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ELECTRIC SHOCK *

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The English, American, German, and French literature contains many isolated reports and abstracts of accidents caused by electricity. Great emphasis is placed on the bizarre effects and playful vagaries of electrical current, less is said about treatment and least about prevention.

LITERATURE

No attempt will be made to give a complete review of the literature dealing with electric shock; that is already voluminous and has been clearly described in a recent paper by Jaffé,[1] who in addition gives an excellent bibliography.

The macroscopic and microscopic observations on the nervous system in death from electric shock vary greatly. Some authors found no changes, others, lesions only when large amounts of electricity passed through the body for a long period of time. Some described small perivascular hemorrhages especially in the medulla oblongata and floor of the fourth ventricle, while others reported marked edema of the brain.

Spitzka [2] in his study of the brain of electrocuted animals found no apparent change in the nerve cells, but described areas of disruption and destruction with capillary hemorrhages. In the pons, medulla and cord curious circular areas were encountered with rarefied centers and peripheral zones of condensation, which he suggested resulted from the electrolytic liberation of gas in perivascular spaces.

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Werner [3] in his article dealing with electrocution as a judicial homicidal agent described the brain at autopsy as being very hot and recorded a temperature in the brain of 145° F. Jaffé feels that this excessive heat offers a sufficient explanation for the vacuolization seen within the brain.

Corradi [4] found chromatolysis of ganglion cells in the nervous system of dogs killed by electricity. Mott and Schuster,[5] in addition to perivascular hemorrhages and chromatolysis of ganglion cells in cortex and medulla, described a peculiar intracellular and intranuclear network. Capogrossi [6] also observed marked variation and destruction of the Nissl bodies. Kawamura [7] described shrinkage and hyaline changes in the nerve cells, and fragmentation of the tigroid bodies within the medulla. Gubler [8] reported a vacuolization of the nerve cells and Jellinek [9] destruction of the cells with extrusion of the nuclei.

The histologic changes in the peripheral nerves have been studied by Bolognesi [10] and Pietrusky;[11] the former found swelling and twisting of the axones, and breaking down of the nerve sheaths, while Pietrusky described a fusing together of the nuclei of the sheaths of Schwann.

Report of Experimental Work

A rather small series of animals, namely, one dog and one cat, and twenty guinea pigs, were used in the study of the histologic changes following electrocution.

These animals were divided into two groups; Group I including the dog and the cat, in which death followed immediately after a single shock, and Group II comprising the guinea pigs in which one to three sublethal shocks were given daily over varying periods of time. Of this second group the first animal was shocked on one day only, the second was shocked for two successive days, the third for three days and so on; the animal receiving the greatest number of shocks was shocked every day for nine successive days. Besides varying the number of shocks for each animal, the interval of time that elapsed after the shocking and before the animals were killed and autopsied was also varied; and this ranged from eighteen hours to fourteen days.

The guinea pig was selected because it is a fairly reliable laboratory animal and especially because it is small. When a large animal

is severely shocked the heart stops during the period the current is being applied and then goes into ventricular fibrillation, but seldom, if ever, recovers. The guinea pig heart behaves in much the same manner but if the shock has not been too prolonged the heart recovers its normal rhythm and the animal lives. Briefly, it is considered that the impulse or impulses that are traveling through the myocardium while the ventricle is fibrillating have relatively fewer excitable muscle groups to stimulate in a smaller heart, and consequently fibrillation is of shorter duration and recovery therefore more probable.

In shocking the guinea pigs the current from a six volt battery was passed through a small induction coil, and the strength of the induced current was varied by adjusting the central core. Tissue from a human came to us from a case of legal electrocution, while the dog and cat were obtained immediately after death following electrocution by the Animal Rescue League, and in this case a current of 110 volts was used.

The electrodes that were applied to the guinea pigs were two bared wires; one was passed through and across the mouth, the other was applied to one leg moistened with saline. The Animal Rescue League used a specially built copper cage and collar. In either case excellent contacts were obtained.

Preliminary to shocking the animals sufficient ether was given to quiet them and to permit fastening of the electrodes. Two of four control animals were given ether by inhalation in much larger amounts than the test animals and over a longer period of time.

As soon as the current was passed through the body there was a general contraction of all the muscles, sometimes accompanied by a shriek, and the animal maintained this rigid tonic state as long as the current was applied. The recovery after the shock varied; when the shock was of short duration and not severe, recovery was nearly instantaneous. On the other hand, with more prolonged shocking and with a stronger current, or in animals which had been shocked on several occasions, the recovery was slower, the animals appeared weaker, tremulous and had difficulty in supporting themselves. In several cases they completely relaxed for several minutes, seemed stuporous and assumed unusual catatonic positions. For hours after the shock many of the animals were unusually nervous and excitable, and in two cases they refused ordinary diet but chewed paper con-

tinually for hours and hours. When a fatal shock was given, the animals remained rigid for several seconds after the current was removed, then slowly passed into complete relaxation.

The current probably plays its greatest part in striking the central nervous system and the many altered physiologic signs which become manifest in the different systems appear to be simply visible expressions of extreme stimulation or marked alteration within the central nervous system.

While the current is passing through the body the animal is rigid, the heart stops beating and respirations cease; and eversion of the anus, defecation, micturition and emission occur almost regularly. Occasionally the animals vomit at the onset, and in two there was a prolapse of the rectum which returned as soon as the current was removed. Certain deranged physiologic signs that appear in the nervous system, the heart and blood vessels and in the respiratory system are unusually interesting and bear a more detailed description. Furthermore, to determine whether defecation, micturition, emission and prolapse of the rectum were merely passive reactions secondary to contraction of the abdominal muscles, or result from the stimulation and response of those viscera independently, we repeated the experiments in another group of guinea pigs with the abdominal cavity opened and with still another group in which the animals had received atropine.

The Immediate Response of the Heart to Electric Shock

The heart was examined in each test by auscultation before and immediately after the current was applied, but during the period of shock the sounds of contracting skeletal muscle were so loud it was impossible to follow the cardiac sounds, and consequently it was necessary to observe the heart's action directly.

With the animal lightly under ether and with the heart exposed, an electric shock of five seconds duration was given in the usual manner. As soon as the current was applied the heart contracted strongly and remained in systole except for two to three irregular beats which probably represent vagus escape. During the entire period of shock and for two to three seconds after, the ventricles showed an almost invisible and constant tremor. This was followed by a definite fibrillation of the ventricles which lasted three to five

seconds and this in turn was succeeded by irregular rather forceful contractions which continued for nearly ten seconds. The rhythm eventually became more regular but the rate was slower. This slowing of the heart was seen only in the animals whose hearts had been exposed, whereas in the unoperated animal which recovered after the shock the heart rate was temporarily greatly increased.

This simple experiment was of interest because we were able to follow the heart simultaneously and by auscultation which enabled us to interpret more clearly the irregularities noted in the animals from day to day.

The Immediate Response of the Heart to Electric Shock after Blocking the Vagus with Atropine

In each of these experiments $\frac{1}{100}$ to $\frac{1}{150}$ gr. of atropine dissolved in 5 cc. of sterile water was slowly injected into the leg veins of large healthy guinea pigs. With the animals lightly under ether, they remained sufficiently quiet to permit constant auscultation of the heart. After about ten to fifteen minutes the heart rate had nearly doubled but retained its normal regularity.

In the first of these experiments the heart was not exposed, so we could not follow the beats while the current was passing through the body, but when the current was shut off there was no pause or irregularity and the heart continued its rapid but regular course.

When the heart was exposed and the same experiment repeated, we watched the heart continue beating while the current was passing through the body and we observed no change in rate; neither was there a pause nor irregularity after the current ceased. This was important for it showed that when the vagus was blocked with atropine, an electric shock, which in a control animal was sufficient to stop the heart, produce ventricular fibrillation, irregularity and acceleration, effected no appreciable response in either rate or rhythm.

The Delayed Response of the Heart following Repeated Shocks

In the course of the experiments it was observed that there was a definite change in the heart which became more marked with each additional shock. When the animal was first shocked it usually made

a good recovery and in a minute or two appeared quite normal, but after shocking an animal for several days less current was required to produce the same effect and the recovery was much slower. In such a case the cardiac irregularity was more easily induced and the irregularity and acceleration lasted several times longer than that following the shocking of a normal animal. Furthermore, great caution had to be exercised in shocking animals that had been shocked previously on several occasions lest they suddenly die. Three examples from our series will make this point clearer. One of the guinea pigs died on the fifth day immediately after five shocks; another on the sixth immediately after six shocks. The third, apparently quite well and with a normal cardiac rate and rhythm, had been given seven daily shocks. The heart of the latter pig was being examined preliminary to the eighth shock when the animal exerted a little effort and suddenly died. In the case of the first two we had used less current and for a shorter period than on the preceding day. The manner of death in all three cases was the same. After the heart first stopped the cardiac sounds were never again audible except for three or four irregular and indistinct beats. The animals lay completely relaxed while the respirations continued for two to three minutes in a slow irregular gasping manner.

These observations indicate that as a result of electric shocks there is a profound disturbance in the cardiac mechanism which may be largely obscured by the presence of a normal rate and rhythm.

THE RESPONSE OF THE BLOOD VESSELS TO SHOCK

There was a change in the peripheral blood vessels following shock which if duplicated by the blood vessels of the central nervous system might have some consequent bearing on the lesions that later appear in the nerve tissue. By placing the ear of a guinea pig before a light the smaller vessels stood out quite clearly, and these were moderately accentuated if the ear was lightly rubbed. During the shock no apparent change was seen in this rich vascular network, but, about thirty seconds after the current was withdrawn, there was a gradual constriction of the vessels and the ear became abnormally blanched and remained anemic for five to ten minutes. The delayed vasoconstriction resulting from sympathetic stimulation simulates the vascular phenomenon in the condition clinically

spoken of as "surgical shock," and in itself would be sufficient to produce a moderate degree of ischemia with resulting oxygen deficiency in the areas involved.

The Response of the Abdominal Viscera to Shock

We have previously mentioned the observations seen in the normal animal which has neither been operated upon nor received atropine, namely, defecation, micturition and emission and occasionally prolapse of the rectum and vomiting. When the current was applied after the peritoneal cavity had been widely opened to remove the factor of contracting skeletal muscle, we were able to observe the contracting seminal vesicles with the resulting emission, and a slow feeble contraction of the bladder and intestines, although in the latter there was no apparent change in the peristaltic movements.

The pressure within the peritoneal cavity was greatly increased during shock as a result of the contraction of the diaphragm and skeletal muscles; this was well demonstrated when only a small opening was made in the anterior abdominal wall preliminary to an electric shock. As soon as the shock was applied the intestines were forced through the opening.

When the unoperated animal was previously given atropine and then shocked, there was neither defecation, micturition nor emission, even though the abdominal muscles contracted in their usual manner. Similarly, when the peritoneal cavity was opened we observed no variation in peristalsis, in the seminal vesicles or in the bladder.

From these observations in the normal animal, the animal with the peritoneal cavity opened and the animal who had previously received atropine, it would appear that the response of the abdominal viscera was due to the combined effect of contracting skeletal muscle and contraction of the individual viscera themselves.

The Response of the Respiratory Center to Shock

The onset of the shock was accompanied by a deep inspiration, with a powerful contraction of the intercostal muscles and diaphragm. This state was maintained throughout the entire period of shock and respiratory movements did not return for several seconds afterwards when they were shallow, irregular and rather difficult to follow.

PHYSIOLOGIC CHANGES IN THE NEUROMUSCULAR SYSTEM

Probably these were the most interesting of all the varied effects which we observed. We have touched briefly on the rather unusual nervous manifestations, namely, the excitability, nervousness and stupor, and have suggested that the responses seen in the other systems are in reality simply indirect manifestations of an altered nervous system. Now in addition to these there were three outstanding changes which belong specifically to the neuromuscular system alone, namely, weakness, paralysis of the posterior extremities and diminished sensation.

The weakness was usually not seen until after the third or fourth daily shock. The animal became less active, did not move about as quickly, appeared wobbly after a shock, and had difficulty in holding himself up.

Paralysis of both posterior extremities occurred in more than half of the test animals and usually in those who seemed rather feeble. In some cases it came on immediately after the shock; in one it appeared at once, lasted a half hour and disappeared, but again reappeared in twelve hours. Others did not become paralyzed till twenty-four to forty-eight hours had elapsed since their last shock. The paralyzed animals were watched and killed at varying intervals. One that lived fourteen days after the onset of paralysis began to regain some strength and motion in the paralyzed legs. When paralyzed the animals drag their hind feet about and appear to have no control of the bladder or anal sphincters.

If the paralyzed legs were lightly stimulated by touch, pressure, pain or heat there was no visible response. A strong electrical stimulation elicited a contraction of the muscles of the body but the normal reflex arc of the paralyzed legs had been interrupted because the leg muscles showed no response.

The left leg was always used in applying the lower electrode, and even after the first shock the animal would move this less than the other, sometimes dragging it along as though paralyzed. This we attributed to a protective mechanism to relieve the muscular pain.

We shall not attempt at this point to consider the possible pathologic conditions that might account for these neurologic findings as these will be discussed after the gross and microscopic lesions have been described. We have merely enumerated our observations to show the important rôle the nervous system plays in electrocution.

Summary of Physiologic Observations

These manifestations in normal animals, animals that had received atropine, and others with the heart and abdominal and pelvic viscera exposed, are simply expressions of the central nervous system including the craniosacral and sympathetic divisions, and may largely be interpreted on the basis of pathologic histology.

Now it is generally assumed that there are no constant demonstrable histologic lesions in the central nervous system of animals following a single fatal shock in which death is instantaneous. The microscopic study of material from cases of sudden death offers many other examples in which one finds no demonstrable lesions. The classic example is the case of chloroform poisoning; the animal that is killed with a single large dose of chloroform shows no gross or microscopic lesions in the liver, but when an animal receives one or more sublethal doses and is allowed to live twenty-four hours or longer, the degeneration, necrosis and hemorrhage in the centers of the liver lobules are startling.

This principle of giving sublethal amounts of electricity, grading the amounts and allowing the animal to live a sufficient time to permit histologic changes to appear was the method that was followed in this short series of experiments.

Pathologic Report

Complete autopsies were performed on all animals immediately after death. The only positive and constant gross finding in the animals that were given a single fatal shock was marked congestion of the lungs, liver, kidneys, spleen and heart. In the guinea pigs this finding was absent because the animals were bled at the time of death to insure better fixation of the tissues. Hemorrhages were frequently seen in the psoas, iliacus and gluteal muscles, and were usually bilateral. The meninges were injected, and two of the animals had petechial hemorrhages in meninges of both brain and cord. The spinal cord of a normal guinea pig that is autopsied immediately after death is pale, firm and homogeneous throughout. In almost every test animal there were portions of the cord that were distinctly swollen, soft and almost diffluent.

The gross findings in one animal must be described separately because this pig had been given only two shocks. Paralysis suddenly

appeared after the second shock and the animal died in eighteen hours. On examination, hemorrhages were found in the psoas and iliacus muscles, along the anterior surface of the vertebral column, in the erector spinal group of muscles, subdurally along the entire length of the cord, within the cord and also in the brain in the region of the right basal ganglia. This animal, which was the smallest animal we used in the tests, had a definite fracture of the second thoracic vertebra. This gives one an idea of the power of contracting skeletal muscle.

Sections were taken from most of the organs, namely, the heart, lungs, spleen, liver, pancreas, kidney, adrenal, testis, bladder, intestine and skeletal muscle; and of the nervous system from the eye, the cerebrum, cerebellum, pons, medulla, different levels of the cord, pituitary, posterior root ganglia and peripheral nerves.

The tissue, apart from the nervous system, was fixed only in Zenker's solution, neutral formalin and alcohol-formalin and was embedded in celloidin and paraffin. In addition to using the above-named fixatives, sections of the nervous system were also placed in Cajal's formalin-ammonium-bromide solution, Mueller's fluid, and a saturated solution of corrosive sublimate.

In staining the tissues other than those from the nervous system we used hematoxylin and eosin, eosin and methylene blue, Mallory's phosphotungstic acid hematoxylin, Mallory's aniline blue collagen stain, Sudan IV, and osmic acid with and without preliminary treatment with a weak solution of potassium bichromate. For the nervous system we employed hematoxylin and eosin, eosin and methylene blue, Mallory's phosphotungstic acid hematoxylin, osmic acid, Sudan IV, Marchi's method for degenerating myelin, Cajal's gold chloride sublimate method for neuroglia astrocytes and Penfield's second modification of the del Río-Hortega silver carbonate method for oligodendroglia.

Microscopic Report

The pathological histology in the tissues other than the nervous system is disappointing. There is congestion of the heart, spleen, kidneys and adrenals, congestion at the periphery of the lobules of the liver and congestion and petechial hemorrhages in the lungs. The lesions in the skeletal muscles that grossly showed hemorrhages and pigmentation are slight and vary with age. Scattered muscle

fibers are ruptured, the free ends are separated and for a short distance they are contracted, wrinkled, swollen, and homogeneous. Surrounding these lesions extravasated red blood cells lie free in the reticulum. After twenty-four hours polymorphonuclear and endothelial leucocytes are seen within the degenerating and necrotic portions of the muscle fibers, lying free in the stroma, and also in the perivascular tissue. In the later stages after removal of the necrotic portion of muscle fibers, regeneration of muscle occurs, showing the typical multinucleate syncytium resulting from the multiplication of muscle nuclei by direct division and the formation of new cytoplasm. Some areas show degeneration and regeneration occurring simultaneously.

No constant lesion is present in the heart. The amount of fat in the myocardium of both the "test" and "control" pigs is extremely slight and shows little variation. With Sudan IV an occasional muscle fiber is shown to contain minute fat droplets, but this finding is as common in the "control" as in the "test" animals.

The petechial hemorrhages and the capillary injection of the pia arachnoid are verified microscopically and also the hemorrhage extending along the posterior column of the cord of the animal whose vertebral column was broken.

The histologic study of the central nervous system of the animals which received a single fatal shock reveals no constant changes other than capillary injection and occasional petechial hemorrhages.

In the guinea pigs we find definite lesions that vary somewhat in the different animals, depending largely on the severity of the shocks, the number of shocks and the duration of time that elapsed before death. The lesions are best studied in the cord and peripheral nerves including the medullated nerves to the diaphragm and heart, but are also seen within the medulla, in the tracts leading from the pons to the cerebellum and also in the lower portion of the internal capsule. It is the white matter of the cord that is most seriously affected, and in the majority of animals the lesion is greatest in the posterior columns. In some it is more generally distributed throughout all the tracts, in others it seems to affect the fibers lying nearest to the gray matter, while in still others the lesion is distinctly peripheral. In the nerves only scattered fibers are affected.

The earliest change consists in a swelling of both the axis cylinders and myelin. The axis cylinder is enlarged to several times its normal

diameter. In some areas it is disintegrating and in others it has disappeared. The myelin distends its sheath and in the ordinary hematoxylin and eosin preparation has an irregular bluish mucoid appearance. As this swelling progresses the myelin gradually becomes paler till finally it disappears leaving vacuole-like spaces among the fibers.

The picture within the cord differs considerably depending on whether it is viewed from a cross-section or a longitudinal section. In the former the gray matter may show little or no change, whereas the surrounding fiber tracts which comprise the white matter may be riddled with different sized vacuoles. The axis cylinders are not present in some of the larger vacuoles, while in others they are large, irregular and eccentric; even in the comparatively normal areas they are much less uniform in size and contour than one normally finds them. In the longitudinal section the cord has the most bizarre appearance. Axones may be followed only for short distances; they are irregularly swollen and tortuous and appear to taper off at the ends as though disrupted. Myelin, in the later stages, has disappeared leaving irregular vacuoles and long spaces around and among the swollen axones.

In the more advanced lesion where the process has progressed farther, there are foci of marked myelin degeneration heavily infiltrated with phagocytic endothelial leucocytes, scattered polymorphonuclear leucocytes and a few extravasated red blood cells.

The nerves show similar though less marked lesions. Individual fibers are swollen and vacuolated with distention of the neurolemma, while the epineurium is infiltrated with numerous endothelial leucocytes.

The nerve cells from various portions of the normal guinea pig's brain and cord are distinctly different. Moreover, in studying any group of nerve cells whether in the cortex, in the medulla or in the cord, it is found that they differ in size, shape, cytoplasmic staining, in the number of their processes and also in the number, size and position of the Nissl bodies. The nuclei and nucleoli also vary in size, contour, position and staining. Because of these variations it is difficult to diagnose the early degenerative changes in the cells. However, in a few of the areas of degeneration of the cord that extend into the gray matter there is actual necrosis of the nerve cells.

So far we have described the lesion only as it appears when we

used the hematoxylin and eosin, eosin and methylene blue, and phosphotungstic acid stains. When the early lesions are stained with Sudan IV or osmic acid, endothelial leucocytes are seen distended with material resembling myelin, whereas the vacuoles in the cord remain unstained.

In both the cord and nerves of animals which lived one to two weeks after the onset of paralysis, the Marchi method for degenerating myelin produces rather remarkable pictures. There is a much more widespread degeneration of myelin than was expected from an examination of the eosin-methylene blue sections. In cross-section of the cord the degenerated myelin appears throughout the tracts in varyingly sized irregular rings, and also as small and large globules, some of which are in endothelial leucocytes. In a longitudinal section of the nerves, the even uniform distribution of the myelin is replaced by droplets lying in the neurokeratin framework.

Sections of the cord and peripheral nerves were impregnated to bring out both the oligodendroglia and microglia. There is little or no change in the oligodendroglia except in an area of degeneration where they are swollen and rather indistinct. As many as eight to ten perineuronal oligodendroglia are frequently seen surrounding an anterior horn cell in a control cord, and we never found a greater number than this in the test animals. The number of microglia is increased in the white matter, and in areas of myelin dissolution all transitional forms are found between these small cells with delicate branching projections and the large, round, phagocytic, compound granular corpuscles.

The neuroglia astrocytes stain well in both the control and test animals, and though there seems to be an increase of these elements in the posterior columns in the animals with older lesions, yet the number is so variable in different animals and in different levels of the cord that it is doubtful whether there is a beginning true gliosis as early as fourteen days.

In summing up the histologic changes we find that they are confined almost entirely to the nervous system and that they involve primarily the nerve cells, the axones and their myelin sheaths.

DISCUSSION

The question of first importance in electrocution is "what causes death?" Now in speaking of electric currents it is usual to call those over 1,000 volts high tension and those below this, low tension currents, while the importance of either type of current in causing fatal shocks is readily shown in the number of accidental deaths occurring annually. Furthermore, individuals vary greatly in their response to electricity and different portions of the body show wide variation in their electrical resistance. The dry calloused palm of the hand may have a resistance of 2,000,000 ohms, while that of the mucous membrane of the same person may be only 100 ohms.

In our experiments we used very low voltage. When the current was applied the heart ceased beating; this, however, did not occur when a sufficient dose of atropine had been given previously. This suggests that the stopping of the heart results from unusual vagus stimulation. It is stated clinically that in death resulting from shocks of low tension the heart ceases first and respiration continues, while in high tension currents paralysis of the respiratory center is said to precede cessation of the heart beats. Death may be produced readily in guinea pigs using a low tension current, provided the exposure is long enough; with short exposure, however, the heart ceases fibrillation and the normal rhythm returns. In larger animals and man, fibrillation of the ventricle is almost always fatal and so death ensues.

It does not appear to be the heart muscle that is affected by the electric current but rather the entire nervous system. It seems reasonable to suppose that the same factors would apply for either currents of high or low tension and hence the production of apparent cardiac death simply depends on whether or not the heart has been thrown into fibrillation.

There are three possible causes which must be considered in the production of lesions within the nervous system, namely, the electric current, mechanical force and ischemia. We have seen from our experiments the relative importance of these and conclude that the electric current itself is probably the most important single factor.

Death in electric shock may occur at the time of the shock, several minutes later, or days later, or may result from complications resulting from local injury. Occasionally a patient dies suddenly hours

or days after an electric shock. This type of death was seen in the experimental animals in whom it was found that while there were no demonstrable lesions in the myocardium, there were definite lesions in the cord, peripheral nerves and nerves to the heart. Hence, we feel that though a patient may appear in normal health after a shock reasonable precautions should be taken for days and weeks to avoid unnecessary stress and strain.

SUMMARY

1. The literature dealing with the pathologic changes in the nervous system of electrocuted animals is reviewed.

2. The observations in normal animals, animals with exposed viscera and animals under atropine which were receiving electric shock are described in detail.

3. The value of repeated sublethal shocks in obtaining histologic lesions is stressed.

4. The rôle of the nervous system in producing changes in other systems is discussed.

5. The gross and microscopic lesions are described in which it is shown that the lesions are confined almost entirely to the nervous system and skeletal muscle.

6. The discussion deals with physical properties of electrical currents, the resistance of the body to currents, the different types of death from electric shock and emphasis is laid on the importance of prolonged rest in cases of recovery.

NOTE: I am indebted to Dr. F. B. Mallory for his many suggestions and helpful criticism, and also for the photomicrographs; and to Miss Marion E. Lamb for technical assistance.

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DESCRIPTION OF PLATES

PLATE 68

FIG. 1. Section of cord removed fourteen days after the onset of paralysis and stained by Marchi's method for myelin degeneration. There is marked myelin degeneration in the lateral tracts, while just beneath and partly involving the posterior horn is an area of "softening" in which the degenerated myelin droplets have coalesced; many of them are in phagocytic endothelial leucocytes. × 100.

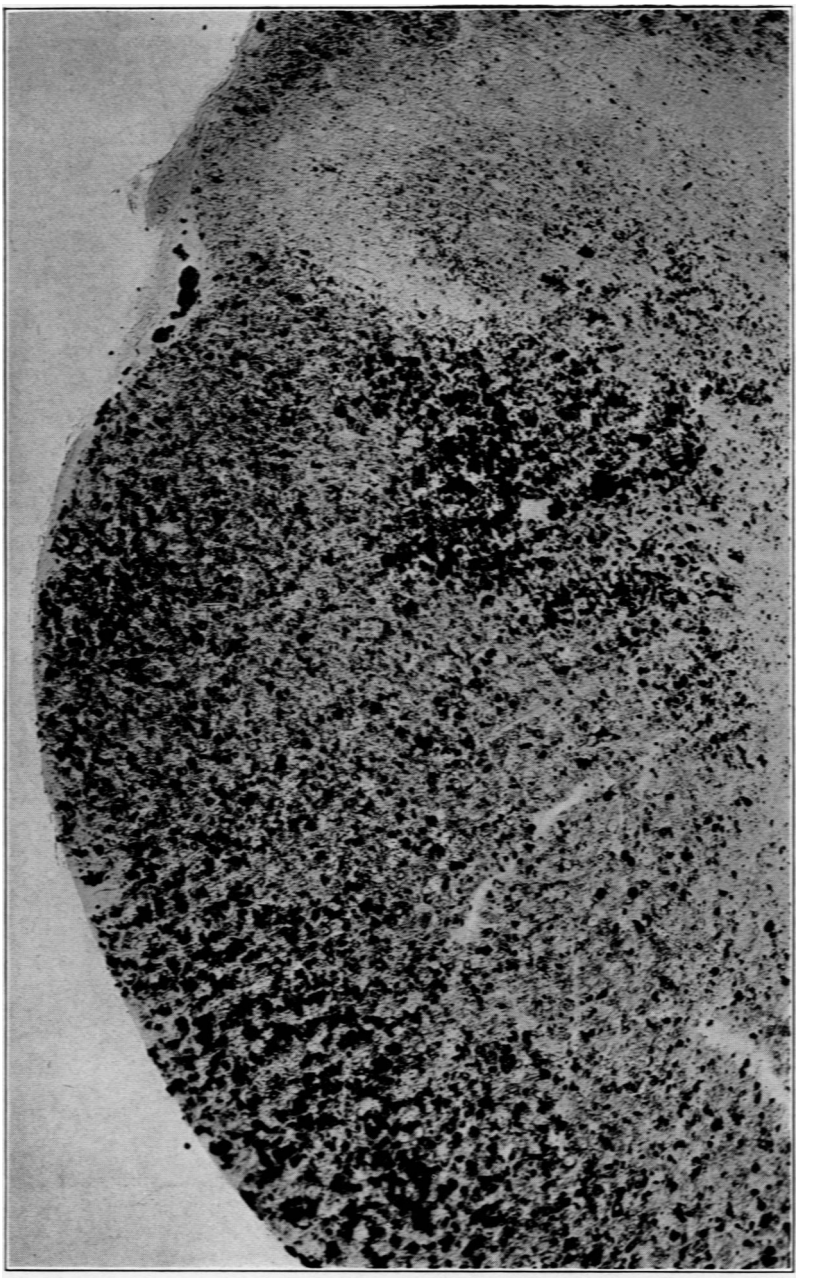

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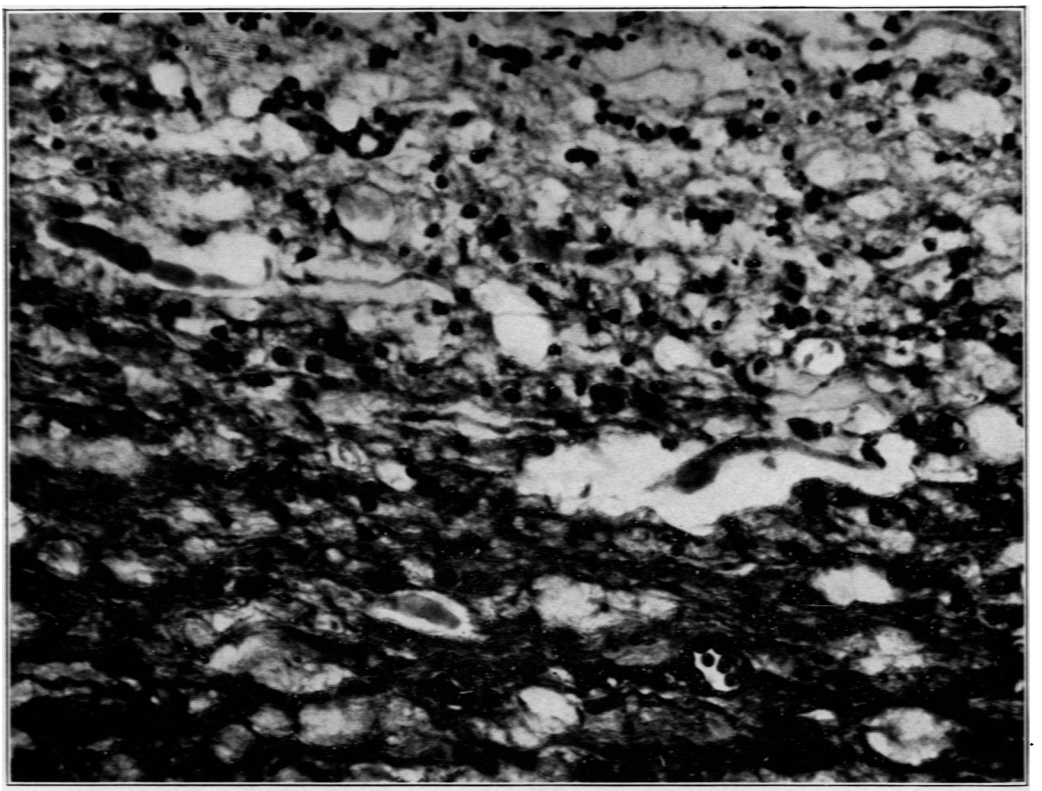
PLATE 69

FIG. 2. Photomicrograph of a longitudinal section of the cord showing in the posterior columns the swollen, tortuous axis cylinders. The clear areas represent swollen myelin or areas from which myelin has entirely disappeared. There is a moderate infiltration of endothelial leucocytes. (Hematoxylin and eosin.) × 300.

FIG. 3. Photomicrograph showing an area of "softening" in the posterior column of the cord. The normal morphology and arrangement of the elements are destroyed. Axis cylinders and myelin have disappeared; the latter is in part phagocyted by endothelial leucocytes. (Hematoxylin and eosin.) × 250.



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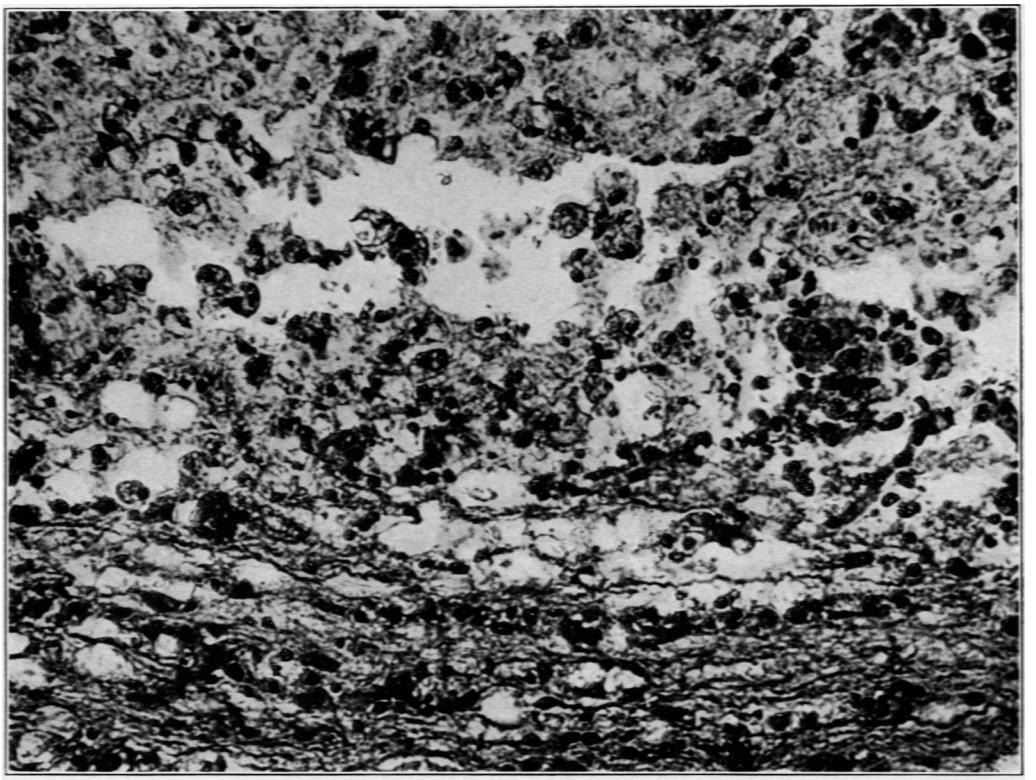

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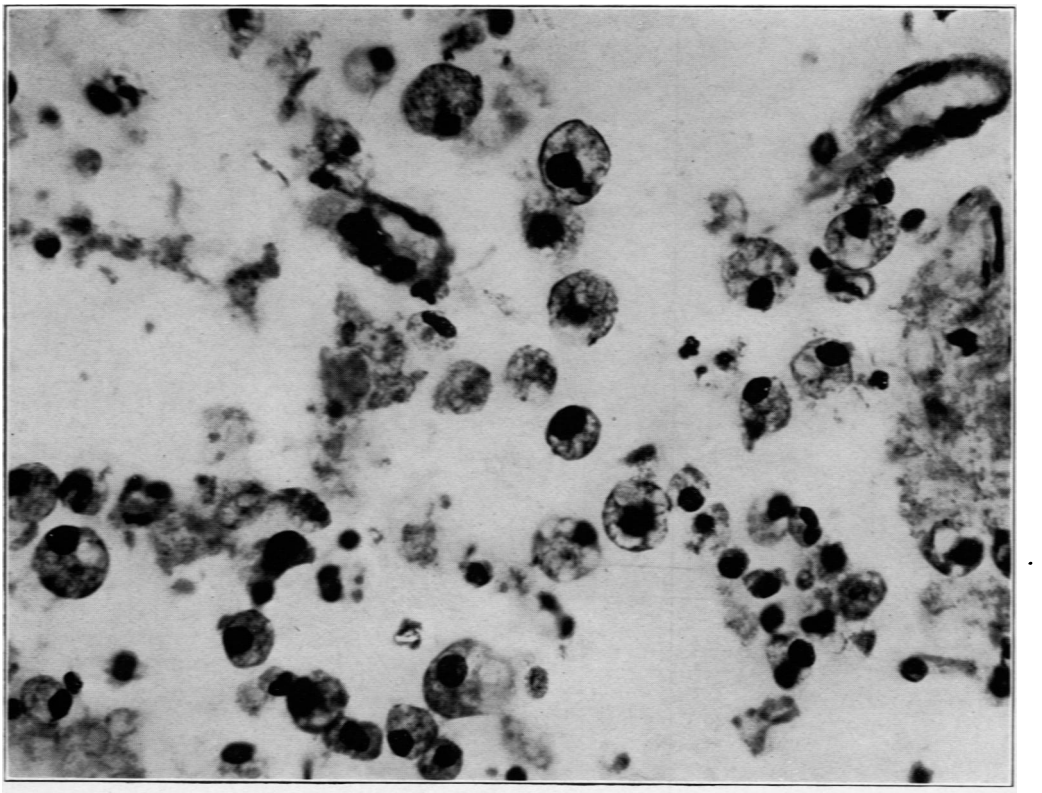
Plate 70

Fig. 4. A higher power photomicrograph from an area of degeneration in the cord, showing endothelial leucocytes many of which are distended with myelin. × 500.

Fig. 5. Longitudinal section of a peripheral nerve traversing bundles of striated muscle. Degenerated myelin droplets of various sizes are held in the neurokeratin framework of the nerve fibers. Marchi stain for degenerated myelin. × 125.



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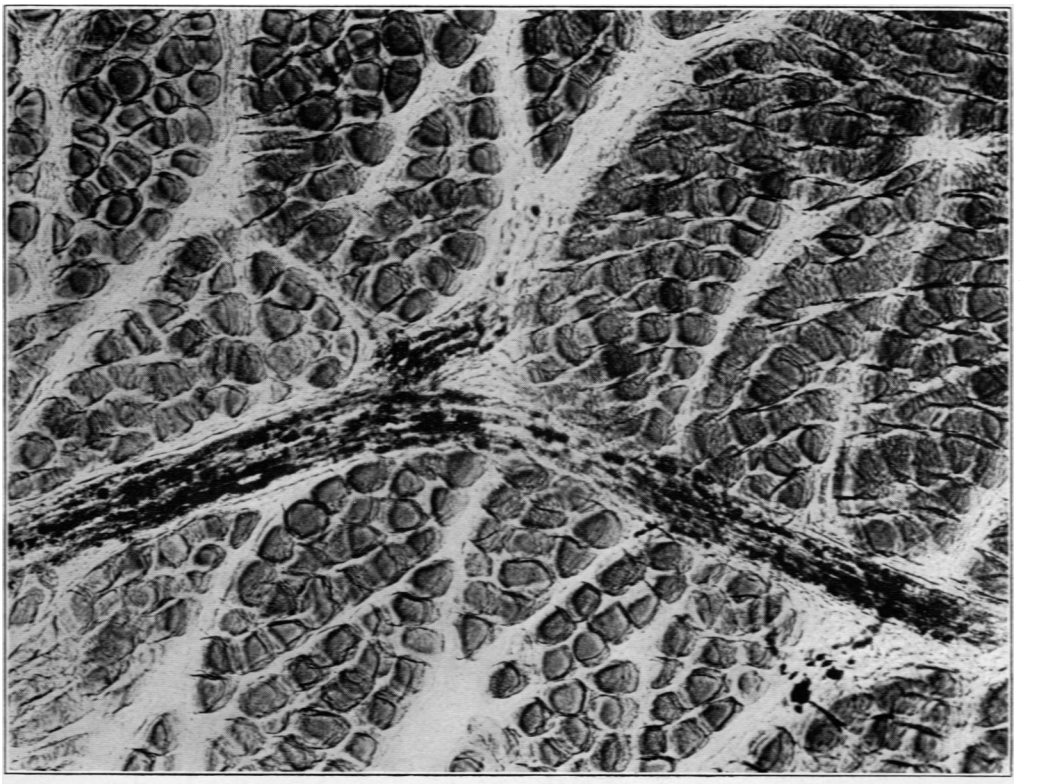

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