicondyle); a: above the sulcus (about 5 cm proximal to the medial epicondyle); d: digit V (the little finger); κv: conduction velocity in fastest conducting sensory fibres; vα: conduction velocity in fastest conducting motor fibres; Latcorr: distal motor latency to muscles of hypothenar eminence, corrected to a distance of 7 cm; Lat a-f: distal motor latency to m. flexor carpi ulnaris, stimulating above the sulcus; * significant difference between first and second investigations (P < 0.05); ( ) - determined from the cumulative distribution.

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FIG. 1. Sensory action potentials in a normal subject aged 54 years (left) and in a patient aged 50 years with a lesion of the ulnar nerve at the elbow before (middle) and 18 months after transposition (right). The clinical and electrophysiological data are in Tables 1 and 2.

The potentials, evoked by supramaximal stimuli to digit V, were recorded at the wrist by photographic superposition or electronic averaging of 500 sweeps, at other sites by electronic averaging. The trace below shows averaging of 500 sweeps with electrodes in place but with stimulus zero. The figure above each potential indicates the conduction velocity in m/sec, $T_s$ the sensory threshold. The temperature near the nerve was 35°C.

Note that after transposition the amplitude of the sensory response had increased at the wrist and that the conduction velocity was uniformly decreased over the length of nerve examined.
velocity across the sulcus in motor fibres was considerably slowed, though less so than in sensory fibres. At the second investigation sensory conduction across the sulcus was normal, potential amplitudes at all levels were only just below normal and the degree of synchronization had increased markedly below- and above-sulcus (Fig. 2).

**Case 32** A 50-year-old carpenter had had no symptoms before a total colectomy for ulcerative colitis. After the operation he remained confused for at least a week, but on recovering normal consciousness he became aware of weakness and paraesthesiae in the left hand. Two months later there were severe hypaesthesia, hypalgesia, and dysaesthesia in the left ulnar distribution and two-point discrimination was impossible. There were gross wasting of ulnar-innervated hand muscles, weakness of the medial

![Graph showing sensory action potentials](image-url)

**FIG. 2.** Sensory action potentials evoked by supramaximal stimuli to digit V in a patient aged 34 years (case 28) before (left) and after 7 months conservative management (right). The clinical and electrophysiological data are in Tables 1 and 2; the procedure was as described in the legend to Fig. 1. The temperature near the nerve was 35°C.

Note that on re-investigation the amplitude and conduction velocity of the sensory responses had increased, and that temporal dispersion below and above the sulcus was lessened.
part of flexor digitorum profundus, and an early claw hand. When re-examined after four months the patient claimed that paraesthesiae were less marked and that use of the hand was improved. On examination there was no sensory improvement except that two points could now be discriminated when 9 mm apart, but wasting and weakness seemed a little less pronounced than formerly. At the first electrophysiological investigation no sensory potential could be recorded at the wrist and no potential evoked in hypothenar muscles by stimulating the nerve at the wrist. On re-investigation a sensory potential of less than 0·1 μV was recorded at all levels and sensory conduction was markedly slowed. A small potential was evoked in hypothenar muscles and motor conduction was somewhat slowed from below- and above-sulcus to wrist.

In brief, the first of these patients was mildly affected and returned almost to normal in seven months; the second was severely affected and began to show signs of recovery within four months.

DISCUSSION

1. Interpretation of changes in sensory and motor conduction The first sign of recovery in both sensory and motor fibres was an increase in conduction velocity in the trans-sulcal segment. In sensory fibres, at least, regeneration at the sulcus preceded that at the wrist, but not until the sensory potential recorded at the wrist had increased in amplitude did marked clinical improvement occur. The increase in sensory conduction velocity across the sulcus after transposition was not associated with an increase in amplitude or a change in shape of the potential above-sulcus, suggesting that at that early stage regeneration had occurred at much the same rate in all surviving fibres and that no blocked fibres had yet started to conduct. By contrast, the increase in sensory conduction velocity across the sulcus in a patient managed conservatively (case 28) was accompanied by an improvement in both amplitude and degree of synchronization of potentials recorded above and below the sulcus.

Sensory conduction velocity after transposition was usually slowed over the whole length of nerve studied—that is, digit V to above-sulcus—but in some instances slowing was confined to particular segments. Thus, in case 25, eight months after transposition, the velocity had increased markedly across the sulcus and from wrist to below-sulcus but not between digit V and wrist, and the amplitude of the sensory potential at the wrist was at least unchanged. In case 22, five months after transposition, an earlier stage in recovery could be seen, an increase in velocity across the sulcus but persisting slowing from wrist to below-sulcus. A normal sensory conduction velocity across the sulcus was reached in only three of 12 cases (25, 22, 28) and a normal motor conduction velocity in only one of 13 (case 34); the mean velocity in the others remained 30% below the normal mean in this segment for both types of fibres.

A 25% reduction below the mean was found by Cragg and Thomas (1964) in rabbit peroneal nerve fibres which had regenerated after crush lesions, and they thought it probable that a normal conduction velocity is never regained by regenerated nerve fibres. However, the slowing in ulnar lesions of the type under discussion is likely to be due to a number of factors in addition to Wallerian degeneration and regeneration, including demyelination and ischaemia, and the survival of only a few fast conducting fibres would explain the normal velocities occasionally preserved or regained. Conduction in immature, regenerating fibres would explain the uniformity of slowing in motor fibres below and across the sulcus in cases 6 (Fig. 3), 4, and 12 after transposition, where pre-operatively there had been either a sharp fall in velocity across the sulcus or no conduction at all. In case 4, for example, the amplitude of the evoked potential increased from 0·1 to 16 mV at the same time that v_m from below-sulcus to wrist dropped from 65 to 32 m/sec.

2. Spontaneous versus post-operative recovery Although the lesion in one of the conservatively managed patients (28) was mild, the changes in sensory conduction were marked once the nerve ceased to be subjected to mechanical insult.

A severe lesion due to compression of the ulnar nerve during a period of unconsciousness may show signs of recovery within a few months whether or not transposition has been performed: initially in neither case 12 nor 32 could a muscle action potential be evoked on stimulating at the wrist, nor a sensory potential be recorded at wrist on stimulating digit V. In the conservatively managed case (32) the period of recovery was shorter but the subjective benefit greater, and although the sensory potential was smaller, conduction velocity in both sensory and motor fibres was faster than in the nerve (case 12) which had been transposed.

In one case (case 1) symptoms had originally been present and equally noticeable in both hands. This was still so at the time of reinvestigation nearly three years after operation on one side, suggesting that such electro-physiological improvement as had taken place was not due to transposition.

Given that spontaneous recovery may take place, can it ever be said that improvement after surgery would not have occurred otherwise? This raises the question of the indications for conservative management. Platt (1926) records that of nine ulnar nerve
lesions complicating recent fracture of the lower end of the humerus seven had recovered spontaneously and completely within eight months of injury. This he ascribes to the pathogenesis of the lesion, contusion without loss of continuity and without alteration of the post-condylar groove to the disadvantage of the nerve trunk. Since spontaneous recovery is the rule in this type of case, he recommends transposition only if the lesion be severe and persistent.

Of 100 patients with ulnar palsy seen consecutively at the Mayo Clinic (Gay and Love, 1947), one was treated conservatively, a 63-year-old accountant with a hand so weak he was unable to button his clothes, shave, or write properly, but no sensory disturbance. He was instructed to avoid his normal posture of resting the elbow on his desk and given exercises to the affected muscles. More than a year later he reported that the hand was functioning normally and the atrophy disappearing. Mcgowan (1950), in a paper devoted to the results of transposition, refers to a 45-year-old labourer who 'felt something go' in the right elbow region while pushing a heavy load and subsequently developed an ulnar palsy. Osteoarthritis of the elbow was found, with roughening of the ulnar groove, but operation was refused and the patient was examined every six months. Two and a half years after onset all affected muscles had regained almost full power. It would appear, therefore, that Brooks' (1950) statement: 'early transposition is desirable; there is no conservative treatment', needs qualification.

3. The indications for transposition. The principle has been clearly stated by Platt (1926):

'When the ulnar nerve reaches the post-condylar groove at the elbow it comes to occupy a position of extreme vulnerability; but it is not the exposed situation of the nerve trunk which alone determines the incidence of traumatic lesions at this level... There are certain special types of ulnar nerve injury which are primarily determined by an alteration in the normal relation between the nerve-trunk and its bed in the post-condylar groove... The nerve lesions produced in this way are ordinarily of the incomplete type, and
may be described under the broad title of traumatic neuritis... There is one simple and effective operation which is universally applicable in all cases of traumatic neuritis of the ulnar nerve in the post-condylar groove, viz., anterior displacement of the nerve trunk.'

In none of our patients was there any reason to suspect 'an alteration in the normal relation between the nerve trunk and its bed'. Nevertheless, lesions of the ulnar nerve may arise in the absence of an obvious injury to the nerve or abnormality of the elbow joint, and some of these have been shown by Osborne (1957) and by Feindel and Stratford (1958) to be due to compression of the nerve by a fibrous band bridging the two heads of flexor carpi ulnaris, a lesion analogous to carpal tunnel compression of the median nerve. Such lesions tend to develop slowly and insidiously, and there can be no point in merely allowing further time to pass in the hope of spontaneous improvement. Though not specifically stated in the operation reports, those lesions the aetiology of which is described as 'Unknown' in Table 1 may have belonged to this group, and they have responded well to surgery (except for case 34, reinvestigated only three months later).

On the other hand, when a single episode of acute trauma or compression, or occupational trauma, has been responsible for a lesion at the elbow there is no indication for surgical intervention, there being no reason to suspect either pre-existing or permanent post-traumatic alteration in the normal relation of the nerve trunk to its bed. When the position of the nerve in the sulcus is no hindrance to its recovery improvement after transposition will be post but not propter hoc (cases 6 and 25, Table I). Similarly, a slow or poor response to transposition in such cases emphasizes that merely altering the course of the nerve can do nothing to promote regeneration (cases 1, 12, 18, and 22, Table I). Mumenthaler (1960) arrived at similar conclusions on the basis of a clinical study of 314 lesions of the ulnar nerve.

FIG. 4. Sensory action potentials (above) and muscle action potentials (below) evoked by supramaximal stimuli to the ulnar nerve in a patient aged 56 years with a lesion of the nerve at the elbow before and 16 months after transposition. The clinical and electrophysiological data are in Tables I and II. The procedure of recording the sensory responses was as described in the legend to Fig. 1. The temperature near the nerve was 35°C.

Note that after transposition the amplitude of the sensory and motor responses was increased, the sensory velocity decreased and the muscle action potential more synchronized.
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SUMMARY

Clinical and electrophysiological recovery has been investigated in 13 patients with a lesion of the ulnar nerve at the elbow, of whom 11 underwent anterior transposition of the nerve. The cause of the lesion was acute trauma, chronic occupational trauma, a period of compression, or was unknown; in no patient was there mechanical abnormality of the elbow joint or spontaneous dislocation of the nerve; all patients had sensory and motor symptoms and signs.

Improvement in both sensory and motor fibres occurred in 12 patients, though most were still far from normal. An increase in conduction velocity across the sulcus was the first sign of recovery in both sensory and motor fibres; restoration of the sensory potential recorded at the wrist occurred later, and only then was there significant clinical improvement. Conduction velocity distal to the lesion was a poor guide to its severity, but the velocity in the fastest conducting fibres across the sulcus, apart from localizing the lesion, indicated its severity and response to treatment.

In the extent and rate of their improvement the patients managed conservatively equalled or exceeded those in whom transposition was performed. It is contended that the operation is being performed more often than necessary: the course of the nerve need be altered only when it lies in adverse relation to its bed, and lesions caused by acute local trauma, avoidable occupational trauma, or a period of compression lack this essential criterion for surgical intervention.

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