ABNORMALITIES PRODUCED IN THE CENTRAL NERVOUS SYSTEM BY ELECTRICAL INJURIES*

By ORTHELLO R. LANGWORTHY, M.D.

(From the Sub-Department of Neurology, Johns Hopkins University, Baltimore)

PLATES 31 TO 33

(Received for publication, February 27, 1930)

The problem of injuries following contact with electric circuits has become an important one owing to the extensive use of electricity in everyday life. In 1913, 670 deaths in the United States were attributed to this cause. Circuits supplying homes and factories are commonly of 110 or 220 volts, either alternating or continuous current. A 100 volt circuit may prove fatal. For this reason it has seemed important to make a careful fundamental study of electrical injuries with a view to saving the lives of those who have received a severe shock. The present paper presents experimental evidence concerning the injuries produced in the central nervous system.

The material serving as a basis for this study consists of some 286 rats, injured by either continuous or alternating electric circuits at different potentials. The experimental technique and the immediate results of the injury have been reported in another place (Langworthy and Kouwenhoven, 1930). The central nervous systems of these rats were preserved immediately after death and typical sections have been cut in an endeavor to correlate the experimental results with actual histological pictures. It will be possible to show that the abnormalities produced by the continuous current differ from those produced by the alternating.

Many fatal accidents from contact with electricity are attributed to the resulting fibrillation of the ventricles of the heart. Ventricular fibrillation is believed to be more common after contact with circuits

* This research was conducted with a fund provided by the Committee on Electric Shock.

at low voltages. Circuits at high voltages, on the other hand, are thought to cause death from respiratory failure due to a central inhibition in the nervous system. Since the present experiments were directed to an elucidation of injury in the brain and spinal cord, an experimental animal was chosen in which the heart recovers spontaneously from ventricular fibrillation. The rat proved to be such an experimental animal; it is doubtful if any deaths in this series can be attributed to cardiac failure. This makes it possible to focus attention upon the central nervous system.

The histological study reported here endeavors to demonstrate evidence of injury in the nerve cell itself as a result of electric shock. These changes are sought in the general appearance of the cell, in the Nissl granules and in the cell nucleus. These criteria of injury of the nerve cell have been used by many investigators.

A good review of the pathological changes produced in nerve cells by poisons, such as arsenic, phosphorus and veratrin, by anemia and by high temperatures was given by Barker (1909). It has been demonstrated that the Nissl granules are formed after death by the coagulation of some substance normally present in the cell. The substance is used in cell metabolism and is greatly decreased in amount by fatigue. As the result of injury, various changes are observed in the cytoplasm; among these may be suggested complete loss of granules, loss of granules at the periphery of the cell or around the nucleus and vacuolization. The cell may be swollen, the granules large and adherent in a compact mass or the cytoplasm stain a uniform blue. Later a contraction of the cytoplasm and pyknotic condition of the nucleus suggest cell death. Buzzard and Greenfield (1925) have more recently reviewed the subject of nerve cell pathology. Here it is important to discuss the changes that have been found in nerve cells following injuries with electric currents and closely allied types of traumata.

Urquhart (1927) felt that the asphyxia accompanying temporary respiratory inhibition was sufficient to produce changes in the cells of the brain and cord that would confuse any picture of injury produced by electricity.

Gomez and Pike (1909) previously found that anemia lasting 8 minutes was sufficient to kill the small pyramidal cells of the cortex. After 13 minutes there were marked changes in the Purkinje cells in the cerebellum. In the case of the medulla, anemia for 20 to 30 minutes produced alterations incompatible with complete recovery. Cannon and Burket (1913) later showed that cells of the myenteric plexus could recover from anemia lasting 6 hours. In the present experiments the periods of apnea were short; the rats that were saved breathed in almost every case within 2 minutes. It seems that the effects upon the nerve cells of anemia may be discounted in the present work.

Mott and Uno (1922) studied the central nervous system of patients that died from surgical shock from 24 to 48 hours after injury. The brains were fixed first in formalin and afterward blocks were placed in alcohol. In the cerebellum very few Purkinje cells showed a normal staining reaction. The Nissl granules were broken up into fine dust; the cell was swollen, the nucleus indistinct and the whole cell only faintly stained. In the neighborhood one could see cells which presented a more normal appearance.

Regarding the changes produced in the nerve cell by electrical injuries, observers do not agree. Small perivascular hemorrhages have been reported, especially in the medulla and floor of the fourth ventricle; these hemorrhages are common after legal electrocution. Chromatolysis of the ganglion cells and rupture of the cells with dislocation of the nuclei have been seen. Huber described a loosening of the glia, vacuolization of the nerve cells and approximation of the nuclei to the walls of the cells. Kawamura says that some cells appear normal while others are shrunken and hyaline. In the medulla oblongata the Nissl substance is broken into small granules. Mott and Schuster (1909) described the lesions in a man who recovered from a shock at 20,000 volt potential and lived for 7 hours thereafter. The autopsy was performed 39 hours after death.

Spitzka and Radasch (1912) sectioned the brains of five electrocuted criminals and found peculiar areas from 25 to 200 micra in diameter around small blood vessels. There was a central rarified zone and a peripheral condensed one. They explained their observations on the basis of a sudden liberation of gas bubbles by the electrolytic action of the current. Since the current used was alternating, it is more likely that the vacuolization resulted from excessive temperatures. In electrocution the brain becomes exceedingly warm, a temperature of 145°F. has been recorded.

MacMahon (1929) has performed experiments similar to those reported in the present paper. Guinea pigs were exposed to continuous circuits of low voltage. Some of the animals received a series of shocks on different days. Following the injury, excitability, nervousness and stupor were observed. There was also weakness, paralysis of the posterior extremities and diminished sensation. The animals were less active and did not move about quickly after the shock. Paralysis of both posterior extremities occurred in more than half the animals. In some cases it came on immediately after the shock; others did not become paralyzed until 2 days thereafter. When paralyzed, the animals dragged their hind feet and appeared to have no control of the sphincters. If the paralyzed legs were lightly stimulated by touch, pressure, pain or heat there was no response.

On examination of the central nervous system of the guinea pigs that had survived the shock it was found that the spinal cord was swollen, soft and almost diffluent. There were petechial hemorrhages in the pia arachnoid. The white matter of the cord was most seriously injured, and the lesion was greatest in the posterior columns. The earliest change in the nerve fiber consisted in a swelling of both the axis cylinder and myelin sheath. The myelin in later stages disappeared

leaving irregular vacuoles and spaces around and among the swollen axones. The peripheral nerves showed similar degeneration. Little information was given concerning changes in the nerve cells.

With this review of the literature, it is important to discuss the peculiar difficulties in interpretation of pathological changes in nerve cells as seen in sections stained with a basic dye. Criteria that are used by investigators as evidence of nerve cell damage have already been presented. But, in order that these criteria may be valid, other very definite postulates have to be filled.

To demonstrate the finer structure of cells, and in particular, nerve cells, it is extremely important that fixation of the tissue be carried out immediately after death. If the autopsy is delayed for only a few hours, the nerve cells stain a uniform, blue color with the basic dyes. Moreover the fixative is important; 95 per cent alcohol should be used if a sharp, clear staining of the granules in the cytoplasm is desired. Formalin is a standard fixative for nervous tissues but it permits only a poor differentiation of the granules. The 95 per cent alcohol has a very low power of penetration; with fairly large brains an arterial injection is important. The central nervous system of the rat is so small that the brain was not injected but was cut in thin slices and immersed in the fixative.

It is often difficult to obtain a good staining of the sections. A good thionin dye becomes slightly metachromatic and imparts to the granules a deep blue tint with a trace of lavender. The sections with this lavender hue do not fade easily and remain in good condition indefinitely. If the sections are overstained the cytoplasm of the cells is a uniform blue and the granules are not distinct; if the stain is light the granules do not receive their full value.

After a sharp staining of the cells, their nuclei and the granules in the cytoplasm is obtained, the interpretation still offers difficulties. Different groups of cells show great variation as to their size, shape and the arrangement of the granules. Characteristic patterns of the granules have been studied by Malone (1912 and 1913). He pointed out that somatic motor cells have large Nissl granules packing the cytoplasm of the cell. Autonomic cells, on the other hand, have large granules surrounding the edge of the cytoplasm and smaller granules in the remaining area. Sensory cells have fine, dust-like granules, scattered uniformly. The possibilities of granule arrangements in the higher centers of the brain are infinite.

To avoid difficulties of telling normal from injured cells by the arrangement of granules, only well known types of cells are discussed in the present report. The somatic motor cells of the anterior columns of the cord were examined in every case. It was most important to study the dorsal nucleus of the vagus because of its important func-

tion in respiration. The Purkinje cells of the cerebellum are characteristic as are the cells of the olives. Finally the sensory cells in the mesencephalic nucleus of the trigeminal were used as a type. No minute examination of the cells of the cerebral cortex was made. The sections of the forebrain were examined for hemorrhage and evidence of burning, as was the rest of the brain and cord.

Methods

The brain and spinal cord of the rats that died as an immediate result of the injury, were removed at once, cut into thin slices and placed in 95 per cent alcohol. After adequate fixation the blocks were imbedded in paraffin and sections stained with thionin. Rats that survived the shock were killed a few hours or days after the injury. In the case of a number of the rats, a microscopic examination of the thoracic and abdominal organs was made.

HISTOLOGICAL STUDY

The experiments are grouped on the basis of different potentials of alternating and continuous circuits. Greater injury was produced by the circuits at high voltages and these marked changes may be described first.

Rats Subjected to a Continuous Circuit at 1000 Volt Potential

It will be possible to demonstrate that the general microscopic picture of injuries produced with the continuous circuit is somewhat different from that produced by the alternating. Since the lesions are more severe and more clear cut with the 1000 volt circuit it is proposed to begin with this group.

Thirty-three rats were subjected to a continuous circuit at 1000 volt potentia for periods from quick manual closure and opening of the switch to 4 seconds. For the purpose of study a contact of ½ second was approximated as closely as possible. The current readings were 600 to 1100 milliamperes. Twenty of the rats died at once, nine recovered and four were paralyzed. These findings need further explanation; thus eight received the current for the time required to open and close the switch. Of these eight animals, five recovered, two died and one was paralyzed. Thirteen were shocked for approximately ½ second; four were normal, three paralyzed and six died at once. No rats survived an application of the current for 1 second or over.

By comparison it is found that the continuous 1000 volt circuit was more injurious to rats than the alternating circuit of the same potential.

This is particularly true if the time element is taken into consideration. But, at autopsy, few gross hemorrhages were found in the central nervous system, even of the paralyzed rats. Study of the sections gives an explanation for the fatalities in this group.

Twenty rats that died immediately will be considered first, in reference to the microscopic findings.

The photomicrographs of abnormal cells that illustrate this paper were made largely from sections of the central nervous system of this 1000 volt group. Large numbers of sections are available and the pathological picture is remarkably uniform. In three of these rats there was a large hemorrhage in the fourth ventricle that must have pressed upon the respiratory center. The brains, in general, were remarkably free from microscopic hemorrhages.

The surface of the cerebral cortex was not as severely injured as in the alternating circuit series. It is true that in several cases large cavities were found near the surface of the gray matter. These have been observed in the brains of electrocuted criminals and are supposed to be due to the collection of gases produced by the generation of heat. It is interesting that in the rats they are found most commonly close to the surface electrode where the concentration of electricity is great, although they do occur in all portions of the nervous system. They are often present in rats that recovered from the shock. In man they are perivascular; this is not necessarily true in the rats. The wall of these cysts is deeply staining due to the condensed brain tissue. In some of the rats the surface of the cortex appears to have actually burned and the remaining edge stains an intense blue.

The greatest interest centers around the changes in the nerve cells that are similar to those produced by the 1000 volt alternating circuit, but more severe. These may be described first in the somatic motor cells of the spinal cord.

The cells are selectively injured; some are grossly damaged and others appear almost normal. The cell in Fig. 4 from a rat of this series has a nucleus that appears practically normal. On one side of the cytoplasm the granules are numerous. On the other, toward the periphery of the cord, few granules are present. The cytoplasm appears shrunken and the outlines of the cell can be made out with difficulty. Since this cell has a normal nucleus it is thought that it has the potentialities of a complete recovery.

In Fig. 5 the cell presents a somewhat different picture. The nucleus stains deeply and the nucleolus is swollen but the nuclear injury is scarcely profound. Granules are numerous around the nucleus but the outer portion of the cytoplasm is relatively free from granules. This cell has suffered a loss of Nissl granules.

A severely injured cell is shown in Fig. 6; this opinion is given because of the

nucleus which is shrunken, and stains a uniform deep color in which no nucleolus or chromatin skeins can be seen. The clear area around the nucleus shows its normal size. The granules are numerous in this cell, although portions of the cytoplasm are quite free from them.

In Fig. 7 are two cells with nuclei slightly less damaged. The nucleous is greatly swollen. The remainder of the nucleus is shrunken and darkly staining although the color is not as deep as the nucleolus. In the upper cell the granules are sparse but scattered uniformly. The cytoplasm has no distinct boundaries in the lower cell and it would appear that the cell has ruptured and the granules are free in the perivascular space. It is doubtful whether these cells have the potential power of recovery.

The deep staining cell in Fig. 8 is similar to that in Fig. 6; the other two cells show severe damage. The nucleus in the lower one, however, appears quite normal. The cytoplasm of both these cells is greatly shrunken and the edges are jagged and irregular. If the nucleus is normal it is probable that the cytoplasm may be regenerated. Similar cells are abundant in all this material and suggest the severe injury responsible for the death of the rats.

The Purkinje cells are particularly vulnerable to injury with electric shocks.

The results of the shock, as seen in the rats that die immediately, are seen in Fig. 12. The injury is again selective although large groups of cells are abnormal as the photomicrograph suggests. The nuclei of the cells stain a uniform, deep color in which the chromatin and nucleolus cannot be distinguished. The cytoplasm likewise stains a lighter but uniform blue. That this injury betokens the death of the cell is clear from the examination of rats that have survived for some time (Fig. 11). Here the cells stain a dark color and are much smaller than normal.

Since sensory ganglia were not stained in every preparation, the cells of the mesencephalic nucleus of the trigeminal were used for illustrating damage to primary sensory neurones.

The nucleus of the cell (Fig. 16) is irregular in outline and small but the nucleolus may still be distinguished. The clear surrounding area was probably filled by the nucleus. The granules show poorly in the cytoplasm which stains a rather uniform blue. These cells are severely damaged. Fig. 15 shows several fairly normal cells and one with a deep staining, pyknotic nucleus. Sensory cells are relatively resistant to injuries by electricity.

It was considered particularly important, in each case, to examine the dorsal nucleus of the vagus since the cells of this nucleus have an important part in initiating and controlling respiration.

These autonomic cells normally have large granules at the periphery of the cytoplasm and smaller granules scattered through the remainder. A fairly typical illustration of the granules is seen in Fig. 17. However, numbers of the cells in these rats, as shown in Fig. 17, have deep staining nuclei suggesting the death of the cells. One of the cells in Fig. 18 has a deep staining nucleus; in another the edges of the cytoplasm are indistinct and a loss of granules has occurred. More severe injury is seen in Fig. 22 where both nuclei and cytoplasm are grossly injured. If many cells of the nucleus are thus damaged not a temporary block but a permanent paralysis of the respiratory center is to be expected.

Four rats were paralyzed following the shock. There was a tendency toward recovery so that the legs became much stronger in one of the preparations and by the fourth day they were used fairly well. Nocuous stimuli applied to the paralyzed portion of the body elicited a response in all of these rats, in contrast with the alternating group. Two of them became incontinent on the second day, although no blood was ever present in the urine. All the alternating group were incontinent and the urine was deeply stained with blood. Moreover, priapism was present in these rats; this suggested an irritative lesion in the spinal cord. Macroscopic examination of the brain and spinal cord revealed no lesions that would account for the marked symptoms.

These rats lived from 2 to 6 days following the shock. On microscopic examination of the cord it was found that many ventral horn cells had recovered from the initial injury and were fairly normal in appearance. The granules, however, were not well defined and the cytoplasm tended to stain a uniform deep blue. Other groups of cells were present which showed practically no granules in the cytoplasm. The third type of injury, found particularly in the lumbar portion of the spinal cord was the most serious and characteristic; it is represented in Fig. 9. The cells are deeply staining and the cytoplasm has irregular contours. The nucleus is distinguished with difficulty; in the upper cells and the one on the lower right, the nucleolus may be recognized, staining a little deeper color than the cytoplasm. The cytoplasm is very shrunken. The two lower cells are being attacked by phagocytes that have penetrated the cytoplasm of the cell. That this is not the nucleus is clear in the lower right hand cell where both the nucleolus of the nerve cell and the phagocytic cell may be distinguished. This phagocytosis is evidence of severe cell injury and death. We have, then, in this group, paralysis of the posterior

portion of the body, not due secondarily to the influence of a large hemorrhage upon the cells and fibers of the cord but to an actual injury of the nerve cells. This injury is not as widespread as in the cases of hemorrhage and only groups of nerve cells are injured. It is important to speculate as to whether the hemorrhage was the sole factor producing the paralysis in the alternating circuit series. Since the tracts were not severely injured in the present group sensation was not abnormal. Irritation of the autonomic cells of the cord produced the priapism and severe damage of similar cells was responsible for the incontinence. The atony of the bladder musculature was not great enough to produce hemorrhages and subsequent hematuria. As the cells tended, to some extent, to recover their functions, the abnormalities became, in some cases, less marked.

Nine of the rats recovered and lived for periods from 5 hours to 3 days. There is considerable infiltration of the ventral columns of the cord with phagocytic cells. The motor cells, in general, stain deeply 5 hours after the injury but the Nissl granules are poorly defined even after a period of days. Many cells are necrotic.

The injury of the Purkinje cells in the group that survived may be seen in Fig. 11. The severely injured cells become shrunken and pyknotic, forming a conspicuous group, contrasting sharply with the normal cells. Others (Fig. 13) not as severely injured show large vacuoles in the cytoplasm Fig. 14 again demonstrates the dead cells. Large numbers of Purkinje cells are injured and the damage is greatest on the dorsal surface of the cerebellum.

Fig. 21 is a photograph of the dorsal nucleus of the vagus from the brain of a rat surviving the shock for 4 days. The shrunken, pyknotic, dead cells are sharply contrasted with the normal autonomic cells. A somewhat different pathological picture in the dorsal nucleus of the vagus is given in Fig. 20. At the lower edge is a fairly normal cell and at the upper edge a shrunken pyknotic one. Between are two cells that have completely disintegrated.

Fig. 19 shows the injury as observed in the superior olive of a rat that survived the injury. Contrasted with normal cells are others with deeply staining cytoplasm and nucleus.

Rats Subjected to Continuous Circuits at 500, 220 and 110 Volt Potential

The histological findings in groups subjected to these voltages will be summarized together. Only one type of abnormality was observed as an immediate result of the injury that has not been mentioned in

connection with the 1000 volt circuit group. Five rats subjected to a continuous circuit at 500 volts exhibited typical clonic convulsive seizures, some 5 or 10 minutes after the shock. These convulsive attacks occurred spontaneously or upon touching the rats, which were definitely hyperirritable. They never continued longer than an hour and the rats were perfectly normal thereafter. No gross lesions were demonstrable in the central nervous system. No histological change was found to explain the convulsions; it is true that these animals lived for some time after the injury and after the convulsions had ceased. The outer portion of the cytoplasm of many of the ventral horn cells was relatively free from granules. No lesion was found in the vestibular nuclei.

The cellular injury in the rats that died immediately was much less severe after shocks with the lower voltages. In like manner, recovery of the abnormal cells was more rapid. Some trauma to the Purkinje cells was observed with the longer applications of the current. There was in every case a definite diminution of the Nissl granules. The impression is gained that prolonged artificial ventilation of the lungs might have saved many of these animals.

Rats Subjected to an Alternating Circuit at 1000 Volt Potential

Twenty-eight rats were exposed to an alternating circuit at 1000 volt potential; only one received the current for 4 seconds and the remainder for 1 to 3 seconds. The current produced a severe injury at the site of the electrodes; in the case of the head electrode there was often a burning of the brain. Of the 28 rates, 11 were paralyzed; 9 died at once and 8 were normal.

At autopsy ten of the eleven paralyzed rats showed a typical hemorrhagic lesion of the spinal cord that could be recognized grossly. They all died within 3 days after the injury. Sections showed a similar hemorrhage in the eleventh so that it may be assumed that paralysis in each case was due to a hemorrhage into the spinal cord.

These hemorrhages occurred frequently in the rats shocked with alternating circuits at all voltages. The bleeding occurred most commonly in the lower thoracic region, the hemorrhage extending in both a cranial and caudal direction. It was most commonly produced by the rupture of the posterior spinal artery and from this point the blood spread out into the posterior columns (Fig. 1). Several

rats that appeared normal during life proved to have a localized injury of this type. With further extension, the blood broke into the central canal of the cord which permitted a greater spread. The anterior horn cells and nerve fibers of the ventro-lateral columns were frequently pressed upon and paralysis ensued. In many cases the spinal cord was necrotic in the thoracic region (Fig. 2), indicating a complete transverse lesion. It is interesting that a spinal ganglion beside a completely necrotic area of spinal cord showed little change in the cells. The cytoplasm of these cells was pale and contained few granules.

The hemorrhage did not always involve the posterior column fibers first; other hemorrhages were observed in the ventro-lateral columns. It was rather common to find blood bilaterally in the ventral columns due to the rupture of the small arteries that travel with the ventral roots. The sensory pathways were injured so that nocuous stimuli applied to the posterior portion of the body elicited no response. The rats were in every case incontinent and, after a few hours, large amounts of blood appeared in the urine.

At autopsy it was found that the bladder was filled with blood clot. The atonic bladder had become so enormously distended with urine that hemorrhages had occurred in the wall and blood escaped into the urine. The paralyzed rats were never active and never ate following the shock. They could only be kept alive for relatively short periods.

The mechanical pressure of the blood had a severe effect upon the motor cells of the cord; this is shown in Fig. 3. The granules in the cells are very sparse. In the largest cell there are a few granules around the nucleus but the outer portion of the cytoplasm is quite free from them. The nucleus of the large cell is displaced toward one edge of the cytoplasm.

Concerning the mechanism of the hemorrhage, one possible theory is this. During the passage of the current an extremely powerful contraction of all the musculature of the body occurs. This is strong enough to tear the muscle fibers and frequent hemorrhages are found, for example, in the deep back musculature. The sudden flexion of the back that occurs when the current is applied may be sufficient to cause a rupture of small arteries in the cord. With a continuous circuit the contractions of the musculature are not as strong and the hemorrhages in the nervous system are few. The hemorrhage may, on the other hand, be attributed to venous stasis during the shock and the high blood pressure that immediately follows the injury.

Developing hemorrhages in the spinal cord were found in four of the rats that died as an immediate result of the shock. Likewise small hemorrhages, involving the posterior columns of the cord only, were present in two of the rats that appeared perfectly normal after the injury.

In all of the rats subjected to the alternating circuit at this potential, there was some injury of the dorsal surface of the brain, due to burning in the region of the head electrode.

This injury was severe over the cerebral cortex. There was a superficial loss of gray matter and the remaining edge was left as a deeply staining seared area. In several of the rats that survived an abscess occurred in this area. Large circular holes occurred just beneath the surface of the cortex without relation to the blood vessels. These are thought to be produced by the great generation of heat. The surface of the cerebellar cortex was, in many cases, injured in a similar manner and the cell injury was greatest close to the surface. In the brain stem there were clear, unstained areas surrounding the small blood vessels. These vacuolated areas have been described in the brains of electrocuted criminals.

The changes in the nerve cells may be described first as they were seen in the nine rats that died as an immediate result of the shock.

The abnormalities are marked and increase in severity in direct ratio with the time that the current is allowed to flow. After a 3 second application the granules in the ventral horn cells are greatly decreased in number. Fig. 4 may be taken as an example of such a cell although the animal was injured with a continuous circuit. The cytoplasm is shrunken and the irregular outlines of the cell are enclosed by a dilated perivascular space. Often it appears that the cell membrane has ruptured and the granules escaped.

The histological appearance of a group of cells, for example the ventral horn cells, is not uniform in a section. While the previous description holds for many of them, others appear shrunken and more deeply stained than normal. In these deep staining cells the granules can no longer be seen and the whole cytoplasm takes the deep blue color. Others cells appear almost normal in appearance. It is clear, therefore, that the nerve cells of the same group are not injured to an equal amount by the electric current; some are killed, some are injured and others are practically normal.

Perhaps the most important information concerning the extent of the cell injury is obtained from an examination of the cell nucleus. This has been illustrated by the photomicrographs from sections of rats injured with the continuous currents.

Thus the cell in Fig. 4 has a relatively normal nucleus with the chromatin arranged in finer skeins and the nucleolus sharply defined. In two cells in Fig. 3, normal nuclei are seen. They appear in the cells as relatively clear areas in a cytoplasm packed with deeply staining granules. In Fig. 7, showing cells severely injured by the current, the nuclei have a different appearance. The whole nucleus

is shrunken and takes a uniform bluish stain with scattered, deeper staining chromatin strands. It contains a swollen nucleolus. In the lower of the two cells in the photograph, the edges of the nucleus are irregular in outline.

The nerve cell in Fig. 6 is even more severely injured. The nucleus is greatly shrunken and is surrounded by a white, unstained area which is free from granules. The whole nucleus stains a uniform bluish color in which chromatin and nucleolus cannot be distinguished. A similar condition is seen in the darkly staining cell in Fig. 8.

From a study of this material, it has been concluded that a prognosis concerning the future recovery or death of a cell can be given from examination of the nucleus. If the nucleus appears normal, it is to be expected that the cell will recover its normal structure and function, no matter how severe the injury of the cytoplasm and granules. Thus, it is thought that the damaged cell in the lower portion of Fig. 8 has a good chance of recovery. On the other hand, a uniform staining, dark, shrunken nucleus betokens a cell that has no potentialities for recovery. Further support for this theory will be given in later pages of this report.

It has already been suggested that histological examination of the nerve cells from the central nervous system of rats that died as an immediate result of the shock shows that not all the cells were equally damaged. A whole range of variation may be observed in one single group. This variation may be due in some way to differences in the chemical composition or relative fatigue of these different cells. It may well be that death or functional damage following the shock is proportionate to the number of cells severely injured.

Changes similar to those described for the somatic motor cells in the cord are found in the brain stem and in particular, in the dorsal nucleus of the vagus. A slight damage of cell groups in the medulla might account for temporary respiratory block; more severe injury means the death of the rat.

The Purkinje cells of the cerebellum are easily injured by toxic poisons, by anemia and similarly by the electric currents. In sections from normal animals, it is frequently observed that a few of these cells are shrunken and pyknotic. The injury is so much more extensive in the present series that there can be no doubt as to its pathological significance.

These changes in the Purkinje cells are more marked in the rats that survived the shock by hours and days and less clearly demonstrable in those that died following the injury. Fig. 10 shows normal Purkinje cells and the light staining, large cells in Fig. 11 are even more characteristic. The cytoplasm contains fine scattered granules that give a foam-like appearance to the cell. In the rats that died, following the shock, the nucleus tends to stain a rather uniform blue color in which the chromatin and nucleolus can still be distinguished (Fig. 12). The granules in the cytoplasm are indistinct and the whole cell stains a light blue.

The animals that recovered were allowed to live for various periods of time; signs of recovery of the cells are clearly seen in the sections.

One rat died at the end of 6 hours. The number of cells with blue staining nuclei suggests that the cell damage had been profound. Even at this time the picture is different from that immediately following the shock. Certain cells have acquired a deeper staining of the cytoplasm, although it does not appear as distinct granules but, rather, as a uniform blue color. This is most clearly seen in the Purkinje cells. The latter are always more severely injured close to the dorsal surface of the cerebellum where the concentration of current was doubtless greatest.

Rats that have survived 2 and 4 days show further repair of the nerve cells.

The anterior horn cells at the end of 2 days are still shrunken and surrounded by a large perivascular space. The cytoplasm stains deeply and the granules are beginning to stand out clearly. Many of the nuclei have a bluish tint and the nucleolus is swollen. Other cells have made no recovery and appear to be disintegrating. After 4 days the cells are not yet completely normal in the structure and arrangement of granules in the cytoplasm.

Repair seems to be slower in the brain stem so that even at the end of 4 days many cells in the dorsal vagus nucleus contain few granules in a cytoplasm that is distinctly vacuolated. Here, deeply staining, dead cells may also be seen.

The picture in the Purkinje cells of the cerebellum is very characteristic at this stage and is represented in Fig. 11. Numbers of the cells, scattered among those of normal appearance, are shrunken and pyknotic, staining an intense color. These cells are found everywhere but are more common near the dorsal surface of the cerebellum. Where many of them are dead there appears to be a great disintegration of nerve fibers so that the Purkinje cell layer is easily pulled away from the granular layer, leaving a marked fissure in the sections.

The histological picture of the nerve cells in the paralyzed rats corresponds closely in general to that described for those that appeared normal when killed.

Due to the hemorrhage and subsequent degeneration of nerve fibers, the cord and brain stem were found to be markedly soft when removed at autopsy. In the completely necrotic portions of the cord the nerve cells are of course completely disintegrated; in other areas they are compressed by the hemorrhage producing characteristic changes. The nucleus is displaced to the periphery of the cytoplasm and there is a distinct loss of granules particularly in the periphery of the cytoplasm. In the areas of hemorrhage numbers of white blood cells are found, a majority of them polymorphonuclear. They wander into the gray matter around the injured cells.

The nerve cells in the brain stem make a slow and incomplete recovery in the paralyzed rats. After 2 days, the length of life of most of this series, the dorsal vagus cells still show an injury almost comparable to that found immediately following the shock.

With these marked changes found in the nerve cells it does not seem strange that numbers of the rats never breathed following the shock and that artificial respiration had to be used in the case of the others. The inactivity for the first 24 hours in those that recovered completely and throughout their lives in the paralyzed rats may be explained on the same basis.

Rats Subjected to Alternating Circuits at 500, 220 and 110 Volt Potentials and at 1000 and 500 Volt Potentials with the Voltage Not Maintained

It is scarcely justifiable to discuss the histological picture found in the central nervous systems of each of these groups of rats separately The relative mortality and paralysis produced at those different potentials have been thoroughly summarized in a previous paper. It is sufficient to point out that the number of rats killed or paralyzed became smaller, as the voltage and the amperage of the circuit were decreased. The shock could be made lethal, however, in every case by increasing the time of current contact.

The changes found in the nervous system were similar to those already described.

Actual burning of the brain occurred only with circuits at 1000 and 500 volts potential. Hemorrhages in the spinal cord were demonstrated in all the rats that were paralyzed following the shock. Hemorrhages were common in the groups in which the potential of 1000 or 500 volts fell considerably, immediately following the initial shock. It must be assumed, therefore, that the initial shock

at high voltage is very important in the development of this lesion. A section from the spinal cord of one of these rats is reproduced in Fig. 1. The conclusion was reached that hemorrhages are seldom responsible for the immediate death of the animal. Except in those cases where a hemorrhage occurs in the fourth ventricle, the respiratory block or paralysis must be due not to hemorrhage but directly to cell damage. This will be clearer when the group of rats shocked with the continuous circuit has been discussed.

As regards the cell changes, they were quite marked in the 500 volt series but decreased markedly in the 220 and 110 volt groups. They could be demonstrated, however, in each group by lengthening the duration of the contact. Recovery of the cells in the rats that survived was more rapid at the low voltages. More of the cells showed the potentialities of recovery. In the groups subjected to the lower voltages, it is probable that death was due, in most cases, to a temporary block in the respiratory center.

DISCUSSION

The injuries produced by the alternating circuits were somewhat different from those produced by the continuous. Hemorrhages in the central nervous system were common after contact with alternating circuits; the damage to the nerve cells was more severe after injury with the continuous circuits.

The hemorrhages following the alternating current shocks occurred not only in the nervous system but in other portions of the body. Blood vessels were found to have ruptured in the mucous membranes and autopsy study showed hemorrhages in the striated muscles. Blood escaped into the subarachnoid space, into the ventricles of the brain and the central canal of the spinal cord. Hemorrhages in the fourth ventricle were responsible in some cases for respiratory paralysis. Other characteristic hemorrhages occurred in the substance of the brain and spinal cord. In the latter position they caused in some cases a complete transverse necrosis and were responsible for the paralysis of the posterior portion of the body. As a result of the cord injury, hemorrhages occurred in the wall of the atonic and distended bladder and blood appeared in the urine. In the cord the pressure of the blood produced characteristic histological changes in the ventral horn cells.

The mechanism of these hemorrhages after injury with alternating circuits is not entirely clear. It has been suggested that it is due to the strong tetanic contraction of the back musculature at the time

the circuit is closed. It might on the other hand be attributed to the marked venous congestion that pertains during the shock or the sharp rise in blood pressure immediately following the injury.

Developing hemorrhages in the central nervous system were found in many of the rats that died at once and small hemorrhages in many that appeared to recover completely. If the hemorrhages involved a large area in the brain stem or cord the animal obviously had no hope of complete recovery. These hemorrhages were common with all voltages although they were more frequent with the higher potentials. They were obviously a large factor in the immediate or subsequent death of rats subjected to the alternating circuits.

With the alternating circuits at the higher potentials of 1000 and 500 volts there was injury of the dorsal surface of the cerebral and to a less extent of the cerebellar cortex. These regions were close to the head electrode where the density of the current must have been great. Indeed injuries of the subcutaneous tissue and bone were evident at autopsy. The surface of the cortex was seared and the edge stained an intense blue. Often large cavities were observed in the gray matter of the cortex and, less commonly, scattered throughout the central nervous system. They are supposed to be due to the collection of gases produced by the intense heat of the injury. They were found in rats that appeared normal after the injury. The cavities were surrounded by a deeply staining, condensed area of brain substance. They were not necessarily perivascular. If the rats survived the shock for several days, an abscess occurred on the surface of the cortex in a number of cases. This is interesting in that marked cellular reaction is believed to be retarded following electrical injuries.

The nerve cells showed marked abnormalities particularly with circuits at 500 and 1000 volts. After study of the sections it was believed that an accurate prognosis could be given concerning the death or subsequent recovery of the cells. If the nucleus was shrunken and stained an intense deep color so that chromatin skeins and nucleolus could not be distinguished, the cell was dead. In slightly less severe injuries the nucleolus was swollen and the whole nucleus was shrunken and stained darkly. A cell with a normal clear nucleus has the potentialities of recovery. In many cases the Nissl granules were greatly decreased in number and the outlines of the cells shrunken and irregular.

The cellular injury appeared to be selective so that a few abnormal cells would be found among a group comparatively normal. The Purkinje cells seemed to be most markedly injured and changes in these cells were found even after contact with circuits at 110 and 220 volts. Likewise injured cells were observed in the dorsal nucleus of the vagus, among the somatic motor cells of the cord and in the olives. The primary sensory neurones are resistant to injury.

In the animals that recovered, the cytoplasm of the cells became rather shrunken and stained deeply within a few hours but it was several days before the granules looked normal and distinct. The dead cells became more shrunken and pyknotic and finally disintegrated.

The cellular damage was even more marked in the rats subjected to continuous circuits at 1000 and 500 volts potential. This explains the fact that at 500 volts the continuous circuit was as dangerous as the alternating for the rats; at 1000 volts it was far more dangerous. Almost no hemorrhages were found after the continuous circuit shocks. The changes in the nerve cells have been adequately illustrated in the photomicrographs.

A small number of rats were paralyzed after contact with the continuous circuit but the picture was not the same as with the alternating circuits. There was a tendency to gradual recovery. Incontinence was not as marked a feature and blood was never present in the urine. Moreover there was no sensory loss in the paralyzed area. Microscopic examination showed that these paralyses were due, not to hemorrhage, but to the injury or death of groups of cells in the cord. These cells appeared shrunken and both the nucleus and cytoplasm stained a deep color. An infiltration of the anterior horns was present and phagocytosis of these dead cells observed. Convulsions and priapism occurred in this series and were probably due to the irritation of cells in the nervous system.

The alternating and continuous circuits at 220 and 110 volts potential produced much less injury to the nerve cells. Hemorrhages were common with the alternating circuit. It seems possible that a respiratory block was produced in these groups which might be relieved with adequate, long continued, artificial respiration. It is true, however, that long continued contacts with these circuits greatly increased the damage in the nervous system.

It is believed that definite abnormalities have been demonstrated in the nervous system that account for the death and abnormalities produced in the rats. With the circuits at lower potential these changes are not as marked and it is quite probable that temporary respiratory block was responsible for death. When a large hemorrhage occurred in the brain or cord or where a large number of nerve cells were permanently damaged, recovery could not be expected.

The fact that nerve cell changes may be demonstrated in animals dying at once following the electric shock indicates the explosive suddenness with which these abnormalities are produced. It is almost as if the cell had been blown to pieces by a charge of dynamite. The electric current must react violently upon the electrolytes producing profound dislocations of the nucleus and cytoplasmic material. While most other pathological changes in cells develop slowly this powerful physical agent produces abnormalities that may be demonstrated at once by means of even the rather gross methods of histological study available at the present time.

SUMMARY

The alternating and continuous circuits produced different types of lesions in the central nervous system. Hemorrhages were common after alternating current shocks and few hemorrhages were observed in the continuous circuit group. With both types of circuits at 1000 and 500 volts potential, severe abnormalities in the nerve cells were observed. These were more marked in the continuous circuit group. A uniformly staining, shrunken, pyknotic nucleus was taken as a criterion of nerve cell death. The Purkinje cells of the cerebellum were most susceptible to the current. Injured cells were studied in the dorsal nucleus of the vagus, in the somatic motor group, among the primary sensory neurones and in the olives. Changes in the histological structure of the cells in reference to recovery have been discussed.

Injury to the cerebral and cerebellar cortices occurred on the dorsal surface close to the head electrode. Small cavities were produced, particularly in the cerebral cortex, as the result of the circuit contact.

With the continuous and alternating circuits at 110 and 220 volts potential less severe changes were observed in the nerve cells although

hemorrhages were common in the alternating circuit group. It must be assumed in these cases that death was due to respiratory block rather than actual death of the cells.

BIBLIOGRAPHY

Barker, L. F., The nervous system and its constituent neurones, G. E. Stechert and Co., New York City, 1909.

Buzzard, E. F., and J. G. Greenfield, Pathology of the Nervous System, Paul B. Hoeber, New York City, 1925.

Cannon, W. B., and I. R. Burket, The endurance of anemia by nerve cells in the myenteric plexus, *Amer. Journ. Physiol.*, 1913, **32**, 347.

Gomez, L., and F. H. Pike, The histological changes in the nerve cells due to total temporary anemia of the central nervous system, *Journ. Exp. Med.*, 1909, **11**, 257.

Langworthy, O. R., and W. B. Kouwenhoven, An experimental study of abnormalities produced in the organism by electricity. *Journ. Industrial Hygiene*, 1930, **12**, 31.

Malone, E. F., Observations concerning the comparative anatomy of the diencephalon. *Anat. Rec.*, 1912, **6**, 281; Recognition of members of the somatic motor chain of nerve cells, *Anat. Rec.*, 1913, **7**, 67.

MacMahon, H. E., Electric shock, *Amer. Journ. Path.*, 1929, **5**, 333.

Mott, F. W., and E. Schuster, Examination of the brain of a man who lived seven hours after receiving a shock of 20,000 volts. *Proc. Roy. Soc. Med.*, Sect. Pathology, 1909, **3**, 140.

Mott, F. W., and T. Uno, Microscopic examination of the brain in cases of surgical shock. *Proc. Roy. Soc. Med.*, Sect. Neurol., 1922, **1**, 25.

Spitzka, E. A., and H. E. Radasch, The brain lesions produced by electricity as observed after legal electrocution. *Amer. Journ. Med. Sci.*, 1912, **144**, 341.

Urquhart, R. W. Ian, Experimental electric shock. *Journ. Industrial Hygiene*, 1927, **9**, 140.

EXPLANATION OF PLATES

PLATE 31

FIG. 1. The rat was subjected to an alternating circuit at 1000 volts for 1 second. A large hemorrhage is present in the posterior columns of the cord. The rat survived the shock and behaved normally until killed 4 days later.

FIG. 2. There is a complete transverse necrosis of the cord following an extensive hemorrhage. The nerve roots appear fairly normal. The posterior portion of the body was paralyzed and there was no sensory response from the paralyzed area. Incontinence followed the shock and large quantities of blood were present in the urine. The animal was inactive and did not eat. The injury followed contact with an alternating circuit at 1000 volts potential for 3 seconds.

FIG. 3. Somatic motor cells of the anterior horn of the spinal cord which have been subjected to pressure from a hemorrhage. The nuclei appear normal except that one occupies an eccentric position. There are few granules in these cells. In the largest cell the granules are more abundant around the nucleus and the peripheral cytoplasm is clear and unstained. This rat was subjected to an alternating circuit at 1000 volts for 1 second.

FIG. 4. This is a somatic motor cell from the anterior gray matter of the spinal cord. The nucleus is normal. The cytoplasm on one side stains deeply and contains many granules; on the other side toward the periphery of the cord the granules are few and the cytoplasm is shrunken and irregular. The rat was shocked with a continuous circuit at 1000 volts for the time required to close and open the switch manually and died at once.

FIG. 5. The nucleus of this primary motor neurone of the cord is normal. Granules are present around the nucleus but the outer cytoplasm is relatively free from them. The rat was injured with a continuous circuit at 1000 volts for ½ second and died at once.

FIG. 6. This somatic motor cell of the cord must be considered damaged beyond hope of recovery. The nucleus is shrunken and stains a uniform black so that the nucleolus and chromatin skeins cannot be distinguished. It is surrounded by a clear area indicating the normal size of the nucleus. The granules in the cytoplasm look fairly normal. The rat died at once following contact with a continuous circuit at 1000 volts for 1 second.

FIG. 7. The nuclei of these two cells are shrunken and the outlines are irregular. The nucleoli are swollen although they can still be distinguished. They represent an intermediate amount of nuclear damage between Figs. 5 and 6. These anterior horn cells have many granules in the cytoplasm. In the lower cell the cytoplasm is shrunken; the outlines of the cell are irregular and indistinct. The rat was injured by a continuous circuit at 1000 volts for ½ second and died at once.

FIG. 8. The deep staining cell resembles that in Fig. 6. The other two anterior horn cells have shrunken, vacuolated and irregular cytoplasm with few granules. The lower cell however has a normal nucleus. The rat was subjected to a continuous, 1000 volt circuit and died at once.

FIG. 9. These are greatly shrunken, pyknotic anterior horn cells. The rat was shocked with a 1000 volt continuous circuit. Following the injury the posterior portion of the body was paralyzed. At the end of 6 days the rat was killed. Nucleoli can be observed in the cytoplasm of the two cells on the right. Phagocytic cells have eaten into the cytoplasm of the two lower nerve cells.

PLATE 32

FIG. 10. These are normal Purkinje cells. The cytoplasm contains delicate flaky granules.

FIG. 11. Normal Purkinje cells are seen in the upper portion of the photograph. The lower cells are shrunken and pyknotic and no distinction between nucleus

and cytoplasm can be made out. This rat survived a contact with a 1000 volt continuous circuit for 4 days.

FIG. 12. All the Purkinje cells in the photomicrograph are damaged. The nuclei are shrunken and stain a uniform black. The cytoplasm also stains deeply. These cells show the immediate effect of the injury since the rat died at once after a 1 second exposure to a 1000 volt continuous circuit.

FIG. 13. These Purkinje cells show a different type of injury in that the cytoplasm is greatly vacuolated, particularly at the edge next to the granular layer. The rat survived 4 days after contact with a continuous 1000 volt circuit.

FIG. 14. This picture again shows shrunken dead Purkinje cells from the central nervous system of a rat that survived 6 days after contact with a 1000 volt continuous circuit.

FIG. 15. These cells are from the mesencephalic nucleus of the trigeminal and indicate changes in primary sensory neurones. Sensory cells are relatively resistant to electrical injuries. One cell however has a shrunken pyknotic nucleus. The rat died at once after shock from a 1000 volt, continuous circuit for 1 second.

FIG. 16. Two cells of the mesencephalic trigeminal nucleus have injured nuclei. The nuclei are surrounded by clear areas indicating their normal size. The granules in the cytoplasm are not sharply discrete. Again the rat was subjected to a 1000 volt continuous circuit and died at once.

PLATE 33

FIG. 17. These are cells from the dorsal nucleus of the vagus. The arrangement of the granules is fairly normal for autonomic cells and the cytoplasm appears to have suffered little damage. The nuclei however stain a uniform dark color and it is thought the cells have been killed. The rat died at once following contact with a continuous, 1000 volt circuit for 1 second.

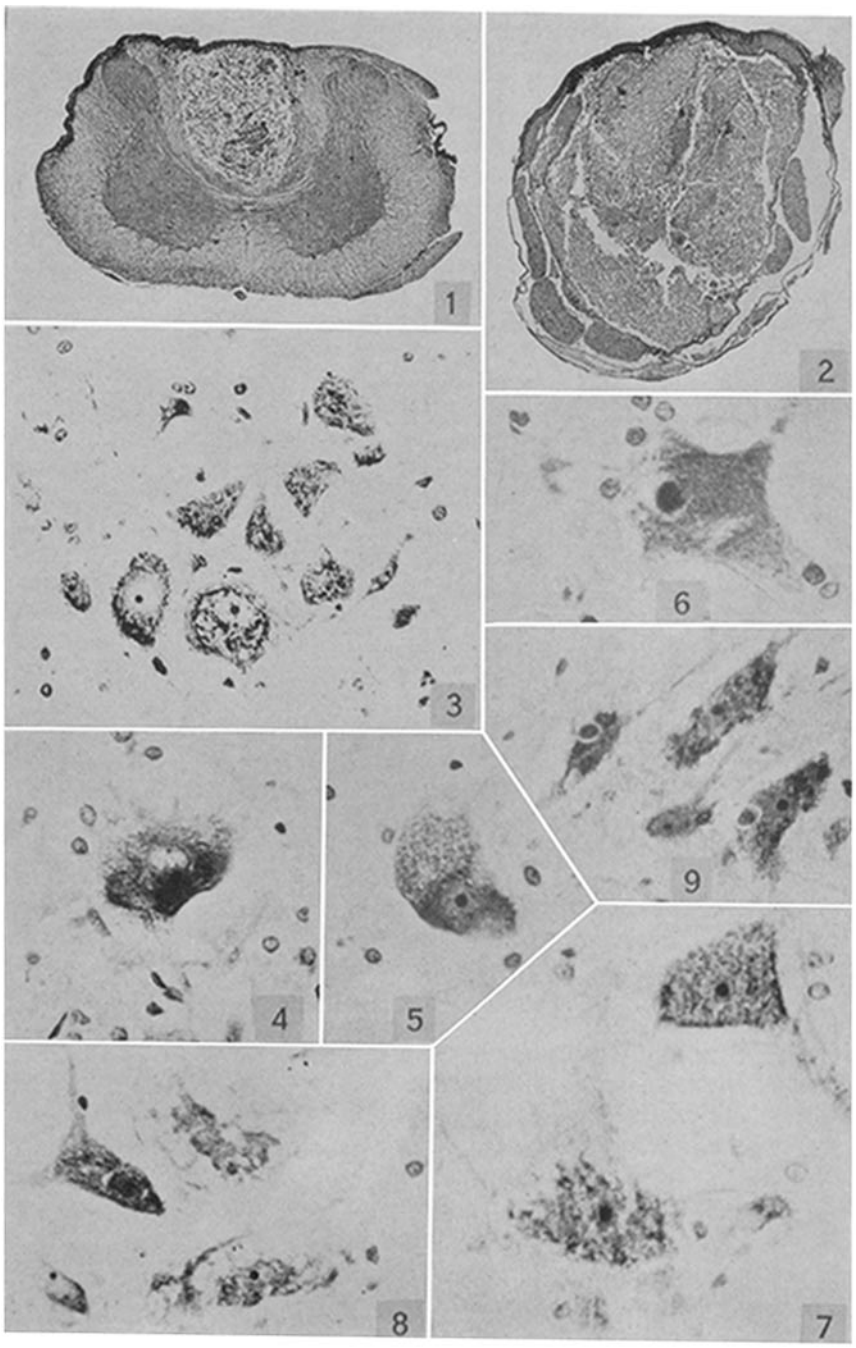
FIG. 18. The dorsal nucleus of the vagus. The upper cells appear normal; the others are damaged. One has a deep staining nucleus. The granules are irregularly arranged and the whole cytoplasm of one cell stains darkly. The rat died at once after a 1000 volt continuous circuit application for 1 second.

FIG. 19. Cells of the superior olive. The rat survived an instantaneous, 1000 volt continuous circuit contact for 4 days. No cell in this group is normal.

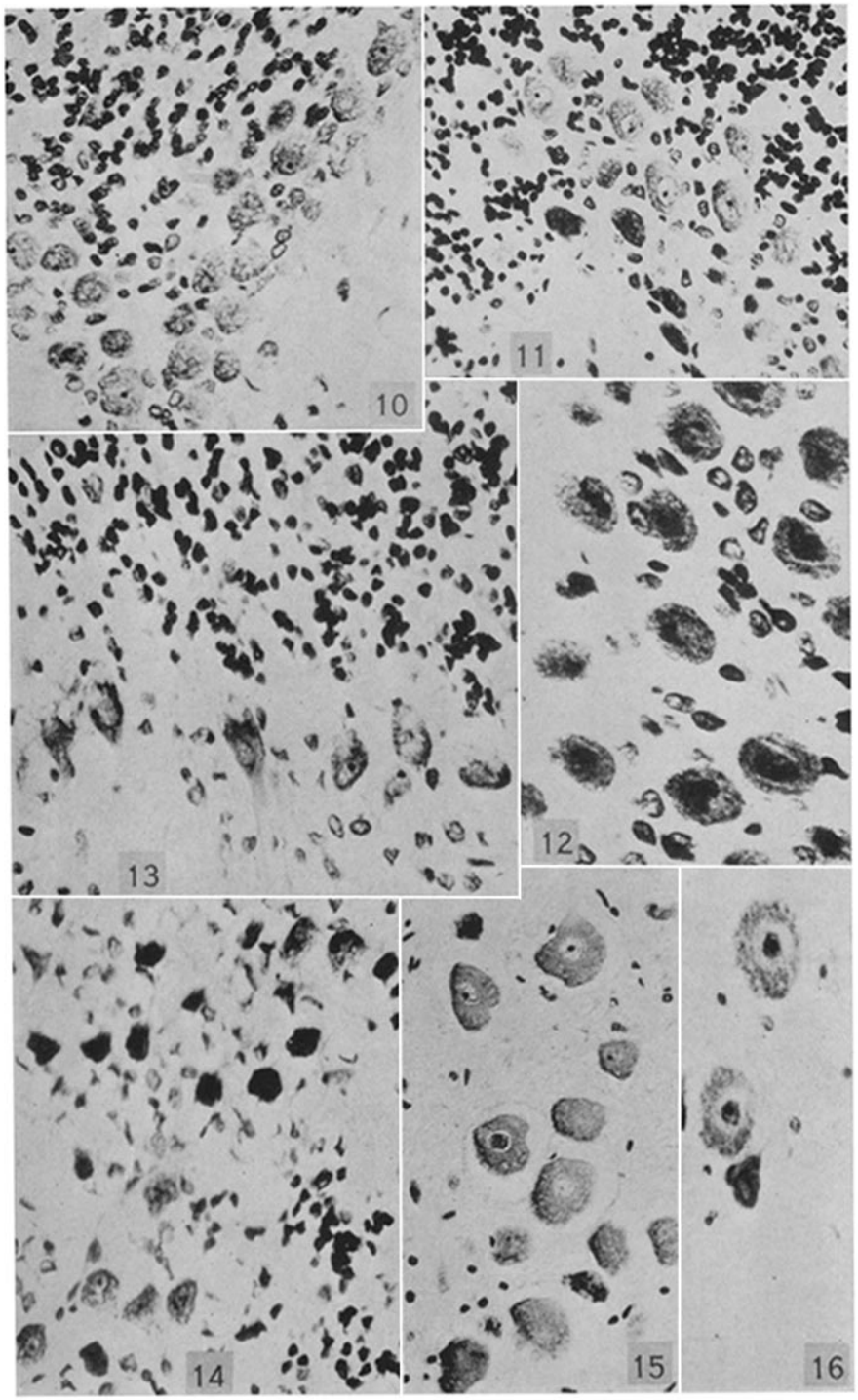
FIG. 20. Dorsal nucleus of the vagus. This rat lived 6 days after an instantaneous, 1000 volt continuous circuit injury. The cell in the lower portion of the field is normal. At the upper edge a pyknotic cell is seen. Between these are shadows of two disintegrating cells.

FIG. 21. The distinction between normal and pyknotic cells in the dorsal nucleus of the vagus is clear. The rat lived 4 days after instantaneous contact with a 1000 volt continuous circuit.

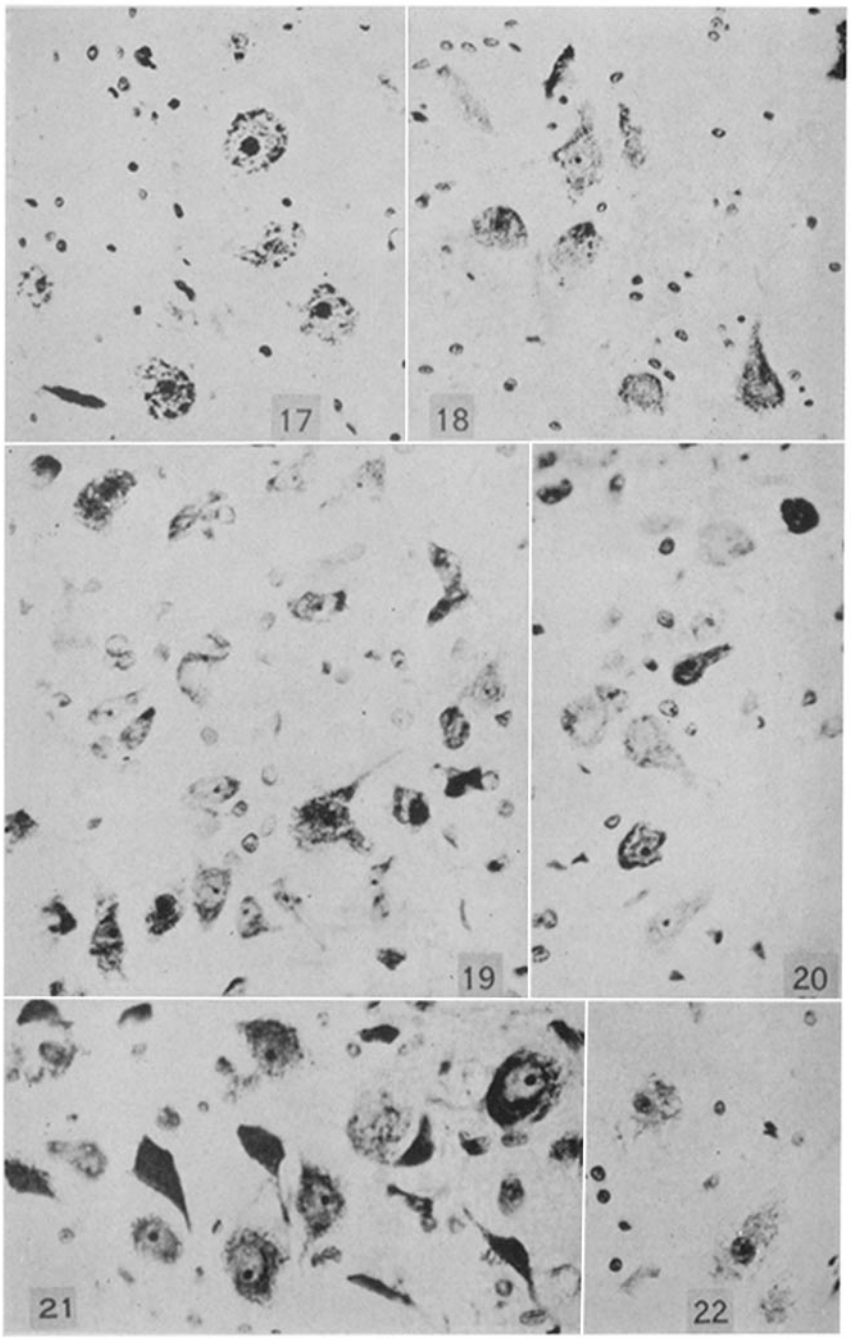
FIG. 22. These cells of the dorsal nucleus of the vagus show severe injury of both the nucleus and the cytoplasm. The rat died at once after contact with a 1000 volt continuous circuit for 1 second.



(Langworthy: Electrical injuries to nervous system)



(Langworthy: Electrical injuries to nervous system)



(Langworthy: Electrical injuries to nervous system)