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THE NATURE OF THE RIGIDITY CAUSED BY SPINAL CORD ASPHYXIATION

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High extensor tone of the hind limbs has been observed by several authors following asphyxiation of the spinal cord for appropriate periods of time (Tureen, 1936; Häggqvist, 1938; Van Harreveld & Marmont, 1939; Rexed, 1940; Kabat & Knapp, 1944; Hochberg & Hydén, 1949; Krogh, 1950; Kosman, Hill & Snider, 1951; Gelfan & Tarlov, 1959). In cats in which the spinal cord had been asphyxiated for 30–35 min by increasing the pressure in the dural cavity two main periods of rigidity were distinguished, in addition to a very slight and fleeting 'initial' tone which developed 5–20 min after the end of the asphyxiation (Van Harreveld & Marmont, 1939; Van Harreveld, 1943, 1944). The first of the main periods of rigidity, the 'secondary' tone, developed from one to several hours after asphyxiation. Simultaneously tendon reflexes returned and somewhat later a small flexion reflex was often observed. The legs remained extended for 48–72 hr and then lost some of their tone, or even became completely flaccid. However, 6–10 days after asphyxiation tone increased again, or redeveloped, often leading to pronounced and permanent rigidity of the legs. This tone will be called the 'late' tone. After longer asphyxiations (45–60 min) an initial and secondary tone developed in the hind legs, but as a rule no late tone. In these animals a late tone was usually observed in the muscles of the tail; nocuous stimuli often elicited tail movements. The evaluation of the late tone was hampered by the development of a permanent shortening of the rigid muscles. This 'myostatic contracture' was first described in asphyxiated preparations by Gelfan & Tarlov (1959), who identified it with a similar shortening observed in muscles fixed at a given length in a plaster cast, after tenotomy and after localized poisoning with tetanus toxin.

Two mechanisms have been proposed to explain the rigidity developing after cord asphyxiation. Van Harreveld & Marmont (1939) suggested that the rigidity is due to over-excitability of the myotatic reflex, that is of the normal mechanism of tone. The myotatic origin of the tone was demonstrated by the effect of stretch and relaxation on the electromyogram of the muscle (Van Harreveld, 1943, 1944; Krogh, 1950), and by unilateral

transection of dorsal roots which caused a decrease or disappearance of the rigidity on the de-afferented side (Van Harreveld & Marmont, 1939).

Gelfan & Tarlov (1959), who asphyxiated the cord of dogs by clamping the thoracic aorta, postulated that the denervation of the motoneurones caused by the extensive destruction of interneurones observed in rigid preparations (Van Harreveld & Marmont, 1939; Kabat & Knapp, 1944; Gelfan & Tarlov, 1959, 1962; Tarlov & Gelfan, 1960; Van Harreveld & Schadé, 1962) would produce a marked increase in excitability of the motor cells, leading to spontaneous firing. This concept was based mainly on the results of rhizotomy of dorsal roots which, although causing an initial loss of rigidity, did not abolish it permanently. In the present experiments these two concepts of the asphyxial rigidity have been examined in preparations of cats showing the late tone in the hind limbs after 30–35 min of cord asphyxiation.

METHODS

The spinal cord of the cat was asphyxiated by increasing the pressure in the dural cavity above the blood pressure. To limit asphyxiation to the lumbosacral segments the dura was ligated at T 10. The cord was severed by the ligature. The day after this operation a hypodermic needle was introduced into the isolated dural sac, and Ringer's solution was forced through the needle under a pressure of 20 cm of mercury. Since damage to the spinal cord due to lack of O_2 is highly temperature-dependent (Van Harreveld & Tyler, 1944) the room was kept at 38° C and the Ringer's solution thus entered the dural sac at this temperature. In addition, an attempt was made to keep the animal's body temperature at this level. When the cat's temperature deviated from 38° C we tried to produce the desired degree of cord damage by slightly adjusting the duration of asphyxiation. The preparations needed considerable post-operative care. Retention of urine was especially troublesome during the first weeks, necessitating frequent emptying of the bladder. Since this is more easily accomplished in female cats, these were preferred in this investigation. To counteract the development of myostatic contractures and stiffening of the joints the hind legs were flexed as far as possible, at least once a day. Although this seemed to retard and alleviate the contractures, it did not prevent their development.

All surgical procedures were carried out under Nembutal (pentobarbitone; Abbott Laboratories) narcosis and with aseptic precautions. Suitable prophylactic doses of penicillin were administered for 3 days afterwards. Electromyograms were led off with steel needles insulated up to the tip, standard recording equipment being used.

Histological preparations were made from the asphyxiated cords and ventral roots. The cords were fixed in 95% alcohol, embedded in paraffin, sectioned and stained with gallocyanin. The roots were fixed and stained with osmic acid. The fibres were counted and measured in a number of ventral roots. These roots were photographed, and on an enlargement small fibre bundles were marked off. The counting and measuring was done by drawing outlines of the fibres in each bundle with the aid of a Zeiss camera lucida at a total magnification of 3000 times. The means of the greatest and smallest dimensions of the cross-sections of the fibres were used.

RESULTS

Function and anatomy of asphyxiated preparations

In twenty-six preparations electromyograms were recorded at regular intervals after asphyxiation of the cord for 30–35 min. Three of the pre-

parations, showing different degrees of cord damage, were studied intensively both physiologically and histologically. The clinical features at the end of a 3-month observation period are shown in Table 1. Preparation 1, which had sustained the least damage, showed no extensor rigidity, but exhibited brisk tendon reflexes of large amplitude. Nocuous stimulation caused marked flexion accompanied by crossed extension. In preparation 2 secondary tone developed which decreased markedly 3 days after asphyxiation, but did not disappear altogether. Five days later the tone started to

TABLE 1

	Leg			Tail		
No.	Extensor tone	Tendon reflexes	Flexion reflex	Tone	Tail reflex	No. of fibres in ventral L 7
Normal						5130
1	−	++	++	−	+	3829
2	++	+	−	−	+	1803
3	+	+	−	+	+	981

The − sign indicates absence of tone or reflexes, the + sign moderate tone and small reflex contractions, the ++ sign indicates pronounced rigidity or reflex contractions of large amplitude.

increase again, and 2 weeks after asphyxiation the rigidity in the hind legs was so pronounced that the leg could be flexed only by exerting considerable force. When held in certain positions clonus was present. Tendon reflexes could be demonstrated, although this was hampered by the extended position of the leg. No flexion reflex was found. This was a preparation with typical late tone. In such preparations myostatic contractures develop which make it increasingly difficult to evaluate active tone. Indeed, when after 3 months preparation 2 was killed, the legs relaxed quite incompletely after death. In preparation 3 the spinal cord was the most seriously damaged. Extensor tone was not very marked 3 months after asphyxiation. Myostatic contracture probably interfered with the evaluation of active tone. However, on bending the knee the resistance was not smooth but clonic contractions were felt. Small but brisk knee jerks were present, but no flexion reflex. This cat showed tone in the tail, and a tail movement on nocuous stimulation.

The ventral roots of L 7 of these three preparations and of a normal cat were fixed in osmic acid. In a root of each animal all the nerve fibres were counted and measured. In L 7 ventral root of the normal cat 5130 fibres were found, which is in agreement with an estimate of the motor neurones in the cat's 7th lumbar segment made previously (Schadé & Van Harreveld, 1961). The histogram of the fibre diameters of this root is shown in Fig. 1*A*. It shows the familiar bimodal shape first described by Eccles & Sherrington (1930) in de-afferented motor nerves. The percentage of large

α fibres (greater than 9 μ) is 63 % and that of the γ fibres 37 %. The numbers of fibres found in L7 ventral roots of the three asphyxiated preparations are given in Table 1. They range from 3829 (75 % of the normal root) in preparation 1, to 981 (19 %) in preparation 3. In L7 ventral root of preparation 2, which exhibited the pronounced late tone, the number of fibres was 1803 (35 %). The clinical features suggested that the asphyxial damage increased from preparation 1 to 3. The fibre counts support this conclusion, since the destruction of motor cells, as indicated by the loss of

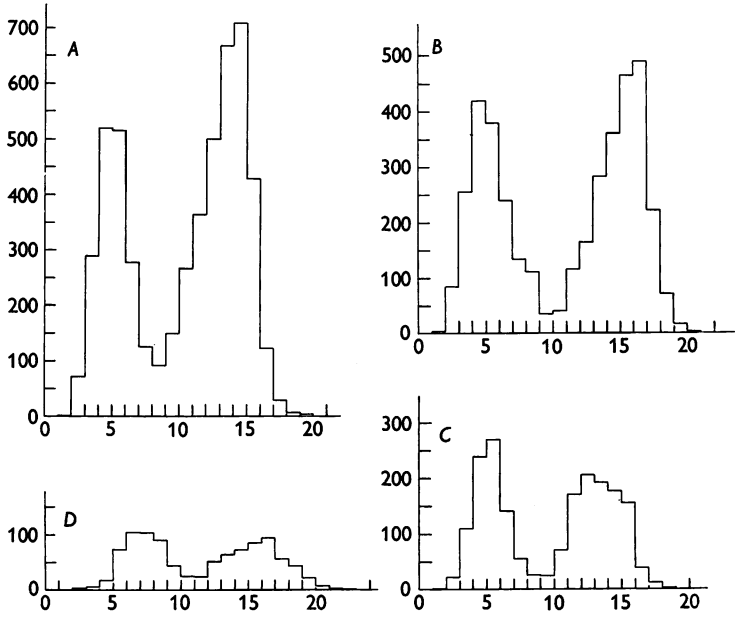

Fig. 1. Fibre diameter histograms of the ventral roots of L7 of a normal cat (*A*) and of the asphyxiated preparations 1 (*B*), 2 (*C*) and 3 (*D*), the clinical features of which are given in Table 1. On the abscissa the fibre diameters are plotted in micra, on the ordinate the number of fibres in each class.

fibres in the ventral roots, increases in severity in the same order. Preparations of the spinal cord showed a more severe loss of large motor cells in preparations 2 and 3 than in 1. It has been shown previously that, with the method of asphyxiation used, no fibre loss is sustained in the dorsal roots (Van Harreveld, 1940).

Figure 1 *B*, *C* and *D* shows the histograms of the diameters of the fibres in the ventral roots of preparations 1, 2 and 3. In preparation 1 the α fibres (greater than 10 μ) comprise 58 % of the total, in 2 and 3 this is 51 and 53 % respectively. It is remarkable that, notwithstanding the differences in the severity of motor cell destruction in these preparations,

the appearance of the histograms, and the proportions of large and small motor fibres are very similar to those of the normal root.

Electromyograms from the quadriceps muscles were recorded at weekly intervals from all three preparations. Figure 2 shows typical records made during bending of the knee, and thus stretching the quadriceps muscle, obtained in the last weeks of the period of observation (3 months). In preparation 1 (Fig. 2*A*), which had no observable tone, such a stretch produced a low-voltage activity in the myogram. Stretch caused more pronounced activity in the quadriceps of preparation 2 (Fig. 2*B*). The activity was greater during the active stretch than during the period in which the muscle was kept in the extended position. This was a consistent

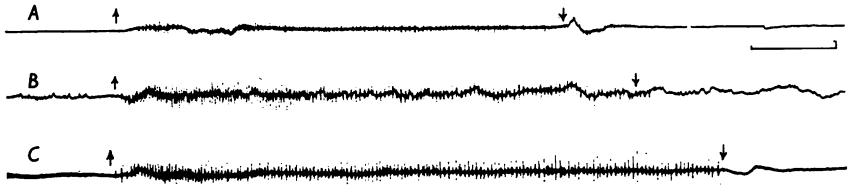

Fig. 2. Electromyograms from the quadriceps muscles of preparations 1 (*A*), 2 (*B*) and 3 (*C*), the clinical features of which are given in Table 1. At the arrow pointing up, the knee is bent from the stretched position. At the arrow pointing down, the knee is stretched again. The horizontal calibration line indicates 1 sec, the vertical 100 μV.

finding. In preparation 3 (Fig. 2*C*) stretch again caused considerable activity. The activity was greater the more the knee of the rigid preparations was flexed. The myograms showed little activity before bending and after the leg was returned to the extended position, indicating that in the extended position the extensor muscles of these preparations were not in a state of contraction. However, in some preparations with pronounced rigidity somewhat more spontaneous activity in the extended position was seen (Fig. 3). Bending of the knee always greatly increased this activity. In all preparations with late tone this effect of stretch of a muscle on its electromyogram has been observed.

Unilaterally de-afferented preparations

In 5 preparations the dorsal roots were severed on one side from L 3 to S 2 after the late tone had developed. This resulted in two instances in an almost complete loss of rigidity on the de-afferented side. Myostatic contractures present in the other preparations made it difficult to evaluate the loss of active tone, but some relaxation was always observed. On the normally innervated side the pre-operative rigidity was found after recovery from narcosis. Tone increased or developed on the de-afferented side in the

ensuing days or weeks, and in all preparations a moderate to rather marked rigidity was present at the end of the period of observation (about 2 months after severing the roots). The rigidity in the de-afferented leg was always less pronounced than on the innervated side, however. Since part of this rigidity was due to a myostatic contracture, it was difficult to evaluate the actual tone at that time. As was to be expected no reflex activity (tendon reflexes or flexion reflex) could be elicited on the de-afferented side. A consistent observation was that bending of the knee on the non-de-afferented side caused stretching of the contralateral leg. This reaction was slight in some preparations, while in others it was quite pronounced.

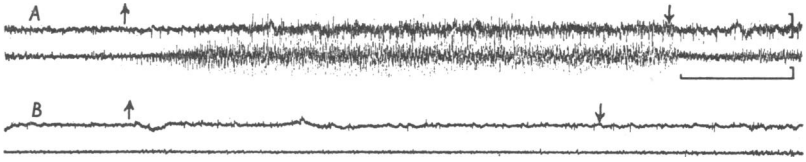

Fig. 3. Electromyograms of the quadriceps muscles on the operated (upper traces) and normally innervated (lower traces) side of a unilaterally de-afferented preparation. In record *A* the knee on the innervated side is bent at the arrow pointing upward, and extended again at the arrow pointing downward. In record *B*, the knee bending and extending are repeated on the de-afferented side. The horizontal calibration line indicates 1 sec, the vertical lines 100 μV for the records of the de-afferented (upper line) and normally innervated sides. Note the difference in amplification used for the two records.

Electromyograms from the quadriceps muscles were in agreement with the observations described above. Figure 3 *A* and *B* shows myograms led off from the quadriceps muscles on the de-afferented (upper traces) and innervated (lower traces) sides of a preparation in which the dorsal roots had been severed unilaterally. Record *A* shows that bending the knee on the innervated side not only produced considerable activity in the ipsi-lateral muscle, but also caused appreciable activity in the contra-lateral, de-afferented muscle. Bending of the knee on the de-afferented side was without effect as shown in record *B*. No flexion reflex could be elicited in this preparation.

In some preparations the crossed reaction was well developed from the first day after severing the roots. In others it was slight at that time, but developed during the ensuing days or weeks.

A crossed contraction was sometimes also observed in non-de-afferented preparations with late tone. It was never pronounced, however. Electromyograms confirmed the presence in these preparations of contractions in the contralateral quadriceps muscle caused by crossed knee-bending.

Bilaterally de-afferented preparations

In seven preparations showing the late tone the cord was de-afferented bilaterally by severing the dorsal roots from L3 to S2. Since an afferent influx could still reach these segments through the spinal nerves cranial and caudal to the de-afferented section, the cord was severed between L2 and L3, and the terminal conus caudal to S1 was removed. All these procedures were not applied in one session, the de-afferentiation and the operations on the cord being performed with an interval of several days to one week to allow development of collaterals. Theoretically the entire afferent influx is excluded in this way from segments L3 to S1. This operation caused an immediate decrease, and sometimes a complete disappearance, of the rigidity, apparently depending on differences in development of the myostatic contracture before de-afferentiation.

In five preparations rigidity reappeared or increased again after a period of several days to 3 weeks. In four of these preparations an enhancement of the activity in the myogram could be demonstrated by bending the legs, suggesting that the de-afferentiation had not been complete. This could indeed be shown. In two preparations in which the rigidity developed within a few days it was possible to perform an autopsy before extensive scar tissue had developed around the spinal cord. In both instances a small dorsal rootlet from L7 was found to be intact on both sides. In these rootlets, which were examined histologically, apparently intact nerve fibres were found. In a third preparation in which the tone developed 2 weeks after de-afferentiation another course was followed, since it was expected that scar tissue would make the dissection of a small intact rootlet difficult. The central stumps of the severed genitofemoral, cutaneous femoris, obturator and sciatic nerves were stimulated on the right as well as on the left side. The femoral nerves were left intact and electromyograms were led off from the quadriceps muscles. Stimulation of the central stump of the right obturator and sciatic nerves elicited muscle action potentials in this preparation, proving that the de-afferentiation had not been complete. The two other cats succumbed unexpectedly at a time at which anatomical examination was not feasible.

Two preparations seemed to have been de-afferented completely. One of these preparations was operated upon about 3 weeks after the development of the late tone. Since a myostatic contracture had developed at that time, the relaxation after the operation was incomplete. This cat was observed for 6 weeks after de-afferentiation. The second preparation was de-afferented as soon as the late tone had developed. This preparation was flaccid after the operation and remained in this state for the entire period of observation (2 months). Reflexes could not be elicited in either of the

animals at any time. However, sudden movements made by the cat showing some rigidity occasionally caused contractions in the hind legs. Also when this animal was made to hang by the front legs movements were observed in the hind legs.

In both cats the electrograms of the quadriceps muscles showed little activity although they were not entirely silent (Figs. 4*A* and 5*A*). Bending of the knees was without effect on the electromyograms. However, maximal backward stretching of the hind legs in the hip joint caused in both preparations activity in the quadriceps muscles (Fig. 4*B*). Forward

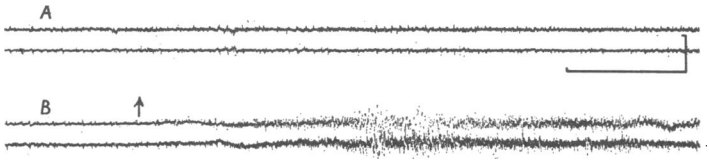

Fig. 4. Electromyograms of the quadriceps muscles of a completely bilaterally de-afferented preparation (which showed a myostatic contracture). Record *A* shows the spontaneous activity. In record *B* the hind legs are maximally stretched backwards at the arrow. The horizontal calibration line indicates 1 sec, the vertical line 100 μV.

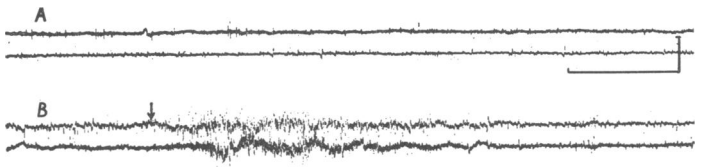

Fig. 5. Electromyogram of the quadriceps muscles of a completely bilaterally de-afferentiated preparation, in which the legs were flaccid. Record *A* shows the spontaneous activity. In record *B* pressure is exerted on the cord at the arrow. The horizontal calibration line indicates 1 sec, the vertical line 100 μV.

bending had no effect. Direct pressure on the cord, which was made possible by the removal of the vertebral arcs for the de-afferentiation, also elicited activity in the quadriceps muscles of both cats (Fig. 5*B*). It would seem possible that maximal backward stretching of the leg (and active movements of the animal showing rigidity) also stimulate the cord directly, either by movements of the lumbar spinal column or by traction on the scar tissue surrounding the cord. Electrical stimulation of the central stump of the peripheral nerves mentioned above did not cause any activity in the electrograms of the quadriceps muscles of the two cats, supporting the conclusion that the de-afferentiation had been complete in both experiments. Stimulation of the peripheral stumps of the femoral, obturator and sciatic nerves caused appreciable contractions in the

relevant muscles, showing that a fair number of motor fibres (and consequently of motor cells in the cord) had survived the asphyxiation. This was confirmed in histological preparations of the ventral roots of L7 which contained in both animals appreciable numbers of apparently normal fibres. Preparations of the cords taken from L7 showed the severe cell destruction described previously in preparations made rigid by asphyxiation for 30–35 min with the method used in the present investigation (Van Harreveld & Schadé, 1962). However, intact motor cells were found in each section of the cords of the two cats.

DISCUSSION

In the present experiments stretching the quadriceps muscles in rigid preparations consistently caused greatly enhanced activity in the electromyograms. In some preparations there was very little activity when the knee was kept in the extended position; in others the spontaneous activity was more marked. The latter could very well have been due to myotatic reflex activity also, especially since it was always enhanced by stretch of the muscle. These findings are in good agreement with experiments in which monosynaptic reflex action potentials were led off from ventral roots of rigid preparations, and in which evidence for the over-excitability of this reflex activity was found (Gelfan & Tarlov, 1959; Van Harreveld, 1962). The hind legs of a preparation showing the late tone thus seems to be kept in the extended position because deviations from this attitude cause reflex contractions which counteract such movements. Eventually the development of a myostatic contracture will cause an anatomical fixation of the leg in the extended position.

Gelfan & Tarlov (1959) suggested that the severe destruction of interneurones observed in rigid preparations causes a partial denervation of the motoneurones, resulting in an increase of their excitability. This is supported by the observations of Drake & Stavraky (1948) and Teasdall & Stavraky (1953) who demonstrated supersensitivity of motoneurones after the severing of dorsal roots, which deprives the motor cells of their monosynaptic innervation. Such an increased excitability of the motor cells could well account for the enhancement of the myotatic activity which was found to be the principal mechanism of the extensor tone described in the present investigation. The initial and secondary tones, which develop within minutes or hours respectively after asphyxiation, cannot be explained by denervation over-excitability of the motoneurones, since the latter takes considerable time to develop (Teasdall & Stavraky, 1953).

The quadriceps contractions caused by bending the contralateral knee, observed in unilaterally de-afferented preparations, resemble Phillipson's

reflex. In decerebrate preparations Sherrington (1909) considered this reaction as a manifestation of crossed extension; the forceful bending of the contralateral knee acting as a nocuous stimulus. Although a large percentage of the interneurones is destroyed in these preparations (Van Harreveld & Schadé, 1962), it would seem possible that a sufficient number remains functional to activate over-excitable motoneurones on the crossed side. The crossed reaction was observed in preparations in which the flexion reflex on nocuous stimulation was very slight or absent. More severe asphyxial damage of the flexor than of the extensor motoneurones, as postulated by Krogh (1950), could explain this finding.

The immediate result of bilateral de-afferentiation was a relaxation of the rigidity as far as the usually present myostatic contracture would allow. In preparations in which the de-afferentiation had not been complete, rigidity re-appeared or increased again after days or weeks. In the one preparation in which complete de-afferentiation had apparently been accomplished before development of a myostatic contracture the hind legs remained flaccid during the period of observation (2 months). The electromyogram of this preparation and of another completely de-afferented cat (with a myostatic contracture) showed little spontaneous activity. In both these preparations anatomical and physiological evidence proved the existence and function of a fair number of motoneurones. It would appear from these findings that the exclusion of the entire afferent influx by de-afferentiation and isolation of the relevant spinal segments can indeed prevent the development of rigidity. Complete de-afferentiation, however, proved to be difficult, and it seems that even a small sensory input is sufficient to cause the redevelopment of rigidity. An immediate relaxation of the extensor tone on the operated side was also observed in the unilaterally de-afferented preparations, followed by the redevelopment of rigidity. A sensory influx may have reached the de-afferented side in these preparations from the normally innervated contralateral side (which is supported by the observation of a contraction in the de-afferented leg on bending the contralateral knee) or from ipsilateral dorsal roots caudal and cranial to the de-afferented region of the cord. Furthermore, it is possible that in some of these experiments the de-afferentiation had not been complete.

Gelfan & Tarlov (1959) attributed the immediate relaxation and eventual return of rigidity after de-afferentiation to recovery of the spinal cord from damage incurred during the operation. The observation that unilateral de-afferentiation does not materially affect the rigidity on the innervated side does not support this explanation. Drake & Stavraky (1948) and Teasdall & Stavraky (1953) showed that de-afferentiation enhances the excitability of motoneurones. Such an effect may also

develop in the asphyxiated spinal cords, in which the excitability of the motoneurones had already been increased by the denervation caused by destruction of interneurones. The slow development of rigidity after incomplete bilateral de-afferentiation, and the sometimes tardy enhancement of the crossed quadriceps contraction after unilateral operations, agree with such a concept, since it takes time for the denervation over-excitability to develop (Teasdall & Stavraky, 1953). The more complete denervation of the motoneurones in asphyxiated cords after severing the dorsal roots may enhance the excitability of the motor cells to such a degree that even a small sensory input is capable of maintaining a measure of rigidity.

It is conceivable that an ultimate increase in excitability results in spontaneous discharges of the motoneurones. One could consider the slight, spontaneous activity observed in the myograms of the quadriceps muscles of completely de-afferented preparations as evidence for such discharges. However, some of this activity could be due to denervation fibrillation of the units of which the motoneurone has been destroyed. Furthermore, since the exposed cord in these preparations could be readily stimulated mechanically, the spontaneous activity might also be due to movements of the animal or to pressure of the scar tissue surrounding the cord.

It has been suggested that small nerve cells are more susceptible to asphyxial damage than large ones (Van Harreveld & Marmont, 1939). The histograms of the fibre diameters in the ventral roots of L7 of normal and asphyxiated preparations do not support this concept. In the asphyxiated preparations the surviving motor fibres, expressed as a percentage of the number of fibres in the root of the normal cat, varied from 75 to 19 %. The histograms, however, were similar in appearance to one another and resembled that of the non-asphyxiated preparation. If the sensitivity to lack of O_2 were based on cell size, and assuming that the dimensions of fibres and their cells of origin are correlated, then one would expect a larger destruction among the group of γ afferents than among the α fibres. This, however, is not the case. The sensitivity to O_2 lack of large and small motoneurones therefore does not seem to differ materially. Since asphyxiation causes a noticeably larger destruction of the small neurones than of large motor cells in the cord (Van Harreveld & Schadé, 1962) the conclusion seems warranted that interneurones are more susceptible to asphyxiation than motoneurones.

SUMMARY

1. High extensor tone was observed in the hind legs of cats after asphyxiation of the lumbosacral spinal cord for 30–35 min. During the first weeks three periods of tone developed in succession, of which the last

one was permanent. The nature of the latter was investigated. The evaluation of this tone was hampered by the development of a shortening of the extensor muscles (myostatic contracture).

2. In all instances stretching the quadriceps muscles of rigid preparations caused marked activity in the electromyogram. In the extended position of the knee the muscles were in many instances almost silent, but showed in other preparations moderate activity.

3. Bilateral de-afferentiation of the cord caused a relaxation of the rigidity. In a preparation in which complete de-afferentiation was accomplished before the development of a myostatic contracture, the legs remained flaccid. In preparations in which de-afferentiation had not been complete, rigidity developed again after the initial relaxation.

4. In unilateral de-afferented preparations bending the knee on the innervated side caused contraction in the contralateral de-afferented quadriceps muscle.

5. Determination of the numbers of fibres in the ventral roots of L7 in three asphyxiated and one normal preparation showed that in the two rigid cats a considerable loss (65 and 81 %) of the fibres had occurred. The histograms of the fibre diameter in normal and asphyxiated preparations were remarkably alike, indicating that similar losses occur among the α fibres and the γ efferents.

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