Original Articles.

ON THE RELATION OF EPILEPSY TO INJURY OF THE HEAD.¹

BY JAMES J. PUTNAM, M.D.

At the recent Congress in Washington the important subject of traumatic epilepsy from fracture of the skull was brought up by Dr. Agnew,² of Philadelphia, in the course of his paper on "The Surgery of the Brain." After speaking of the unsatisfactory results of operative treatment after the neurosis has once established itself, he expressed the opinion that the improper surgical treatment of the case at the time of the occurrence of the fracture is responsible for much of the subsequent mischief, and, went on to say: "Whenever, therefore, in my judgment, the profession can accept the doctrine that all depressed fractures of the cranium, however slight may be the depression, and entirely irrespective of pressure symptoms, are proper subjects for trephining, then will traumatic epilepsy largely disappear from the list of surgical diseases; indeed, I am sure that he who shall propose to tabulate, at the end of the next twenty-five years, the cases of epilepsy, will find, as compared with the present time, a meagre supply for his purpose. It is not improbable, indeed, in view of the greatly diminished risk from trephining, that the operation will be extended even to cases of simple fracture or fissure of the skull."

It will be fortunate, indeed, if this optimistic view turns out to be well founded. Since, however, it is of the highest importance for us to know, in detail, on just what principles we ought to proceed, when called to a case of fracture, and how much immunity from epilepsy or insanity we can promise our patient in return for his submitting, though free from symptoms, to trephining and perhaps to opening the dura, it seems worth while to re-examine the evidence as to the frequency and the causes of the unwelcome result and the possibility of avoiding them.

The adoption of this view assumes that the cause of the epilepsy is the irritation at the seat of principal injury, and especially the irritation due to displaced fragments of bone,³ and the failure of late operations for removal of the bone to effect a cure is considered as due to the epileptic habit having become established.

It is difficult to test the correctness of this explanation, but it is not an altogether satisfactory one. It is common enough to see cases where trephining, undertaken after a certain number of fits have occurred, do check their recurrence for several years, long enough, one would think, to break up the epileptic habit, if there were nothing else besides the irritation starting from the depressed bone to maintain it. But not only do the fits almost always recur in the end after trephining, but they usually recur when, besides the bone, portions of the cortex, which the symptoms indicated to be the centres of origin of the attacks, have been removed likewise, as Dr. Agnew himself points out.

Moreover, the recent excisions of motor areas which were apparently responsible for outbreaks of ordinary

epilepsy of the localized type, also seem to be far less effective than was hoped. The attacks are usually arrested, sometimes for one or two years, or even longer, and in exceptional cases they may never return; but this is hardly more than seems occasionally, (though less often) to happen after other operations where neither cortex nor diseased bone are removed, so that, important as both trephining and cortical excision may be as a means of treatment, where we wish to gain a temporary respite, or to take every chance of cure, they must for the present count rather as excellent inhibitory measures, than as proving the nature or origin of the disease.

If Dr. Agnew's opinion, which represents that of many surgeons, cannot be sustained by evidence, and the patient's prospects are not improved by treatment of the local injury to anything like the degree indicated, it is eminently important that we should not let ourselves be turned aside by it from the search for other influences that may turn out to be equally important in the causation of epilepsy. If, on the other hand, that opinion can be sustained, and if trephining carries with it so little risk of inducing either immediate or subsequent mischief, then, I think, it should be seriously considered whether we ought not to trephine, also, in cases of healed fracture that come to us not too long after injury, but before epileptic symptoms have appeared.

There is, of course, no question as to the propriety of trephining, in many cases of fracture even without symptoms, on general surgical grounds. The indications for doing this have been clearly laid down by various writers, and among others by Wagner, in an excellent address published in 1886 as Nos. 271, 272, of Volkmann's *Klinische Vorträge*. The treatment there advised is based almost wholly on the importance of preventing or curing local infection, though with the secondary aim of getting rid of irritating fragments of bone. Simple depression is considered as dangerous, not as a cause of intracranial pressure, but only in that it is liable to interfere with the union of the broken bones and lead to suppuration by disturbing the circulation in the diploë, and he recommends that when the fracture is not compound a moderate depression should be left untreated. Linear fracture without symptoms, even when compound, is considered as calling for treatment only when hairs or other foreign substances are caught in the crack, and then he uses the chisel so far as is necessary for removing them.

Wagner recognizes at the same time that trephining is a relatively trifling operation.⁴ He urges it for localized meningitis, or to secure a more aseptic condition of the broken bone and underlying parts, as in perforating wounds, and even, in case of doubt, for exploring the condition of the tissues beneath the bone, especially when suppuration of the dura is suspected.

Although Wagner considers it important to remove broken fragments of the inner table when feasible, he calls attention to the fact that they generally heal smoothly if left alone, and this opinion gains some support from the fact that the osseous plates which so often form in the dura usually do no harm, though they are considered by some pathologists⁵ as of inflammatory origin.

⁴ Dr. Manley, of Brooklyn, demurs to the view that trephining after injury is so safe, and quotes cases to show the danger of hernia cerebri.

⁵ See Ziegler's *Handbook of Pathological Anatomy*.

¹ Read before the Boston Society for Medical Improvement, November 9, 1891.

² Published in the *University Medical Magazine*, Philadelphia, October, 1891.

³ Dr. Agnew (loc. cit.) speaks particularly of the irritation of the sensory nerves of the dura by depressed or broken bone, "an irritation propagated to the meninges, and, later on, to the cortex and brain-ganglia."

Do we know with any approach to accuracy how much immunity from epilepsy is secured by early trephining,⁶ or in what class of cases immunity is secured at all?

The absolute evidence of the character necessary for answering these questions is small in amount. No systematic attempt has been made by any writer to compare, as regards the subsequent occurrence of epilepsy, any large series of cases in which the skin, bone and underlying parts were treated early by the best modern methods of surgery, with a similar number which were not so treated; and, on the other hand, there are plenty of individual cases on record where, under such treatment as was adopted, the parts healed satisfactorily and showed later no sign of disease, and yet epilepsy supervened.

Among the cases of fracture, especially of gun-shot fracture, occurring in our late war,⁷ this happened repeatedly, though of course the methods then followed were not as thorough as those in use to-day.

An analyses of these cases made for me by Mr. F. Coggeshall, shows that five per cent. of the patients trephined at the time of injury subsequently had epilepsy, and that thirty-two per cent. of all the patients who became epileptic later had been trephined at the time of injury.

It is not enough to point out that cases of epilepsy following properly treated fracture do not present themselves in our dispensaries and consulting-rooms, for the whole number of cases that would be admitted by the best surgeons as having been properly treated is extremely small, and the proportion of cases in which epilepsy occurs after fracture, treated or not treated, is also a small one, though the danger is apt to loom up prominently when we are first called to such a case.

Does the simple persistence of a depression in the bone, for example, without detached fragments or signs of inflammatory processes, at the end of a number of years, prove that the case was not properly treated at the outset? From the point of view of surgical treatment we should perhaps have to answer yes, but, in fact, it is doubtful whether the chances of epilepsy are really much increased by such a condition, except where the displacement prevents the bone from healing properly, as sometimes happens.⁸

Not only is it common enough to see depressions in the skull without epilepsy, but it is difficult to see why epilepsy should result unless the depression is the cause or accompaniment of osteitis or meningitis, or changes in the nutrition of the brain, and that these results usually occur is not shown by the records of subsequent operations or post-mortems, or by the inspection of the specimens in our museums. Microscopic investigations might tell a different story, but at any rate there is no reason to think that the simple increase in intracranial pressure from a moderate depression causes symptoms of cerebral compression, except sometimes at the outset. If depression in itself is a cause of epilepsy it must be because, as Dr. Agnew suggests, it sets up an irritation of the dural nerves, and, in some way not known to us, possibly by way of neuritis, an impairment of the cerebral nutrition.

It must, I think, be admitted, with regret, that the view which looks for so much security in the treatment of the fractured and depressed bone is not based

upon a sufficient recognition of the obscurity which still surrounds the origin of epilepsy, and of the variety of the possible causes liable to be set in action by severe injuries to the head, some of which are amenable to early treatment, while others are not.

The study of epilepsy as it occurred among the German troops in the late⁹ Franco-German war is full of interest in this connection.⁹

It is, in the first place, worthy of note, that by far the greater number of cases occurring in the German army and due to army life were of other than traumatic origin,¹⁰ because it reinforces the view that epilepsy is usually a general cerebral neurosis.

That it may arise from purely local cerebral disease seems unquestionable, especially when we consider that it may be excited in animals by localized faradization of the cortex. But in many of the traumatic cases, and especially where the outbreaks do not occur until years after the injury, influences are present affecting the nutrition of large areas of the brain, and these certainly constitute possible causes of the disease.

Of the sixty-three traumatic cases (omitting a large number of epileptiform vertigos, etc., and a few unclassified cases) forty-six followed injuries of the head, seventeen, injuries of the body and limbs.

Again, epilepsy occurred out of

8,985 injuries of the head	28 times = 0.31 %
11,091 " " neck and trunk	5 " = 0.04 %
30,700 " " arms	5 " = 0.016 %
35,670 " " legs	7 " = 0.02 %

The greater number of the epilepsies from head injury appeared to be from sensitive scars of the scalp, combined with the effects — difficult to estimate — of cerebral contusion and concussion; but that these latter were of themselves sufficient causes (and causes which are present in almost all cases of fracture) was shown by the history of seven cases where the head sustained no local injury, but only suffered from general concussion.

The sensitiveness of the scars of the scalp seems to have been due to the bruising of the skin against the hard underlying surface by bullets or fragments of shell; and the scars following sabre-cuts were generally non-sensitive. The avoidance and removal of such scars would, of course, be eminently in place, and the same may be said of irritations of the dura.

It may be easily conceived that, besides fractures with displacement, cases of *healed* fracture would occasionally present themselves where — as a protection against epilepsy — trephining for the removal of suspected irritation of the dura would be justifiable.

Local injuries of the brain are recognized in these reports, as well as by all surgeons, as a possible cause of epilepsy, and these local lesions are liable to be beyond the reach of such surgical treatment as will remove the danger of epilepsy.

Dr. Keen¹¹ has, to be sure, suggested the importance of the systematic removal of disorganized brain tissue, and this may mark a decided advance over Wagner's method,¹² which consisted in removing only such parts of the brain as protruded beyond the dura, but it is too early to tell how thoroughly this plan can be carried out or with what results.

It is quite possible that we attribute too much im-

⁶ This term is to be taken, of course, as covering all the measures used in exploring the injury of the bone and underlying parts.

⁷ Surgical History of the War of Rebellion.

⁸ Compare Wagner (*loc. cit.*).

⁹ Vol. vii, *Erkrankungen des Nervensystems*, Berlin, 1886.

¹⁰ Fatigue, acute disease, excitement, over-exertion, fright, cold, general conditions.

¹¹ *American Journal Medical Sciences*, September, 1891.

¹² Compare Wagner, *Volkmann's Klinische Vorträge*, Nos. 271, 272.

portance to the irritation at the seat of fracture as the cause of the subsequent epilepsy, instead of considering it as only one, though usually the chief of several contributive causes. At any rate the following facts should be borne in mind before we commit ourselves to a definite conclusion.

(1) Traumatic epilepsy is by no means always of the localized, so-called "Jacksonian" type. In many cases the loss of consciousness is as early and as sudden, and the first convulsive symptoms as widespread, as in ordinary epilepsy, and although the irritation of the dura at the seat of fracture might account for such attacks, we have no right to consider that it is the only or even the principal cause, so long as other possible causes are present, and this, in severe head injuries, is usually the case. As a rule, whether the skull be fractured or not, the brain is injured in a number of different parts, and the lesions of the brain near the seat of injury to the bone are liable to be much more extensive than the osseous fracture, and more or less independent of it.¹³

Either one of these various lesions, as well as the general injury which the brain sustains from concussion, are adequate causes of epilepsy, and it is not improbable that they may work together as contributive causes. Of course, even if this be true, we are not relieved from the duty of treating the original wound so as to secure a healthy and rapid repair of all the injured tissues, including the brain, so far as this is possible, but even after doing this we should be guarded in our prognosis. I shall refer again below to the important subject of the contributive action of several partial causes in inducing epilepsy even of the Jacksonian type.

(2) In many cases there is a considerable interval, often years in length, after the injury and before the epilepsy appears. Are we to assume that, during this period of latency, the hidden danger is silently drawing nearer, or is it more probable that while the irritation remains the same some new cause comes in to precipitate the outbreak? If the former is probably true, have we a right to assume that the series of nerve and brain changes resulting in the epilepsy are alone those which have the injury at the seat of fracture as a focus? Is it improbable that the inflammations and irritations starting from other foci of injury to the bone, membranes or brain, play their part in inducing the result? The answer to this question must depend upon the evidence with regard to the contributive influence of partial causes.

The immediate causes of epilepsy in these traumatic cases are so obscure and baffling that we ought to keep ourselves in a very catholic spirit during our search for them.

The only way to answer the question as to what are the significant alterations that occur before the attacks appear would be to tabulate all the morbid changes which do take place during this interval and that could possibly be causes of epilepsy, and then try to discriminate between their effects.

The physiological characteristic of the epileptic brain is generally believed to be that a greater or less portion of its ganglionic matter has acquired the habit of discharge under less than the normal stimulus; and

from what we know of the physiology of the nervous system it is fair to assume as probable that this liability to premature discharge might be brought about in three general ways: (a) by modifications of the nutrition of some particular centre or area of the brain; (b) by impairment of the inhibitory relations between different parts of the brain;¹⁴ (c) by an unnatural increase in the number or force of the stimuli tending to provoke the discharge of a given part of the brain. It is in this latter way that we may suppose "reflex" epilepsy to be caused, and we might class as "reflex" — at least for purposes of investigation — those epileptic attacks which are produced by the action of one part of the brain upon another.

In seeking the cause of epilepsy from injuries of the head, such as fracture with depression, we are too prone to look only at the lesion indicated under *a*, and fail to recognize sufficiently the more distant and obscurer lesions falling under *b* and *c*.

I think there is reason to suspect that even when the "signal symptom" of an epileptic attack, in a case of head injury or cerebral disease is sharply localized, the responsibility for the outbreak should not always be laid exclusively at the door of the centre especially correlated with this "signal symptom," but that it often implies an abnormal condition of other parts of the brain, or of the brain at large. Certainly a number of cases have been reported¹⁵ of epilepsy of the Jacksonian type where careful examination has failed to reveal any lesion of the corresponding centres in the cortex.

We do not yet know the anatomical correlates of the epileptic state with enough exactness to make it worth while to describe them, but we may hope to arrive at them eventually by a conscientious study, macroscopic and microscopic, of the changes occurring in epileptic brains, especially during the pre-epileptic stage.

We do know already that during the latent stage of traumatic epilepsy the morbid changes developed by the injury (which may have several centres) are liable to spread laterally (Ziegler) as well as to become locally more intense; and we know further that localized epilepsy is liable to occur in brains which have been for a long time the seat of extensive and progressive morbid processes, as in the case of the post-hemiplegic epilepsy of children, and the epilepsy in parietic dementia. Again, we know that influences acting on sensory centres in the brain, such as fright, or fatigue, or toxic substances in the blood, may precipitate an attack, or even cause the disease. Löwenfeld¹⁶ calls especial attention to the fact that epilepsy having its starting point in the irritation produced by a tumor or other gross lesion, may remain latent until some other general cause comes in to increase the effectiveness of the local cause.

It is reasonable provisionally to suspect, therefore, that in the traumatic cases, multiple and spreading lesions which often exist, may contribute to increase the injurious effect of a predominant local injury.

It is undoubtedly true that purely local irritations of the cortex may excite epileptic attacks, just as these may be excited by localized electrical stimulation; but there is a body of facts, to which I think too little at-

¹³ Widespread hemorrhages and softening often ending in adhesive meningitis, are extremely common. See Byrant, *Hunterian Lecture*, *Lancet*, June, 1888; also Duret, *Traumatismes Cérébraux*; and an interesting case reported by Walter Channing in this Journal, July 8, 1886.

¹⁴ See Hare's recent monograph on epilepsy.

¹⁵ Charcot and Pitres: *Rev. Mensuelle*, 1878, xi. See also Löwenfeld: *Arch. für Psych.*, etc., vol. xxi; and Kramer: *Jahrbuch für Psych.*, 1891, 1.

¹⁶ Loc. cit.

tention has been paid, which indicate that both in so-called idiopathic epilepsy and in some forms of traumatic epilepsy, the special and "localized" auras and convulsions are sometimes only the expression of a morbid condition involving a large cerebral area, or a whole hemisphere, or the whole brain, just as the feeling of anger may cause habitually, in one person a clenching of the fists, in another a frown or a paleness of the face, in another a sense of intellectual inhibition.

The character of the first, and, through the establishment of habit, of the subsequent outbreaks, would then be referable to a greater physiological excitability of one or another part of the brain, either native as in the case of the hand and face centres, which, as Hughlings-Jackson long ago pointed out, are so prone to be the first to discharge in ordinary epilepsy, or due to local disease or irritation of the brain or of the peripheral nerves as in traumatic and reflex epilepsy.

This principle has been abundantly established by both physiological experiment and clinical observations,¹⁷ but its practical significance as necessitating a modification in the application of the doctrine of the localization of functions in the brain,¹⁸ has not been sufficiently dwelt upon. The character of the most important pieces of evidence may be briefly indicated as follows:

(1) The experiments of Ferrier,¹⁹ of Bubnoff,²⁰ and Heidenhain, and other physiologists have shown that the movements of certain parts, such as the face, may often be excited by minimal stimulations of the cortex which are ineffective as regards other movements.

(2) It is asserted by Bubnoff that if an animal, in the course of an experiment, makes an accidental movement of a limb, the same movement is especially likely to be called out by the subsequent electrical stimulations of the cortex; or that if one limb is stroked or moved, movement is more likely to occur there than in other parts under a stimulation of given strength applied successively to all parts of the motor area.

(3) It is a remarkable fact, noted by Dr. Hughlings-Jackson, that the primary or "signal" (sensory or motor) symptoms of an epileptic attack in man occurs far oftener in the hand or face than in other parts, evidently because of the great physiological mobility and sensitiveness of these parts, so much so that I think such symptoms are of less value as indicating a localized irritation of the brain than the similar symptoms when affecting other less mobile parts, such as the leg or shoulder; that is, the hand or face may be thrown into convulsion alone, or primarily alone, by an inflammatory process covering a large area. This principle applies both to traumatic and inflammatory, and to so-called idiopathic cases. I say so-called, because I believe that, pathologically considered, the traumatic and non-traumatic cases are often closely analogous.

Thus, to take one case out of many that could be cited, Dr. J. C. Warren operated recently, at my request, on a patient who, in consequence of a meningeal hæmorrhage or meningitis which had occurred during a typhoid fever eight or ten years before, had had a transient hemiplegia followed by epilepsy.

He had distinct auras, both sensory and motor, in the left hand, frequently without loss of consciousness, but at the operation the lesion was found to cover a good part, if not the whole, of the arm and face area. We snipped out a bit of the cortex, simply guessing at the hand centre, since no electrical responses could be obtained, and for the next week the hand was paralyzed, obviously because its cortical arrangements were especially sensitive to damage, as they had previously been to irritation.

CONCLUSIONS.

(1) The causes of epilepsy are numerous; and we cannot hope in most cases to remove them all by early trephining and care of the wound, though these measures, and especially the removal of fragments, are probably very important.

(2) The local and the general injury of the brain are probably of prime importance, as causes of epilepsy, and are to some extent independent of fracture. The former may perhaps sometimes be treated as suggested by Keen, but the latter can only be reached by general treatment.

(3) The treatment of the general conditions of the brain, by cold and by prolonged, absolute mental rest is probably of great importance, and might perhaps be reinforced by other measures directed to the same end.

(4) The occurrence of localized convulsions or auras does not necessarily indicate limited local disease, to be removed by operation; since, on the theory that the disease is a general one, it would have to find some local expression. It is especially true that convulsions or sensory auras beginning in the hand or face are untrustworthy indications of the extent of the cerebral lesion, because the cortex corresponding to these parts has a high degree of irritability.

(5) Considering the long period that usually elapses after an injury to the head, before epilepsy declares itself, a period during which the patient may be perfectly well, it is reasonable to seek for some better term than "local irritation" to express the connection between the two events. We may fairly suspect that in most such cases a degenerative process goes on, which increases in extension and perhaps in intensity. Sometimes a neuritis, starting from the seat of injury seems the important link ["History of Franco-German War;" Nothnagel].

We know that such a progressive degenerative process as this is capable of giving rise to epilepsy of the localized type, independently of any gross local irritation, as in the case of epilepsy complicating general paresis, of ordinary epilepsy of certain types, and perhaps of epilepsy in cases of head injury without fracture, and of diffuse cerebral sclerosis. We know, further, that the removal of gross irritations in case of an already existing epilepsy of traumatic origin usually gives only a temporary relief. It is, therefore, reasonable to suspect that the state of impaired storage power on the part of the ganglion cells, which we call the epileptic state, is liable to be more widespread than the signal symptoms of the fit would suggest; that we have as often an instance of impaired inhibition or mutual support between different cerebral centres as of locally impaired storage power; that the fit is local in its first expression because the centre corresponding to the initial symptom was an especially irritable centre (for physiological or pathological reasons), and that the impulse to its discharge may originate else-

¹⁷ Hughlings-Jackson and others.

¹⁸ I have called attention to some of its relations in a paper published in the Transactions of the New York State Medical Association in 1890.

¹⁹ Localization of Functions in the Brain. ²⁰ Pflüger's Arch. 1889.

where, or be inoperative unless reinforced by other influences.

How near the epileptic condition may be to apparent health, that is, the latency of the epileptic state, is shown by the fact that epilepsy interchanges with other neuroses, and that the attacks may be brought on by slight causes. This, of course, is a reason for trying first to inhibit attacks for a time so as to check the habit, and, next, to keep away all possible exciting causes. This may constitute a sufficient reason for trephining even when we cannot believe that we can remove the cause of the disease.

(6) May not operation by trephining, for the sake of exploring the parts, be called for in case of patients presenting themselves for the first time after the fracture has healed, but before the outbreak of epilepsy — say within six months or a year? In some respects the tissues are perhaps in better condition for exploration, with the prospect of finding a delimited and removable lesion, a short time after the injury, though it is also true that degenerative processes may already have been initiated. This is a matter for further study.

TWO CASES OF TREPHINING FOR TRAUMATIC EPILEPSY.¹

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At the first Congress of American Physicians and Surgeons the subject of brain surgery excited much interest; at the second Congress the interest was as great, but three years' additional experience rendered the views of those who took part in the discussion distinctly less hopeful. This was especially the case with the question of trephining for epilepsy. The hope of cure from trephining in traumatic epilepsy, or from excision of the cortex in Jacksonian epilepsy, is now regarded as slight, although relief is not infrequently obtained. Some men, indeed, in the discussions took, without warrant, a thoroughly pessimistic position; the majority were not disposed to hope greatly from surgical interference. It therefore seems not unjustifiable to report the following cases, partly because failures should be reported as well as successes, and partly because they illustrate one or two points of interest.

Kate F., sixteen, single, a nursery-maid, presented herself at the out-patient department of the Boston City Hospital, February 17, 1890. She was born in Ireland. Her mother had been insane for eight years, her older sister was rather nervous; otherwise no hereditary taint could be discovered. The girl herself had always been in good health. The catamenia had been established two years before, and since then had been regular and without disturbance. Five years before coming to the hospital she was struck in the forehead with a stone, and was rendered unconscious by the blow. She was carried to a hospital in Dublin and lay unconscious for three days. She then recovered her senses, but she remained in the hospital for three months. After the injury she had strabismus for a time, but that passed away. Ever since the injury she has been subject to headaches; the pain being referred

to both sides of the forehead and to the top of the head, but, as a rule, it starts from the seat of the injury. She has a headache nearly every day, and it lasts several hours. She has also had a slight cough for some time.

In November, 1889, she had a convulsion. There was no aura; the head and eyes turned to the right, and the limbs worked. She frothed at the mouth, could not speak, began to stutter, something came up in the throat and then the head turned and she fell. She said that in some attacks she was conscious of what was going on, but she could not control the attack; in other attacks she lost consciousness. After the first attack she vomited. After the attacks she was sleepy and foolish. A month later, in December, she had a second attack. Since then she has had six more, about one attack a week, the attacks coming on in the morning.

Physical examination showed a stout, sturdy, rosy-cheeked girl with a small stellate cicatrix on the forehead, a little to the left of the median line, about an inch from the beginning of the hair, beneath which was an area of rough, irregular bone with some depression. Careful examination revealed nothing else remarkable. She was given fifteen grains of potassic bromide three times a day, and ordered a restricted diet. This was continued until March 5th, when it was omitted for two days, without orders. No indiscretion in diet was committed. March 9th she had a fit, and March 11th another. On the 11th she had a bad headache and cramps in the stomach. The bromide was ordered to be taken four times a day.

March 13th, she had another fit, and on the 19th another, in which the head and eyes turned to the right. She again, on the 19th, had headache and cramps. She was ordered to take thirty grains of bromide, three times a day.

March 26th, she reported an incomplete attack. The attacks have been preceded by marked vertigo.

April 9th. Three fits since last report.

The bromide having apparently failed to control the fits, I thought it advisable to lose no more time, and I advised her to enter the hospital for operation, which she consented to do, and on the 11th of April she was admitted to Dr. Post's service.

April 13th. Last night and this morning she had convulsions in which the head, eyes and mouth were drawn to the right. She was conscious, but she could not control the attack.

April 21st. She has been kept quiet in bed, and, aside from a slight tonsillitis, she has been comfortable and there has been nothing to attract attention. This morning she had a convulsion, followed by another at noon. These convulsions were general, the legs and arms becoming rigid, the muscles of the face and jaw twitching violently. They lasted four or five minutes. The last catamenia ceased a week ago; the convulsions have never been more marked at the menstrual period.

She had now had twenty convulsions, and bromide seemed powerless to control them. In many of them the signal symptom seemed to be turning of the head and eyes to the right. The centre for this movement is the motor centre nearest the seat of the injury. I thought it possible that a discharge, starting from the old wound, might extend first to this centre and then give rise to a general convulsion. The thing to be done was clearly to trephine over the seat of injury,

¹ Read before the Boston Society for Medical Improvement, November 9, 1891.