Radiofrequency cordotomy for the relief of spasticity in decerebrate cats¹

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SUMMARY The effectiveness of radiofrequency (RF) cordotomy of segmental motoneurone pools of the lumbosacral cord in reducing spasticity of decerebrate cats is evaluated. The need for a new form of therapy for clinical spasticity is based upon the limitations of contemporary methods, including surgical and pharmacological techniques. In man, spasticity of spinal origin may be treated effectively by intrathecal administration of hyperbaric phenol solutions. The advantages and disadvantages are described. Difficulty in controlling the lesion is emphasized. Tension and EMG-length curves of the spastic triceps surae muscle in acute and chronic animals show that RF lesions (fixed amperage and duration) of the segmental motoneurone pools reduces myotatic reflex activity in accordance with the number of segments cordotomized. Clinical examination including cinematography and electromyography complement the physiological interpretation. RF lesions of the internuncial pool induce spontaneous EMG discharges. This finding is related to similar observations of EMG discharges and alterations in muscle tone after asphyxiation of the spinal cord.

Neurological disorders often lead to exaggerated muscle tone (spasticity or rigidity) which subsequently may result in disabling patterns of motor behaviour and/or deformity.

Until recently, surgery was the principal method employed to reduce spasticity of spinal—that is, paraplegia—origin. Numerous surgical procedures, such as partial and total neurectomy, anterior and posterior rhizotomy, transverse myelotomy, lumbosacral cordectomy, hemicordotomy, dorsal column cordotomy, myotomy and tenotomy, to name but a few, were performed. However, the results of these surgical procedures were not, for the most part, successful in relieving spasticity. Moreover, they often led to further complications usually associated with major surgery, and frequently contributed to enhanced morbidity.

Recently, the pharmacological approach to the clinical management of spasticity has received wide attention and practice. One group of pharmacological agents are referred to as muscle relaxants.

These are centrally acting, rather than peripherally acting, drugs that at least in the experimental animal suppress brain-stem centres and/or the internuncial pool of the spinal cord. Such central nervous system depressants initially influence the internuncial pool, considering the large number of nuclei and neuronal chains that are available to drug action (Wright, 1954). This alteration in polysynaptic activity often releases its inhibitory effects upon the extensor motoneurons, resulting in enhancement of the monosynaptic reflex (Brooks and Koizumi, 1953). Suppressants of this nature, given in large amounts, may directly inhibit cells of the motoneurone pool, which are fewer in number and, therefore, less likely to be affected by relatively smaller amounts of the drug (Wilson, 1958). It appears that spinalization abolishes tonic inhibition of the internuncial pool leading to enhanced background of motoneurone activity and increased flexor response (Eccles and Lundberg, 1959; Herman, 1964). Thus, by altering internuncial pool excitability, these drugs can apparently suppress this background activity, thereby decreasing the excitability of the motoneurone pool (Wilson, 1958). It may be that the drugs act on the Renshaw system as well and inhibit the spinal cord by removing recurrent facilitation

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or by weakening the latter sufficiently so that it is overcome by recurrent inhibition (Wilson and Talbot, 1960). Since muscle relaxants act primarily upon the interneuronal pool (including the Renshaw system), they should have greater effectiveness in patients with enhanced polysynaptic activity—for example, subjects with spinal cord lesions—(Teasdall, Magladery, and Ramey, 1958; Magladery and Teasdall, 1958; Herman, 1964).

Considerable experience, both clinical and physiological, with various muscle relaxants has substantially reinforced the principle that these drugs are more effective in subjects with spinal cord lesions; such drug induced alterations in myotatic reflex activity are attributed to a reduced flexor response. However, sedation and other side-effects often accompany changes in reflex activity. Further, there is no scientific evidence that muscle relaxants enhance motor behaviour or directly depress feedback from muscle afferent nerves (Pedersen, 1962; Schwab, 1964).

An ideal muscle relaxant is a drug which, when administered orally, gives the desired reduction of exaggerated muscle tone without simultaneous decrease in voluntary power, and without side-effects such as sedation. No such substance is known at this present time.

In patients with spinal cord lesions, the application of phenol in a hyperbaric form has been found to be a very effective agent for reducing exaggerated reflex activity and improving reciprocal behaviour between agonist and antagonist motor groups (Nathan, 1955; Dimitrijević and Nathan, 1967). When used judiciously in selected cases it is considered the optimal method. Phenol solutions have both a short-term (local anaesthetic) and a long-term (destructive) effect on nerve tissue. The destructive effect is non-selective in that nerve fibres of all diameters are destroyed (Smith, 1964; Nathan, Sears, and Smith, 1965). In subjects (usually paraplegics) with severe disabling spasticity of the flexor and adductor muscle groups, intrathecal phenol solutions applied to nerve roots (preferably sensory roots) reduce exaggerated local and general reflex activity. Changes in reflex activity occur as a result of an adequate number of fibres being destroyed. The advantages of this technique are: (1) it is easy and rapid to perform, (2) it causes only minimal discomfort, (3) it can be repeated, (4) it effectively relieves spasticity in proximal motor groups for a prolonged period of time. Bladder and sphincter involvement are major disadvantages. These complications restrict its use to a selected group of patients.

The need to avoid such complications and to provide a more controllable technique for the relief

of spasticity led to this investigation of radiofrequency cordotomy for the treatment of spasticity in decerebrate cats. The introduction of percutaneous cervical radiofrequency cordotomy for the relief of chronic pain has spurred much interest in stereotactic spinal cord surgery (Rosomoff, Carroll, Brown, and Sheptak, 1965; Rosomoff, Sheptak, and Carroll, 1966; Gildenberg, 1969). Both lateral and anterior approaches to the cervical spinal cord have proved successful and a similar means of access to the lumbosacral cord has been found feasible.

The studies described below were carried out to determine the feasibility of utilizing percutaneous radiofrequency cordotomy to the lumbosacral cord for the treatment of spasticity of spinal origin.

METHODS

In this study, 25 cats of both sexes, weighing between 3.0 and 4.0 kg, were decerebrated under ether anaesthesia at the intercollicular level. They were separated into two groups; 11 animals served as acute preparations and 14 as chronic preparations.

ACUTE PREPARATIONS Lumbosacral laminectomies were performed following the development of decerebrate rigidity (approximately three hours after surgery). The dura mater was incised to expose the nerve roots as they emerged from the spinal cord. The roots were identified and followed centrally to the corresponding spinal cord segment. A needle electrode, which served as both a stimulating and a lesion-inducing instrument, was introduced in the area of the ventral horn. The electrode (0.020 in. stainless steel wire, insulated by no. 50 polyethylene with 1 mm bare at the tip) was then inserted through an 18 gauge thin-walled 3.5 in. short bevel spinal needle and connected to a radiofrequency generator (Radionics 3A). The position of the electrode was monitored by stimulating the ventral horn zone with a low intensity square wave electrical impulse of 0.1 msec duration. The induced action potential (EMG) was recorded by means of a pair of silver disc electrodes placed on the surface of the gastrocnemius muscle, amplified by a Teca high gain amplification system, and recorded on a Grass oscillograph or a Tektronix storage oscilloscope. After identification of the appropriate ventral horn(s), a radiofrequency (RF) current of 40 mA was applied through the electrode for 30 to 60 seconds to *only* the right side of the lumbosacral cord. The ventral horn segments on the left were not disturbed. Thus, in these animals, rigidity was maintained in the left lower extremity and reduced in the right lower extremity. In control animals, the electrode was placed into similar segments but current was not applied (sham procedure).

After the RF cordotomy, the tendons of the triceps surae and tibialis anterior muscles were dissected free. The animal was placed in a prone position with the ankle and knee of each lower limb fixed. The tendons were secured to a Satham strain gauge transducer which was connected to a Grass 5P1 amplifier and oscillograph. The transducer was also fixed to a micromanipulator

which was used manually to stretch the muscle 12 to 14 mm at 2 mm increments. After each 2 mm increment, tension and EMG activity were recorded. Sustained stretch after each step was maintained for five seconds. Three recordings of *total* tension- and EMG-length relationships were made for each muscle in both lower extremities. After this procedure, the sciatic nerve was sectioned and the tests repeated. The resultant tension-length curves represented the passive, viscoelastic properties of muscle. The difference between passive tension and total tension indicated the magnitude of active or reflex tension induced by slow stretch of the muscle. Each animal was killed after these procedures; the spinal cord was removed and submitted for histological studies—for example, Nissl stains.

CHRONIC PREPARATIONS Parallel preparations were conducted; laminectomy and RF cordotomy were performed six weeks before decerebration. During this six week period, clinical observations of gait, sensory, motor, and bladder function were documented. Diagnostic electromyography and cinematography served to complement the clinical findings. Tension- and EMG-length findings were determined before and after the decerebration. Histological examinations of the spinal cord were made and compared with the clinical and physiological data obtained.

RESULTS

In both the acute and chronic group of animals, RF lesions were created in selective segments—for example, L7, L7-S1, L6-S1—so that each group had comparable procedures performed. Sham and bilateral cordotomy procedures served as control experiments.

A 40 mA radiofrequency current of 30 to 60 seconds duration produced lesions with coagulative necrosis and localized haemorrhages as seen in histological sections. Nissl stain of the segment showed a reduction of neurones in some areas about the anterior horn, and partial to complete absence of neurones in the anterior horn (Fig. 1). The lesions often extended to the intermediate grey matter and, at times, to some portion of the white matter. The size of the lesions was between 1 to 2 mm in the transverse plane, and 2 to 3 mm in both the longitudinal and anterior-posterior planes. Multiple lesions were usually ellipsoidal in shape in the longitudinal plane. A specimen from the lumbosacral portion of the spinal cord with a bilateral lesion demonstrated an almost completely selective lesion of the anterior horns.

The tension- and EMG-length relationships of the triceps surae muscle were determined three hours after decerebration of the animals in both the acute and chronic preparations. The results consistently showed that tension and EMG induced by stretch of the right triceps surae muscle were less

FIG. 1. Nissl stain of L7 segment of the spinal cord (see text for description).

than those of the left triceps surae muscle. The observed tension and EMG decreased as the number of segments destroyed increased (Figs. 2, 3; Table). The tension-length measurement of the left triceps surae muscle (control) always demonstrated a 'spastic' curve, in which both terminal tension and EMG were marked and in which the tension-length curve shifted to the left. In the sham operations, spastic curves were observed in both lower limbs (Fig. 3). After bilateral cordotomy, tension-length curves of the triceps surae muscles approximated those obtained by stretch of the same muscle after peripheral sciatic nerve section (Fig. 2d).

FIG. 2a. Tension-length curves after RF cordotomy of right L7 segment.

FIG. 2b. Tension-length curves after RF cordotomy of right L6, L7 segments.

FIG. 2c. Tension-length curves after RF cordotomy of right L6, L7, S1 segments.

FIG. 2d. Tension-length curves after bilateral cordotomy (L5, L6, L7, S1); note the marked alteration in tension-length curves and in EMG activity.

FIG. 2. Tension-length curve of left (—) and right (— · —) triceps surae muscles after RF lesion of motor neurone pool segments. The difference between the left or control curve and the right or treated curve represents alteration in total stretch reflex (myotatic plus visco-elastic behaviour of the muscle). The tension-length curves of the left (—) and right (— · —) triceps surae muscles reveal the passive, visco-elastic response of the muscle after complete transection of the sciatic nerve. The degree of myotatic activity of the respective triceps surae muscles is then viewed as the difference between the total and passive response. EMG firing patterns at 12 mm of stretch are shown for the left (A) and right (B) triceps surae muscle.

TABLE
OBSERVATIONS OF CATS AFTER VARIOUS VENTRAL HORN CORDOTOMIES (SIX WEEKS POST-CORDOTOMY)

<i>Cat. no.</i>	<i>Segmental lesion</i>	<i>EMG findings</i>	<i>Clinical findings</i>	<i>T/L measurement</i>
RF 26	Right L5-S1	R. triceps surae + + + + R. anterior tibialis + + + L. side negative	Marked weakness of foot flexor and extensors. Sensation preserved	Right limb tension reduced 92% at 12 mm. Almost flaccid curve
RF 27	Right L7	R. triceps surae + R. anterior tibialis + + L. side negative	Minimal weakness of foot extensor. Other muscle groups normal. Sensation preserved	Right limb tension reduced 28% at 12 mm length
RF 28	Right L6 and L7	R. triceps surae + R. anterior tibialis + + + R. quadriceps + R. hamstring L. side negative	Moderate weakness of knee flexors and foot extensors and flexors. Sensation preserved	Right limb tension reduced 30% at 12 mm
RF 29	Right L3-S1 (unilateral)	R. triceps surae + + + + R. anterior tibialis + + + R. hamstring + + + + R. quadriceps + + + + L. side negative	Flaccid entire right hind limb. Sensation preserved	Right limb—flaccid curve (similar to curve after sciatic nerve section)
RF 30	Right L6, L7	R. triceps surae + + + R. anterior tibialis + + R. quadriceps ± R. hamstring + + L. side negative	Marked weakness of foot extensors and moderate weakness of knee and ankle flexors. Sensation preserved	Right limb tension reduced 40% at 12 mm
RF 31	Right L5, L6, L7	R. triceps surae + + + R. anterior tibialis + + R. hamstring + L. side negative	Marked weakness of foot extensor and moderate weakness of foot flexors and knee extensors. Sensation preserved	Right limb tension reduced 72% at 12 mm
RF 32	Right L6, L7	R. triceps surae + + + R. anterior tibialis + + + R. quadriceps + R. hamstring ± L. side negative	Moderate weakness of the foot extensors and flexors. Sensation preserved	Right limb tension reduced 42% at 12 mm
RF 33	Bilateral L3-S1	L. and R. triceps surae + + + + L. and R. anterior tibialis + + + + L. and R. quadriceps + + + L. and R. hamstrings + + +	Marked weakness of both hind limbs. Sensation preserved. Transient bladder dysfunction	Flaccid bilaterally
RF 34	Sham operation	All muscles negative	No weakness	No change in tension right and left limbs
RF 38	Bilateral L3-S1	All muscles + + + +	Flaccid hind limbs. Sensation preserved. Transient bladder dysfunction	Flaccid bilaterally
RF 39	Right L6, L7, S1	R. triceps surae muscle + + + + R. anterior tibialis + + R. quadriceps + + R. hamstrings + L. side negative	Moderate weakness of foot flexors and extensors and knee flexors. Sensation preserved	Right limb tension reduced 70% at 12 mm
RF 40	Right S1	R. triceps surae + R. anterior tibialis — R. quadriceps — R. hamstrings ± L. side negative	Minimal weakness of foot flexors. Sensation preserved	Right limb tension reduced 20% at 12 mm
RF 41	Right L3-S1 (unilateral)	R. muscle + + + + L. side negative	Flaccid R. hind limb. Intact L. hind limb. Sensation preserved	Right limb—flaccid curve
RF 43	Right L2-L3	R. triceps surae — R. anterior tibialis — R. quadriceps + R. adductors + + L. side negative	Weakness of R. thigh flexors. Sensation preserved	

When the tension-length curves were compared with the number of segments cordotomized, some quantitative values (peak tensions, shift of the tension-length curve) could be established with respect to alterations in stretch reflex activity. A lesion of one segment, either L7 or S1, usually diminished reflex activity of the triceps surae muscle

to a minimal degree—that is, 20-28%. Cordotomy of two segments (L6, L7) diminished the response to stretch further (30 to 42%) while lesions of three and four segments (L6-S1, L5-S1) showed considerable reduction in reflex activity (72 to 92%).

The magnitude in fall in active tension as a function of stretch was directly associated with a reduc-

FIG. 3. Sham operation; note the relatively minimal alteration in the tension-length curves of the left (—) and right (---) triceps surae muscles and of the EMG response.

tion in EMG discharge. The voltage of electrical activity of the triceps surae muscle of the cordotomized hindlimb was considerably less than that of the spastic, non-treated hindlimb, particularly in lesions of two or more segments (Figs. 2b, c). An EMG response was barely discernible in the completely cordotomized hindlimb (Fig. 2d).

In chronic animals, the disturbance in gait performance corresponded with the segments denervated (Table). Destruction of any one segment of the lumbosacral spinal cord resulted in clinical weakness in muscles related to the segmental distribution. One animal with an upper lumbar (L2-L3) lesion was observed to have weakness of the thigh flexors—for example, iliopsoas muscle. Sensation to pinprick remained intact even in the bilaterally denervated animals (Table). In the two chronic animals with bilateral denervation, bladder incontinence was noted for only one week. In both acute and chronic preparations extensive unilateral lesions of the right lumbosacral cord produced temporary relaxation in both lower extremities; however, rigidity of the left lower limb returned after 15 to 90 minutes. There was electromyographic evidence of denervation in muscles supplied by the cordotomized segments. The degree of fibrillation activity was directly correlated with the extent of the lesion.

Electromyographic records of the resting left tibialis anterior and left triceps surae muscles in the chronic preparations were usually silent. This electrical silence was also observed after a sham operation of the lumbosacral spinal cord. When the RF lesion was confined to the motoneurone pool (confirmed by histological sections) of L6, L7, or S1 segments fibrillation potentials in the triceps surae and tibialis anterior muscles were noted. However, in two animals, when the lesion was inappropriately placed in the interneuronal area with sparing of most motoneurons, sustained spontaneous action potentials appeared in both muscle groups (Fig. 4). These action potentials were continually observed during routine electromyographic examination. They were not influenced by muscle stretch. After decerebration, RF lesions were applied again to the appropriate ventral horn segments in order to suppress these spontaneous EMG discharges.

DISCUSSION

Apart from the effects of feedback from muscle and other peripheral receptors, excitability of the spinal motoneurone pool depends upon the integration of suprasegmental and intersegmental pathways upon the anterior horn cells. Suprasegmental control of spinal reflexes varies according to the anatomical level. This control of motoneurons is most likely mediated through the corticospinal and brain-stem reticular systems which apparently influence the

FIG. 4. Spontaneous EMG action potentials observed after a RF lesion of the interneuronal pool of L7 segment.

interneuronal processes of the spinal cord (Magoun and Rhines, 1947; French, 1960). Lesions involving these descending systems withdraw the inhibitory effects of the interneuronal link between the peripheral afferent (IB, II, and III fibres) and the motor nuclei of the ventral horn (Eccles and Lundberg, 1959; Kuno and Perl, 1960). The intersegmental system, consisting of the ascending and descending propriospinal neurones (long and short) interconnects the spinal segments and enables the cord to integrate supraspinal and intraspinal influences, particularly at the interneuronal level (Gernandt and Gilman, 1960; Gernandt and Shimamura, 1961).

Several investigations dealing with spinal cord lesions in man suggest that segmental interneuronal activity is altered (Magladery and Teasdall, 1958; Herman, 1964, 1970). It would appear that the response of the motoneuronal pool fluctuates with the level of external influences of background activity, such as the threshold of the interneurons (Hunt and Perl, 1960). Interneuronal excitability results from withdrawal of tonic inhibition of the interneurons and from the development of recurrent facilitation in the segmental axon collateral system (Wilson and Burgess, 1962). As a result, the excitability level of both extensor and flexor motor discharges are enhanced, although flexor activity is usually most pronounced. While in some animals most descending systems influence the ventral horn cells through the internuncials, in monkey, and probably in man, the corticospinal tracts also terminate directly upon these motor cells (Lloyd, 1941; Kuypers, 1958, 1960; Landgren, Phillips and Porter, 1962). Therefore, it is possible that, in spinal cord lesions, the inhibition of motor discharges may be removed directly as well as indirectly. The effect of spinalization upon the propriospinal system also modifies the excitability between neighbouring segments and interlimb reflexes. While these connections (interneuronal) are normally depressed by the propriospinal system, they are released by spinal cord transection, thus contributing to a spread of enhanced facilitation throughout the cord (Gernandt and Gilman, 1960; Gernandt and Shimamura, 1961).

The medical management of spasticity of spinal origin must, therefore, be orientated towards attenuating motor neurone pool discharges either directly or indirectly. Indirect reduction of motor neurone pool excitability can be accomplished by suppressing background interneuronal activity—that is, by direct action on the interneuronal pool and/or by altering brain-stem reticular discharges—and by restoring the balance between recurrent facilitation and recurrent inhibition (Wilson and Burgess, 1962). Centrally acting muscle relaxants most probably function in this manner; however,

their effect is not usually pronounced in patients with severe disabling spasticity in which flexor or extensor reflex behaviour is markedly sustained. When higher concentrations of these drugs are administered, anterior horn cells are suppressed directly. At such doses, however, side-effects limit their usefulness.

The enhanced level of segmental and intersegmental background activity is also maintained by peripheral afferent discharges. Spinal cord lesions suppress the inhibition of interneurons which transmit impulses segmentally from group II and group III fibres (Eccles and Lundberg, 1959). Further, after spinal cord transection, there is facilitation or removal facilitation of intersegmental and interlimb reflexes, which are activated by group I afferent fibres, leading to enhanced activity throughout the spinal cord (Gernandt and Shimamura, 1961). This altered response of the spinal cord to peripheral afferent input leads to changes in the reciprocal behaviour between muscle agonists and antagonists during stretch and during voluntary activity. The lack of reciprocal inhibition is a feature of the spinal form of spasticity as contrasted with the hemiplegic form of spasticity in which reciprocal inhibition is marked (Herman, 1970). The effect of afferent discharges on spinal cord reflexes can be modified by the intrathecal administration of hyperbaric solutions of phenol. Such solutions of phenol can lead to a non-selective, partial destruction of a number of sensory roots (Nathan, 1966). By reducing sensory inflow, background segmental and intersegmental excitation is reduced, thus decreasing motor neurone pool discharges. Alterations of these external influences on spinal cord activity often improves agonist-antagonist motor behaviour with considerable increase in reciprocal inhibition (Dimitrijević and Nathan, 1967; Herman, 1970). Additionally, the decrease in sensory input also reduces the level of autonomic hyperreflexia which may be marked in subjects with high cord lesions.

Partial destruction of the motor root with phenol is not such an effective form of treatment. It usually does not improve reciprocal behaviour between agonist and antagonist and does not sufficiently suppress peripheral afferent support of the interneuronal systems.

Peripheral neurolysis of mixed peripheral nerves with aqueous phenol solutions is not generally utilized in severe spasticity of spinal origin unless a considerable number of mixed peripheral nerves are blocked—for example, bilateral obturator and tibial nerves. Utilizing the latter technique, some investigators have successfully treated a number of patients with a moderate degree of spasticity; how-

ever, this form of treatment is not generally recommended as a routine measure unless reduction of reflex activity of a specific motor group is desired (Morrison and Herman, 1967).

Thus, while intrathecal phenol injection is currently the preferred method of treatment for spasticity of spinal origin, there are a number of limitations which restrict its application to a select group of patients (see introduction). These difficulties are primarily due to problems in controlling both the degree of neuronal damage of any one root lesion and the number of segments involved.

The technique of radiofrequency cordotomy of the motor pool is capable of overcoming some of the objections attributed to intrathecal phenol administration. In decerebrate cats, a review of the myotatic reflex response—that is, tension-length curves, EMG activity, etc.—of the spastic triceps surae muscle and of the clinical examination, before and after RF cordotomy of the lumbosacral segments, reveals that regulation of motor outflow can be better achieved by means of an RF lesion with respect to the control of the degree of destruction of any one motor neurone pool and to the ability to select the number of segments necessary to reduce myotatic reflex activity. Additionally, the RF cordotomy procedure will not interfere with sacral reflex pathways of the viscerosomatic system. Despite the apparent success with RF cordotomy of the motor neurone pool, an RF cordotomy which reduces sensory inflow to segmental and neighbouring segmental motor neurone pools should be a more effective technique in suppressing exaggerated reflex activity (for reasons described above). These experiments are presently in progress.

EMG examination of the lower limbs was utilized to describe the presence and degree of denervation activity and the magnitude of stretch induced reflex activity. Spontaneous action potentials did not appear when the muscle was at rest or at a slack position. However, in two chronic animals, when an RF lesion was created in the internuncial pool rather than the motor neurone pool, spontaneous sustained EMG discharges appeared in the resting triceps surae and tibial anterior muscles. These discharges were not influenced by muscle stretch. Similar findings were observed experimentally following asphyxia of the spinal cord.

Gelfan and Tarlov (1959) developed a concept of asphyxial rigidity. This rigidity would be caused by spontaneous discharge of motoneurons made hypersensitive by the destruction of interneurons. The loss of interneurons induces a change in the subliminal fringe of the motoneuronal pool, thus creating high excitability of motor neurone discharge. According to Van Harreveld (1964), the

discharge activity from the anterior horn cells can be viewed as a result of denervation hypersensitivity. Van Harreveld demonstrated that, in dogs, spinal cord asphyxia maintained for 30 to 35 minutes induces extensor tone in the hind limbs. The asphyxia is accompanied by marked destruction of the interneurons. In Gelfan and Tarlov's (1962) preparations (asphyxiation of the cord by clamping the thoracic aorta), the interneuronal destruction was not accompanied by destruction of large motor cells. It was postulated that the smaller cells of the interneuronal pool (and possibly the gamma efferents) are more sensitive to the lack of oxygen than the larger alpha cells of the motor neurone pool.

These changes lead to a clinical rigidity of the limb in both animal and man. This form of rigidity is not supported by muscle stretch nor markedly influenced by deafferentation and thus has been classified as an alpha type rigidity resulting from release or disinhibition of large motor cells (Wright, 1964).

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