ELLIOT (J.W.)

INTRACRANIAL HEMORRHAGE: TWO CASES TREPHINED.

Read before the Surgical Section of the Suffolk District of the Massachusetts Medical Society.

BY

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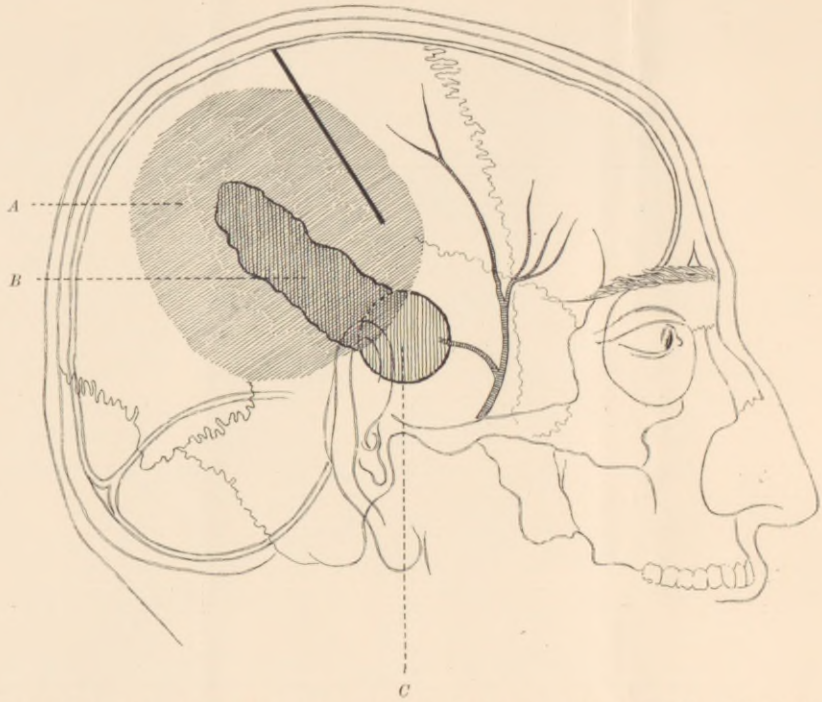

FIG. 1.—MIDDLE MENINGEAL HEMORRHAGE, operated upon July 5, 1892, by J. W. Elliot, M.D.—*A*, hæmatoma; *B*, extension of trephine opening; *C*, trephine.

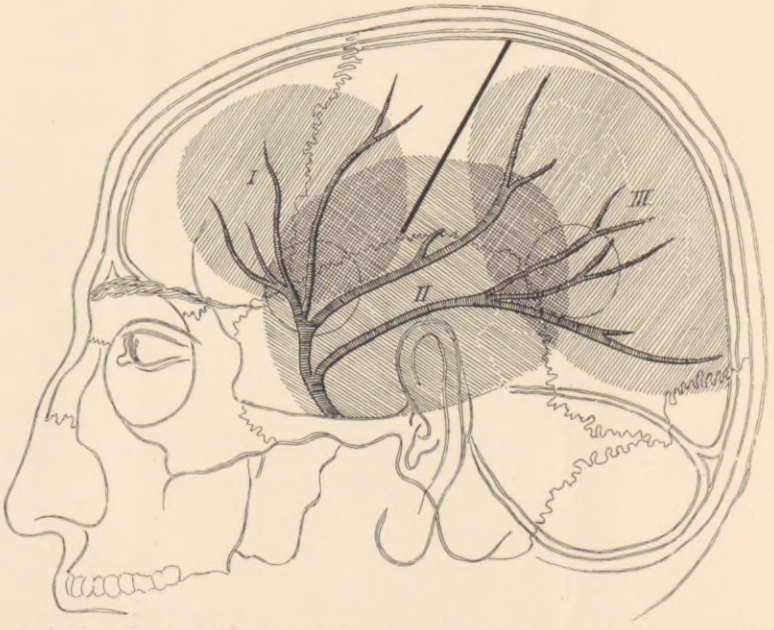

FIG. 2.—*I*, hæmatoma, fronto-temporal; *II*, hæmatoma, temporo-parietal; *III*, hæmatoma, parieto-occipital. (Krönlein.)

INTRACRANIAL HEMORRHAGE: TWO CASES TREPHINED.

CASE I.—MIDDLE MENINGEAL HEMORRHAGE IN A CASE OF EXTENSIVE FRACTURE OF THE RIGHT SIDE OF THE SKULL, INVOLVING THE BASE; TREPHINING AND LIGATURE OF THE ARTERY.—RECOVERY.

On July 5, 1892, the patient, a sailor, aged sixteen years, fell from his ship on to a wharf, a distance of about twelve feet. He was picked up unconscious, but partially regained consciousness on the way to the Massachusetts General Hospital, where he was brought about half an hour after his injury. On being pinched (at 7 P.M.), and on supra-orbital pressure, he said, indistinctly, "Don't." He could be roused when shaken, and looked up with enough intelligence to recognize a friend, immediately dozing off again. The pulse was fairly strong at 68; respiration quiet, 20 to the minute. There was hemorrhage from the right ear and from the nose and mouth. There was no paralysis, but he responded more slowly when pinched on the left foot than on the right. The reflex at the knee was slightly more exaggerated than on the left. There was a small scalp-wound near the right parietal eminence. The ear was syringed with a solution of corrosive sublimate (one to four thousand), and the patient was left on the accident-room table for observation.

At 7.45 the pulse is 56, and somewhat halting. The patient is roused with a little more difficulty. The right pupil responds more slowly; breathing is a little more noisy. The tongue is protruded to the left side; the patellar and plantar reflexes are diminished on the left side.

8.15.—Pulse 52, and somewhat retarded. The right pupil responds very slowly to light. Respiration shows distinct signs of expiratory difficulty. The cheeks are puffed out on expiration. The patient can be aroused only with extreme difficulty.

8.30.—The right pupil does not react. Pulse is 46, and much retarded. Respiration more noisy, though not yet stertorous. He moves all his limbs.

8.45.—Neither pupil responds to light; the right is slightly more dilated than the left. Pulse is 42. Respiration beginning to be stertorous. The patient cannot be roused.

8.55.—For ten minutes there has been restlessness of the limbs, most marked in the left arm. Pulse 40. Distinct expiratory stertor. The right pupil is dilated one-half, the left slightly dilated. The patient's head was shaved and a surgeon sent for.

9.15.—The stertor is becoming very well marked. The patient is in deep coma. Respiration 28, and pulse 40. He has general adductor convulsive movements of the arms, his legs kick out irregularly. Right pupil is dilated,—much larger than the left.

[The above symptoms were recorded by the house pupils, who were quick to recognize the importance of the case.]

I arrived at the hospital at 9.45 P.M., about four hours after the accident had occurred, and found the patient with a small scalp-wound over the right parietal eminence and bleeding from the right ear and mouth. As stated above, he had been picked up unconscious, but had partially regained consciousness by seven o'clock, when he arrived at the hospital. Between seven and nine o'clock his pulse had fallen from 68 to 40; his respiration had risen from 20 to 28; his pupils had ceased to respond to light, and the right one had become dilated. He had had convulsive movements and had sunk into deep coma. These were very evident symptoms of progressing brain-compression. The dilatation of the right pupil and paresis of the left side suggested that the pressure was on the right side. The bleeding from the ear and mouth suggested fracture of the base on the right side. Middle meningeal hemorrhage was therefore thought probable, and severe brain-laceration seemed also probable.

Operation.—Ether was unnecessary, owing to the deep coma. Mucus had already begun to impede respiration and was sponged from the throat. An incision was made from the scalp-wound to a point just in front of the right ear. This revealed a linear fracture of the skull, running from near the parietal eminence down through the temporal bone, which was comminuted above the auditory meatus, but not depressed. The scalp was freely reflected back. An inch trephine was so placed that, while including the comminuted part of the temporal bone, one portion of the disk would cut half an inch above the zygoma, thus giving access to the lower part of the middle fossa, and another portion of the disk would nearly reach a point one inch and a half above the zygoma and one and one-half inches behind the external angular process of the frontal bone, the point of election of the middle meningeal artery. The trephine opening was fortunately placed, for as soon as the disk was removed a quantity of liquid blood gushed out, and I saw at once the black edge of a large blood-clot and a branch of the middle meningeal artery spurting from the dura, which had been extensively torn from the skull. The artery was tied by passing a curved needle through the dura. The opening was enlarged with rongeur forceps, and a clot as large as a fist was removed with the fingers and a spoon. The brain was compressed more than an inch below its normal level, and did not return when the clots were removed, consequently this large space between the dura and the skull immediately refilled with blood and clots. The skull was then chipped away with forceps along the line of fracture as far as the parietal eminence, in the hope of finding another bleeding branch of the middle meningeal artery. The bleeding was, however, found to be a general oozing from the whole surface of the dura, which had been dissected up from the bone by the blood-pressure. The dura was slightly lacerated at one point near the parietal eminence. The hemorrhage continued to be serious until the whole space between the dura and bone was tamponed with iodoform gauze and a drainage-tube.

The finger was passed under the brain into the middle fossa, and could distinctly follow the line of fracture through the petrous portion of the temporal bone. The scalp was replaced and stitched, except at one point left for the removal of the gauze.

During the operation the pulse rose from 40 to over 100. The respiration became less stertorous and the pupils less dilated, but the right was still larger. The patient became somewhat sensitive to pain and moved both arms and legs. During the following day (July 6) he remained in profound coma, with a pulse of good strength and somewhat stertorous respiration. He moved the right arm and leg, but rarely the left, and did not open his eyes. He passed urine and had an occasional involuntary dejection. The tubes were taken out and most of the gauze was removed. His pupils were contracted,—the right being the largest. In the afternoon the temperature rose and the patient perspired freely. He became restless and attempted to remove the dressing, his movements being chiefly on the right side. The reflexes were increased.

CASE II.—Appearance of patient subsequent to the operation. The area to which the trephine was applied and the relative position of the fracture are well shown.

On the next day (the 7th) the pulse and temperature fell, the patient became less restless, and could open the left eye. The pupils were equal. He apparently understood questions, and in the afternoon said he was hungry. He took milk and lime-water by the mouth. The wound healed very rapidly. The gauze was all taken out on the 8th, and the stitches on the 9th, leaving only two small, superficial, granulating areas. The patient could by this time understand and answer questions with some intelligence, but was apt to mutter when spoken to. He kept his eyes closed, and was apparently asleep most of the time. The pupils were equal and reacting, there was ptosis of the right lid, and slight facial paralysis.

During the three weeks following his mental condition greatly improved, and the memory of his life up to the time of the accident returned. He was sometimes drowsy, at other times noisy and unreasonable, but was conscious and intelligent. There was well-marked facial paralysis on the left side, which gradually improved, and by August 10 had almost entirely disappeared. The patient could by that time walk, and was perfectly rational, although still rather childish.

On November 13 he went to the Convalescents' Home, where he made himself very useful as a dresser, and seemed to be perfectly sound in mind. A letter from his sister on December 13, in answer to our inquiry as to his condition, states that he is in splendid health and is learning the steam-heating business.

CASE II.—SUBARACHNOID HEMORRHAGE IN A COMPOUND DEPRESSED FRACTURE OF THE SKULL.

On November 25 the patient, a man thirty years old, was kicked by a horse, and was unconscious for about ten minutes. He was brought to the Massachusetts General Hospital three hours after the injury. When seen there at 5.30 P.M. he was conscious, but had difficulty in speaking. The tongue turned to the left side when protruded. Pupils equal and reacted quickly. Pulse 96. There was no other paralysis. There was a compound depressed fracture on the right side of the skull, about two and one-half inches above the ear, running up and backward for a distance of two and a quarter inches, the depression being one inch wide and three-quarters of an inch deep.

Under ether, the crushed edge of the scalp-wound being cut away, and the scalp reflected back so as to lay bare the fractured bone, an inch trephine was placed just below the fracture, and the depressed bone was easily removed. The dura appeared white and uninjured, but it bulged into the wound and did not pulsate. Distinct fluctuation showed the presence of fluid under the dura. On opening the dura a quantity of clear fluid escaped, but the pulsating brain was still not visible. Another fluctuating membrane, which had the color of muscular tissue, pushed into the opening of the dura. On incising this membrane, a little clear fluid and about an ounce of liquid blood ran out. The pulsating brain-convolution then became visible for the first time. Blood continued to pour out of the subarachnoid space, and was only controlled by packing gauze deep in between the convolutions of the brain. A dry gauze dressing was applied with moderate pressure. The operation was well borne. The bandage was quickly stained with blood, and required constant reinforcing for two days. On the third day the gauze packing was removed, and the bleeding was found to have entirely stopped. A gauze dressing was then placed outside the dura.

The patient was seen by Dr. J. J. Putnam on that day, who made the following report: "There is paresis of movement of the upper lip, especially noticeable in talking. The tongue protrudes to the left, but lies straight in the mouth. There is no loss of sensibility. There is very slight imperfection in the formation of some letters, and difficulty in saying the alphabet rapidly; no paralysis of the legs or arms. Knee-erks exaggerated, but not different from each other."

On the fifth day the temperature fell to normal and the pulse was 70. The

tongue came out straighter, and there was less difficulty in talking. The patient thought his voice had sounded unnatural ever since the accident. December 6, eleven days after the accident, the pulse and temperature were normal; the patient felt bright and well; the tongue came out straighter; he was still unable to say the alphabet rapidly; the wound was closing up. Recovery was complete.

The hemorrhage in this case was undoubtedly over the lower part of the fissure of Rolando.

TRAUMATIC INTRACRANIAL HEMORRHAGE.

This is a very important subject from a surgical point of view, and I am surprised that we hear so little about it. It will, no doubt, be found, like appendicitis, to be of frequent occurrence in communities where it is understood. I happen to have been called to see three cases in the Accident-Room of the Massachusetts General Hospital in the last five months. Two of the cases are here reported in full. The other was a child whose head was so smashed that the bone fragments could be moved past each other. The scalp in the temporo-parietal region was tense with blood, which had found its way through the fracture. The patient was so nearly pulseless that no operation was done. The diagnosis was middle meningeal hemorrhage, with laceration of the brain, and probable rupture of the lateral sinus. The child died in a few hours, and an autopsy showed the diagnosis to be correct.

From the practical surgeon's stand-point we have three sources of intracranial hemorrhage to consider, which are, in the order of their importance,—

1. Middle meningeal hemorrhage,—extradural.
2. Hemorrhage from the pia-mater,—subdural.
3. Hemorrhages from the sinuses.

Hemorrhages into the brain-substance will not be considered here, as this paper is intended to deal only with the more common surgical emergencies.

1. MIDDLE MENINGEAL HEMORRHAGE.

It is of practical importance that the squamous part of the temporal bone, traversed by the posterior branch, and the anterior inferior angle of the parietal bone, which is grooved or tunnelled by the anterior branch of the middle meningeal artery, are the thinnest parts of the vertex of the skull. This accounts for the fact that a very slight blow has often caused a fatal hemorrhage. The grooves for the artery in the bones cause such weakness as to invite the line of fracture to follow them. Indeed, fracture of the base often extends upward in these grooves, and is accompanied by middle meningeal hemorrhage, as in Dr. Warren's case.[1]

The artery and its branches can in a general way be said to lie behind the parietal and squamous portion of the temporal bone. In this region is also the fissure of Rolando with the motor area, which, of course, accounts for

[1] Amer. Journ. Med. Sciences, May, 1890.

the paralysis in these cases, and makes a more or less accurate localization of the clot possible.

Middle meningeal hemorrhage often occurs without a fracture of the skull, and may even occur on the opposite side from the blow from *contre-coup*. In such cases the blow detaches the dura from the skull, which process tears the artery. Jacobson[1] found that in eight cases out of seventy the hemorrhage occurred without fracture, and that in thirty-eight out of sixty-two cases the base of the skull was involved. He also found that the main trunk of the artery was rarely injured.

According to the same authority, the extravasation will vary according as the injured branch is large or small. It may—

(*a*) Be rapid and quickly fatal.

(*b*) Be delayed for some time.

(*c*) Take place in two stages, the first slight and producing no coma.

The clots are nearly always dark or black, and disk-shaped, thick in the middle, with a definite margin. Old clots are granular and adherent, and difficult to remove.

The amount of violence required to cause the hemorrhage may be slight; for example, a blow from a cricket-ball, or from a poker, or with a spade, or a slight fall of two feet six inches has been sufficient. It is, however, nearly always caused by a severe fall.

Symptoms.—According to Jacobson, the conditions to be considered are:

(*a*) Interval of consciousness or lucidity.

(*b*) Condition of limbs as to hemiplegia, paraplegia, rigidity.

(*c*) Condition of the pupil.

(*d*) Character of the pulse.

(*e*) Unconsciousness increasing and passing into coma.

(*f*) Character of the respiration.

(*g*) State of the scalp.

"It is well known that between the time of the concussion or stunning of the patient by the original violence and the supervening of symptoms of compression due to the blood which is now effused, there often exists *an interval of more or less marked consciousness*, the value of which has long been recognized." This is not always present, and not always well marked when present. It varies from fifteen minutes to ten days. It is not marked when the concussion is very severe and lasts a long time; for during this time the brain becomes compressed, and thus consciousness may not intervene. During the interval a child has been known to eat its dinner. Patients have had scalp-wounds dressed and walked away from the hospital by themselves. In this way one of the most fatal of head injuries may be overlooked, owing to the slight violence and the delay in the symptoms of compression. Patients have been allowed to sleep, and have become comatose without waking. Frölich mentions the case of a patient who was hit on

[1] Guy's Hospital Reports, vol. xliii.

the head with a cane, and walked for an hour and a half. When he arrived home he vomited and became comatose, and died in five hours. At the autopsy there was no fracture, but a large clot from middle meningeal hemorrhage was found. The interval of consciousness, when present, is considered the most important point in the diagnosis, but Jacobson found it absent in one-third of the cases collected by him.

Hemiplegia, or *paralysis* of the limbs on the opposite side to the injury, is a very common condition in middle meningeal hemorrhage, but is not necessarily present, and when present varies greatly in degree. The absence of all paralysis is, however, according to Jacobson, a rare thing in these cases.

Rigidity of the limbs, combined with convulsive movements, usually indicates contused and lacerated brain-substance.

If the *pupils* are natural in reaction, the compression is not severe. If the pupils are insensitive and dilated, the compression is extreme. If one pupil is widely dilated and the other is natural or contracted, and if the dilated one is on the injured side, it is considered to be a sign of great importance. Mr. Hutchinson explains this by pressure on the trunk of the third nerve. This explanation is open to doubt; certainly in Case I., here reported, the clot could not have pressed on the third nerve, and yet the symptom was present.

The *pulse* will vary according as the brain is compressed, or lacerated and contused. In uncomplicated pressure cases it is usually about forty. In serious cases of brain-laceration or contusion it is rapid and feeble.

The state of *coma* varies with the size of the clot effused. It may come on rapidly or slowly, according to the size of the artery ruptured. Commencing coma may be taken for natural sleep, and the patient may be allowed to lie until it is too late. It has also been taken for drunkenness. If coma comes on late, it is usually sudden and rapidly fatal.

The *respiration* is usually stertorous, is sometimes slow and sometimes rapid, but usually ceases suddenly.

The condition of the *scalp* is important when no history is known. Ecchymoses and puffy or pulpy spots over the parietal or temporal region would, of course, suggest an injury in that region sufficient to cause middle meningeal hemorrhage. Sometimes hemorrhage may make its way through a crack in the skull, and be felt tense and fluctuating under the scalp. When there is doubt, such pulpy places may be incised to see if the skull is fractured beneath; and even if no fracture is found, the trephine should be used, if other symptoms make the diagnosis of middle meningeal hemorrhage probable.

The treatment should consist in trephining and cutting away as much of the bone as may be necessary to remove the clots and arrest the hemorrhage.

Where shall we trephine? Of course, if a fracture is present, we must trephine in the line of the fracture. But with no outside clew to the seat of the rupture, where shall we trephine?

Wiesmann[1] has suggested that the symptoms will vary according to the location of the hæmatoma, and that we should trephine accordingly. But we cannot always observe the patient from the beginning. He is often brought to the hospital in deep coma or in a condition of drunkenness, and the case may be complicated with brain-laceration. Vogt and Beck have suggested trephining at a point one and one-half inches above the zygoma and one and one-half inches behind the external angular process of the frontal bone. An inch trephine so placed is certain to expose the anterior branch of the middle meningeal artery. But removal of the clots which cause the compression is much more important than finding the artery. Indeed, the bleeding itself—*i.e.*, the amount of blood lost—is rarely important, and is usually easily controlled by pressure,—in fact, it has often entirely stopped before the surgeon sees the patient.

Krönlein[2] makes the suggestion of trephining twice, if necessary, in the positions where the chances of finding the clots are good. He says that a large hæmatoma may fill the whole side of the vault as far back as the tentorium, but usually the hæmatomæ are less extensive, and may be divided in a general way into three classes,—

1. Fronto-temporal,
2. Temporo-parietal,
3. Parieto-occipital,—

according to their situation. (See figure.) He suggests trephining first at the point suggested by Beck, because that position would expose either a fronto-temporal or a temporo-parietal hæmatoma; if no hæmatoma is found there, and the indications still point to middle meningeal hemorrhage, to trephine again on the same horizontal line just below the parietal eminence, because an opening in this position would expose either a temporo-parietal or a parieto-occipital hæmatoma.

Having found the hæmatoma, it should be removed with the finger, or washed out, and the bleeding stopped.

II. SUBDURAL HEMORRHAGE.

Dr. S. W. Gross, quoted by Jacobson, says, "Extravasation into the arachnoid sac coexists with nearly all of the severe contusions of the cortex of the brain, when it is due to laceration of the vessels of the pia mater with simultaneous rent of the visceral arachnoid. It is also occasioned by rupture of the cerebral veins, or of the great sinuses, or it may depend upon a wound of the dura mater. . . . The symptoms of this accident vary in intensity. In some cases there is no evidence whatever of cerebral disturbance; and even when the effusion is very considerable, provided that it be diffused or spread over both hemispheres, the phenomena are vague and masked by those of laceration and contusion. When, on the other hand, the fluid is

[1] Deutsche Zeitschrift für Chirurgie, Bd. xxi.
[2] Deut. Zeit. für Chirurgie, Bd. xxiiii.

more circumscribed, or the compression is limited to one hemisphere, the symptoms are decided, but they are identical with those produced by effusion between the bone and the dura mater from injury of the middle meningeal artery."

According to Hutchinson, the bleeding in these cases usually takes place not from one large vessel, but from many small ones, and hence it is poured out with much less compressive force, and ceases much short of the results which a rupture of the middle meningeal artery can produce. A subdural clot will usually, to a greater or lesser extent, bruise and even tear the brain surface. Spasm due to irritation of the motor cortex may be present. Paralytic symptoms may be definite and pronounced, if the lesion is in the motor area, as in Case II., here reported.

Commonly the symptoms are identical with those of middle meningeal hemorrhage,—viz., more or less marked coma, dilated pupils, slow, full, and labored pulse, hemiplegia, or diminution of temperature of the opposite limbs, and slow, noisy, or stertorous respiration. In the majority of cases there is also the interval of consciousness, but absolute insensibility may exist from the first.

III. HEMORRHAGE FROM THE SINUSES.

This may be extra-dural or subdural, according to the location of the rent in the sinus.

It is popularly supposed that hemorrhage from the sinuses must necessarily be fatal. This is by no means true. I have myself on two occasions been able to control hemorrhage from a sinus by pressure with antiseptic gauze. In one case the hemorrhage was external,—that is, was pouring outward through the fracture; in the other, the hemorrhage was from the lateral sinus and intra-cranial, causing brain-compression. I was able in this case, by removing a loose fragment of bone, to allow the escape of the intracranial extravasation and to plug the sinus. Both of these cases died several days later of extensive brain-injury, but not of hemorrhage. This plugging of sinuses is no uncommon procedure. The rent in a sinus may also be closed with a fine needle and suture. Indeed, Bergmann entirely removed a part of the superior longitudinal sinus in one of his cases. The researches of Schellmann have shown that the integrity of one sinus at least may be destroyed without any serious effect upon the brain itself.

With our accumulated experience with extra- and subdural hemorrhage I see no reason why brain-compression following laceration of one of the large sinuses should not be successfully diagnosed and treated by trephining, even where there is no external evidence of fracture of the skull.

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