Electrophysiological findings in pressure palsy of the brachial plexus

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SUMMARY Two patients with signs and symptoms of paralysis of the brachial plexus, caused by compression during surgery in one (case 1) and by a knapsack in the other (case 2), were examined. The characteristic electrophysiological findings were: (i) severe attenuation of amplitude of motor and sensory nerve action potentials evoked or recorded above the site of nerve injury compared to those evoked or recorded below, and (ii) slowing of motor and sensory conduction across the damaged area. Case 1 made a complete recovery clinically and electrophysiologically; EMG in case 2 suggested the presence of Wallerian degeneration. The palsies were classified as a local demyelinating block alone (case 1) or combined with axonal loss (case 2), and, if possible, to contribute to the pathogenesis by means of nerve conduction studies across the presumed site of nerve injury.

Case reports

CASE 1 (EMG 17322) A 50 year old housewife was operated upon for a lumbar disc under general anaesthesia. The operation lasted about three and a half hours. When the patient woke up from anaesthesia she was unable to move her left arm and her right arm was weak. A neurological examination the day after the operation showed paralysis of the whole left arm and moderate to severe paresis of all muscles of the right arm. There was anaesthesia of the left arm but normal sensation on the right. The tendon reflexes were absent. The palsies were thought to be due to compression by shoulder braces used during the operation. Seven days later there was practically normal force and mobility of the right arm, but only very faint movements of the fingers on the left side. There was anaesthesia-analgesia of the whole left arm. An electrophysiological examination was performed one month after the onset of the palsy when the left deltoid, biceps and triceps brachii, the brachioradialis, the extensor and the flexor muscles of wrist and fingers were still paralysed, and the force of the small muscles of the hand was 0-1 (Table 1). At that time there was hypaesthesia-hyalgesia corresponding to the axillary and musculocutaneous

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nerves, hypaesthesia in the distribution of the radial, median, and ulnar nerves. Two months after injury the force had improved considerably, especially that of the forearm flexor and extensor muscles, as well as of the small muscles of the hand. There was still a moderate to severe paresis of the deltoid, brachial biceps, and triceps muscles (Table 1). After two more months recovery was complete.

**CASE 2 (EMG 19127)**

A 29 year old man developed paralysis of the left arm when carrying a shoulder pack of 40 kg weight. He experienced transient paraesthesia in both the left and right hand about half an hour after he had started to carry the weight. Approximately 15 minutes later the left arm became paralysed. Nevertheless the patient continued to carry the knapsack for the next four to five hours. He was seen the next day by a doctor who described the arm as paralysed. Eleven days later on admission to a local hospital he could abduct the arm about 60° at the shoulder joint. There was no movement of elbow joint in either flexion or extension; wrist and finger extendors were paralysed, wrist and finger flexors severely paretic. There was moderate weakness of the small muscles of the hand (Table 1). He complained of dyæsthesia of the three radial fingers.

About two months after the onset of palsy he was referred for an electrophysiological examination. There had been a slight recovery since the onset, but there was still marked atrophy and weakness of the supra-and infraspinatus, the del-

toid, biceps, and triceps brachii as well as of forearm and wrist extensor muscles. There was still a slight paresis of the forearm flexor muscles and of the small muscles of the hand (Table 1). There was hypaesthesia along the radial margin of the left arm and two radial fingers. When discharged three months after the onset of the palsy the force was only mildly reduced in the previous severely affected muscles and there were no sensory disturbances.

**Methods**

Case 1 was examined electrophysiologically one, two, and four months after the onset of paralysis. Case 2 was examined only once, two months after the onset of paralysis.

**ELECTROMYOGRAPHY**

The criteria used were (1) the pattern of activity at full effort, (2) the amplitude of its envelope curve, (3) the number of sites at which fibrillation potentials and positive sharp waves were recorded outside the endplate zone, and (4) the mean potential duration and amplitude of at least 20 randomly sampled motor unit potentials. Findings were evaluated by comparison with normal muscles matched for age (Buchthal, 1957; Rosenfalck and Rosenfalck, 1975).

**CONDUCTION STUDIES**

Motor and sensory conduction in the radial and musculocutaneous nerves were determined according to methods previously described (Trojaborg

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**Table 1 Muscle testing**

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Time after onset</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Postoperative palsy</td>
<td>Case 1</td>
<td>Case 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 mo</td>
<td>2 mo</td>
<td>4 mo</td>
<td>11 d</td>
</tr>
<tr>
<td>Rhomboid</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Supraspinatus</td>
<td>0</td>
<td>2</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Infraspinatus</td>
<td>0</td>
<td>2</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Deltoid</td>
<td>0</td>
<td>2</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Biceps brachii</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Triceps brachii</td>
<td>0</td>
<td>4</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Extensors of forearm</td>
<td>0</td>
<td>4</td>
<td>5</td>
<td>0-1</td>
</tr>
<tr>
<td>Flexors of forearm</td>
<td>0</td>
<td>4</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Abductor pollicis brevis</td>
<td>0-1</td>
<td>4</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Abductor digiti minimi</td>
<td>0-1</td>
<td>4</td>
<td>5</td>
<td>3</td>
</tr>
</tbody>
</table>

*Graded according to Medical Research Council (1943). d = days, mo = months.
and Sindrup, 1969; Trojaborg, 1976). In short, the radial and musculocutaneous nerves were stimulated by needle electrodes placed at the elbow, axilla, and supraclavicular fossa. Muscle responses were evoked in the triceps brachii, brachioradialis, extensor digitorum communis, and biceps brachii by supramaximal stimulation. When stimulating in the supraclavicular fossa responses were also evoked in the infraspinatus and deltoid muscles (Gassel, 1964).

Sensory fibres of the radial and musculocutaneous nerves were stimulated by needle electrodes placed at the wrist and elbow respectively. Sensory potentials were recorded over the radial nerve at the elbow, and over the radial and musculocutaneous nerves at the axilla and in the supraclavicular fossa. Findings were compared with normal values matched for age.

Results

ELECTROMYOGRAPHY

The electromyographic findings are presented in Table 2. In case 1 there was discrete activity of low voltage on voluntary innervation in all muscles tested one month after the onset of palsy. Both fibrillation potentials and positive sharp waves (denervation potentials) were abundant, and too few motor unit potentials could be evoked voluntarily to determine a mean duration and amplitude. One month later muscle force had improved, although in muscles innervated by the superior trunk the force was graded as 2-3 (Table 1). Electromyographically, there was more activity during full effort, and the amplitude of the pattern had increased. Denervation potentials occurred in fewer sites (Table 2). The mean duration and amplitude of motor unit potentials determined in the brachial biceps was normal (12.4 ms, SD 3.2 ms, 250 μV, SD 140 μV, n=20), and the incidence of polyphasic motor unit potentials was 10% which is within the normal limit (Fig. 1). When the patient was tested four months after the onset of palsy there were no signs of denervation in the brachial biceps muscle.

In case 2 there were electromyographic findings similar to case 1—that is, discrete activity at full effort in most of the muscles tested and pronounced signs of denervation with the exception of the small muscles of the hand. However, the mean duration of motor unit potentials in the brachial biceps and extensor digitorum communis muscles was slightly increased (24% and 22% respectively), and the mean amplitude was markedly increased (150%). The incidence of polyphasic potentials was 36% in the brachial biceps and 38% in the extensor digitorum communis—that is, about 10 times more frequent than the average in normal muscles.

MOTOR NERVE CONDUCTION

The findings are summarised in Table 3. In both cases, muscle responses evoked by nerve stimulation at the axilla, one and two months after nerve injury were normal in spite of severe muscle weakness. However, when stimulating in the supraclavicular fossa, there was a severe attenuation in amplitude of all muscle responses with the exception of that evoked in the infraspinatus muscle in case 2. Thus, in case 1 the amplitude of the potential in the brachial biceps evoked by proximal stimulation was only 3% of that evoked by distal stimulation (axilla, Fig. 2). The conduction velocity along the musculocutaneous nerve was 38% in case 2 and 39% in normal muscle.

Table 2  Electromyographic findings

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Postoperative palsy</th>
<th></th>
<th></th>
<th></th>
<th>Pack palsy</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mo</td>
<td>2 mo</td>
<td>Full effort</td>
<td>Amplitude (mV)</td>
<td>Den†</td>
<td>Full effort</td>
<td>Amplitude (mV)</td>
<td>Den†</td>
</tr>
<tr>
<td>Deltoid</td>
<td>0</td>
<td>1.0</td>
<td>DA</td>
<td>0</td>
<td>0</td>
<td>1.0</td>
<td>DA-RR</td>
<td>1.0</td>
</tr>
<tr>
<td>Biceps brachii</td>
<td>DA</td>
<td>1.0</td>
<td>DA-RR</td>
<td>0.3</td>
<td>0.3</td>
<td>DA</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>0</td>
<td>1.5</td>
<td>DA-RR</td>
<td>0</td>
<td>0</td>
<td>1.5</td>
<td>DA</td>
<td>0.5</td>
</tr>
<tr>
<td>Triceps brachii</td>
<td>0</td>
<td>1.5</td>
<td>DA-RR</td>
<td>0</td>
<td>0</td>
<td>1.5</td>
<td>DA</td>
<td>0.5</td>
</tr>
<tr>
<td>Extensors of forearm</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>0.5</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>1.0</td>
</tr>
<tr>
<td>Flexors of forearm</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>0.5</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>1.0</td>
</tr>
<tr>
<td>Abductor pollicis brevis</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>0.5</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>1.0</td>
</tr>
<tr>
<td>Abductor digiti minimi</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>0.5</td>
<td>0.5</td>
<td>1.0</td>
<td>DA</td>
<td>1.0</td>
</tr>
</tbody>
</table>

*Pattern at full effort, DA = discrete activity, RR = reduced recruitment.  
†Fibrillation potentials and positive sharp waves, each + denotes a site in which denervation potentials were recorded, 0 = absent, — = not examined.
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Fig. 1  Histograms of action potential duration in brachial biceps (left) and extensor digitorum communis muscles (right). Above (left) from case 1 (mean duration: 12.4±0.7 ms, n=20), middle from seven normal subjects aged 25-34 years (mean: 11.1±0.7 ms, n=166), below (left) from case 2 (mean: 13.1±0.6 ms, n=28). Above (right) from 12 normal subjects aged 33-47 years (mean: 9.1±0.1 ms, n=353), below (right) from case 2 (mean: 12.0±0.6 ms, n=34). The arrow above each diagram denotes mean duration, black columns polyphasic potentials, ± mean error of SD.

from the supraclavicular fossa to the axilla was 30% decreased and the latencies to deltoid, infraspinatus, and brachial triceps muscles were about 50% prolonged. Two months after the onset of palsy, the degree of recovery of conduction was 60% as estimated from the attenuation in amplitude of the response in the brachial biceps when stimulating proximally and distally. The conduction velocity was 75% of the normal values and the latency to the deltoid and brachial triceps muscles was 30% increased, but the amplitude of the responses was within the normal range. Four months after onset of the paralysis recovery was complete, clinically and electrophysiologically.

In case 2 the conduction in motor nerve fibres to the extensor digitorum communis muscle was blocked between the supraclavicular fossa and the axilla, but they conducted normally from axilla and distally. Only few of the fibres to the brachioradial and the brachial triceps muscles conducted impulses when the radial nerve was stimulated in the supraclavicular fossa. The velocity in the conducting fibres was decreased in the proximal nerve segment (26% and 40% respectively) compared to the velocity in the segment from axilla to elbow. There was no response in the deltoid muscle to stimulation in the supraclavicular fossa, but an action potential was evoked in the infraspinatus muscle with a 50% increased latency and an amplitude half the normal mean.

SENSORY NERVE CONDUCTION
The conduction velocity in the musculocutaneous and radial nerves distal to the axilla was normal in both cases 1 and 2 (Table 3). The amplitude of sensory nerve action potentials at the axilla was
normal on all three examinations in case 1 (24-28-33 μV respectively, Fig. 2), whereas the amplitude of the potential recorded at Erb’s point was markedly reduced one month after the onset of the palsy, less so one month later, but normal on the third examination four months after the onset. The conduction velocity from axilla to Erb’s point was 50% reduced on the first examination, 37% reduced on the second, and normal on the third, compared with the velocity distal to the axilla. There were similar findings in case 2 with respect to decrease in amplitude of sensory nerve potentials recorded in the supraclavicular fossa. The sensory conduction velocity was 36% decreased in the radial nerve across the clavicle and 26% in the musculocutaneous nerve. Moreover, the amplitude of the sensory nerve potential recorded over the musculocutaneous nerve in the axilla was only half the normal mean.

**Discussion**

The two cases reported here with signs and symptoms of a brachial plexus lesion caused by compression during surgery in the one and by a knapsack in the other showed electrophysiological similarities. On account of clinical findings and changes in motor and sensory conduction the palsy could be classified as neuropraxia (Seddon, 1942), or as a local demyelinating block alone in case 1 and combined with axonal affection in case 2 (Wallerian degeneration, Gilliatt, 1975). This was based on the following criteria: (1) fast recovery clinically; (2) normal amplitude and shape of muscle responses evoked by stimulation distal to the site of compression in spite of severe paresis or paralysis as shown in animal experiments (Denny-Brown and Brenner, 1944; Fowler et al., 1972) and in man (Bauwens, 1960); (3) marked diminution of the amplitude of motor and sensory responses or block of conduction when stimulating or recording proximal to the site of compression; and (4) slowing in nerve fibres across the block as shown in rat and cat (McDonald, 1963; Meyer and Denny-Brown, 1964), and in motor and sensory nerve fibres in man (Trojaborg, 1970, 1977).

In case 1 recovery was completed within four months, clinically as well as electrophysiologically, and thus resembles the so-called ‘Saturday night palsy’ (Trojaborg, 1970). In case 2 the electromyographic findings of increased motor unit potential duration and amplitude, as well as increased number of polyphasic potentials, suggested reinnervation due to peripheral sprouting similar to the findings in a case of prolonged conduction block described recently (Trojaborg, 1977). This is in contrast to the findings in case 1 and in patients with Saturday night palsy, where there were no changes in the properties of the motor unit potentials during recovery.

In both cases 1 and 2 the distribution of motor paresis or paralysis was widespread and suggested an affection of the brachial plexus localised to the trunks, distally to the branching off of the nerve fibres to the rhomboid muscle which was clinically intact. Fifty cases of postoperative brachial plexus paralysis from the literature were classified, whenever possible according to the degree and distribu-
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Fig. 2  Sensory (above) and motor (below) action potentials evoked by supramaximal stimulation of musculocutaneous nerve. Sensory potentials were evoked by stimulating the lateral cutaneous nerve of the forearm at the elbow, and recorded at the axilla (first horizontal row), and at 'Erb's point' (second row), where the potentials were recorded using electronic averaging of 250, 500, and 1000 responses. The musculocutaneous nerve was stimulated at the axilla and at Erb's point, and the responses were recorded from the brachial biceps muscles (two lower rows); a = one month after onset of brachial plexus paralysis, b = two months after, and c = four months after onset of nerve injury. Figures above the traces denote maximal conduction velocity (m/s) in the segment distal to point of recording. Broken line (second row) indicates the use of a delay before sampling.

Table 4  Distribution of involvement and degree of recovery in 50 cases of postoperative brachial plexus palsies

<table>
<thead>
<tr>
<th>Degree of palsy</th>
<th>Distribution of involvement</th>
<th>Recovery*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All trunks</td>
<td>Upper trunk only</td>
</tr>
<tr>
<td>Complete</td>
<td>24</td>
<td>9</td>
</tr>
<tr>
<td>Incomplete</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>36</td>
<td>9</td>
</tr>
</tbody>
</table>

*In four patients (Büdinger, 1894) the degree of recovery could not be evaluated as they died shortly after the operation.
described existence in incomplete ported in the within less four months and, restored palsy. It may reasonably be assumed, on account of clinical and electrophysiological findings, that the mechanism of injury is the same in the postoperative as in the pack palsy. The fact that both the supra- and infraspinatus muscles were affected in the two cases presented here, as well as in most of the cases published, places the lesion proximal to the branching off of the suprascapular nerve from the superior trunk. Thus, the shoulder braces used during the operation in case 1 and the straps of the rucksack used in case 2 might have caused the compression of the nerve roots towards the transverse processes of the cervical column. A compression here could also account for the co-existence of a Horner's syndrome which has been described in five cases of postoperative brachial plexus injuries (Büdinger, 1894; Clausen, 1942).

A bilateral affection as observed in case 1 has also been reported previously after operation (Brichner, 1901; Clausen, 1942; Dhuné, 1950; Ewing, 1950; Petrick, 1955), and also among patients with rucksack paralysis (Sternberg, 1917; Bom, 1953; Daube, 1969).

The prognosis for complete recovery of function is good (Table 4). The rate of recovery is faster for sensory than for motor function, and muscles innervated by the lower nerve roots seem to recover before those innervated by upper nerve roots. The mode of recovery suggests that the highest degree of compression is exerted at the C5, 6 roots or the upper trunk. Motor deficit resolved within less than five months in about two-thirds of the cases published. Recovery took more than five months in about one-quarter, and was incomplete in 6% of postoperative brachial plexus palsy. In case 1 recovery was complete within four months and, although sensory deficit was restored before motor function, on clinical testing the rate of recovery electrophysiologically was the same for motor and sensory fibres in the musculocutaneous nerve. Similarly, in pack palsy the prognosis is good. Among the 30 cases reported in the literature, 26 made a complete recovery within less than three months, and only four had a prolonged convalescence (Daube, 1969) possibly due to partial axonal damage as in case 2 reported here.

References


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