The Brain Changes Associated with Electrical Shock Treatment: A Critical Review

Bernard J. Alpers, M.D.*
Philadelphia, Pennsylvania

Since the introduction of electrical shock treatment as a means of combating psychiatric disorders, great interest has been manifested in the brain changes which occur as the result of treatments by this method. Sufficient time has transpired so that it is possible now to evaluate the results of experimental and clinical reports. For this reason, it has seemed desirable to review critically the brain findings in experimentally produced electrical shock, as well as in the human wherever this has been possible. A survey of recent reports relative to this problem would lead one to believe that the matter is settled and that there is nothing further to be said. There are differences of opinion, however; but even if the matter is settled, it is well for us to recognize that this is so, in a problem which was not so long ago, quite controversial. Moreover, electrical shock treatment is a severe shaking-up process, the prescription of which should not be ordered lightly, despite its efficacy in some forms of psychosis. Recognition of what occurs in the brain during the course of shock treatment may well make us pause before adding injury to insult too promiscuously in the course of shock treatment. Though the method has been used widely in the treatment of psychiatric disorders, it has not been without its opponents who look with horror on its use and who regard it as an insult to the nervous system.

With these few words of apology, let us proceed to a review of the record in the problem of the brain changes in electrical shock treatment.

Review of Experimental Literature

In order to clarify the approach to a rather involved problem, I think it may be advisable first to summarize the reports in experimentally induced electrical shock and then to survey those pertaining to the human. In this fashion it may be easier to visualize the changes in the two categories.

Since the report of brain changes in the cat after experimentally induced electrical shock was the point of departure for a number of subsequent controversial studies, it may be well to begin with a survey of reports in which changes have been demonstrated in the nervous system.

Electrical Shock with Associated Brain Changes

In a group of 30 cats given electrical shock, Alpers and Hughes [1] found evidence of damage to the nervous system in a high percentage of cases. Of the 30 cats studied, 14 had subarachnoid hemorrhage in some degree and 9 had hemorrhage within the brain substance itself. The subarachnoid hemorrhage was not extensive, except in a few instances. It was usually found scattered over the cerebral hemispheres, but in a few instances it was located around the medulla. The cerebral hemorrhages were all punctate except in two instances, in one of which there was a hemorrhagic infarct and in another a fairly extensive cerebral hemorrhage with hemorrhage into the ventricles. The hemorrhages varied widely in number and size. They were for the most part scattered, appearing at times in a single area of the cortex and nowhere else, or occurring as scattered punctate hemorrhages elsewhere in the brain or brain stem. All parts of the brain were vulnerable—the cerebral hemispheres, the cerebellum, third ventricle, and hypothalamus.

Similar results were recorded in rabbits by Heilbrunn and Weil.[2] The outstanding feature of their experiments was the presence of localized hemorrhages in the pia-arachnoid at the base of the brain and over the cerebellum and spinal cord. These were combined with small pericapillary and perivenous hemorrhages, localized chiefly in the brain stem and spinal cord. Organization of the hemorrhages was clearly evident in those animals which survived for a sufficiently long period of time, thus eliminating the possibility that the hemorrhages were agonal. Similar changes were evident in the areas of hemorrhage in the meninges, where a mild proliferation of the pial tissue could be seen. Astrocytic proliferation of mild degree was seen around the hemorrhages within the brain stem and spinal cord. The ganglion cells in the immediate vicinity of the hemorrhages were shrunken and pyknotic.

Studies carried out on dogs by Neuberger, Whitehead, and Ebaugh [3] indicate that changes occur in the brain following electrical shock treatments, but in the opinion of these investigators, they are not serious. The nerve cells showed widespread damage, sometimes to the point of ischemic cell changes and severe damage. Satellitosis and neuronophagia were found occasionally. In some small areas only pale, ischemic, ghost-like cells remained. Many cells showed the changes typical of chronic cell damage, the cells being small, dark and shrunken. Slight proliferative changes were present in the astrocytes and microglia. Myelin sheath damage was found in a few animals. Vascular dilatation and minute hemhorrhages were found in the cerebral cortex, in the meninges, and around the ventricles in some of the brain.

The observed changes, though definite, were not regarded as serious. Most of the nerve cells and nuclei were well preserved; hence the description of widespread damage of the nerve cells must be regarded to mean widespread in distribution but not in number. The changes described in the nerve cells were regarded as reversible.

A study of the effects of electrical shock treatments in rats by Heilbrunn [4] reveals the production of hemor-

*From the Department of Neurology, Jefferson Medical College, Philadelphia, Pennsylvania.

rhages both in the pia-arachnoid and the brain substance. The meningeal hemorrhages were most numerous and extensive at the base of the brain and considerably less frequent over the cerebral hemispheres and the cerebellum. The hemorrhages into the brain substance were found in all the lobes indiscriminately, in the hypothalamus and cerebellum. They had a particular predilection for the pons and medulla. The hemorrhages were petechial in character. Organization of the hemorrhages was observed both in the meninges and brain substance.

There appears to be some evidence therefore from experiments in rats, rabbits, cats and dogs, that there is brain damage with the use of the electrical current for treatment purposes. I shall not discuss here the validity of these findings or the objections which have been raised to them. It seems best simply to record them here and to leave the controversial aspects for general comment. Hemorrhages have been found in the meninges, especially over the brain stem, in the cerebrum, and in the cerebellum, associated with relatively little glial reaction, but showing indications of organization.

Opposed to these findings are several studies which cast considerable doubt on the validity of the observations recorded.

ELECTRICAL SHOCK WITHOUT BRAIN DAMAGE

In a study of three dogs treated in a fashion similar to that of humans, Lidbeck [5] found in one animal a recent perivascular subcortical hemorrhage in the frontal lobe, three capillaries filled with fibrin thrombi, and shrunken nerve cells with a reduction in the number of stainable granules; in two other animals there were occasional areas in which the nerve cells showed a greater degree of shrinkage than normal. Lidbeck regarded the findings as insignificant and looked upon the results as indicating that electrical shock treatment was not dangerous.

In an effort to determine the path of the current in electrical shock, as well as to study the effects of the current on the brain tissue, Alexander and Lowenback [6] studied 23 cats, 19 of which received only single electrical shocks. It was pointed out that if changes were present, they were confined to the path of the current and were not observed beyond its calculated path. "Significant, morphologically recognizable tissue reactions, vascular or otherwise, were limited to that part of the brain which was within the path of the current; that is in our experiments they were limited to the frontocruciate lobes or parts of them. In one animal which died after multiple shocks, there were, in addition to the changes within the path, diffuse changes obviously related to the general circulatory disturbance prior to the death of the animal. In all other animals, those parts of the brain which were outside the path of the current . . . showed no morphological or histological changes, neither immediately nor at times varying from a few minutes to nine days after the shocks. Here even temporary vascular reactions were absent. The parietal and occipital lobes, the bulk of the temporal lobes and the brain stem from the thalami backwards showed in all these animals not only a perfectly normal picture of the neural parenchyma, but also a perfectly normal picture of the vascular pattern."

"Within the path significant changes could be produced wtih definite regularity. But the threshold for the production of changes which were morphologically and histologically recognizable at times varying from one half hour to seven days after shock, were rather high. No such changes were observed in animals which had received shock from 60 to 450 m.a. for times varying from 5 to 10 seconds; that is, in animals in which the current density within the path had not exceeded 0.6 to 4.5 m.a. per square millimeter of the cross-section of the path through the brain. However, in one animal which had been given a 300 m.a. shock but which was killed only 4 minutes after the shock, blanching of the anterior suprasylvian gyri bilaterally within the path of the current was noted."

It seems clear therefore from the work of Alexander and Lowenback that changes in the brain in electrical shock, when present, are confined to the path of the current. What changes were observed under these circumstances? Of the 19 cats who were given a single electrical shock 9 were described as showing blanching of the cortex, 4 had vasoparalytic stasis, and 6 were described as having no changes. In the majority of cases those animals with blanching had no changes in the nerve cells, axis cylinders, or myelin sheaths. In true instances of blanching swelling and vacuolation of the nerve cells were observed and there was swelling and effilochement of the axis cylinders, with swelling and decreased intensity of staining of the axis cylinders. In the majority of cases with blanching there were therefore either no changes, or alterations of a minor degree in the nerve cells, axis cylinders, or myelin sheaths.

Vasoparalytic stasis was found in animals which were shocked with currents of 2000 m.a. for 5 to 10 seconds with a maximum current density of 20 m.a. per square millimeter of the cross section through the path of the current. It developed therefore in animals shocked by higher currents. By vasomotor paralysis is meant congestion and extreme dilatation of the capillaries, arteries and veins, with or without, but usually with, perivenous hemorrhages.

The threshold for changes in the nerve cells, axis cylinders and myelin sheaths was found to be higher than that for vascular reactions. No changes in these structures were found in animals given single shocks of from 60 to 1500 m.a. of 3 to 10 seconds' duration and survived from 4 minutes to 7 days. No significant changes were found in an animal which received six shocks of 1500 m.a., each of which lasted two-fifths of a second.

Significant changes could be produced with single shocks of higher amperage. As in the case of the vascular reactions, the observed changes were limited to the path of the current. Reversible cell changes such as swelling and vacuolation appeared in animals which had received single shocks of 1800 m.a. for two to four seconds. After single shocks of 2000 m.a. for five seconds and more, irreversible types of nerve cell changes, predominantly severe degrees of pyknosis with bizarre cell deformities were found in cortical areas which

showed vasoparalytic stasis and where current density was great. In the marginal areas where current density was less the nerve cell changes were reversible.

Axis cylinder threshold changes were found at the 1800 m.a. level. These too were reversible in type, consisting of swelling and unraveling of the fibrillae and in a few instances fragmentation. Animals shocked with 2000 m.a. showed irreversible changes in the axis cylinders, consisting of bizarre formations, irregular swelling and shrinkage, and fragmentation.

Myelin sheath changes followed similar rules.

A further study of 13 cats by Winkelman and Moore[7] reveals no changes in the meninges and no evidence of subarachnoid or cortical hemorrhages. Changes were found in the nerve cells of the cerebral cortex in layers II and III, in the frontal and parietal cortex. These consisted of moderate pyknosis of the ganglion cells with hyperchromia of the smaller nerve cells. The changes were not different from those of the control animals. No damage was found in the basal ganglia, hypothalamus or ammon's horn. Pyknosis of the perkinje cells was found at the summit of the cerebellar folia. The spinal cord was normal. Winkelman and Moore conclude that permanent changes do not occur in electrical shock, but that intracellular and biochemical changes take place because of passage of the current and the resultant convulsion.

A study of adult guinea pigs by Windle, Krief, and Arieff[8] reveals no visible hemorrhages of neurocytological changes after single shocks of alternating current of 45 volts and 225 to 240 m.a. for ¼ to ⅜ seconds or of 100 volts and 650 to 725 m.a. for 6 to 12 seconds.

A study of the brain changes in the monkey (macacus rhesus) was made by Barrera, Lewis, Pacella and Kalinowsky.[9] The conditions of treatment were made to simulate as closely as possible those in the human. Seizures were induced three times per week with voltages varying from 70 to 135 with current times of .10 to .15 seconds. Neuropathological findings were surprisingly meagre. There were no hemorrhages, either petechial or gross. The blood vessels were normal. There were no changes in the myelin sheaths, axis cylinders, neuroglia or microglia. "The nerve cell changes were spotty in distribution and not localized to any particular portion of the brain. In the areas involved some of the nerve cells appeared shrunken with pyknosis of the nucleus, paling of the cytoplasm, and disappearance of the Nissl substance. Some of the cells were only shadow cells . . . Changes of this type occurred in small areas and the nerve cells immediately surrounding these areas were usually entirely normal . . . The incidence of such "pathological" changes bore no direct quantitative relation to any of the characteristics of the series of seizures administered, i.e., frequency, number of seizures, voltage or current time passage, type of resulting seizures." Similar changes were found in the brains of untreated animals. "The changes could therefore not be related to the electrically induced seizure and their significance in the general behavior of the animal seems relatively insignificant." Barrera and his collaborators state that "in the macacus rhesus monkeys subjected to electrically induced seizures administered at frequency, voltage, and current times definitely within the range as utilized in human treatment, there is no evidence, on the basis of our work, to indicate a relation between electrically induced seizures and histopathological changes."

Evidence is offered therefore to indicate (1) that electrical shock treatment is not dangerous, (2) that, if given within safe limits comparable to those used in the treatment of humans, it is not associated with permanent brain damage, (3) that the changes which can be detected subsequent to shock treatments are reversible and functional, that they are confined to the path of the current, and that changes when seen in nerve cells, axis cylinders and myeline sheaths are reversible in character.

I shall leave for subsequent discussion the criticism of these assertions. For the present it seems best to complete the collection of evidence by a survey of the changes which have been recorded in the human cases dying in connection with electrical shock treatment.

REVIEW OF HUMAN MATERIAL

The findings in the few human cases which have come to necropsy are almost as conflicting as in experimental animals. Alpers and Hughes[10] reported brain changes in a woman of 45 who had received 62 electrical shock treatments over a period of 5½ months, and who died 7 months after the last treatment, of cardiac failure and bronchopneumonia. The brain in this case revealed pronounced congestion in many portions of the cerebral cortex, perivascular hemorrhages, and perivascular edema. The perivascular hemorrhages were fresh in some instances, but in others there was evidence that the hemorrhage was old. Hemorrhages were seen in the thalamus, medulla, and cerebellum in addition to the cerebral cortex and white matter. Punctate hemorrhages were found under the ependyma of the fourth ventricle.

In a second patient, a woman of 79, who had had six shock treatments and died five months later there was found generalized arteriosclerosis, arteriosclerotic heart disease, sclerosis of the cortical arterioles, ischemic and chronic cell changes of the cortical ganglion cells, and an occasional perivascular hemorrhage. All the changes are probably attributable to the vascular disease of the brain.

Two additional human cases studied at necropsy were reported by Ebaugh, Barnacle, and Neuberger.[11] The first was a patient of 57 years who received 13 electrical shock treatments (85 volts and 900 m.a. for 0.15 seconds) and who died 1½ hours following the last treatment. The heart showed a soft moist discolored area in the upper part of the anterior wall and the interventricular septum, and calcified plaques in the left coronary artery. In the frontal and temporal lobes were several small areas of devastation, entirely devoid of ganglion cells and containing some ghost cells. The astrocytes in these areas were swollen and there was some proliferation of the microglia with fat granules in their processes. Diffuse degeneration of the nerve cells in the cortex was present, consisting chiefly of shrinkage and sclerosis of the cells. Ischemic cell changes were seen elsewhere in the cortex. The hippocampal area revealed ischemic cell

change in scattered nerve cells, with swollen astrocytes and in some places loss of nerve cells. No changes were seen in the vessels of the cortex.

The second case concerned a patient of 57 who received the same dosage as the preceding patient and died following the third treatment. No changes were observed in the heart or other organs. The changes were present throughout the cortex. Areas of ischemic cell change were seen. The neuroglial reaction was slight and was particularly noticeable in the polymorphic layer of the hippocampus. The thalamus contained occasional pale and poorly defined nerve cells with vacuolated cytoplasm and somewhat distorted nuclei. The small cells of the striatum showed occasional satellitosis and changes similar to those observed in the thalamus. The cell changes were patchy. The dorsal vagal nucleus in the medulla revealed occasional pale cells and ghost cells with neuronophagia, enlarged glial nuclei, and small glial rosettes.

Ebaugh and his collaborators believe that the nerve cell changes may be a part of the seizure reaction and that all the lesions in the brain were brought about by the electrical shock treatment.

The problem is elaborated further by Gralnick [12] who reported death following electrical shock in a negro of 38 years who developed syphilis in 1939 but was reported to have no clinical evidence of the disease in 1942. Death occurred after the second electrical shock treatment, two days after the shock. Necropsy revealed edema of the lungs and hypoplasia of the circulatory system.

The brain revealed diffuse congestion of the blood vessels, thickening of the vessel walls, and endarteritis involving the smaller blood vessels. Diffuse degeneration of nerve cells of varying types was seen in the cerebral cortex, chiefly of the ischemic variety. Scattered areas of cell loss were found and some disturbance of the cortical architecture. A considerable degree of neuronophagia was found. The oligoglia cells of the white matter were increased. Glial nodules were found in the medulla and cerebellum. The glial nuclei were considerably increased in the region of the auditory, vagus, and trigeminal nuclei. Vascular changes were pronounced in the basal ganglia, some of the vessels showing hyaline degeneration and calcification. Amyloid bodies were found in the occipital lobes around the posterior horns of the lateral ventricles. No fresh hemorrhages were seen, but blood pigment was seen occasionally around the blood vessels.

The significance of the case reported by Gralnick is obscured by the possible complication of cerebral vascular syphilis, for which reason it seems best not to emphasize it in an evaluation of the brain changes associated with electrical shock.

Levy [13] reports brain hemorrhages in a patient who died of heart failure after electrical shock treatment. "There were a considerable number of dilated capillaries with hemorrhages which undoubtedly antedated the acute myocardial failure, as indicated by the pressure of blood pigment."

Attention to the role of circulatory failure in death from electrical shock treatment was directed by Jetter [14] who reported death in three cases following shock treatment. His first patient was a man of 61 who died in 12 minutes following his eighth shock treatment. The heart revealed extensive obliterating coronary arteriosclerosis, a recent myocardial infarct, and hypertrophy and dilatation. In the brain were moderate sclerosis of the arteries and arterioles, occasional acellular areas in the cerebral cortex, moderate hyperemia and occasional petechial hemorrhages in the white matter. The second case concerned a patient of 70 years who died 12 minutes after the sixth treatment. The heart revealed obliterating coronary sclerosis, an old myocardial infarct, and hypertrophy and dilatation. The kidney was the seat of arterial and arteriolar nephrosclerosis. The brain revealed moderate sclerotic changes in the arteries and arterioles, occasional acellular areas in the cortex, slight rarefaction of the myelin around the blood vessels, recent small infarcts in all the lobes of the brain, with gitter cells, etc., and minor hemorrhages in the white matter. The third case concerned a young subject of 23 who had had one course of eight treatments and two months later was given another course with death ensuing about twelve hours after the eighth shock. Necropsy revealed severe pulmonary edema, an acutely dilated heart, acute diffuse glomerulonephritis and acute hyperemia of the brain.

The death in Jetter's cases was attributed to heart failure. The petechial hemorrhages found in the white matter in two cases were regarded as a manifestation of agonal anoxemia associated with cardiac collapse.

Six deaths following electrical shock treatment have been recorded in England and Wales (Napier[15]). The situation in three cases may be summarized as follows: (1) hemorrhage into both thyroid lobes following a single shock treatment in a subject of 46 years. The brain showed no significant findings; (2) death from pulmonary tuberculosis in a subject of 52 who had two shock treatments and died two months later; (3) hemorrhagic staining over the right cerebrum in a patient of 62 who died 30 minutes after the fourth shock treatment.

The occurrence of fat embolism as a possible factor in death following electrical shock treatment is reported by Meyer and Teare.[16] Their patient, a man of 63, collapsed following a single treatment and died twelve hours later. Study of the brain revealed many capillaries blocked by fat emboli which were present diffusely throughout the brain and cerebellum, and were more frequent in the gray matter. No other changes were found.

A further case is reported by Gralnick.[12] It concerns a man of 61 who died two days following his second electrical shock treatment. Autopsy examination revealed a large meningioma lying in the subfrontal region, petechial hemorrhages in the mesencephalon, the pons, cerebellum and white matter. Larger hemorrhages were seen in the pons.

The findings in the few reported cases of death following electrical shock are conflicting, but they give us at least some concept of the conditions encountered at necropsy. On the one hand are reported (1) hemor-

rhages of small size and varying age scattered throughout the brain (Alpers and Hughes); (2) scattered areas of cell loss and ischemic cell change (Ebaugh, Barnacle and Neuberger); (3) no brain change of significance except for minor petechial hemorrhages in the white matter associated with acute cardiac failure and attributed to agonal anoxemia (Jetter); (4) fat emboli (Meyer and Teare).

Not only is there no unanimity of opinion concerning what occurs in the brain but there is not even uniformity of findings.

DISCUSSION

It is obvious that there is no agreement on the brain changes encountered in the course of electrical shock treatment either in animals or in the human. The problem however is the same in the two groups—the nature of the findings and their meaning. In animals the circumstances can be varied according to the plans of the investigator, whereas in man the circumstances are usually beyond the control of the physician. It is precisely the circumstances of the experiments and the autopsy studies which have aroused criticism and doubt and it is to these to which I should like to direct attention for the moment.

In an effort to ascertain whether brain changes occur in the course of shock treatment, emphasis has been too heavily placed on the fatal features of whatever damage has occurred. Clinical experience has long since taught that electrical shock treatment is safe and in the vast majority of instances without danger. It has been estimated that it was a cause of death in 0.05 per cent (Kolb and Vogel) of 7,207 cases and 0.8 in 11,000 cases (Impostate and Almansi). The problem is obviously not whether electrical shock is a cause of death, but whether it is associated with brain changes of any sort, and if so what these changes may signify. That this is an important problem can hardly be denied in view of the shaking up which patients receive during the course of a treatment which is now in common use and which depends for its effectiveness on stimulation of the cerebral cortex. I shall attempt therefore to approach the evidence with this issue in mind, and shall make an effort to determine what we can from the data now available.

EXPERIMENTAL DATA

1. *The problem of dosage.* That the problem of comparable dosage is one of great importance, cannot be denied. If the results obtained in experimental animals and in humans are to be evaluated properly, the conditions of dosage and density of current must be similar. Thus far, no such comparable study has been made to my knowledge. The dosages used have either been in excess of those used in humans, or the conditions of the experiment have differed along other lines. It seems certain now that the original dosages used in the cats reported by Alpers and Hughes were greater than those used in humans and the same is probably true also of the experiments of Weil. One of the major obstacles to agreement on the brain changes in shock lies in the fact that it has been claimed that in those instances in which irreversible brain damage has been found, that the dosage in animals is considerably greater than that used in humans. Neymann, in commenting on Weil's experiments in rabbits, estimates the fact that if the electrodes used were equated for use in human cases, one would have to use electrodes 100 to 211 cm.^{2} in area. The currents of 130 volts and 300 m.a. were strong enough to produce electrical convulsions in practically any human subject weighing 50 Kg. In the experiments of Alpers and Hughes disc electrodes 5 mm. in diameter were used and currents of 150 to 200 m.a. were applied to the scalp.

2. *The problem of actual brain damage.* It is doubtful whether the conditions of experiments in other reported series are comparable to those found in the human. In the majority of the experiments of Alexander and Lowenback (19 out of 26 animals), only single shocks were used. The same is true of Windle and his collaborators who reported no changes in the nerve cells following electrical shock. The conditions therefore do not simulate the actual circumstances encountered in treatment in the human and the reported findings are of value only in relation to single shock studies. They give valuable information concerning the functional changes following single shocks, but they do not reproduce the conditions produced in man.

On the other hand, there have been several groups of experiments in which such conditions have been reproduced. Here, too, the results are open to criticism. In four of the animals studied by Alexander and Lowenback, "vasoparalytic stasis" was found even with a single shock, the findings consisting of dilatation of capillaries, arteries, and veins with or without, but usually with, perivenous hemorrhages. In one animal which received 52 shocks of 1400 m.a. for a total time of 33 seconds, severe pyknosis of nerve cells was produced in parts which were limited to the central core of the current. Of the three dogs reported by Lidbeck with negative results, dog 1 (16 treatments, 250-300 m.a. 0.2 seconds) showed a small perivascular subcortical hemorrhage, with shrunken nerve cells in all the sections; dogs 2 and 3 (16 treatments, 350 m.a., 0.3 seconds) had a greater number of shrunken cells. In 13 cats Winkelman and Moore found moderate pyknosis and hyperchromia of the smaller nerve cells of Laminae II and III and pyknosis of the purkinje cells. Their conclusion is that *permanent* morphological changes do not result from electrical shock, but that intracellular and biochemical changes take place from passage of the current and from the resulting convulsion. Similar changes were found in monkeys by Barrera and his associates, but the changes in the nerve cells were not regarded as significant because of the disclosure of similar findings in control animals.

The argument which I am laboring is that brain changes have been disclosed even in those cases in which the experiments have been regarded as negative. They have not consisted of perivenous hemorrhages as a rule, though these too have been found, but they have been characterized by changes in the nerve cells themselves, usually without glial reaction. The problem of para-

mount significance is whether changes of any sort occur. The answer to this must be in the affirmative. Whether the changes are permanent or transitory is open to investigation. If hemorrhages develop, the possibility of permanent damage must be conceded. If sclerosis of the cells develops, the problem of irreversible change is not so readily settled, since it is difficult to determine from fixed specimens alone whether irretrievable damage to a nerve cell has been done.

Possibly the factor of greatest significance is that changes of some sort do develop in electrical shock treatment, and it is therefore not a form of treatment to be regarded lightly or to be used indiscriminately. From the experimental evidence alone it is not possible to assert dogmatically that no brain damage is done by the passage of repeated electrical currents through the brain. More data is still necessary.

HUMAN DATA

Unfortunately, the missing data and the answer are not to be found in the cases of death in human subjects following electrical shock. A variety of findings have been disclosed: perivascular hemorrhages, areas of cell loss, diffuse ganglion cell disease, sclerosis of ganglion cells, and subarachnoid hemorrhage. The subjects in many instances have fallen within an age range in which the type of ganglion cell disease recorded could be normal except for one patient reported by Gralnick in a subject 38 years of age in whom, unfortunately, the problem of syphilis complicated the histological picture.

This much is certain: that electrical shock as administered to the human is not in itself fatal. Nor is the cause of death to be found in the brain damage. On this, all are agreed. Death is usually the result of cardiac or cardiovascular collapse in subjects with coronary disease, but isolated instances of death with hemorrhage into the thyroid gland and in uremia have been recorded.

The problem of vital importance is not whether the procedure is safe, but whether it is in any sense harmful by the production of changes of any sort within the nervous system. The answer is not yet available from human material. All instances of death following electrical shock treatment are extremely important and require recording until a more complete picture of what occurs in the human brain can be elucidated.

MECHANISM OF ACTION OF SHOCK

Though the problem of brain damage is still unsettled, other vital problems concerning the mechanism of action of electrical shock have been more or less clarified. It seems clear that only a small percentage of the electrical current delivered by the ordinary apparatus is conveyed through the nervous system. Currents such as those in routine usage—70-150 volts, 300-1200 m.a.; 0.1-0.5 seconds—"would probably be exceedingly dangerous and probably fatal if such currents in their entirety passed through the cortex or other parts of the central nervous system. But such considerations become less significant when it is realized that probably only a small portion of the current flowing between the electrodes actually passes through any one portion or even the entire brain. . . . Most of the current appears to pass through the scalp" (Barrera).

It seems definite also that whatever brain changes occur, whether they are transitory or permanent, depending upon the circumstances of the experiment, they occur only in the path of the current or at its immediate periphery. This has been demonstrated by Alexander and Lowenback. They state that their experiments demonstrate that "changes were produced only within the path of the current, but that these changes were not always present throughout the entire path." On the other hand it is doubtful whether it is possible to state definitely that the path of the current can be delineated by the changes which developed between the electrodes. Brain tissue is not the ideal conductor of electricity, and from the standpoint of physics it would be possible to determine the paths of the current only in the case of a known good conductor surrounded by a poor conductor. It is questionable whether brain tissue fulfills these requirements. It seems to be more accurate to speak of diffusion of the current than of concentration. Since it is possible also that other factors besides the electrical current are operative in the brain developments during shock, it is difficult to be certain which changes are the result of the direct action of the current and which are due to other factors. A second factor in the possible production of brain changes is found in the excessive stimulation of the vagus-vasomotor centers in the medulla causing in turn generalized circulatory disturbances interfering with the circulation to the brain tissue. Finally, possible changes in the brain tissue must be attributed to the effects of the convulsion itself.

SUMMARY

A survey of the brain changes found in experimental electrical shock and in reported human cases, reveals a wide diversity of opinion. In the experimental animal, on the one hand, are reported petechial hemorrhages which probably represent the results of greater dosage and density of current than that used in the treatment of human beings. In contrast to this are reported scattered cell loss and cell changes which have often been interpreted as being reversible. Even in instances in which no significant changes are recorded, there has been observed an occasional petechial hemorrhage which has been attributed to overdosage. When such hemorrhages have been disclosed in the study of human cases they have been regarded, as a rule, as agonal.

The results in human cases have been less conclusive than those reported in experimental animals, since, in almost every instance, some extraneous factor has entered into the situation and made analysis of the direct effects of electrical shock difficult to evaluate. Among such factors are: advanced age which has introduced doubt whether the recorded cell changes are due to electrical shock or to unrelated vascular disease; cardiac complications which introduce the element of anoxia as an explanation of the brain changes; long latencies between the termination of shock and the death of the patient; and complicating syphilis of the brain.

Despite these obfuscating factors, the suspicion per-

sists that changes of some sort occur as the result of electrical shock treatment. The probabilities are that these are functional in nature in the ordinary case and are unattended by permanent or irreversible brain damage. Clinical correlations would tend to support this contention, since the confusion, anxiety, memory loss, and other effects of shock disappear in the course of time. The possibility of damage is present, however, under two conditions: (1) in the presence of a large number of treatments, even in young and healthy subjects; (2) in the presence of existing brain damage. I have under my care at the present time a young lawyer who received elsewhere over 50 shock treatments, and who, after a year, still complains of enough memory loss to interfere with his work, though his hypomania has not recurred. It is doubtful, in my opinion, whether he will ever regain his normal memory capacity. The rare indicate also the procedure is not entirely benign, and that damage may ensue sufficient to cause serious sequelae.

In an effort to determine whether electrical shock was a safe procedure, emphasis was placed primarily on whether it caused irreversible brain damage and whether it could be regarded as a cause of death. Experience has shown amply that it is not a cause of death by virtue of brain damage, and that where death occurs it is usually the result of cardiovascular disease. The problem, as I have stated elsewhere, however, is not whether it causes death, but whether it causes damage and, if so, how frequently. We are not in possession of the facts which can answer this question, so that, for the present, electrical shock must be regarded as a form of treatment to be used judiciously and sparingly, for those conditions which can definitely profit by its application.

Though the study of human material has not revealed what happens to the brain in electrical shock, it has thrown some light on the types of cases which are likely to develop harmful effects. Autopsied cases suggest that brain damage is likely to occur in conditions associated with pre-existing brain damage, as in cerebral arteriosclerosis. It may be advisable therefore to prescribe shock treatment with caution in instances with known brain damage.

I realize how indefinite have been my conclusions concerning the effects of electrical shock on the structure instances of convulsive seizures following electrical shock of the nervous system, but the available facts have forced this position upon me. If I have been able to indicate only that more studies are necessary concerning the problem in question, and that security in the application of shock treatment is ill-founded, I shall not apologize too profusely for leaving you in a state of ferment.

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A DOCTOR'S MISSION

Jean Jacques Rousseau, in *Emile, or Education* (1762), Book I, says, "Medicine is all the fashion in these days, and very naturally. It is the amusement of the idle and unemployed, who do not know what to do with their time in taking care of themselves. If by ill-luck they had happened to be born immortal, they would have been the most miserable of men; a life they could not lose would be of no value to them. Such men must have doctors to threaten and flatter them, to give them the only pleasure they can enjoy, the pleasure of not being dead."—From *Army Medical Library News,* July 1946.