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ELECTROMYOGRAPHIC STUDIES OF PERIPHERAL NERVE BLOCK WITH PHENOL

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ABSTRACT. Peripheral nerve block with dilute phenol solution has been used for several years in spastic conditions. As regards the neurophysiological mechanism of peripheral phenol block, clinical observations and certain experimental studies on animals have given rise to the hypothesis of selective gamma fibre block. It was the purpose of the present study to analyse the immediate and the long-term effect of dilute phenol solution on the function of the peripheral nerve in patients with spasticity. Twenty-five nerve blocks were performed in 16 patients. Hypotonic or isotonic 2% phenol solution was injected intra- and perineurally. The electromyographic examination included a study of nerve fibre conduction, of the muscle response elicited mechanically and electrically, and of the myoelectric activity at rest and at attempted maximal voluntary contraction. In addition, the results were evaluated clinically. The findings indicate that the immediate effect of dilute phenol solution on spasticity cannot be ascribed to an absolutely selective block of gamma efferent fibres. Alphamotor and afferent group I a fibres were found to be blocked to a varying degree. As regards the longstanding effect on the exaggerated stretch reflex, however, this appears mainly to be due to a persistent depression of gamma fibres causing a decreased sensitivity of the muscle spindles. Electromyographic findings indicating degeneration of alphamotor fibres appeared to some extent after blocking, especially in cases injected with hypotonic phenol solution. Clinical evaluation showed a beneficial effect on spasticity in 16 out of 25 blocks.

Spasticity in brain damage is in most cases assumed to be caused by an increased activity of the fusimotor system as the result of decreased supraspinal inhibition. This view is based upon extensive experimental studies in the decerebrate cat. It is strengthened by reports on differential gamma fibre blocking with dilute procain solution in spastic patients (1). Thus a rational treatment of spasticity would aim at a selective depression of the hyperactive reflex arc.

In 1959 intrathecal injection of phenol was reported to reduce spasticity (2, 3). A dilute solution of phenol was assumed to block small fibres in the spinal roots selectively (4). However, injection of 5 to 20% solution in glycerin or ethiodan gave no selective fibre damage (5–7). In 1964 Khalili and co-workers (8) reported results of peripheral nerve blocks with 2 and 3% phenol solution. The alleviation of spasticity in certain cases without interference with motor or sensory function was ascribed to a selective block of gamma fibres (9).

Since experimental evidence for this interpretation has not yet been provided, it was the purpose of the present study to analyse the neurophysiological effect of dilute phenol injected intra- and perineurally in spastic conditions.

MATERIAL

The material comprises 25 nerve blocks in 16 patients. The cause of spasticity and other clinical details are given in Table I. Fifteen patients had a spastic hemiplegia due to traumatic or vascular lesion, 1 patient with multiple sclerosis had a spastic paraplegia. Spasticity was a major problem in all patients. Most patients had a severe clonus which interfered with activities of daily living. There was usually no appreciable voluntary movement of the spastic muscle groups. Position sense was preserved in 6 patients.

METHODS

The neurophysiological functions studied were

1. activity at rest with special regard to the presence of denervation potentials after phenol injection;
2. myoelectrical pattern at attempted maximal voluntary contraction;
3. myoelectrical pattern during rapid passiv stretching of the muscle group (clonus);

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Table I

Age: $m = 39$ y. (22–57). Duration of disease: $m = 3$ y. 8 mo. (7 mo.–9 y.)

	N
Traumatic brain lesion	6
Cerebral thrombosis	9
Multiple sclerosis	1
	16

4. amplitude of the mechanically evoked reflex response (tendon jerk);

5. muscular response to nerve stimulation proximally and distally to the point of phenol injection with special regard to (*a*) latency and maximal amplitude of direct muscle response, (*b*) latency and maximal amplitude of muscle response to stimulation of afferent fibres (Hoffman-reflex).

The electrically induced reflex (H-reflex) by-passes the muscle spindles. The response is a measure of motoneurone excitability. The mechanically evoked reflex, however, depends additionally upon the dynamic sensitivity of the muscle spindles. The sensitivity of the spindles is controlled by the central nervous system via the gamma efferent fibres. After intra- or perineural phenol injection, the ratio of the direct motor response at proximal and distal stimulation provides a measure of the extent of alphamotor block, since the amplitude of the potential is related to the number of excitable nerve fibres. The ratio of the H-reflex elicited above and below the site of phenol injection measures the extent of afferent (I *a*) block. A block of efferent gamma fibres will result in a depression of the mechanically evoked response. The depression is greater than might be expected from the extent of the afferent block.

EMG-recording. Recordings from the triceps surae muscle were made with non-insulated subcutaneous needle electrodes placed over the belly and the tendon of the muscle group. In other muscles action potentials were picked up by means of concentric needle electrodes. When the muscles were searched for spontaneous activity concentric needle electrodes were used in all muscles.

The potentials were amplified and displayed on one of the three channels of a DISA electromyograph (type 14A30). Continuous and interrupted recordings were taken. The film speed at continuous recording was 5 and 20 cm/sec. Interrupted recordings were taken with a time base of 1 and 2.5 msec/mm respectively.

Electromyography was performed before and after phenol injection, and in 12 cases the examination was repeated after 3–4 weeks.

Nerve stimulation. The subject was placed in a position as comfortable as possible. The nerves to be blocked were stimulated with pairs of non-insulated needle electrodes placed close to the nerve, proximally and distally to the point of phenol injection. In order to avoid a spreading of the phenol solution to the site of stimulation, the electrodes were placed as far as possible from the point of injection (3–5 cm). The needles were kept in place throughout the procedure. The stimulus was a rectangular pulse of 0.5 msec duration from a DISA Multistim. The stimulus frequency was about one per second. The amplitude of the maximal response was measured.

Ankle jerk. The ankle jerk was elicited manually with a reflex hammer and with the subject in the prone position. The foot was held dorsiflexed in zero position or, in most cases, in slight plantarflexion because of pronounced spasticity. Exactly the same position was used after nerve blocking. At least 4 reflex responses were recorded electromyographically (continuous recording, paper speed 20 cm/sec). The amplitude of the mechanically evoked action potentials showed a rather small variation when the tendon was tapped repeatedly with a constant strong blow (S.D. = 5.1%, 10 subjects). The mean value of 3 responses with the highest amplitudes was considered representative.

Phenol injection. The nerve to be injected was located *percutaneously* by means of an injection needle which served as a stimulus electrode. This approach was intro-

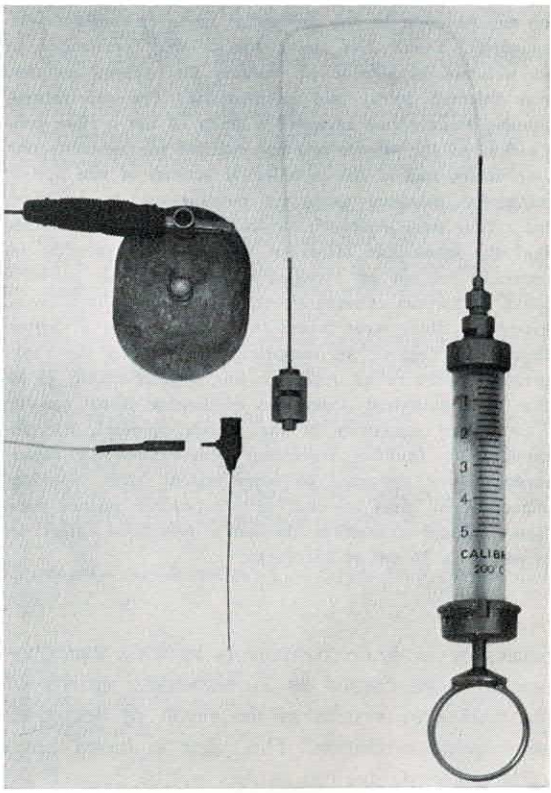


Fig. 1. Equipment for peripheral nerve blocking with phenol. The injection needle (0.6 mm diameter) serving as a stimulation electrode is insulated except at the bevel. The needle has a small screw-thread attachment to the cable. A very thin plastic tube connects the syringe with the needle.

duced by Highet in 1942 (10). It has been modified and applied to phenol injection by Khalili and co-workers. The needle, which had a diameter of 0.6 mm was insulated except at the bevel. It was connected with the syringe via a very thin plastic tube (Fig. 1) which made it more easy to handle. The dispersive electrode was placed opposite to the point of needle insertion. The nerve was localized by placing the tip of the needle in such a position that a minimal voltage of the stimulation impulse (usually below 1 V) produced a maximal amplitude of the evoked action potential (Fig. 2).

The tibial nerve was injected in the popliteal fossa, the ulnar nerve just proximally to the sulcus and the median nerve at the level of the medial epicondylus. The musculocutaneous nerve was approached according to Khalili & Betts (11).

The amount of phenol injected varied from 1.5 to 6 ml.

In the beginning of this study a 2% phenol solution in distilled water was used as recommended in the literature. This solution is hypotonic. However, because of a high frequency of denervation in these cases after injection we preferred an *isotonic* 2% solution in the majority of injections (21 blocks). The isotonic solution is prepared by adding 0.25 g% sodium chloride.

RESULTS

Clinical evaluation of spasticity after phenol injection showed a beneficial effect in 16 blocks (Table II). In 12 cases the spasticity disappeared completely, i.e. there was no clonus, the tendon jerk was absent or subnormal and muscle tone was judged to be reduced. After blocking of the posterior tibial nerve, the patients usually showed an improvement of voluntary contraction of the pre-tibial muscles. In 4 cases a slight exaggeration of the stretch reflex persisted. Reduction of spasticity with a duration of a few days only was recorded as a failure.

Table II

Nerves	*N*	Reduction of spasticity after phenol injection: Complete	Partial	Slight or none
Tibial	14	9	2	3
Median	9	1	2	6
Ulnar	1	1		
Musc. cutaneous	1	1		
	25	12	4	9

The *electromyographic findings* were as follows. Fibrillation potentials were not seen in any case before phenol injection. Re-examination 3 to 4 weeks after injection was performed in 12 cases. All cases (4) injected with hypotonic solution showed a high frequency of denervation potentials

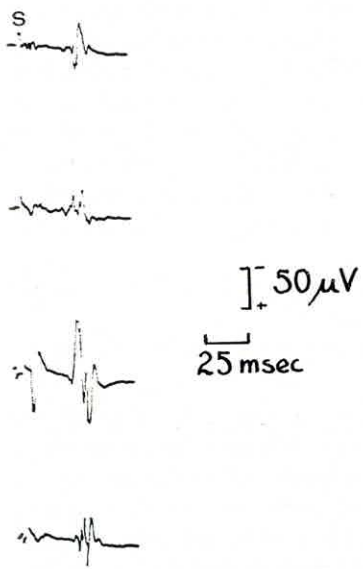


Fig. 2. Muscle response to repeated stimulation via the searching injection needle. The needle is moved slowly while the stimulation intensity is kept constant. The amplitude increases when the tip of the needle comes close to the nerve. Sweep recording. Subcutaneous needle electrodes. *s*, indicates the stimulus marking.

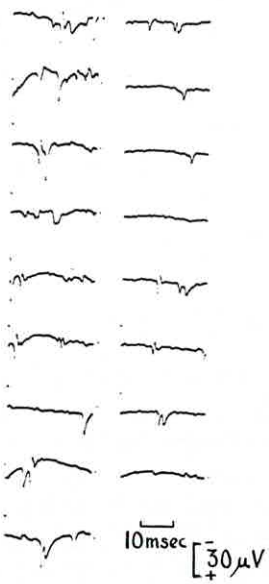


Fig. 3. Spontaneous fibrillation activity recorded 3 weeks after intra- and/or perineural injection of hypotonic 2% phenol solution. Concentric needle electrodes. Sweep recording.

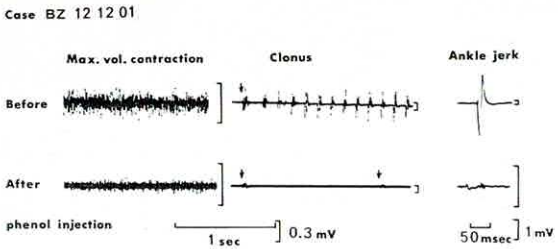


Fig. 4. The effect of phenol on the pattern at maximal voluntary contraction, on the clonus and on the action potential of the ankle jerk. Continuous recording from the calf muscles. Subcutaneous needle electrodes. The arrow indicates the moment of rapid passiv stretching of the muscles to evoke a clonus.

including positive monophasic potentials (Fig. 3). In the remaining 8 cases, moderate fibrillation was observed in 2 only.

The pattern at maximal voluntary contraction was reduced in all cases except 3 after phenol injection (Fig. 4). In 2 patients a transient complete flaccid paralysis was produced. In these cases voluntary function had reappeared after 3 weeks (Fig. 5).

The alteration of the evoked muscle response after phenol injection is illustrated by Fig. 6.

A detailed analysis of nerve function as described above was performed in 12 nerve blocks (tibial 7, median 4, ulnar 1). Ten to 20 min after injection the following alterations were observed.

Alphamotor fibres were found to be blocked to a varying degree in 11 nerves. Figs. 7 and 8 show a decrease of direct muscle response to proximal stimulation as compared with distal stimulation.

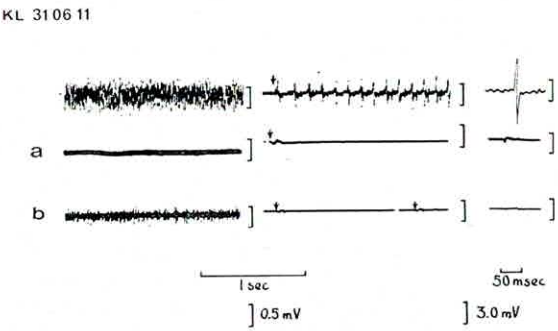


Fig. 5. Case of complete paralysis immediately after phenol injection (*a*). Three weeks later (*b*) voluntary activity has reappeared while clonus and ankle jerk are still absent. Continuous recording from the calf muscles. Subcutaneous needle electrodes.

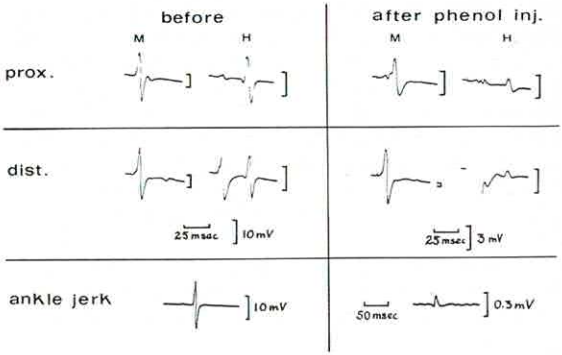


Fig. 6. Action potentials from the calf muscles. Subcutaneous needle electrodes. Maximal motor response (M), reflex response (H) induced by electrical stimulation proximally and distally to the point of phenol injection, and mechanically elicited motor response. After phenol the distal motor response is unchanged while the amplitude of motor response to proximal stimulation and the H-reflex is reduced to different extent. The most pronounced depression is seen in the mechanically evoked response.

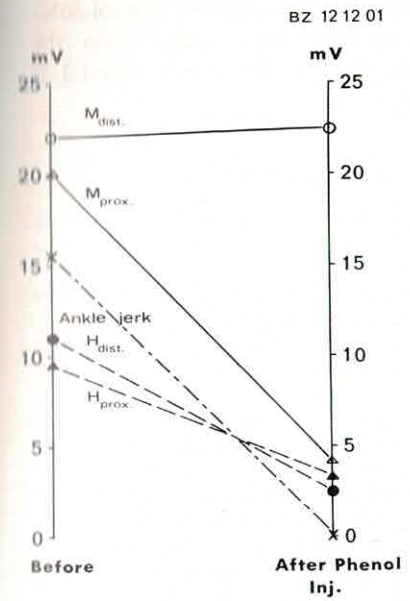
The amplitude of the H-response was reduced in all cases, partly due to the loss of motor fibres of the reflex arc. In 6 cases the findings suggested a partial block of afferent fibres (Fig. 7). The alteration of the tendon jerk was studied in the 7 tibial nerve blocks. The decrease of the ankle jerk exceeded considerably the degree of alphamotor and afferent block (Fig. 8). The amplitude of the mechanically evoked potential was on the average reduced to less than 20% of the pre-block value as compared with a 50% decrease of the H-response. The difference argues for a block of fusimotor fibres.

Figs. 9 and 10 illustrate the result of an incomplete nerve block.

Re-examination after 3 to 4 weeks showed in successful blocks a persistent depression of the stretch reflex while the block of alphamotoneurones and of afferent fibres was less pronounced or could not be demonstrated any longer.

A comparison of the latency of the response before phenol injection with that after injection did not show any consistent deviation.

Complications. Painful paresthesias were noticed in 8 our of 25 nerve blocks. The occurrence of this complication appeared not to be related to the amount of phenol solution injected. These symtoms disappeared usually within a few days



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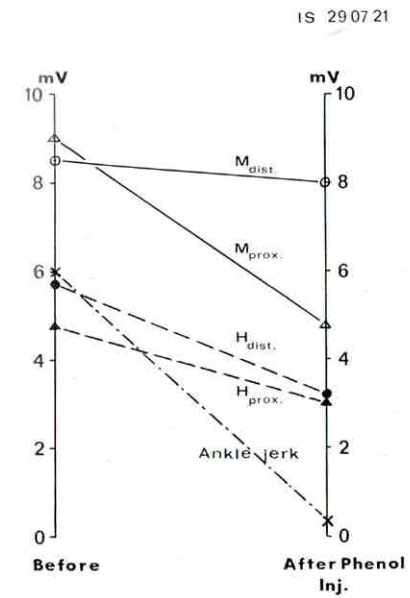

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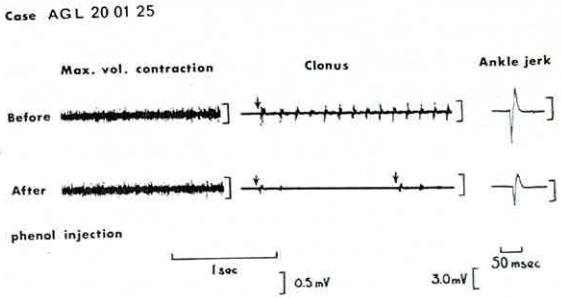
Figs. 7 and 8. Phenol block of peripheral nerve fibres as indicated by the decrease of amplitude (mV) of the electrically and mechanically elicited muscle action potentials.



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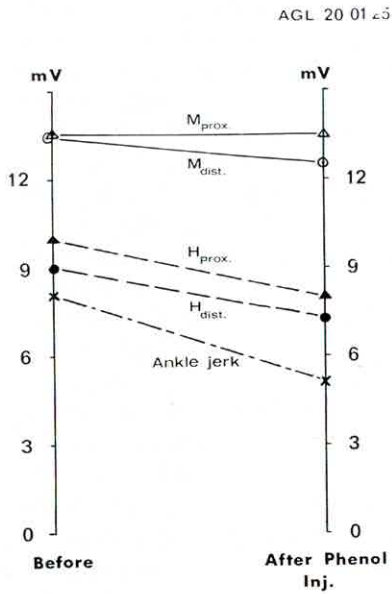

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Figs. 9 and 10. Partial effect of phenol. The clonus was depressed and the ankle jerk slightly reduced. A minor block of afferent group I *a* fibres. One week later spasticity had reappeared completely.

or weeks. The longest duration observed was 3 months.

DISCUSSION AND CONCLUSION

In conformity with other reports, blocking of peripheral nerves with dilute phenol solution was found to be a valuable method in the management of spasticity. Functional improvement was noticed especially in the lower extremities with regard to standing position and ambulation. In the upper extremity the functional gain was less marked. Decrease of spasticity, however, in the flexor muscles especially of the forearm was of great value for prevention from contracture and deformity. Relief from marked flexion positions very often improved the patient's ability for certain activities of daily living such as dressing and undressing. Voluntary movement was not improved to any functional degree in the upper extremity.

Since the follow-up time was relatively short an

evaluation of the duration of the effect is beyond the scope of this study.

The immediate effect of phenol solution on spasticity could not be ascribed to an absolutely selective block of gamma efferent fibres as it has been suggested. Alphamotor and afferent group I *a* fibres were found to be blocked to a varying degree. This was also the case when isotonic phenol solution was used. The results of this study indicate, however, that the longstanding effect on spasticity is mainly due to a persistent depression of gamma fibres causing a decreased sensitivity of the muscle spindles. A persistence of muscle weakness despite absence of demonstrable alphamotor block might be explained by the lack of feed-back from the muscle spindles during voluntary contraction against resistance. Apart from cases who developed paresthesias after injection sensory function was not affected beyond the defect caused by the brain damage.

A comparison of the direct muscle response to distal stimulation before and after phenol injection did not reveal any consistent variation. This finding indicates that there was no appreciable spreading of phenol solution to the part of the nerve where the excitability was tested.

Recent studies have thrown doubt upon the hypothesis of gamma neuron hyperactivity in spasticity (12, 13). However, in any case an intact reflex arc appears to be of fundamental importance for the clinical appearance of spasticity as has been demonstrated by the effect of dorsal root cutting in spastic conditions. Studies are in progress in this laboratory on the effect of peripheral phenol block on anterior horn cell excitability in patients with cerebral and spinal cord lesions respectively.

Re-examination after 3 to 4 weeks revealed denervation potentials in 6 out of 12 blocks, mainly in cases injected with hypotonic phenol solution. The fibrillating activity was not distributed evenly among all muscles belonging to the nerve injected, the usual finding being denervation in one or two muscles only. This might be explained by the position of the injection needle in relation to the topography of the nerve bundles and fibres.

The incidence of paresthesias after phenol injection was relatively high as compared with other authors (14). Our recording of even slight discomfort may partly explain this discrepancy. On the other hand, it might be possible that phenol solution gives rise to neurotoxic agents from the plastic tube, especially if the plastic material is used several times.

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