Bremer (L) & Carson (N. B.)

Aphasia Due to Sub-dural Hemorrhage Without External Signs of Injury;

Operation; Recovery.

BY

L. BREMER, M.D., AND N. B. CARSON, M.D.,
OF ST. LOUIS.

FROM
THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES,
FEBRUARY, 1892.

Extracted from
The American Journal of the Medical Sciences for February, 1892.

APHASIA DUE TO SUB-DURAL HEMORRHAGE WITHOUT EXTERNAL SIGNS OF INJURY; OPERATION; RECOVERY.

By L. Bremer, M.D., and N. B. Carson, M.D.,
of St. Louis.

The history of the patient, H. T. K., a healthy and well-built man of about twenty-one years, is as follows:

There is neither heart nor kidney disease. Two weeks previous to my seeing him he went to a wedding, got intoxicated, and on his way home fell between the joists of a new building. This was his statement subsequent to his recovery after the operation. He went home and was found asleep in the kitchen of his parents' house the following morning. Except what appeared to be the effects of the liquor, he seemed in his usual health. In fact, nothing was mentioned by him as regards the fall. Being out of work, he stayed at home and rarely left the house, complaining off and on of a dull headache on the left side of the forehead, with exacerbations in the afternoon. Occasionally he vomited, but had generally a good appetite. All this while there was not the slightest suspicion on the part of his family of any serious trouble.

About one week after he had begun to stay at home, while walking on the street, one block away from his home, he suddenly became unconscious and fell. This attack did not last long, however, and he was assisted home by a person who happened to be near at the time. When he arrived at the house he was able to undress himself and went to bed. Shortly afterward it was discovered by his family that he had some difficulty in speaking. He now for the first time intimated to his family that he met with an accident on the night of the wedding. Dr. H. F. Hendrix was called in, who, in addition to the dysphasic disturbance, noted other symptoms, especially a slow, laborious pulse indicative of brain lesion. He observed that the patient was more or less speechless in the afternoon, when a moderate fever of about 101° would set in, whereas in the morning, when free from fever, the difficulty of speech would be much less, and he had many more words at his command than in the afternoon.

When I saw him for the first time, thirteen days after he first commenced to complain, it was stated that for the last three days he had been entirely unable to speak. On the day previous to my visit Dr. Hendrix had found his pulse to be 54.

The patient seemed to be quite rational, judging from the looks of his eyes and the expression of his face. There was no trace of an injury to his head. He understood every word that was spoken to him, every question that was asked. Unfortunately, although not entirely illiterate, the patient was not possessed of sufficient education to render the

examination of this form of aphasia very profitable. Only the most elementary questions could be asked of him, the scope of his intellect being limited.

In order to test his mental calibre and ascertain the nature of the trouble of speech, a number of questions were put to him. The principal ones were:

Do you know what this is (showing him a glass)?

Ans. Zer—

Q. Is it a glass?

Ans. Yes.

When a pitcher is shown him, he calls it a "tipper;" a "pen" he calls "riglah;" a spittoon "sempen;" a hat "sem."

Q. Do you call this (the hat) "sem"?

Ans. No.

Q. Is it a hat?

Ans. Yes.

Q. What is this (showing him a match)?

Ans. "Ses."

In order to demonstrate that he knows what it is, he makes the movement of striking a match. A book he calls "pok;" handkerchief, "sempence;" suspender, also "sempence;" for pocket-knife he gives the correct name; but when shown a bunch of keys, he also says "pocket-knife." After this he calls everything that is shown him pocket, *e. g.*, a watch and a button.

When ordered to repeat a word that is spoken to him, he is unable to do so.

He understands perfectly what he reads. He is handed a newspaper, and an advertisement of an entertainment in the Exposition Building is pointed out to him. By putting a variety of questions, some of them misleading, I convince myself that he is familiar with the location of the building, and the purposes it is built for.

He is asked to read the advertisement of a boxing-match. I point out the name of the prize-fighter, and ask him: What is he? Is he a preacher? This causes him to laugh.

In short, there is no flaw in his perceptive and reasoning powers as far as can be ascertained by a necessarily limited conversation, and as far as a short acquaintance will permit.

The most prominent of the other symptoms is a beginning obliteration of the naso-labial fold on the right side; on showing his teeth, the left naso-labial fold becomes much more marked than the right, and the left angle of the mouth is drawn considerably to the left; during an effort at whistling the right cheek puffs somewhat. The tongue deviates to the right. He cannot well draw the right angle of the mouth to the right, or make the right platysma muscle contract. On the latter symptom, however, not much stress is laid, because an effort on the other side is not very successful, and many people, even in absolute health, have not the power of contracting this muscle either singly or together with its fellow.

The grip of the right and left hands seems to be almost equal; he moves his arms with absolute freedom, and nothing abnormal can be seen in his walk. He stands on the right leg with the same ease as on the left.

On being told to alternately flex and extend the right index finger,

there is an associated movement of the other fingers; and on trying to move the right thumb, the right index also moves in a rather clumsy, erratic manner. All such movements of the fingers on the left side are executed with precision, no associated movements of the others being noticeable.

Sensation (tested with a pin) is somewhat dulled on the whole of the right side. The main dulness is in the fingers, the palm and back of the hand, and the wrist; it is less higher up to the elbow and shoulder, and much less in the face. But, as just stated, the whole of the left side, including the leg, shows a defect in common sensibility. The same is true of the sense of temperature and pain. Passive movements of the fingers of the right side are not so well perceived as those of the left, showing a lowering of the muscle sense. The passive movements of the toes on the right side, however, are correctly stated. There is no ataxia in the right arm or hand; without hesitancy he carries his right index to the tip of the nose, the eyes being closed, and puts with precision the tip of the finger on the point of a pin.

But on being told to write, he holds the pen in an awkward manner, and drops it repeatedly. He never has been much of a penman, but has been able to write simple letters. It is now impossible for him to express his thoughts in writing, and even the most commonplace and every-day expressions, when dictated, he fails to fix by letters.

The effort at writing his name is more of a success. While his inability to write words, even the most familiar ones, is very marked, he puts figures with comparative ease. Thus in writing what is meant for "April 28, 1891," he writes 28 and 1891 without the slightest hesitation. This facility of writing figures and difficulty, amounting often to impossibility, of penning words was tested in different ways, always with uniform results. There is no visible abnormality about the eyes; no inequality of the pupils, no hemianopsia. Nothing of a spastic character is observed in any of the muscles of the affected side. Patient is right-handed.

From the foregoing data the diagnosis was made: Blood-clot (probably extra-dural) pressing principally on the foot of the third frontal (Broca's) convolution and the foot of the second frontal (probable centre for writing), impinging also on the face and tongue centres of the left hemispheres.

The next day all the symptoms were more marked; the grip of the right hand was weaker than that of the left; pulse 43.

The operation of trephining was now set for the following day, and the patient transferred to the Mullanphy Hospital. While the preparations for operation were in progress, the patient was once more examined as to the general and localizing cerebral symptoms. As regards the latter, it was found that they had become more vague and indistinct. The patient did not answer questions as readily as on the preceding days; it took him a longer time to comprehend their import. While during the first half-hour of the examination he tried to read from the questioner's mouth, he grew listless and inattentive later on. Although there was no outspoken hemiplegia, he dragged the left foot,

when told to walk, which had not been the case on the previous day. The grip of the right hand was also much weaker, the paresis of the right side of the face and the deviation of the tongue more marked. The dulness of sensation had increased in proportion to the motor weakness. How much, however, this was to be attributed to a want of attention and increasing mental hebetude was difficult to decide.

There could be no doubt that the pressure on the brain was rapidly increasing. During the last hour consumed in the examination, the patient grew more and more confused and listless; this was not entirely due to the fatigue attending upon keeping his waning mental faculties at work, for it could be distinctly ascertained that the grip of his right hand became more feeble, and that his right leg became unable to bear the weight of the body—so much so that he was unable to walk to the operating-room, and had to be carried there on a stretcher.

The pulse when last examined was 56 as against 43 on the day previous, this being the lowest figure ascertained.

The operation, by Dr. N. B. Carson.—H. T. K. was admitted into the St. Louis Mullanphy Hospital, April 29, 1891, with the history given above, and the symptoms calling for an operation.

The head having been rendered aseptic, a semicircular flap, with its base forward and its convexity backward, was raised together with the pericranium so as to expose the antero-lateral portion of the skull, with the pterion as a centre. The temporal vessels bled freely, and had to be clamped before the operation could be continued.

An inch button was then removed with the trephine an inch and a quarter behind the external angular process, and the same distance above the base line.

Upon exposing the dura, it presented a dark, cloudy appearance, and the vessels were empty and flattened. All evidence of pulsation was wanting; the trephine opening was enlarged in every direction, more posteriorly, however, than otherwise, until an opening deemed sufficiently large had been made. The dura was then opened to full size of cranial opening, and a clot extending in all directions beyond the cranial opening was exposed to view.

Upon raising the dura, a stream of dark, semi-liquid blood forced itself through the superficial layer of the clot, and spattered myself and assistants two and three feet distant. The thickest part of clot seemed to correspond to the centre of trephine opening, and was immediately under anterior branch of middle meningeal artery, between the dura and arachnoid., With dull-edged curette the greater part of the clot was removed, and the smaller portions subsequently taken away with a very fine flat sponge held in forceps. In this way I wiped out the entire cavity occupied by the clot, which almost reached to the longitudinal sinus above, the base of the cranium below, and at least an inch and a half anteriorly and the same distance posteriorly.

Being satisfied that there was no more bleeding, the dura was replaced over a horsehair drain, but not sutured. A rubber drain was then placed under the flap, which was replaced and sutured, and over all a

dressing applied, and the patient returned to bed in good condition. Before I left the hospital, an hour and a half later, the patient had returned to consciousness apparently none the worse for the operation.

6 P.M. (day of operation). Temperature, 98.4°; pulse, 86; reacted very nicely.

April 30 (first day after operation). Temperature, 99.3°; pulse, 70; respiration, 22; clonic spasms in right side of face and right platysma myoides. Is stupid and unable to speak; complete motor aphasia. 6 P.M., temperature, 99.3°; pulse, 70; conscious, but unable to use the right word.

May 1 (second day). Temperature, 98.3°; pulse, 72; entirely free from spasms. In all efforts to speak prefixes "shay" to words. 6 P.M., temperature, 99°; pulse, 76; brighter look; tongue slightly deflected to the right when protruded; elevation of lip improved. Can answer "Yes" and "No" correctly, yes having the "sh" sound very marked. A watch was pronounced "swatch;" keys, "shkeys;" half-dollar, "shalf-dollar."

2*d* (third day). Temperature, 98.4°; pulse, 66. In answer to questions, said, "he felt well," that "he liked the hospital." Can speak words without sibilant sound. 6 P.M., temperature normal; pulse, 68; doing well.

3*d* (fourth day). Temperature normal; pulse, 66; dressed, horsehair removed. Union by first intention. All words spoken correctly. Replied to questions by answers of three words correctly. Sensation in right arm still impaired. Sharp points on right arm recognized as two points one and a half inches apart, on fingers two inches apart. Unable to feel blowing of breath on hands or arm. 6 P.M., temperature and pulse normal. Improving in all respects.

4*th* (fifth day). Temperature normal; pulse, 54; 6 P.M., temperature and pulse unchanged.

5*th* (sixth day). Temperature normal; pulse, 62; sensation improving. Can speak and write as well as before the accident.

6*th* (seventh day). Temperature and pulse normal.

7*th* (eighth day). Temperature, 98.4°; pulse, 56. 6 P.M., temperature and pulse normal.

8*th* (ninth day). Temperature and pulse normal. 6 P.M., temperature and pulse normal.

9*th*. Discharged cured May 18th, nineteenth day after operation.

June 10. Returned to clinic to consult about a numbness or tired feeling in right foot, only after exertion and when fatigued.

The question so often asked, In what cases of intra-cranial hemorrhage are we to operate, and what are the symptoms that should make us decide to interfere in these cases? is, in my opinion, still unanswered.

Keen[1] says: "The importance of operative treatment is best shown by Wiseman, who collected 147 cases treated expectantly; out of this number 89.1 per cent. died, while, on the contrary, out of 110 cases treated actively only 32.7 per cent. died. This certainly points very decidedly in favor of active treatment."

[1] Handbook of the Medical Sciences, page 227.

But what surgeon is there that has not seen many cases where the symptoms pointed conclusively to an intra-cranial bleeding, that, after a varying lapse of time, had recovered as effectually as if the trephine had been applied?

A very interesting case bearing upon this subject was reported by Dr. Warren in a discussion following a report of a case of trepanation for sub-dural hemorrhage, reported by Drs. Homans and Walton recently.[1]

In this case a diagnosis of clot having been made, and while preparations for an operation were under way, the patient began to move the paralyzed limb, and eventually made a complete recovery without an operation.

It is to be remarked, however, that although the diagnosis of clot was probably correct (the reasons for the diagnosis are not given), it was by no means certain. Without going into details, I will simply suggest that hemiplegia without focal lesions does not count among the impossibilities.[2]

Again, in rapidly improving cases of hemiplegia without operation, with result of complete restoration, a diagnosis of clot, if it has been made, should be subject to reconsideration, and if the symptoms have not been cogent and unequivocal, the possibility of a transient thrombosis (or embolism) of the sylvian artery should be thought of as being best calculated to explain such cases.

I make this remark on general grounds, and without any special reference to the case which has given rise to it.

Another case, nearer home, has just come to our knowledge, and is reported further on. Here, too, blood-clot was the diagnosis, by Dr. Bremer. For the reason that decided signs of improvement are demonstrable, operation was delayed. The patient is now making a good recovery.

On the other hand we see cases—and I think most often—presenting not any more decided symptoms, that die because their friends will not consent to an operation.

In our case there was no doubt as to the propriety of an operation: first, for the reason that the symptoms of hemorrhage were so very plain; and, second, because the symptoms were rapidly growing worse. While two hours before being brought to the table the patient walked with only a slight halt, he could not move the limb when the time for the operation had come.

[1] "A Case of Successful Trephining for Sub-dural Hemorrhage produced by Contre-coup," Boston Medical and Surgical Journal, February 12, 1891.

[2] Pillet: "Hémiplégie sans lésions en foyer de l'encéphale," Progrès Médical, 1890, No. 7.

In selecting cases for operation, we should not be too hasty in rushing into the cranial cavity, as we have seen some of these cases recover completely, and that, too, without the sequelæ that are said to be dependent upon head injuries.

Unless the condition of the patient demands immediate action, he should be treated with a view of controlling the hemorrhage and causing absorption of the clot.

A careful watch ought to be placed over him, and so soon as there is an evident increase in the symptoms, then, and not until then, should the operation be undertaken.

To one point of paramount importance in the management of intracranial hemorrhage I should like to call attention. It is based on the observation that patients of this class are prone to assume the horizontal position and go to sleep; often this is a sleep from which there is no awaking. Owing to a simple mechanical law, renewed bleeding is apt to take place much more readily in the horizontal than in the erect position. The patient, therefore, as soon as the nature of the trouble has been established, or is only suspected, should be propped up by pillows, and kept in that position as long as possible. I am certain that many an unlooked-for catastrophe might be averted in this manner. My experience is, that this simple precautionary measure is only too often neglected by physicians and laymen.

(By Dr. Bremer.)

Considering the present advanced state of cerebral localization, the diagnosis was comparatively an easy matter. The case presented a group of the clinical focal symptoms which form, so to speak, the very groundwork of local diagnosis in brain disease. That a blood-clot was the cause of the trouble could not be doubted considering the manner of development of symptoms and the history of the case.

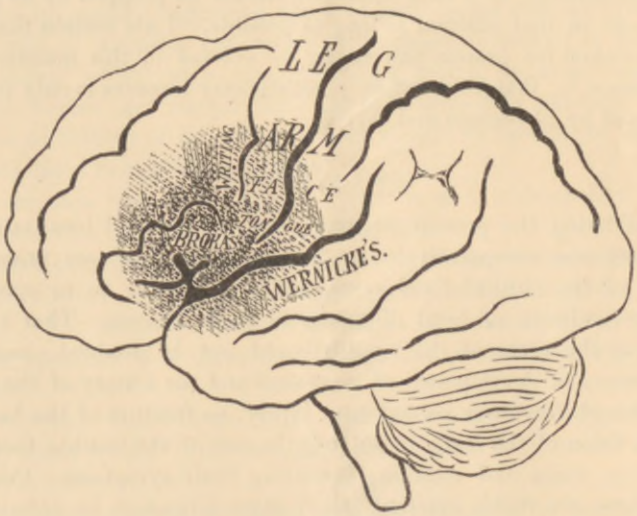
Although there was no external injury, no fracture of the bone, not even a lesion of the scalp, to point to the seat of the trouble, there were the more exact and unfailing localizing brain symptoms. Compared with these, the visible external injury plays nowadays an inferior *rôle*. This is well illustrated by cases in which the external injury exists but the site of the symptom-producing clot is opposite to that of the injury.

Such a case has lately been reported by Homans and Walton,[1] in which a hemorrhagic clot was diagnosticated on the opposite side of the injury (this being on the right side). Aphasia was also one of the localizing symptoms. Contrary, however, to our case, the brain substance itself was involved, which gave rise to Jacksonian epilepsy. This was, as remarked above, entirely absent in our case. In Homans' and

[1] Loc. cit.

Walton's patient it was probably a branch of the middle cerebral artery that was ruptured, implicating the cerebral cortex directly and causing discharging symptoms; whereas, in our instance, the smooth and shining arachnoid intervening between the clot and dura could be made out very plainly.

Keen[1] says: "No diagnosis can be made at present between hemorrhage from the middle meningeal and the middle cerebral." This is probably true for most cases. It is the generally accepted opinion among competent observers. Possibly, however, the presence or absence of irritative (spastic) phenomena may enable us, in a limited number of cases, to differentiate between cerebral and meningeal hemorrhage. In the former, localized and serial spastic disturbances are most likely to appear, owing to the direct involvement of the cortex; in the latter, provided the arachnoid is not torn, as in our case, the paralytic symptoms will be the first to appear. General epileptic convulsions, as the result of diffuse brain pressure, will, of course, not count as a differential diagnostic factor.



The extent of the clot covered a larger area than had been expected. It is outlined in the accompanying figure, and shows the various centres pressed upon, and the outline is, of course, approximate. It did not extend to the top part of the Rolandic region—the leg centre. The fact that the latter became implicated is probably to be explained by a pressure through the brain matter on the corresponding part of the internal capsule. It is seen that the clot extended down to the first

[1] Reference Handbook of the Medical Sciences, vol. viii. p. 226.

temporo-sphenoidal convolution, which would account for the difficulty on the part of the patient, on the morning before the operation, to understand what was said to him (word-deafness). The darkest shade denotes the greatest thickness of the clot, and is over Broca's convolution, the foot of the second frontal and the lower end of the anterior central, which is in keeping with the prominent symptoms—motor aphasia, a gradual paralysis of the face and tongue. The arm and wrist centres are less implicated.

A brief allusion to a few points bearing on this and similar cases will, perhaps, not be out of place. How is the fact to be explained that, during the first week after the supposed occurrence of the hemorrhage, there were almost no symptoms of brain pressure, and that suddenly, a week later, alarming cerebral symptoms developed, which gradually grew in intensity almost to a fatal issue?

The concurrent testimony of observers of this class of cases goes to show that the bleeding has ceased before the operation has been resorted to. Whether arterial or venous in origin, the blood is found in the coagulated, not in the fresh or fluid, state.

In our case the clot was old; no distinct layers could be made out. Such layers, if present, would indicate different successive hemorrhages. There was a discharge of tarry blood breaking through the clot on opening the dura, but nowhere was there any evidence of recent hemorrhage or a recent coagulum. The anterior main branch of the middle meningeal artery was empty, and pressed flat like a ribbon. The same was true of two of its anterior subdivisions that came within the field of operation. The probability, therefore, is that the hemorrhage was stopped long before the operation; that the clot had acted as a sort of tampon, compressing the ruptured branch and the main artery, and that by this "auto-tamponade" any further hemorrhage was prevented.

It will be remembered that the diagnosis of extra-dural clot had been made. This was done on account of the insignificance of the initial symptoms and of the comparatively slow development of the graver ones. *A priori*, it stands to reason that a hemorrhage between the skull-bone and the tough and unyielding dura is apt to be more limited, and to produce less rapidly cerebral symptoms than sub-dural hemorrhage, although it is a well-established fact that just in the neighborhood of the meningeal arteries the dura is less firmly attached to the bone than elsewhere. Besides, in the vast majority of ruptures of a middle meningeal artery or its branches the hemorrhage takes place upon the dura. For these reasons the diagnosis of extra-dural clot seemed to be the more likely one. This was a mistake, and, I believe, a mistake that could have been avoided on the ground that probably all meningeal hemorrhages due to indirect violence are sub-dural. This point will be enlarged upon further on.

Now, taking the sequence and course of the symptoms into consideration, one would, basing one's reasoning upon simple mechanical laws, be apt to conclude that, one of the smallest anterior branches having burst, the hemorrhage was at first extremely limited and insignificant, and pressing moderately on the left frontal lobe, anteriorly to the well-known centres, no positive symptoms developed, and that only the general ones of mental dulness (the pulse was not examined during the first week) and vomiting were present; that gradually, or in turns, slight fresh hemorrhages ensued, pressing successively upon the centres for articulate and written speech; on those of the angle of the mouth, tongue, arm; then upon the centre for the interpretation of words heard (first temporo-sphenoidal convolution). Thus, *e.g.*, the fall and short attack of unconsciousness one week after the accident might be due to a recurrent hemorrhage. Again, in this manner might the inability of the patient to understand what was said to him shortly before the operation be accounted for, if one does not prefer to ascribe it to a lowering of the intellect preceding a gradually approaching coma.

Much more likely, however, to my mind, is another explanation. In meningeal, as in cerebral hemorrhage, there are two factors which have to be considered as productive of brain pressure. The one is the mass of blood extravasated, the other is the amount of reaction which is bound to arise in consequence of pressure-irritation.

The brain will tolerate a certain amount of local pressure, according to the nervous make-up of the individual, as has been established by the clinical histories of a number of cases of cerebral tumors which may exist without symptoms, regardless of their size, until suddenly their presence becomes manifest by paralytic or discharging symptoms. For there is a limit of tolerance in cases of clot as well as of tumor.

Thus, in our case, the at first gradual, and afterward rapid, supervention of paralytic symptoms might be explained in this way: There is a clot extending over a large part of the left hemisphere, covering a number of cortical centres. The brain mass accommodates itself to the pressure when gradually developed, and there are only slight symptoms until the branches of the vessels that enter at the base of the brain—viz., the anterior perforated space—respond to the irritation coming from above. It is, to my mind, conceivable that after a certain amount of pressure has been borne by the cortical and sub-cortical substance, the branches of the lenticulo-striate artery and other neighboring ones react according to the old pathological maxim, *ubi irritatio ibi affluxus.*

This afflux of blood means increased pressure from the opposite (under) side of the clot—a pressure which not only bears upon the cortex, but also on the conducting fibres below, including the internal capsule. The rapidly-developing complete hemiplegia involving the

leg, which for a long time was intact, may be accounted for in this way.

Considering this rapid progress of alarming symptoms, the operation was performed in the nick of time, and may, without doubt, be set down as a life-saving act.

The establishment of the exact source of inter-meningeal hemorrhage is often a matter of impossibility. Whenever there is a fracture of the skull, the trunk of the middle meningeal artery or vein may, of course, be lacerated, together with other soft parts, by direct violence. It is also said that hemorrhages of the middle meningeal artery have been observed without injury either to scalp or bone. Merkel[1] points out that it is principally the anterior branch of the artery that is ruptured. But it is hard to understand how an artery or vein of the dura mater can be ruptured by concussion due to indirect violence, unless there is a hemorrhagic diathesis, a disease of the vascular wall, a miliary aneurism, for instance. Such abnormal vascular state we have no right to assume in a young man of twenty.

Judging from the clinical course of our case we must, I think, dismiss the idea of arterial hemorrhage, whether of a smaller or a larger branch, at once. The sudden increase of pressure such as would be likely to result from arterial bleeding would make more pronounced and violent symptoms at the start. A slow oozing from a vein and the gradual formation of the clot, allowing the brain sufficient time to accommodate itself in a measure to increased pressure, is the more probable process.

But how are we to conjecture the cause and nature of venous meningeal hemorrhage? Is it probable that from indirect violence, from mere concussion (for it is not at all likely that in the fall the head of our patient was struck) a healthy vein should burst?

Perhaps the interesting researches and observations of Dr. Mittenzweig[2] will, if enlarged and confirmed, throw some light on this vexed question.

Mittenzweig quotes Bergmann, who, in his book on Injuries of the Head, says (§ 260):

"The hemorrhages into the so-called arachnoidal sac are principally derived from those veins which pass from the upper and lateral parts of the cerebral hemispheres out of the pia to the longitudinal sinus. These are simply torn off, a tearing which, of course, can take place only by a considerable displacement of the brain *in toto*. Since it has been observed in some cases even without injury of the bone, it proves the very considerable change of form the bony cranial capsule is capable of, before its limit of elasticity is passed."

[1] Handbuch der topograph. Anatomie, 1885, vol. i. p. 68.

[2] "Subdurale Blutungen aus abnorm. verlaufenden Gehirnvenen," Neurolog. Centralbl., 1889, p. 193.

Now, from a series of observations Mittenzweig arrives at the conclusion that in a number of persons abnormal anastomoses between cerebral and dural veins exist, being the remnants of fœtal conditions which are normal. It is easy to understand how such abnormal venous anastomoses will tear on even slight displacements of the brain mass, and that an absolutely healthy and young individual may suffer from sub-dural hemorrhage as a result of comparatively little violence.

This probably happened in our case, and if anatomical research and clinico-pathological experience should bear out Mittenzweig's investigations and conclusions, an additional differentially diagnostic point may have been furnished us as to extra- and sub-dural hemorrhages.

In Dr. Carson's report it is stated that on the day following the operation muscular twitchings about the face and mouth were observed. I believe that these post-operative spastic phenomena are to be ascribed to capillary hemorrhages in the cortex of that part of the brain previously compressed by the clot. Continued pressure will produce a weakened, possibly a more or less atrophic condition of the cortical capillaries, or possibly venules and arterioles. The sudden removal of the pressure and the sudden rush of blood will then give rise to minute hemorrhages referred to. In a case (to be published by us in the near future) of a large sarcoma of the dura mater, pressing upon the motor area, such hemorrhage took place throughout the cortex directly and indirectly involved by the pressure as could be demonstrated by a post-mortem held two weeks after the operation. That capillary hemorrhage will result from even so slight an insult as light pressure of the surgeon's finger for the purpose of ascertaining the consistence of underlying cerebral structures, I could demonstrate in a case of epilepsy operated upon by Dr. Prewitt, of this city. The wrist centre was excised and the ablated piece of cortex showed throughout recent hemorrhages into the perivascular lymph spaces, and the brain substance itself. Thus, I think, the twitching, which was absent before the operation, might be accounted for after it.

The long interval between the accident and the appearance of any decided pressure symptoms is noteworthy. In one of Wiseman's cases the interval was still greater, being eleven days.

Owing to the shortness of time allowed for examining into the dysphasic troubles of the patient, and chiefly owing to his want of education, the yield in this respect is rather meagre.

In conformity with the topical lesion, the aphasia was of the ataxic variety, combined with agraphia. The latter was somewhat obscured by clumsiness of movements of the hand and fingers, owing probably to the simultaneous pressure upon the hand and finger centres. As usual, there was, in addition to motor aphasia, paraphasia and paragraphia.

It is rather singular, that, after the patient had tried to pronounce a

number of simple and short words, he all at once was able to say "pocket-knife." The tendency to call everything "pocket" after he had succeeded in pronouncing this word finds its analogue in other reported cases of motor aphasia. A beaten path for certain words and syllables remains intact amidst many others that are destroyed or obstructed, and every effort at speech is bound to travel this path.

The meaningless monosyllables of "zer," "sem," and "shay" must also be looked upon in this light. In prefixing these easily pronounceable syllables to other words he "oils," so to speak, the machinery of speech. With some people such meaningless and nonsenical prefixes are physiological; they not only fill out a gap in the thinking process, but they act as starters in the speech-mechanism.

In his efforts at writing he succeeds best, as is also usual in these cases, with his own name and with figures:

At the last (fifth) French Congress of Surgery, held in Paris (April, 1891), Dr. Michaux presented a case of non-traumatic meningeal hemorrhage cured by trephining. The topical diagnosis was made from focal symptoms. The clot was under the dura over the Rolandic region. It occurred in an alcoholic, and Dr. Michaux thinks the rupture due to alcoholism and uræmia.

In the case under discussion the alcohol certainly played, pathologically, a very subordinate part, and it is, to my mind, very doubtful whether there ever has been a well-authenticated case of idiopathic meningeal hemorrhage of the type exemplified by our case. If such is said to occur in an alcoholic, it must be remembered that a toper is more liable to accidents which are forgotten than anyone else.

This is not the place to discuss the necessity or justifiability of trepanation for blood-clot. In our case there was a vital indication for operative interference. Whether in a number of other cases it would be safer to trust to the healing and absorbing powers of the membrane and adopt the expectant plan, is a question as difficult to solve as the propriety of operating in given cases of appendicitis and ileus. But the concurrent testimony of the surgeons of to-day is in favor of operation, even where less urgent symptoms are present. Even with much less extensive hemorrhage than in our case the outlook as to complete absorption is far from favorable. Epilepsy and mental impairment are the usual sequelæ of neglected blood-clot in the brain, and it is certainly much more in keeping with the principles of conservative surgery to operate early and remove the possible and even probable cause of epilepsy, than to wait until the latter has declared itself, and the epileptic change has taken place in the brain.

A case of undoubted meningeal hemorrhage in which an operation was desisted from is the following:

On June 23, 1891, Mrs. L. fell, with her two-and-one-half-year-old child, down a flight of stairs and landed on a pile of bricks. She had managed to hold the child in her arms and believed that it had escaped injury. After her arrival home the child went to sleep. When it woke up, it vomited, lost consciousness, and passed into general convulsions. It remained unconscious for six hours, during which time it had a number of general convulsions, while it jerked continually with the right side in the intervals.

After the spasms had ceased, it remained unconscious for four hours. On waking up, it was paralyzed on the right side. After a few hours, however, the right leg moved slightly, whereas the arm remained perfectly motionless. At the same time the child had lost its speech.

An examination, on June 25th, revealed a slight depressed fracture large enough to hold the tip of the forefinger, and situated about three inches above the meatus auditorius externus. The child was aphasic, right leg somewhat paretic; knee-jerks absent on the right, normal on the left. Right arm paralyzed. Pupillary reaction normal. Pulse 70 and 65. Diagnosis: Depressed fracture of the parietal bone on the right side; meningeal hemorrhage by contre-coup on the left, involving the arm and motor speech centres. No paralysis of facial muscles could be made out.

On the 26th, she spoke a few words; an occasional slight voluntary movement of the right arm had been observed. June 30th, the child was able to stand on the leg and moved the arm much better. She had been able to walk a few steps. From this on the improvement was steady and rapid. July 10th, all paralytic symptoms had disappeared. The mother claims that the child talks much better than before the accident.

The question in this case would arise, whether it would not be a safe plan to at least elevate the bones of the depressed fracture, and, Is not this fracture, situated in the binauricular line, apt to lead to epilepsy later in life?

That such is not always the case I can prove in a young man twenty-two years of age, who, when five years old, had meningeal hemorrhage, from the effects of which (left-sided hemiplegia) he recovered in the course of six weeks. There is now a very perceptible depression about two inches above and a little anteriorly to the right auditory meatus. He has never shown any sign of epilepsy.

October 15, 1891.—The patient H. T. K. has been hard at work in a brick-yard for a couple of months. He is in his usual health with all his faculties intact, and a steady worker.