THE DURATION OF NEUROMUSCULAR FUNCTION
AFTER NERVE SECTION IN MAN

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Conduction in the distal segment of severed peripheral nerve disappears after a maximum period of 3 to 5 days in a variety of experimental animals: rat, rabbit, guinea pig, dog, cat, and monkey. In contrast, Erb believed that the peripheral segment in man may remain functional for 1 to 2 weeks and Adrian and more recent workers have concluded that peripheral neural conduction may survive up to a month after nerve section in man. In better agreement with the results in animal studies are the recent observations by Murphey and Sunderland. The former states that peripheral excitability in sectioned human nerve is lost in 2 to 4 days; the latter, in 90 hours. A subject of such disagreement was considered worthy of special study.

Direct measurement of the nerve action potential is the most nearly accurate method of studying degeneration of conduction when the nerve can be dissected out for recording. This, of course, is impossible in man and percutaneous recording of nerve action potentials would be too insensitive to record small numbers of functional fibers.

Another complication is the fact that neuromuscular conduction fails a day or so before disappearance of the nerve action potential in some species of mammals. Since this dissociation has not been studied in primates, observations upon neuromuscular conduction after nerve section in the monkey were made for comparison with the nerve action potential data of Heibecker, Bishop, and O'Leary.

Visible and palpable muscle contraction in response to electrical stimulation of nerve has been used in this study as an index of function in the peripheral segment of severed nerve in man.

MATERIAL AND METHODS

Most of the lesions in human subjects studied were the result of lacerations. Direct stimulation of exposed proximal and distal nerve segments was carried out at the time of primary closure in most cases. Subsequently the peripheral segments were stimulated percutaneously at a distance from the muscles innervated. Since most lesions in this study were in the median and ulnar nerves, the intrinsic hand muscles were usually observed with stimulation at the wrist.

A small portable thyratron stimulator was used to deliver 0.1 mfd. condenser charges about once a second. Stimulation at such low frequencies is

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well tolerated (pain fibers respond only at several times maximal intensity for motor fibers^{10}), and the identification of direct responses was distinct from the reflex contractions that may be confusing with high frequency stimulation. The indifferent pad electrode was placed conveniently and a 5 mm. disc was the stimulating cathode.

RESULTS

The results in man are summarized in Table 1. Following complete nerve section (Cases 1–5) diminished maximal contraction strength on stimulation of the distal segment was usually apparent 48 hours after the lesion was made. Complete loss of excitability was seen between 91 and 128 hours after section.

### TABLE 1

<table>
<thead>
<tr>
<th>Case History</th>
<th>Nerve</th>
<th>Last Observation of Neuromuscular Response (hours)</th>
<th>First Observation of Complete Inexcitability (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Median and medial antibrachial cutaneous nerves cut in upper arm by glass. Major vessels spared.</td>
<td>Median</td>
<td>94</td>
<td>117</td>
</tr>
<tr>
<td>2. Median nerve and brachial artery cut at elbow with razor. Extensive blood loss. Tourniquet on arm for 3 hours.</td>
<td>Median</td>
<td>96</td>
<td>107</td>
</tr>
<tr>
<td>3. Buccal branch of facial nerve cut in the parotid gland by glass.</td>
<td>Facial</td>
<td>66</td>
<td>91</td>
</tr>
<tr>
<td>4A. Median and ulnar nerves cut in middle third of forearm</td>
<td>Median</td>
<td>109</td>
<td>121</td>
</tr>
<tr>
<td>4B. by razor. Moderate blood loss from radial artery.</td>
<td>Ulnar</td>
<td>121</td>
<td>128</td>
</tr>
<tr>
<td>5. Ulnar nerve and artery cut in lower third of forearm.</td>
<td>Ulnar</td>
<td>72</td>
<td>86</td>
</tr>
<tr>
<td>Moderate blood loss.</td>
<td>Ulnar</td>
<td>95</td>
<td>119</td>
</tr>
<tr>
<td>6A. Median nerve cut about 9/10 through by razor. No motor response to stimulation above lesion at time of operation. Moderate blood loss.</td>
<td>Median</td>
<td>95</td>
<td>119</td>
</tr>
<tr>
<td>6B. Tourniquet on arm for 3 hours. Ulnar deficit noted postoperatively, presumably caused by tourniquet pressure.</td>
<td>Ulnar</td>
<td>95</td>
<td>119</td>
</tr>
<tr>
<td>7. Ulnar nerve cut 4/5 through in lower third of forearm by glass. Muscular response to nerve stimulation above lesion at time of operation. No motor response to stimulation above lesion at elbow the next day.</td>
<td>Ulnar</td>
<td>71</td>
<td>85</td>
</tr>
<tr>
<td>8. Median nerve cut almost 1/2 through at elbow with knife. Clinical deficit incomplete.</td>
<td>Median</td>
<td>168</td>
<td>No definite deficiency</td>
</tr>
<tr>
<td>Macaque. Nerves aseptically severed in arm.</td>
<td>Ulnar</td>
<td>72</td>
<td>89</td>
</tr>
<tr>
<td></td>
<td>Median</td>
<td>54</td>
<td>72</td>
</tr>
</tbody>
</table>
In one patient (Case 6B) ulnar paralysis apparently resulted from tourniquet pressure on the arm during surgery. The patient was in a schizophrenic panic state and reliable examination was difficult. During exploration and primary suture of the median nerve in this patient (Case 6A) the intact ulnar nerve was identified beneath a partially severed flexor carpi ulnaris muscle. Postoperatively an ulnar deficit was noted. The ulnar became inexcitable at both elbow and wrist after 119 hours. It was impossible to follow this patient closely, but it is certain that the ulnar did not recover in the next 2 or 3 weeks. Nine months later ulnar function was clinically perfect; there was a moderate median deficit. It is inferred that the ulnar nerve underwent complete Wallerian degeneration and subsequently regenerated.22,24

The ulnar and median nerves were aseptically severed in the upper arm in one adult macaque monkey (Table 1). The median was unresponsive at 72 hours and the ulnar, at 89 hours. This correlates fairly well with the disappearance of the alpha spike in the nerve action potential of the macaque and indicates that failure of endplate conduction occurs at approximately the same time as loss of nerve conduction. It would appear likely that this relationship is similar in man.

**DISCUSSION**

The results indicate that neuromuscular function in man as in other mammals disappears 3 to 5 days after nerve section. Denervation is certainly complete by the end of the 6th day. Therefore the distinction between complete Wallerian degeneration and less severe injury can be made on the basis of the disappearance of excitability in the peripheral nerve segment at this time.

It is probable that when stimulation has to be applied close to the site of traumatic or surgical manipulation, premature loss of neural conduction may occur. In these cases such local effects of trauma are considered insignificant since there is no positive correlation between functional survival time and the distance from the lesion to the point of stimulation. Three lesions were more than 30 cm. from the area tested.

The disagreement with earlier workers may be attributed to a difference in the clinical material they studied. Apparently Erb's data are largely based upon observations in nontraumatic neuropathies and late traumatic lesions. Similarly Adrian's cases did not include acute traumatic nerve sections. His often reproduced diagram of the progressive change in motor point strength-duration curve after nerve section is derived from a case of Bell's palsy, in which degeneration can scarcely be assumed to be as acute or as complete as in nerve severance. Moreover, his conclusions are based not upon nerve stimulation, but upon a single discontinuity in the motor point strength-duration curve, and recent observers have not found the single discontinuity uniformly.

It follows from the present study that all changes in motor excitability
from the end of the 6th day onward must be the result of progressive excitability changes in the muscle fibers alone. Since the question of clinical significance is whether or not nerve function exists, it seems unnecessary in most cases to study secondary changes in muscle excitability in order to establish the diagnosis of denervation.

The technique described here requires any simple source of current and no special training. With the addition of a shielded needle electrode, study can be extended to nerves that are not readily accessible to superficial stimulation and to nerves that are injured near their terminations. The loss of muscle twitch where contraction was previously present is a sharp endpoint. By contrast it may be noted that the most elaborate muscle stimulation apparatus still depends upon the determination of threshold contraction by the fallible eye and hand of the observer. Moreover, changes of excitability as time goes by are gradual rather than distinct.

Electromyography and muscle excitability changes are reported to be of predictive value in recovering nerve injuries, but their complexity prevents their wide application. Direct stimulation of the proximal portion of regenerating nerves has been combined with electromyographic recording by Harvey and Kuffler and by Hodes, Larrabee, and German. It may be that such synchronous activation of nerve would give perceptible movement of predictive value before gross clinical signs of recovery appear.

SUMMARY

Electrical stimulation has been applied to the peripheral portions of human nerves after traumatic section, and to macaque nerves after surgical section. As in other mammals, motor function is completely abolished 3 to 5 days after the lesion is produced. The muscle is therefore certainly completely denervated by the end of the 6th day.

The technique used in this study is proposed as a simple direct method of establishing the diagnosis of complete Wallerian degeneration after nerve injury.

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REFERENCES