ON INGRAVESCENT APOPLEXY.

A CONTRIBUTION TO THE LOCALISATION OF CEREBRAL
LESIONS.

BY

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SIR THOMAS WATSON following Abercrombie and corroborating by his own observation the distinctions laid down by Abercrombie describes three forms of apoplectic attack.

“In the first form of the attack, the patient falls down suddenly, deprived of sense and motion, and lies like a person in a deep sleep; his face generally flushed, his breathing stertorous, his pulse full and not frequent, sometimes below the natural standard. In some of these convulsions occur; in others rigidity and contraction of the muscles of the limbs, sometimes on one side only.

“Now, respecting persons seen in this condition, the immediate prognosis is uncertain. Some die in a short time and much blood is found extravasated within the cranium. Some die after a rather longer interval, and

then we often find serous effusion only. And in some that die early, no effusion either of blood or of serum can be detected. Some recover altogether without any ill effect of the attack remaining. Others recover from the coma, but are left paralytic of one side, and with some imperfection of speech, or of one or more of the senses. And this paralysis and imperfection may disappear in a few days, or gradually depart, or remain for life.

In the second form of attack, the coma is not the earliest symptom. The disease generally begins with sudden and sharp pain in the head. The patient becomes pale, faint, and sick, and usually vomits, and sometimes, but not always, falls down in a state of syncope, with a bloodless and cold skin, and a feeble pulse. This also is occasionally accompanied by some degree of convulsion. Sometimes he does not fall down, the sudden attack of pain being accompanied by slight and transient confusion. In either case he commonly recovers in a short time from these symptoms, and is quite sensible and able to walk ; but the headache does not leave him. After a certain interval, which may vary from a few minutes to several hours—and Dr. Abercrombie records cases in which it was even much longer—the patient becomes heavy, forgetful, incoherent, and sinks into coma, from which he never rises again. In some instances, paralysis of one side occurs ; but perhaps more often, there is no palsy observed.

The disease when it comes on in this way, is much more uniform, and of much worse omen, than when it commences after the former fashion. Upon inspection, says Dr. Abercrombie, we find none of those varieties and ambiguities, which occur in the apoplectic cases, the coma being present from the first ; and the second class which we are now considering, he calls cases not primarily apoplectic.

The third form of attack described by Dr. Abercrombie can scarcely be said to be an apoplectic attack at all ; indeed he himself includes this form in the class of

paralytic cases. It is characterised by sudden loss of power on one side of the body, and frequently by loss or impairment of speech, without loss of consciousness."

To the second form of attack the name *Ingravescent Apoplexy* has been given. This I now proceed to consider. Its features are the absence of loss of consciousness at the outset, the gradual accession of symptoms, the almost invariably fatal result, and the large amount of blood found to be extravasated. I ought to add that vomiting invariably occurs at an early period of the case. My observations show, that in these cases not only is the hæmorrhage large, but that its situation is within certain limits constant, and I believe that an anatomical explanation can be given of all the phenomena characteristic of the attack.

The seat of the extravasation is on the outer side of the extra-ventricular corpus striatum (*Linsenkern*, lenticular nucleus) between this ganglion and the external capsule, a plane of fibres which separates it from the convolutions of the island of Reil, and it has appeared to me that the large amount of hæmorrhage is explained first by the size of the vessel or vessels ruptured, and secondly by the very slight resistance to extravasation. Also that the absence of loss of consciousness in the early stage of the attack is explained by the fact, that until the quantity of blood poured out is considerable there is no extensive rupture of fibres or ploughing up of grey matter, and probably no compression of brain substance.

A hemisphere in which hæmorrhage has occurred in the situation and to the amount here in question will present the usual external appearances suggestive of internal pressure, flattening of the convolutions, exclusion of blood from the minute vessels on the ridges, and obliteration of the sulci. If the examination is made simply by the usual sections many interesting points will escape observation. The fissures of Sylvius should be freely opened out, when less moisture will be found in that of the side on which the lesion has occurred than in its fellow, and the *pia mater*

will contain less blood; more remarkable, however, will be the condition of the convolutions of the insula, which will be flattened out and obliterated, so that the island of Reil has the appearance of a smooth oval elevation. Usually an incision into it at once exposes the clot. Occasionally, indeed, the thin layer of nervous matter over the effused blood is torn in the examination, especially when the clot extends under the anterior perforated space. I have never seen hæmorrhage into the fissure in these cases, though I have known the blood enter and fill the ventricles, burst through the floor of the third ventricle into the interpeduncular space and travel beneath the arachnoid to the Sylvian fissures, finding its way far into the one on the opposite side from the original lesion, but being excluded by pressure from the one near which it was originally effused.

After exploration of the Sylvian fissures the examination may be continued by the usual sections from above downwards. The blood may have penetrated the ventricle or it may have ploughed up the hemisphere on the outer side of the ventricular cavity. Occasionally in the latter case it even reaches the surface of the brain; in the only instance in which I have seen this, the blood had split up the ascending gyri on each side of the sulcus of Rolando (it never, so far as I know, escapes into the bottom of a sulcus). In either case, whether the clot is intra or extra ventricular, unless special care be taken it will probably be concluded that the corpus striatum is destroyed, or at least torn by the blood, whereas it will usually be found entire and intact, or only penetrated by a process of the clot, but completely dissected off from the hemisphere proper and displaced inwards. A detail of some little interest may here be added, namely, that when the blood has entered the lateral ventricle, while all the ventricles may be filled and the cornua distended by clot and fluid, the descending cornu on the side of the lesion will be all but empty, the blood being excluded from it by antecedent pressure on it by the earlier extravasation.

It will now be necessary to refer to the anatomy of the part of the brain in which the lesion occurs, and to describe in some detail the relation of parts, the course of fibres, and the distribution of blood-vessels. For this purpose drawings of sections of the brain are shown, which I venture to interpret from my investigations on the course of the fibres of the hemispheres.

The internal capsule, as it has been absurdly called, is constituted by the ascending and diverging fibres which enter the cerebrum below as the crus between the extra-ventricular corpus striatum and thalamus, and emerge above from the region of the central ganglia to enter the hemisphere proper as the corona radiata. If any fibres of the crus pass uninterruptedly to the hemisphere it is in this tract. As the crus passes into the interval between the ganglia its two divisions, presumably motor and sensory, the crusta and tegumentum, are distinct one from the other, but this separation is soon lost and in the corona radiata there is a complete intermingling of fibres, so that no distinction can be made between those which in origin and function are different. We are not here concerned with the rearrangement of fibres which takes place as the crus becomes corona radiata, but the increase in number is obviously very great. To this increase the thalamus contributes conspicuously, the corpus striatum less conspicuously, though probably more abundantly. It is not easy to trace fibres arising in the intra-ventricular corpus striatum and proceeding onwards to the hemispheres, but the radiating fibres in passing between the head of this ganglion and the extra-ventricular division of the corpus striatum separate into bundles between which the soft grey matter of the two portions dips in and comes into communication, probably giving off fibres. Between these bundles also M. Duret has followed to the intra-ventricular corpus striatum arteries which enter at the anterior perforated space, and far more numerous and larger veins which pass from the extra-ventricular corpus striatum and the hemisphere towards the ventricle, to appear there as the venæ Galeni.

The external capsule will also require a detailed description. It is seen in the various sections as a thin layer of white fibres separating the grey substance of the corpus striatum externum from the cortex of the insula, and it includes a plane of grey matter, the *claustrum*, which appears in the drawings as a thin streak. The convolutions of the island of Reil lying upon the outer wall of the corpus striatum have absolutely no connection with this ganglion, and the claustrum and all outside of it can be removed without disturbing a cell of the ganglion or a fibre arising in it. The corpus striatum then has only a very thin lamina of fibres of its own, forming its proper capsule externally; an equally thin lamina of fibres arising in its cells encases it on its inferior aspect. About these further particulars must be given.

When by careful dissection the proper wall or capsule of the extra-ventricular corpus striatum has been exposed, the temporo-sphenoidal lobe will almost have disappeared and the fissure of Sylvius will be converted into a wide hollow or groove curving round the ganglion which, as the brain lies base upwards, has the appearance of a smooth elevation embracing the crus. The fibres of distribution forming this capsule radiate forwards, backwards, and outwards in all directions from two patches of the grey substance of the ganglion which have become exposed at the summit, one beneath the uncinate lobule, the other just at the outer side of this point. Those passing outwards, forming the proper "external capsule," instead of crossing the Sylvian hollow, as would be expected from the apparent continuity of fibres across it, to the remains of the marginal convolutions of the fissure, dip in between other larger masses of fibres which are those of the corona radiata emerging from the "internal capsule," so that a crossing or intersection here takes place almost at right angles.

A more important anatomical fact relating to this plane of fibres forming the external capsule is that, as Duret has shown, the large arteries which enter the hemisphere at the anterior perforated space for the nutrition of the corpora

striata pass upwards between the capsule and grey matter. To this further reference will be made in the concluding remarks.

CASE 1. *Hemiplegia of left side. Lateral deviation of head and eyes. No loss of consciousness. Sensation greatly impaired. Vomiting. Sleep or stupor. Seven or eight hours after the attack, sudden access of stertor and profound coma. Death in twenty minutes.*—*Hæmorrhage between the grey substance of the corpus striatum externum and the plane of fibres separating it from convolutions of insula cutting off hemisphere from central ganglia, and ultimately bursting into lateral ventricle. Pulmonary, splenic, and hepatic hæmorrhages.*

This case, though not the first of the kind observed, is placed first because I was able to watch it from an early period of the attack, and to recognise the seat of the lesion.

Wm. S—, æt. 50, foreman of some lime-works in the immediate neighbourhood of St. Mary's Hospital, was admitted into the hospital about 2.15 p.m. on Feb. 9th, 1871, and at once seen by me.

He was an intelligent, steady, sober man, had never had any serious illness, but had lately suffered occasionally from headache, and his appetite had been variable.

On the above-named day he had just gone to work after dinner, when he felt giddy and sat down. He appeared to be confused, but never lost consciousness. Within a few minutes he was seen by a friend to whom he said he felt giddy, but should soon be better; his friend, however, noticed that his head seemed to be turned to the right, that the left arm hung powerless, and that he did not appear to know he had a left arm.

He was at once brought to the hospital, where he arrived about 2.15 p.m. and, being at hand, I was called to him.

He answered questions, gave an accurate account of the

attack, and expressed anxiety about some money which was in his purse, but his manner was excited and he called his friend by name and gave him directions, though he was no longer present. He tried also to take hold of the paper on which notes were being made, and resisted when his hand was restrained.

The face was not perceptibly distorted, scarcely so even when he spoke, and he could whistle though not so well as usual. The head was turned so that the face looked over the right shoulder, and the eyes were carried to the extreme right of the palpebral fissures, the deviation of head and eyes being most marked. He was unable to look straight before him, still less to the left.

Left arm and leg quite powerless and relaxed; there was slight reflex action when the sole was tickled.

Sensation was greatly diminished in face, body, arm, and leg of this side.

Face slightly flushed; arcus senilis present; pupils natural.

Pulse 108, rather long and firm, but not strong. Heart-sounds normal; no reduplication; no evidence of hypertrophy. Respiration natural.

A tendency to sleep gradually manifested itself, and having by careful observation come to the conclusion that the case was one of those in which a fatal termination is almost certain and in which a large clot is invariably found, I determined to bleed. My resident medical officer, Mr. Boone (now of St. Kitts), opened in succession three veins, two in one arm and one in the other, but was unable to obtain more than a very trifling flow of blood. That the veins were really opened was shown by passing a probe along them upwards and downwards. I then proposed to bleed from the jugular, but it was impossible to bring about the necessary fulness of the vein. While considering the desirability of dividing the temporal artery the condition of the patient underwent a change, the face became bedewed with perspiration, the pulse was softer, and very shortly he vomited violently, bringing up large quantities

of undigested food, and during the effort urine and fæces were passed involuntarily. He repeatedly provoked fresh acts of vomiting by putting his fingers into the throat in spite of remonstrances. This was about 2.45 p.m.

I did not think it desirable to persist in the attempt to bleed, and a dose of calomel and castor oil was ordered, cold being applied to the head.

The patient continued to sleep during the afternoon, but could be roused, and when awake was quite sensible; he took a little milk and vomited occasionally.

At 10 p.m. stertor suddenly set in, and he was found to be quite unconscious. The stertor continued though he was turned on his side as recommended by Dr. Bowles, of Folkestone (a proceeding which is usually perfectly successful in eliminating the element of danger which stertor adds in these cases), and the respiration was catching. The heart was acting violently, at the rate of 150 beats per minute. During this final attack the lateral deviation of the head and eyes ceased. This was observed for fifteen minutes. The right pupil was large, the left small.

At length, twenty minutes after the stertor set in, the breathing suddenly stopped, but the heart continued to beat and the pulse could be felt for seven and a half minutes afterwards, becoming gradually more feeble. At the moment of death the left pupil dilated.

Post-mortem examination.—Brain,—a little flattening of convolutions of right hemisphere; a superficial, slight, meningeal hæmorrhage of the anterior extremity of frontal lobe on this side. Blood seen to have issued from fourth ventricle, between medulla and cerebellum.

Atheromatous patches in arteries, generally with dilations.

When fissures of Sylvius opened out, convolutions of island of Reil on right side seen to be flattened out; the entire insula being large and soft.

When hemispheres sliced nothing abnormal met with above level of corpus callosum. When lateral ventricles opened, blood-stained fluid and a small clot found in left,

a rupture in septum lucidum; blood in third ventricle, iter, and fourth ventricle, and in all the cornua of both lateral ventricles. In right lateral ventricle a large black clot, and this being removed a fissure seen (to the outer side of the corpus striatum and thalamus) extending along the entire length of the ventricle, crossed by venæ Galeni and processes of the ependyma left untorn. From this fissure, which was wide and gaping, black clot protruded, on removing which a cavity was found, two and a half or three inches long, which extended along the entire length of the two central ganglia on their outer side. The outer wall of this cavity was for the most part formed by the plane of white fibres which, arising from the grey matter of the corpus striatum externum, separates this grey matter from the convolutions of the insula (external capsule). The inner wall was formed mainly by the grey substance of the extra-ventricular corpus striatum, which was not ploughed up or penetrated by the blood or softened, but was left entire, and was simply dissected off from the fibres. The lower edge ran parallel to the descending cornu of the ventricle along its whole length, and was close to it; superiorly the rupture into the ventricle had taken place by tearing of the fibres of the corona radiata issuing from the central ganglia (internal capsule).

The hæmorrhage had taken place from vessels running from the artery in the Sylvian fissure to the ganglionic substance of the corpus striatum, between the grey matter and the plane of fibres arising from it to pass to the hemisphere. The blood had followed this line of cleavage, and after separating the fibrous capsule from the grey substance had torn through the corona radiata and burst into the ventricle.

Heart not large; valves fairly healthy.

Lungs.—Extensive hæmorrhage into both, the blood black and coagulated; the extravasations had neither the appearance nor distribution of ordinary pulmonary apoplexy.

Considerable hæmorrhage had taken place into substance of spleen, and small scattered hæmorrhages were found in the liver.

Kidneys healthy.

I am of opinion that the pulmonary, splenic, and hepatic hæmorrhages took place during the act of dying, and were due to the mode of death, *i. e.*, by suffocation; that, in fact, the extravasation was caused by the prolonged and violent action of the heart after the suspension of respiration.

CASE 2.—*Hemiplegia, left. Sudden in access. Frequent vomiting during next twelve hours. Sopor for three days. Sensation greatly diminished. Rapid formation of bedsores. Death in three and a half weeks.—Extensive hæmorrhage between extra-ventricular corpus striatum and plane of white fibres separating it from insula. Mitral disease. Contracted granular kidneys.*

Mary B—, æt. 45, married and multiparous. Admitted into St. Mary's Hospital, February 2nd, 1871, died February 5th.

Last child six years old; catamenia regular, but diminishing. No previous illness, but liable to pain in the right side of the head and sickness at menstrual period.

On January 8th she had a slight attack of paralysis, but soon recovered.

On the 11th, *i. e.* three weeks before her admission, while at the house of a friend, she suddenly lost the use of her left limbs. There was no loss of consciousness at the time. She was taken home; vomited six or seven times in the course of the night; slept almost continuously for three days, waking at intervals, however, when she appeared to be quite sensible, and took food. The urine and fæces were passed in bed, and this continued to be the case after the patient recovered from her stupor. Bedsores formed rapidly, and a large anthrax came at the back of the neck where a blister had been applied.

On admission there were extensive sloughs on the left hip (the paralysed side) and over the sacrum; a large abscess in the right buttock and the anthrax mentioned above.

The left limbs were paralysed and relaxed; the mouth drawn to the right; not much deviation of the tongue. Sensation greatly impaired over right half of body generally (face, limbs, and trunk). The patient usually lay on her right side; the face and eyes turned to the right, but she was able to look to the left. She answered questions rationally, but wandered a good deal in the night. Pupils equal, of natural size, and acting well. No ophthalmoscopic examination (the ward lamp being out of order, and the patient dying before a second visit could be made).

Heart's action attended with thrill, perceptible to the hand below the left nipple; the thrill led up to the impulse. No murmur over any part of the heart. Apex beat below normal situation.

Legs slightly oedematous. No urine could be obtained for examination. Temperature 101.3° .

Post-mortem examination.—Brain. Nothing remarkable on surface of hemispheres; no embolism; no marked disease of arteries.

On section of hemispheres, just above level of corpus callosum, a blood-stain seen on right side to the outer side of the corpus striatum and thalamus. On further examination a large clot exposed, outer part brown, central portion black; walls of cavity ragged and soft. The cavity extended nearly the whole length of both ganglia, but scarcely reached the anterior end of the corpus striatum, and was between the corpus striatum externum and the plane of white fibres separating it from the convolutions of the island of Reil. The blood only invaded the ganglion at one point, about the middle of its length, where a process of the clot penetrated the grey substance nearly up to the fibres running between the intra-ventricular and extra-ventricular portions of this body. Opening out the fissure of

Sylvius the thin lamina of nervous matter formed by the convolutions of the insula was easily torn through and the clot or cavity exposed. The thalamus was not involved, but was dissected off from the hemisphere equally with the corpus striatum.

The ventricles were empty, and apparently compressed by the clot which had displaced the corpus striatum and thalamus inwards.

Heart weighing $10\frac{1}{4}$ oz. Mitral valve contracted and thickened, and chordæ tendineæ shortened. The degree to which the orifice was narrowed not noted. Left auricle much dilated.

Kidneys small; much of cortical substance came off with capsule; surface coarsely granular and soft; a few cysts. Many purple points and patches, which on section looked like embolic infarctions. Cortex not greatly diminished.

Embolic patches in spleen.

Posterior zone of one eye removed. Apparently normal.

CASE 3. *Apparent drunkenness. Left hemiplegia coming on gradually with hemianæsthesia. Later unconsciousness. Hæmorrhage between corpus striatum and external capsule penetrating into lateral ventricle.*

William F—, æt. 60, cabman, admitted May 27th, 1875. Patient was brought in by police at 4 a.m. in an insensible condition. He had always been a steady man, but two years before had a pecuniary misfortune which preyed much on his mind.

Nine months ago he had a fit which lasted from 2 to 8 p.m. In this attack his wife says he was from the first unconscious (meaning probably delirious), but kept springing off the bed and rushing about the room, not knowing where he went, striking his head against the wall. He foamed at the mouth and bit his tongue, but did not speak until 9 p.m.; was considered epileptic by the practitioner who attended him. Ever since he has appeared "vacant," and complained of pain in the back of the head.

This was one of the "drunk or dying" cases about which sensational articles were written. He was noticed by a policeman about 1 a.m. sitting on the box of his cab apparently drunk. Was charged with being drunk and unfit to drive, and ordered to get down. He answered "all right" in a drunken sort of way, got down and into his own cab, in which he was driven to the police station. His wife, a most respectable-looking woman, said he did not drink.

The surgeon of the police called to him stated that he was brought in by the police between 1 and 2 a.m. He vomited, bringing up large quantities of undigested food smelling of liquor. He remained with him two hours, during which symptoms of paralysis of left side gradually came on. On which he sent him to the hospital.

Condition on admission.—Lies insensible, breathing noisily, respirations 32 per minute; occasionally making a sort of moan and tossing on the bed, using, however, only his right limbs. Lies rather on the left side, the limbs of which lie motionless. There is also a loss of sensation on the left side. If pinched on the right thigh he at once rubs the irritated spot, whereas he makes no such motion when pinched on the left side, though the spot be equally within reach of his right hand. There is considerable flexor rigidity of the left arm, and some rigidity of the leg; reflectorial movements may be produced by irritation of left limbs. Through his parted lips the tongue can be seen pushed very much to the left side. Both pupils react sluggishly to the light. Scarcely any perceptible difference between the two sides of the face. Eyes never move to left of orbit. Is able to swallow with difficulty. Vomited twice this morning.

Pulse rather weak, full between the beats. Temperature: left axilla, 100.8° ; right axilla, 100.9° Urine, sp. gr. 1017, light-coloured, alkaline reaction, contains some albumen. This was drawn off with catheter: passes it under him. Five grains of calomel were given, an enema was administered, and an ice-bag was applied to the head.

May 28th.—Patient lies in much the same condition, but has as yet shown no signs of consciousness. Respiration 24; temperature 100.5° ; pulse 80, irregular. Heart's action weak, no morbid sounds. Pupils much contracted. At 12.30 opened his eyes, and said, "The back of my head aches, so do not touch it." Lies in the same condition. Passes everything under him. Right extremities so restless that it became necessary to confine them, to prevent his throwing off the clothes. Respirations 44 per minute, less noisy; pulse 120, full, but weak and irregular; temperature 102.6° . Is perspiring very freely, much strong rigidity of left arm. Died at 5 p.m., no change having taken place up to that time.

Post-mortem examination, June 1st.—Encephalon. Considerable extravasation into meninges of posterior part of left hemisphere, and over back of both hemispheres of cerebellum. Bulging and flattening of convolutions of posterior part of right hemisphere. Membranes in both Sylvian fissures rather blood-stained, but no distinct extravasation. Bulging of island of Reil on right side, and slight extravasation into the surface of the right side of pons. The Sylvian and basilar arteries present evidence of not very extensive disease. A small aneurism on right vertebral cut across. Dura mater abnormally adherent to left hemisphere near longitudinal fissure. There was a very large clot in right lateral ventricle and its anterior and posterior horns, but not in the descending cornu. The blood reached the ventricle by an aperture which was at line of junction of the corpus striatum with hemisphere, dividing the body of the corpus striatum from the tail, and completely dissecting it away from the hemisphere. With this was a very large cavity in the hemisphere along outer side of ventricle, extending forwards nearly to the frontal extremity of hemisphere, and separated from the Sylvian fissure merely by the thickness of the gyri operiti. The corpus striatum and thalamus were completely separated from the convolutions except by a small bridge close to descending horn. The clot in

addition penetrated into the corpus striatum, separating it from the thalamus along the line of the tænia semi-circularis. There was also some hæmorrhage into the lower part of the optic thalamus, and streaks of extravasation into the body of the corpus striatum. There was merely some bloody serum in the left ventricle. The left hemisphere and the cerebellum appeared quite healthy.

The heart was large, the left ventricle being very thick and of good colour; the valves healthy.

The liver was fatty and slightly congested.

The kidneys were small, contracted, and granular; cortex much wasted; capsule adherent.

The spleen appeared normal.

CASE 4.—*Giddiness and staggering; left hemiplegia; ingrarescent in access; loss of consciousness; vomiting. Hamorrhage between corpus striatum and convolutions of insula.*

Mary R—, æt. 56, admitted August 5th, 1873. Died August 7th. Quite well up to time of attack; had been going about a good deal in underground railway. On leaving Edgware Road Station began to drop parcels she was carrying. Said she was giddy and staggered, but with the assistance of her daughter walked to John Street (quarter of a mile), where she sat down in a shop, and ten minutes later became insensible. Brought to the hospital an hour afterwards apparently quite insensible; left limbs paralysed and rigid; right arm constantly in convulsive motion; she opened her eyes when roused, but soon relapsed into coma; pupils equal and small; pulse full and bounding 80; respiration slow, left cheek puffed out in expiration; froth about mouth; tongue bitten on left side; vomited freely before admission.

Ice to head; sinapisms to calves; calomel gr. v; leeches to right temple; castor-oil enema.

August 6th, a.m.—Spoke distinctly this morning, told

her daughter she was in St. Mary's Hospital, having heard this over night when apparently unconscious.

2.30 p.m.—Apparently insensible; eyes closed; pupils small, sensitive to light; no distortion of face, but left cheek puffed out in expiration. Left arm relaxed and motionless; has seemed to move it a little. Left leg in similar condition; movement more decided; this leg rigid after arm had become relaxed; reflex action when soles tickled, more right than left. Swallows with difficulty. Heart sounds normal; pulse rather resistant; respiration very abdominal; inspiration sudden; expiration long (subject to bronchitis).

When roused answers very intelligently and clearly; no pain except at back of neck when moved; does not open eyes when requested; after speaking, pupils larger; urine and fæces passed in bed; temperature 7 p.m. 103.6° . She gradually became worse; much twitching of body; conjunctiva insensible; temperature 102.4° . Died.

Post-mortem examination.—Body remarkably stout and fat.

Brain.—Some congestion of surface and superficial blood-stains on left hemisphere. Right hemisphere softer to the touch. When about to open the fissure of Sylvius a rent appeared in anterior perforated space, exposing blood clot. This was found to extend far forwards into frontal lobe, and backwards along outer side of corpus striatum, between it and convolutions of insula, dissecting corpus striatum completely away from hemisphere, leaving it entire; ploughing up hemisphere to near thalamus and bursting into ventricle.

CASE 5.—*Hemiplegia with marked hemianæsthesia; ingravescant mode of attack; hæmorrhage between corpus striatum and thalamus and external capsule at posterior part of insula, with penetration of blood through thalamus into ventricle.*

Benjamin E—, æt. 64, a coachman, was admitted into

St. Mary's Hospital, December 11th, 1875, and died December 17th.

He was steady and sober; had enjoyed good health, but chalk stones were present in his fingers, and during the summer of 1875 he complained at times of pain in the back of the head.

He had not been sleeping well for a fortnight, but had been otherwise in his usual state of health, when, on the evening of December 10th, while grooming a horse, felt a numbness down the right arm, and experienced a peculiar general feeling which alarmed him. He found himself no longer able to use the brush, and sent his wife, who was holding the horse, for a medical man. He did not fall or lose consciousness, but on his wife's return could not walk upstairs, though he could still move the right arm barely. He answered the questions put to him by the medical man, but afterwards muttered to himself. At 8 p.m. he vomited and gradually went off into a heavy doze, which continued till he was brought to the hospital.

On admission there was conjugate deviation of the head and eyes to the left; the head, however, could be turned to the right, but not the eyes; pupils equal; slight distortion of face; tongue carried to right when protruded; paralysis of the right limbs; very great impairment of sensation over entire right half of body and head. Not easily made to answer; speech indistinct and rapid, exactly like that of a drunken man; mind confused; did not know his wife. A systolic murmur was heard at apex of heart.

On the 13th, after a purging, he was less drowsy; answered rationally and distinctly, and talked about home affairs with his wife; pulse 64, high tension; slight inequalities in the course of the artery.

14th.—Says he is better and answers intelligently; is, however, unable to protrude the tongue, and failing to do this when asked tried to drag it out with his fingers. Lateral deviation of eyes still present; motions passed in bed; pulse 90; temperature 100.8° .

15th.—Answers “yes” to everything; unable to put out tongue, and uses fingers for the purpose; pulse 96; temperature now rising and much higher on the paralysed side; a.m., right axilla 103°; left 101·6°. 2.30 p.m., right 103·8°; left 102°. At 4 p.m. right 104°.

Ice-bags were applied to the scalp, and the temperature was reduced on the 16th to right 102·8°; left 101·4°; pulse 98. He got gradually worse, however, and on the 17th the pulse was 132; respiration 60; temperature, right 104·6°; left 103·2°. On this day he died.

Post-mortem examination.—Head,—dura mater firmly adherent to the skull; no thickening of either.

Arachnoid over top of hemispheres on left side thick and opaque. Much yellow subjacent fluid. Wasting of convolutions.

Arteries of base very atheromatous.

In right fissure of Sylvius much blood-stained fluid beneath membranes; gyri operi well marked.

In left, less fluid; posterior convolutions flattened out, and bulging. Clot not reached, however, by a slight incision. (*Anatomical note.*—A greater thickness outside proper wall of corpus striatum posteriorly, as fibres pass from temporo-sphenoidal wall of fissure of Sylvius to parietal wall beneath convolutions of island.)

On section, brain substance firm. In right lateral ventricle much blood-stained fluid; in left, clot and fluid. Section exposing left lateral ventricle also exposed the main clot, which lay outside posterior end of thalamus. Thalamus displaced inwards and forwards; intra-ventricular corpus striatum crowded forwards, its tail being turned outwards, so that the clot had pushed apart the two ganglia. The rupture into the ventricle had taken place at the inner side of the posterior part of the thalamus, and the effused blood had also penetrated between the posterior end of the thalamus and the *tegumentum*, tearing the thalamus and raising it up from the *tegumentum*. It had also carried away the anterior edge of the left superior corpus quadrigeminum.

The clot altogether appeared to be about the size of a walnut; it was flattened from above downwards. Anteriorly, the cavity from which it was dislodged did not pass forwards beyond the anterior end of the thalamus; its outer and lower wall was formed by the spread-out posterior convolutions of the island.

The hæmorrhage must have taken place much farther back than usual, and from the branch of the Sylvian artery described by Duret as passing to the thalamus, and the course of the blood inwards must have been determined by the process of the thalamus, which turns round the posterior edge of the crus. The lesion might have been described as involving the posterior part of the internal capsule, but this tract of fibres was little, if at all, damaged; a natural dissection had been performed by the blood raising the thalamus from the tegumentum.

The cases just related furnish sufficient evidence of the association of the ingravescens form of apoplectic attack with hæmorrhage of large amount between the lenticular nucleus and the external capsule. The occurrence of extensive extravasation of blood in this situation has already been noted by Charcot, Bouchard, Bourneville, Purvost, and Duret, and explained by the large size of the arteries here found. It is well known that the grey substance of the nerve-centres is much more freely supplied with blood than the white fibres, and that large vessels pass from the middle cerebral artery into the foramina of the anterior perforated space to the grey mass of the corpus striatum. Duret has shown by sections of injected brains that these arteries do not at once plunge into the grey substance of the ganglion and ramify in it, but run, some of them, one of large size in particular, round nearly its whole external periphery, between the grey matter and the external capsule—a fact which I had observed independently in the course of my dissections. Duret has also shown that they end in tufts and do not give off many branches like other arteries. It is obvious, as

pointed out by this observer, that in the large size of the vessels and in their mode of termination, we have an explanation of the liability to rupture and of the profuse hæmorrhage. A further reason not previously taken into account why extravasation of blood in this situation is liable to be extensive is the absence of resistance. I have found that the plane of fibres forming the inner surface of the external capsule, though the fibres no doubt arise from the cell processes of the corpus striatum, is detached from the grey substance with extraordinary facility, so that the blood only performs a natural dissection in following the line of cleavage between them. In thus separating the capsule from the ganglion it will, moreover, probably cause rupture of other arteries running here.

Not only is there an absence of the obstacle to extravasation which would be offered by interlacing fibres, but the proximity of the fissure of Sylvius and of the descending cornu of the lateral ventricle diminishes the resistance to displacement and consequent pressure which solid brain substance would oppose. The Sylvian fissure is one of the provisions found in the brain for meeting variations in the vascular turgescence of the cerebral substance by corresponding variations in amount of meningeal fluid, and the room it affords for expansion of the island of Reil, can only be fully appreciated when the gyri operati of the latter are seen, as they are in these cases, not merely flattened but completely obliterated, so that the insula appears as a smooth, unconvoluted eminence. Further space is yielded by the compression of the descending cornu of the lateral ventricle, and by the displacement of the corpus striatum inwards.

The ready mobility of the parts surrounding the seat of hæmorrhage, besides facilitating the extravasation, will also prevent any general or local pressure on brain substance till the mobility is exhausted, and to this I am disposed to attribute the late and gradual oncome of coma. The relation between pressure and coma, it is true, is a

question still in dispute, and it cannot here be adequately discussed. It is, however, generally admitted that the brain will bear without symptoms the gradual application of pressure, which, applied suddenly, would give rise to coma, and that sudden injury produces the unconsciousness of shock, or, as it was named by Trousseau, *cerebral surprise*, and it is clear that sudden pressure and forcible laceration are avoided in consequence of the anatomical facts here pointed out.

The extravasation is, however, rarely confined to the space between the extra-ventricular corpus striatum and its external capsule. When this is the case, the hemiplegia will probably be slight and temporary, and a minute account of the symptoms would be of extreme interest and value. It will easily be understood how the effusion of a considerable amount of blood before resistance is encountered favours the further progress of the extravasation; since, according to the laws of fluid pressure, the distending force is the same at every point of the surface of the blood poured out as at the orifice of the bleeding vessel, and is therefore multiplied indefinitely. It will thus readily make its way, but always in the direction of least resistance; and here, as in all cases of cerebral hæmorrhage, the further course of the blood will be determined by the arrangement and relations of the fibres. When the ruptured vessel is at the anterior part of the corpus striatum, the blood travels forwards, and may penetrate into the frontal lobe; when on the lateral aspect the blood is directed upwards. The hæmorrhage never breaks through into the fissure of Sylvius, nor does the blood ever, so far as I know, enter and plough up the temporo-sphenoidal lobe; to do this it would have to penetrate continuous planes of fibres, which in the absence of antecedent softening never happens. At the upper angle of the extra-ventricular corpus striatum the plane of fibres which forms its proper external capsule, passing upwards, meets the mass of fibres of the corona radiata proceeding outwards, and an interlacement nearly at right angles takes place between them. At the line of intersection, too, the few arteries

which are sent from the middle cerebral to the intra-ventricular corpus striatum and to the centrum ovale of the hemisphere pass upwards and inwards, and also the far more numerous and larger veins conveying blood from the extra-ventricular corpus striatum and the mass of the hemisphere to the venæ Galeni. It might have been supposed that the intersection of fibres would oppose a barrier to the further extension of the hæmorrhage, but apparently interlacing fibres offer less resistance to rupture than a smooth continuous plane. No doubt, also, the vessels with the perivascular spaces afford a guide and open a way to the blood, since it almost invariably happens that the corona radiata is torn through partially or completely along the line of its emergence from the region of the central ganglia. The extravasation then either reaches the ventricle or ploughs up the hemisphere; in the latter case it may be directed into a longitudinal course in the substance of the parieto-frontal lobes by the mass of fibres of the great longitudinal commissural system, or may follow the central radiating fibres into the ascending parietal convolutions, which it splits up in the direction of their length.

It is the ingravescient mode of attack which constitutes the distinguishing character of the class of cases I have endeavoured to describe, and which I associate with hæmorrhage between the corpus striatum and external capsule. With the progress of the hæmorrhage new symptoms arise, which will differ according to the course taken by the blood. So far as the cases here related throw light on the symptoms attending different degrees and direction of further structural damage, it would appear that in all there were hemiplegia, hemianæsthesia, vomiting, and sopor. A noteworthy point is the very slight degree of facial hemiplegia observed, attributable perhaps to the distance of the lesion from the first or internal part of the lenticular corpus striatum, which is said to be specially concerned in the movements of the face and eyes. At the same time lateral deviation of the eyes was nearly always present. The patient in Case 2 survived for three weeks,

complete separation of the extra-ventricular corpus striatum and thalamus from the hemisphere. The rapid formation of bedsores indicated great impairment of the nutritive functions in the paralysed parts. In all the other cases the blood reached the ventricles, and a fatal termination occurred within a few days. In Case 1 laceration of brain fibres was attended with the symptoms enumerated and marked conjugate deviation of the head and eyes, while rupture into the ventricle caused profound coma and speedy death. In Cases 3 and 4 extensive ploughing up of the hemisphere with penetration into the ventricle at an indeterminate period gave rise to early rigidity of the paralysed parts, and constant movements of the limbs not paralysed. There was gradual loss of consciousness in both, but less profound and less durable in the latter, in which the blood took a direction forwards. In Case 5 the lesion was further back, and the injury to the thalamus greater than in any other case, and the impairment of sensation was very decided, while the loss of consciousness was not for some time so profound. The size of the clot was, however, much smaller.

It will be noticed that loss of sensation to a certain and indeed considerable degree has accompanied the hemiplegia, and this not only when the thalamus has been damaged, or when the posterior part of the internal capsule so called has been involved in the lesion, but when the hæmorrhage has been too far forwards for either occurrence. To this I beg to call special attention as hemianæsthesia has recently been too exclusively referred to lesion of the *posterior* part of the "internal capsule." There can be no doubt that injury of the fibres at this part will cause hemianæsthesia as observed by Charcot, Bourneville, and others, and shown experimentally by Carville and Duret and others. I desire simply to point out that impairment of sensation can be induced by lesion elsewhere, and especially in the situation I have spoken of outside the central ganglia and beyond the internal capsule.

This does not constitute a valid objection against the sensory function of the thalamus. The impairment of sensation is caused in the same way as the motor paralysis, which in these cases is due not to destruction of the corpus striatum, or to a lesion cutting it off from communication with the medulla and cord, but to separation of this ganglion from the hemisphere by rupture of the fibre of the corona radiata, and the same lesion which divides the communication of the corpus striatum with the hemisphere divides that of the thalamus. The fibres of the thalamus do not go merely or even mainly to the posterior part of the hemisphere, but pass forwards and outwards beneath the intra-ventricular corpus striatum to emerge from the region of the central ganglia in the corona radiata of which they everywhere form an important part. They are thus implicated when the corona is torn, and the cases show that separation of the thalamus from the hemisphere is attended with impairment of sensation as well as damage to the ganglion, or its separation from the sensory tract of the cord.

The object of this communication is to utilise the more extended and exact knowledge of the structural arrangements of the cerebral hemisphere in localising lesions occurring in different parts. It would have been more satisfactory to myself if I could have produced a parallel series of cases in which hæmorrhage had taken place in other situations than the one here considered, but opportunities for this have not fallen to me, and I have only the present contribution to offer to the notice of the Society.

DESCRIPTION OF PLATE XIV.

Figs. 1 and 2.—Transverse section of brain. Fig. 1, at anterior perforated space, and Fig. 2, in front of optic commissure, showing (Fig. 1) origin, and (Fig. 2) further distribution of arteries of corpus striatum. After Duret.

CC, corpus callosum; *Sv*, ventricular corpus striatum (nucleus caudatus); *Sz*, extra-ventricular corpus striatum (nucleus lenticularis); *rcR*, fibres entering below *rc* from crus cerebri, emerging above *R* as corona radiata, constituting so-called internal capsule; *FS*, fissure of Sylvius; *IR*, island of Reil; *c*, thin layer of grey matter, *claustrum*, between which and grey substance of corpus striatum is thin plane of proper white fibres of corpus striatum called *external capsule*.

Fig. 3.—Vertical section passing obliquely from crus cerebri to fissure of Sylvius on outer aspect of hemisphere. *Th*, thalamus.

The representation of fibres is diagrammatic.

Fig. 2.

Fig. 1.

Fig. 3.