LECTURES

ON THE

PRINCIPLES OF SURGICAL DIAGNOSIS:

ESPECIALLY IN RELATION TO SHOCK AND
VISCERAL LESIONS.*Delivered at the Royal College of Surgeons of England.*

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LECTURE V.

Diagnosis of Hæmorrhage within the Skull: its Different Positions.—Reactionary Hæmorrhage.—Consequences of Hæmorrhage.—Diagnostic Indications of Concussion and Compression Compared.—Temperature in these Conditions.—Contused, Punctured, and Incised Wounds: Laceration of Brain-tissue: their Relation to External Injury.—Effects of these Lesions.—Abscess of Brain.—Accidental Complications in Injuries of Brain.—Pyæmia.—Diagnosis of Special Lesions of Encephalon.

MR. PRESIDENT AND GENTLEMEN,—There are certain effects of injury to the head, apart from lesions of the encephalic centre, to the diagnosis of which I will now briefly advert, before discussing the symptoms of direct injury to the brain itself.

Although I have verified, from repeated observation, the impunity with which fissure of the skull may be sustained, my own experience has likewise confirmed the observation of others, that blows, unattended by fracture of the skull, may be succeeded by very grave symptoms, and even a fatal result. Some of these sequences are remote, others are immediately associated with the accident as their exciting cause. Extravasation of blood is, probably, less infrequent than is generally supposed; of course, I mean in very limited quantity, and diffused over the surface of the brain. Certainly this organ has the property of accommodating itself to such pressure as would be hereby exercised upon it, where vascular lesion alone occurs; and the *post mortem* appearances which present themselves in fatal cases that have been carefully watched during life, would seem to support the opinion that such extravasation of blood may be absorbed, whilst its presence probably accounts for the protracted symptoms which precede recovery.

Some hæmorrhage must always accompany fracture involving both tables of the skull, and those which extend into the base. But we know that this consequence of injury may also occur without any fracture.

Viewed by itself, hæmorrhage between the dura mater and skull is manifested by unequivocal symptoms, which also indicate, in many cases, its position. The circumscribed nature of this extravasation, which is due to the close adherence of the dura mater to the skull, is at once suggestive of the nature of the lesion and constitutes its dangerous feature. In some instances, no doubt, a short interval may elapse between the receipt of the injury and the occurrence of symptoms indicating pressure. Where such is the case, the surgeon has a valuable aid in forming his diagnosis. He may reasonably conclude that, as the collapse of shock is succeeded by renewed vascular activity, blood has been poured out, so as to press upon the brain; and if fracture co-exist, even without depression, an effort should be made, with the trephine, to save his patient. But, if there be no fracture, he cannot be certain where the blood is to be found. Profound coma is diagnostic of this form of extravasation, and the presence of such extravasation is probable when this condition exists and where no fracture can be detected; or hemiplegia is indicative of the side affected. Certainly the same quantity of blood may be diffused over the surface of the brain, at its base, or discharged into the ventricles, without producing the same symptoms of simple and deep coma. The same condition may coexist with fracture; and, even under these circumstances, hemiplegia of the same side of the body will point to the opposite hemisphere as the seat of pressure. Unfortunately these diagnostic manifestations are not generally such as can be acted on; for, although there can be no doubt that the seat of extravasation, when distant, is sometimes diagonally opposite to the spot where a blow had been inflicted, this is by no means invariably the case: blood may be poured out over any part of the opposite hemisphere, in the course of the meningeal artery or its primary branches. Where the extravasation in the situation referred to is abundant, profound coma ushers in speedy death. The following is a typical example.

A middle-aged man was admitted under my care, immediately after

he had fallen from a height of not more than ten feet on to his head. He was quite insensible. On examination, there was found a scalp-wound on the *right* side of the head, towards the vertex; it was about an inch long, and the periosteum was removed at one point, but no fracture could be detected. The breathing was stertorous, with groaning. The pupils quite inactive, the left being much contracted, the right somewhat dilated. He died in forty-eight hours, without rallying in any respect. There was no fracture; but, on removing the skull-cap there was found to be a large mass of dark-coloured coagulum, weighing upwards of four ounces, beneath the left parietal bone, outside the dura mater, to which it was rather firmly adherent, and causing a marked lenticular depression on the surface of the middle lobe of the *left* hemisphere, (the contusion was on the right side). There was no hæmorrhage into the arachnoid cavity, but a small amount beneath the arachnoid, over the left hemisphere of the cerebellum; and at the base of the anterior extremity of the right cerebral hemisphere, the grey substance of the brain was bruised and ecchymosed. The white substance of the left cerebral hemisphere, beneath the depression caused by the clot, was studded with small blood-points looking like minute extravasations. (Illustrated by preparation.) I may remark, respecting this case, that it further illustrates conditions of which I was speaking in my last Lecture, in relation to the effects of vibration communicated through the brain-substance. The extravasation was not limited to the membranes and surface of the brain, but extended through its substance, involving the cerebellum and even the under surface of the cerebrum. But I shall presently return to this subject.

I have not on record any case in which extravasation of blood between the dura mater and *vault* of the skull was not primary, *i.e.*, indicated by the immediate or early presence of symptoms of pressure on the brain. No doubt these symptoms are, as I just now remarked, in some cases, less positive at first, and become confirmed by further effusion, as the heart recovers from the first shock of the injury; but the cases are, I think, exceptional and rare where the symptoms of compression are *entirely* absent in the early stage of shock, and supervene only when reaction takes place. I must, however, qualify the preceding remarks on these effusions, by observing that extravasation, having the same relations in the *base* of the skull, may exist without entailing necessarily the same consequences. I have met with several such instances, which admitted, *post mortem*, of no other interpretation than that the blood was deposited between the fibrous membrane and bone some time prior to death, and yet the symptoms were not such as to indicate its presence during life. Probably the pressure in this direction is more readily diffused, or the extravasation takes place more slowly if the lesion be not arterial. Usually in these cases of extravasation, external to the dura mater, this membrane is not ruptured or torn; but, when there is fracture, and that fracture is accompanied by displacement, the dura mater may exhibit the lacerated appearance in proximity to the clot, which is presented by the accompanying preparation of fracture, with extravasation in the base of the skull. (Preparation shown.)

Extravasation of blood into the cavity of the arachnoid is commonly seen in fracture of the base of the skull; but, in my experience, it is less frequent than subarachnoid effusion, on the *upper* surface of the hemispheres, which sometimes extends into the interstices of the convolutions. It is also seen in some instances diagonally opposite to the seat of injury. In these situations probably reactionary hæmorrhage often occurs, as there is less resistance to be overcome by the current of the blood. The following case exemplifies the condition which I regard as diagnostic of this form of limited reactionary hæmorrhage.

A labourer fell from a scaffold two stories high, and was brought to the hospital immediately. He was stupid, and answered questions confusedly, and complained of pain over the right eye-brow, which was contused and swollen, and in the back; he was pale and suffering from shock. Two hours afterwards he became rapidly comatose. I ordered cold lotion to the shaved head, a brisk purge, and leeches to the spine. On the following day he was sensible; his pulse was quiet, and all symptoms were improved. But on the third day the pulse had risen to 120, his breathing was laboured, and coma appeared again imminent. The scalp was freely leeched, and small doses of grey powder and ipecacuanha were given. From this time he rallied, and subsequently quite recovered.

The succeeding case is a fatal one of the same class; I quote it as characteristic, though, unfortunately, I was unable to verify the diagnosis by *post mortem* examination. A livery-stable keeper was thrown from his gig, and fell on the right side of his head. When brought to the Hospital he was in a state of collapse, with contracted pupils, slow and feeble pulse, and cold skin. The only injury detected was a trifling contusion on the right side of the head; he was in a state of semi-consciousness. The usual means were employed to restore warmth. On the following morning the pulse had risen in number and

volume, the skin was warm, and he had vomited. He answered questions rationally, and complained of most pain at the back of the neck. A purgative was ordered, and acted in the course of the day. On the second day it was more difficult to rouse him; the pupils were dilated, especially that on the side opposite to the injury, and there was some hemiplegia on that side also. The nape of the neck was blistered and a dose of calomel was ordered. Profound stupor afterwards ensued, and he died on the morning of the third day. As a *post mortem* examination was not permitted, it is impossible to say that the base was not fractured; and the suffering he complained of at the back of the neck suggested the possibility of this complication; but the symptoms under which he sank indicate extravasation of blood and consequent pressure. The memorandum I affixed to this case is to that effect; and that probably such extravasation was at the base of the skull, associated with fracture. I have no record of any ecchymoses being observed at the nape of the neck in this instance.

That extravasation of blood may exist in this diffused form without producing symptoms of a nature to excite alarm, I think there can be little doubt; but of secondary consequences under these circumstances we have not infrequent instances. The following case is interesting in many respects, in a diagnostic point of view, both negatively and positively; exemplifying the above remarks and other points to which I shall presently refer.

A cab-driver, of middle age, was thrown from his box, and taken up insensible. When admitted into the Hospital, it was found that he had a severe scalp-wound over the left temple, and a bruised face. He gradually rallied from his state of unconsciousness, and on the following day was quite sensible, with moderate reaction, the pulse being 80. He continued free from suffering and any head symptoms; there was denuded bone, and a fetid discharge escaped from the scalp-wound. At the end of a month he was convalescent, the wound in the scalp healing, and the part entirely free from tenderness; he was permitted to walk about, and would soon have been discharged, when, on the twenty-ninth day after the accident, he complained of having caught cold, and remained in bed; he had a flushed face, hot skin and quick pulse, and was inclined to sleep. In the evening of the same day he became insensible. There was no swelling nor inflammation of the scalp around the wound, but it adhered to the edge of the exposed bone, which was dry and white. The trephine was applied on the same night, and a small quantity of pus escaped where the bone was raised; the dura mater was sound. As no amelioration of the symptoms resulted, I punctured the dura mater on the following morning, permitting the escape of a considerable quantity of serum, but no pus. Towards the afternoon he became sensible, but was unable to speak, though he attempted to address his wife on several occasions. At night the circulation again became more excited, and he died comatose about fifty-six hours after the first indication of relapse. The following are the *post mortem* appearances recorded.

There were the remains of a partly healed scalp wound over the left temple, with a circular opening in the skull about an inch in diameter; the dura mater thus exposed was blackening. There was no fracture of the skull, which was very thick, with but little diploe. The dura mater was separated from the skull for a short distance in the neighbourhood of the trephine aperture, and the interval contained some recent clot. The dura mater was healthy over the part corresponding to the aperture; but a short space in front of this, in the frontal region, it was sloughy and presented a small perforation. Beneath the dura mater, and forming a thin layer over the left cerebral hemisphere, was a coagulum partially adherent to the dura mater by its outer surface, but softening within; the corresponding portion of the surface of the brain being also softened and covered with pus. The grey matter of the greater part of the hemisphere was softened and discoloured; and a portion of that of the middle lobe was in a similar condition, and, together with the adjacent portions of the white matter, much congested. The right cerebral hemisphere and the base of the brain appeared healthy.

The purulent deposit is often more circumscribed than in the preceding case, and the local pain more manifest; but the relief obtained by the application of the trephine is, as I formerly observed, rarely permanent in this class of cases.

When hæmorrhage takes place into the ventricles of the brain, it is usually in sufficient quantity to produce coma, but this is not invariable; and the record of such cases in my possession affords no diagnostic characteristic of this special form of extravasation, on which dependence can be placed.

The causes which operate in producing functional disturbance or material injury of the brain-structure are so frequently complicated with those lesions of which I have already spoken, that it is often, I may say generally, impracticable to draw anything approaching to a sharp line of definition between these several conditions. The diagnostic signs of concussion and of compression are, no doubt, distinct in a certain sense;

yet compression rarely exists, as a consequence of violence, without concussion, and both are complicated with shock. Further, symptoms of simple concussion may become developed, at a later period, into those which indicate some more serious lesion; and it is in exceptional cases only that we can identify, with any degree of certainty, the efficient and sole cause of compression. The practical lesson to be learned from these facts is, caution in prognosis, and the importance of carefully investigating all the circumstances presented to our notice, in connexion with the causative accident; and of justly estimating their bearing and value in each individual case.

Some of the symptoms which characterise *Concussion* are positive, but the most valuable are relatively negative. In the stunning effects which succeed a blow on the head, we attach more importance to the absence of those consequences which manifest compression or other material injury to the brain, than to those which indicate what we are accustomed to call concussion. Thus, if we can rouse a patient from his state of unconsciousness, even for a few moments, if the breathing is calm and noiseless, if the pulse is feeble, the pupils are contracted, and reflex action can be excited, we conclude that the condition is one of concussion; because the above symptoms, collectively, are not consistent with a state of compression of the brain. But very often the diagnosis is by no means so simple; for, on the other hand, much that we witness in concussion is due to, and possessed in common by, shock; indeed, in some instances, the more alarming symptoms are those of general prostration, and these may survive the functional disturbance of the brain, which is, so to speak, an accidental concomitant of the shock. On the other hand, the disturbing effects of concussion or violent commotion of the brain, may be such as produce irreparable organic change in its structure, without palpable laceration or contusion of any particular part. The intensity of the effects of so-called concussion are marked by the character of the symptoms and by their duration. Yet, these two elements in the diagnosis of such an injury, do not always bear a direct proportion to each other. I have already suggested that the probable explanation of protracted somnolence and other evidence of brain disturbance is, the presence of diffused extravasation of blood over the surface of the hemispheres, which is subsequently gradually absorbed.

The milder forms of concussion present the general indications of shock, with such cerebral derangement only, as is frequently present in the latter state, and is manifested by giddiness, confusion of intellect, a tottering gait, functional disturbance of the senses, and sympathetic irritability of the stomach. In fatal concussion, if the existence of such a condition be admitted, death occurs from shock rather than from direct injury to the brain; unless, indeed, such injury imply organic lesion, which places it out of the pale of so-called concussion. The symptoms in such cases are those of fatal shock, with suspension of the functions of the brain. Whether, in a supposed instance of this kind, the brain would ever be found devoid of material injury I am not prepared to say; I should think not: certainly I have never made nor witnessed a *post-mortem* after speedy death from a blow on the head, where there was not palpable physical lesion of the brain. Yet, I would not deny the possibility that concussion, without organic lesion of the brain, but accompanied by severe general shock, may prove speedily or even suddenly fatal; especially if the heart be feeble, or the presence of some other accidental circumstance favour this result. This question, indeed, presents a very apt exemplification of the erroneous conclusions that may be reached by disuniting different conditions, which, conjointly, are competent to entail consequences that, separately, they would not produce. The diagnosis does not involve the dilemma of selecting concussion, or shock, or extravasation, as the cause of death; it is rather concussion, *plus* shock, and it may be also *plus* other predisposing conditions or concomitant injuries which have been overlooked, or were beyond the reach of observation.

The symptoms of traumatic *Compression*, when well-marked and uncomplicated, are those of apoplexy. The pulse marches slowly, with a full beat; the pupils are fixed and generally dilated; and voluntary movement and sensation are partially or entirely suspended; the intellectual functions are in abeyance. The soft palate partakes of this general paralysis; and the stertor which marks the apoplectic condition is due to the vibration of the velum, when the recumbent position of the patient favours this result; but change of posture may modify or remove this diagnostic sign. The unresisting cheeks flap loosely, and are inflated at each expiration; the distended bladder retains the urine; and the still active intestine overcomes the feeble resistance of the sphincter, and discharges its contents. This condition is met with in cases where a circumscribed portion of bone or any foreign body is driven into the brain, or where a large clot of blood is suddenly extravasated between the dura mater and skull, or into the substance or ventricles of the brain.

The state just described is well-marked and readily distinguishable,

by the symptoms presented, from cases of simple and uncomplicated concussion. Moreover, the more serious, indeed almost invariably fatal lesion, is not infrequently accompanied by those indications of shock which always, in a greater or less degree, characterises the milder injury. The temperature is maintained and the pulse is well-filled for a time, often when the stertor and flaccid limbs, and profound insensibility betoken the fatal pressure to which the brain is subjected. The force applied has been expended on the shattered skull and in the lacerated brain; it has been less diffused through the system, or the special injury is too exclusive and certainly mortal, to arouse the sympathy of the organic nerves. Be this as it may, the temporary existence of this state in compression of the brain, as contrasted with the, often protracted, organic prostration in concussion, would seem to point to the conclusion that simple shock has more share in producing the symptoms of the latter condition than is generally assigned to it.

But these typical cases of cerebral injury so often merge in each other or are combined, or they are so often complicated with contusion or laceration of the brain-substance, whereby the symptoms are materially modified, that much disappointment necessarily awaits the inexperienced surgeon who anticipates that he will realise in practice the sharp line of definition in diagnosis which he has learned theoretically.

Just now I remarked that, in compression, the temperature is maintained. I should express myself more correctly if I say that the registered temperature in cases of severe head-injury seems to be no measure of the amount of lesion sustained by the brain, in those instances in which it has been tested in my hospital cases. Thus, in two instances of simple concussion in which the temperature was taken, half an hour and an hour respectively, after the accident, it was found to be 93.5 deg. and 96.2 deg.; yet both these patients recovered without any reaction, beyond the normal standard. In another case of hæmorrhage into the brain, with total unconsciousness, the temperature was noted as being 95.2 deg., half an hour after the injury, and never sank below 94.9 deg. In another remarkable instance, however, of fractured base, with laceration of brain, the temperature fell as low as 87.4 deg. in one and a half hour after the accident. I am not aware of any lower recorded temperature. This patient survived about nine hours, but the temperature scarcely attained 90 deg. just before death.

I may take this opportunity of saying in anticipation, that the records which have been kept at St. Thomas's Hospital, of spinal and of other visceral injuries, seem to point to a different conclusion as regards the diagnostic value of temperature in these lesions. It has been found more uniformly low than in head-injuries, the depression corresponding with the amount of injury received. Moreover, there seems to be, in this class of cases, a more steady fall in temperature after the injury, lasting for a greater length of time, than in head-cases, before reaction occurs. The observations on which these remarks are founded, are too limited to permit me to generalise upon them; I throw them out as suggestive rather of the value of further research in this subject.

The brain is obnoxious to the various forms of mechanical injury to which other more accessible textures are liable. It may be bruised on the surface, or have blood extravasated into its substance or interior; it may be punctured, cut, or lacerated. These lesions are not necessarily fatal, though most of them are usually so; sometimes death ensues without reaction; in other instances it is the indirect consequence of renewed vascular action; or it may be deferred until inflammation is developed. In exceptional cases recovery takes place, even after loss of texture. Punctured and incised wounds are of comparatively rare occurrence in civil practice. When a sharp fragment of bone penetrates the dura mater and enters the brain, we have an exemplification of the former kind of injury. It has never been my fortune to witness a case in which a foreign body has been thrust through the nostril or orbit into the brain; but several such cases are recorded. Incised wounds occur in military practice. There is a specimen in the Museum of St. Thomas's Hospital which shows that the original proprietor of the skull had been the subject of a sabre-wound, which probably penetrated the brain. The edges of the bone are rounded off.

The symptoms consequent on penetrating wounds of the cerebrum, when unaccompanied by pressure, are frequently not developed until inflammation ensues; and therefore the diagnosis is necessarily obscure, or the nature of the lesion is overlooked, until alarm is excited by the indications of brain-disturbance, after an interval which may extend to three or four days or even longer. The same remark applies to the far more common injury of contusion or laceration of the hemispheres. That the brain may be bruised, without any symptoms to show that such is the case, there is no doubt. The history of the cab-driver, details of whose accident I just now narrated, together with his death and the *post mortem* examination more than a month afterwards, exemplifies this fact; and I apprehend we may conclude, from the occurrence of such instances, where we have an opportunity of verifying the actual condition

after death, that similar mischief may happen, without the development of any symptoms of importance at any time. But recovery in such instances is probably limited to cases in which the texture of the brain is not seriously damaged. I say seriously, because extravasation into the substance of this organ must, more or less, break up or disintegrate its tissue. Extravasation is sometimes diffused, in the form of a series of small blood-points, over various parts of the brain-structure; or the bruising may be circumscribed. In either case the result must be occasioned by the vibration transmitted through and across the brain. When the contusion is at a distance from the spot on which the blow was received, the mode of its production must be the same; it must be due to shock directly transmitted, and not to vibrations travelling round the skull.

It is difficult to separate, for purposes of diagnosis, these cases of contusion manifested by extravasation, from laceration of the brain. They are, in fact, essentially the same; and, as far as my experience has enabled me to judge, they are characterised by similar symptoms, which vary only in accordance with the degree and locality of the lesion. Whatever may be the correct explanation of the circumstance, it is certain that the middle lobes of the cerebrum are most frequently contused, and the posterior lobes least frequently so; but I am unacquainted with any diagnostic indications by which we may know with certainty where the bruising has occurred. We may suspect that it is near to the seat of injury, or opposite to it; but whether far forward or toward the back of the hemisphere must, so far as we depend on symptoms, be matter of conjecture in most cases.

Of bruising of the brain on the opposite side to the part of the skull which was struck, I select from my notes the following case, which exemplifies the above remark. A lad of 18, having fallen from a height into some water, struck, in his descent, his head and right elbow. There was a compound fracture of the latter, and a scalp-wound, without fracture, on the right side of the occiput. He was stunned at the time, but rallied when he was admitted into the hospital. He had no stertor nor other symptom of compression, but was very restless, dozing at times, and then throwing his arms about, but not involuntarily. He lived for thirty-six hours, remaining conscious to the last. Blood was found extravasated on and in the brain, at a point diagonally opposite to where the blow was received, viz., in the left temporal region.

As regards the relative liability of the different lobes to contusion, of which I just now spoke, I apprehend this may be explained by their several relations. The consequence of transmitted vibration would be greater where it falls upon that part of the brain which is beneath the vertex, and is resting on that portion of the base of the skull which is in nearer proximity to the spine; whereas, on the other hand, the transmitted shock would be broken and diffused by the tentorium on which the posterior lobes rest.

I have said that we possess conclusive evidence, that a certain amount of contusion of the brain may be unattended by other symptoms than those which are usually described as pertaining to concussion; but when the bruising is extensive, or there is actual laceration of texture from violence or extravasation of blood, not only are the symptoms and signs of concussion more intense and prolonged, but we have others superadded, which I have observed to be present usually, though not invariably; and when not present, I believe their absence is generally due to the co-existence of such an amount of pressure from effused blood as to produce coma. The indications I refer to are, more or less constant restlessness, accompanied by spasm, in which some particular member is affected, or amounting to general convulsion. If capable of giving expression to his feelings, the patient will point to some particular part of the head as the seat of pain. In some instances, noisy incoherency and obtuseness of intellect accompany this condition from the first. When these symptoms are well marked, they usually culminate, after three or four days (provided the patient survive so long), in fever, wild delirium, and more violent convulsion, in which he may die; or he sinks into a state of coma, which is the precursor of speedy death. These are the symptoms of fatal inflammation supervening on the mechanical lesion.

In some instances of laceration or severe contusion, accompanied by extravasation of a large clot, hemiplegia indicates the hemisphere opposite to the paralysed side as the seat of injury. But such hemiplegia may be accompanied by convulsion of the opposite side of the body; the interpretation of this complication is, that both hemispheres are injured, but that pressure exists on one side, viz., that opposite to the paralysis, and laceration or contusion on the other. It is scarcely necessary to add that the cross effect is equally uniform, whether the mechanical condition be that of pressure or irritation.

I have remarked that the upper surface of the hemispheres may sustain considerable injury without the immediate presence of symptoms to indicate it. Therefore, the absence of such symptoms must not be con-

sidered as conclusive evidence that the brain has escaped material lesion. The same remark applies, though with less force, to the substance of the hemispheres at their upper part; and it is especially remarkable what extensive lesions may occur in the anterior lobe of either hemisphere, without serious primary disturbance, either physical or mental.

The tendency of these mechanical injuries is similar to that of wounds in other tissues; but as reparation requires increased vascularity, this hyperæmic condition or inflammation is almost uniformly fatal. This result appears to be due to the delicacy of the organ involved, and to the absence of any vent for disorganised tissue and the superfluous products of the reparative effort; and probably, also, to the facilities afforded for fresh extravasation of blood. Thus, the period at which such indications may be looked for, is after the lapse of three or four days, though they may supervene after a much longer interval. General increase of vascular action accompanies the indications of local hyperæmia. The patient complains of pain more or less severe, and a sense of fullness or distension at some particular part of the head; and there is general feverishness, with disturbance of rest; this is followed either by stupor, or by the signs which I have just mentioned as characterising, at an earlier period, contusion or laceration of the brain; local or general convulsion, hemiplegia, delirium; and, after a varying interval, coma succeeds. If abscess form, it is usually, but not invariably, indicated by a well-marked shivering fit; and this is not necessarily accompanied or followed by coma. I have witnessed many instances in which the intellect has been unaffected till within a very short period of death, but where, after death, a large abscess has been found in the substance of the brain. I should, however, observe that the cases to which I refer were not directly consequent on cerebral lesion. Indeed, I believe circumscribed traumatic abscess in the substance of the brain to be of rare occurrence as an *immediate* sequence of injury to the head.

In one case, of which I have the notes, the patient, a young man of 23, had previously been the subject of severe headache, terminating in abscess of the left ear, on which side he was deaf; but in the intervals between these periodical fits of suffering, he was tolerably well. Two weeks before his admittance into the hospital, he had one of his usual attacks, but this did not terminate as on previous occasions. When admitted, he complained of extreme pain over the left temporal region, and a small quantity of pus was liberated by incision of the temporal fascia. The febrile disturbance, however, continued, and he became delirious, frequently pointing to the head as the seat of suffering. In this condition he remained until his death, forty-six hours afterwards. Pus was found within the sloughing temporal fascia, and over the corresponding portion of the bone within, separating it from the dura mater; also in the tympanum; the auditory passage was blocked with fungoid granulations. Nearly the entire middle lobe of the left hemisphere was occupied by an abscess, containing dark and fetid pus, which had found its way into both ventricles, but seemed to have no communication with the surface of the brain.

The succeeding case exemplifies the occurrence of abscess after injury, and the latent nature of the mischief during a considerable period, a circumstance which has been observed and commented on in other cases.

Not long since, I was consulted respecting the probable cause of abscess in the brain, occurring in a schoolboy who had previously enjoyed robust health. I inquired whether he had been the subject of any injury, and was informed by his father that he had been struck violently by a cricket-ball on the back of the head; but that occurrence preceded the illness about five weeks. He had been stunned by the blow, but recovered apparently in less than a week, pursuing his studies as previously, until he was suddenly, and without any suspected assignable cause, attacked by his fatal illness. The first symptom was a sense of cold, attended by shivering. On the second day, he complained of pain in the back, but continued to be tolerably cheerful. On the third day, he complained for the first time of pain in the head, pointing to his forehead as the seat of suffering; he was feverish, and became rather sick. Later in the day, the pain increased, and he became convulsed and delirious. The night was passed in sleepless delirium, frequent and incoherent talk, with occasional intervals of apparent consciousness. The fourth day was passed in the same state, and he seemed to suffer acutely in the head. On the fifth day, he was weaker; and he died, without coma or paralysis, on the sixth day. An abscess was found in the anterior part of the cerebrum, about large enough to contain a walnut, and at a point diagonally opposite to where the blow was received. There was no trace of tubercle found in the brain.

The following case appears to belong to the same category, and ran its course in a remarkably short time. An apparently robust youth of 17, a sailor, applied for admission into the hospital, stating that he had been attacked suddenly on the previous day with pain in his head. He did not account for this by referring to any injury which he had received. The pain was diffused, and he frequently shivered; he had an injected

eye, a quick and small pulse, and a coated tongue. He passed a restless night, and on the following morning the headache was more intense, and located in the right temple; he lay on this side with his head low—the position he preferred. Through the day he appeared very drowsy but quite rational, desiring to be left alone that he might sleep. In the afternoon he became convulsed, and speedily died; little more than forty-eight hours having elapsed, according to his account, from the commencement of the attack. On examination of the head, a small quantity of pus was found over the right orbital ridge; and at this point was an irregular indentation, presenting the appearance of an old fissure; there was pus on the corresponding part of the cerebrum. On the same side, above the frontal sinus internally, two small tubercles or blunt spicula of bone were found projecting inwards; they were cylindrical and rough, and about half an inch apart; and in the interval a small squamous edge of bone overlapped the neighbouring part of the inner table; this corresponded to the indentation or fissure externally, but there was no communication between the interior and the exterior of the skull. Within the anterior lobe of the right cerebral hemisphere, superficially situated and opposite to the ossific projections, was a small circumscribed abscess, containing dark-coloured, unhealthy pus. The dura mater was entire. The comment which I attached at the time to this case is, that the accident, of which the patient had evidently been the subject, had been forgotten; and that the mischief, which proved fatal, had probably been excited by intemperance and irregular habits when he came on shore.

I shall presently refer to another case of traumatic abscess occurring concurrently with other complications; and I may take this opportunity of remarking, that the absence of symptoms of compression in this condition is probably due to the fact that brain-abscess is, usually, devoid of any new element in the composition of its ingredients, which are found to consist of nerve-matter in a state of degeneration and decomposition: pus-globules and other reproductive or reparative elements are almost or entirely absent. The surrounding cerebral structure is generally in a state of inflammatory softening, similar to that observed in traumatic lesion; and the extension of this mischief to some vital part explains the symptoms that accompany the sudden and fatal change which precedes death.

In a patient, of whose case I have notes, though he was not under my own care, three weeks elapsed from the time that he received a blow on the forehead before *any* head-symptoms declared themselves. He had, in fact, left the hospital, when a succession of fits was followed by left hemiplegia; subsequently, agonising pain, convulsion, and delirium preceded death about forty-eight hours. At the *post mortem* examination, the skull was found to be fractured, and an abscess occupied the right anterior lobe of the cerebrum.

Independently of the complications which are, so to speak, inherent in injuries of the head, there are others of an accidental nature which are occasionally a source of additional perplexity to the surgeon. Drunkenness is so often instrumental, indirectly, in causing accidents, that we are frequently called upon to discriminate between this predisposing condition and its more serious consequence. Under such circumstances, I am not acquainted with any conclusive evidence by which we may measure accurately what is due to intoxication, and what to brain-injury, assuming that they coexist. The safest course is to wait until the effect of the drink has passed off spontaneously, and then the indications of brain mischief become apparent from standing alone.

Again, delirium tremens may be excited by injury of the head, as by any other traumatic cause, and thus give rise to perplexity in diagnosis as well as treatment. I have the notes of one case under my care which proved fatal. A middle-aged man had fallen on his head and sustained a scalp-wound. Three days afterwards, symptoms of delirium tremens supervened; and before the opium which was given had tranquillised him, an epileptic fit closed the scene. The *post mortem* examination revealed neither injury to the skull nor abnormal condition of the brain. In another of my patients, of whose case I have kept a record, the injury to the head was more severe. The man had fallen and struck his head against a lamp-post; he was taken up, bleeding from a scalp-wound, and insensible. When admitted into the hospital, he rambled and gave disconnected and irrational answers. In the course of three or four hours he became violent and noisy. He was sleepless and had the other well-marked symptoms of drunken delirium. Opium was given cautiously; and, after profound sleep, he awoke rational, and ultimately made a good recovery.

The diagnosis of drunken delirium when the head is injured, is all the more important, in that we find, in some cases of such injury, the patient manifests symptoms in many respects resembling that condition. I have several cases recorded which exemplify the association to which I allude. In some, the incoherence, want of intelligence, and noisy, rambling talk, or violent resistance requiring restraint, date from the

period of recovering from the first shock of the accident; or this symptom may appear synchronously, with others, indicative of inflammation, and be thus more readily identified.

Two patients were admitted under my care, into the hospital, within a few days of each other, whose cases exemplify all these several states. One, an omnibus-driver, was thrown from his box, and suffered severe fracture of the base of the skull, with laceration of both hemispheres. Though almost in a state of collapse when admitted, he was rambling about his horses, and repulsed the attempts made to examine his injuries. He survived four days, and continued to ramble and vociferate loudly from time to time, being always engaged ideally in his usual occupation; this seemed to be his one mental impression. He did not appear to understand questions put to him, and gradually passed into a state of muttering delirium before he became comatose just prior to his death.

The other patient was an engine-driver, whose head was struck by a locomotive at Woking. The left parietal bone was comminuted and driven into the brain, the middle meningeal artery being wounded. After removing an overhanging angle of bone with Hey's saw, I raised the depressed portion, and removed several loose fragments imbedded in the hemisphere; a compress was then applied to the torn artery. The patient was perfectly conscious and collected during the operation, and afterwards recounted how it occurred. On the second day he became feverish, and complained much of pain in the head, and his articulation was impaired; but it was not until the third day that he began to vociferate, and soon afterwards he became violent and convulsed. Coma preceded death a short time. Inflammatory softening had extended from the upper surface through to the base of the brain, and there was considerable extravasation of blood.

I will mention the particulars of another case, as illustrative of the difficulties which sometimes attend the diagnosis in doubtful cases of head-injury. An excavator was buried beneath fallen earth in a drain. He was soon disinterred and brought to the hospital. He was insensible; his pupils were contracted and sluggish; his breathing was stertorous, and he was convulsed. His respiration became intermittent, and at shorter intervals; and he died comatose after six hours, his heart continuing to beat for some time after respiration ceased. There was not any injury found in the brain or spinal cord; but a part of the contents of the stomach, identified by comparison, had found its way into the trachea, and had blocked up many of the bronchial tubes. The case was one of apnoea, complicated, probably, with concussion.

Occasionally it occurs that an antecedent condition is mistaken for a consequence, just as patients so often ascribe fracture of the patella to a fall on the knee, whereas the fall is caused by the previous fracture of the bone from muscular action. The cases to which I refer are those in which apoplexy occasions a fall. An instance in point recently occurred in one of my hospital patients. A middle-aged woman was admitted, who said she had fallen downstairs, and her symptoms were assigned to injury of the head. At first she was insensible, and all her limbs were paralysed and without feeling, as if she had been the subject of head-injury; and I was induced, when I first saw her, to take the same view as the house-surgeon, without making any special inquiry as to the particulars of her accident. But in a few days, her condition induced me to question her more particularly, and I found that she could not account for her fall. She was standing still at the time, and suddenly found her limbs give way beneath her. Her symptoms at this time were also considerably modified. There was feebleness in the right limbs, but complete paralysis of the left; sensation in the right limbs was considerably impaired, but scarcely perceptibly so in the left. The expression of the face, the speech, and the eyes were unaffected. This patient made a slow progress towards recovery.

The history of another of my patients points more distinctly to a similar explanation of the circumstances of his case. He had fallen downstairs, and was insensible for some time after the accident; on recovering consciousness, he was unable to move either left arm or leg. A careful examination failed to detect any injury to the head or spine; but, on inquiring into his previous history, it appeared that he had been suffering for some time from giddiness and headache, with a sense of fulness about the head, especially aggravated in stooping; and that he had suffered from slight sun-stroke, eight or nine years before in India. The conclusion I arrived at was, that the fall was occasioned by the condition of the brain, whatever that might be. He speedily recovered the use of his paralysed limbs, and left the hospital, still suffering from frontal headache, ten days after his admission.

The occurrence of pyæmia, in association with injuries of the head, has been noticed by writers on the subject. This form of blood-poisoning, with purulent deposits in distant organs, presents no peculiarities in the relation referred to; but it may introduce an additional perplexing element in the diagnosis, where obscurity previously existed. In my experience this is not a common occurrence. I think I may say that not

more than two cases have been observed during the last five or six years at St. Thomas's Hospital, and not one in the many cases of head injury of which I have records, in my own patients. During last year a single case occurred in the practice of one of my colleagues; in this instance there was no fracture; the trephine was applied for secondary intracranial suppuration, and the patient died with brain abscess and purulent deposits in the lower lobe of the left lung. The pyæmic symptoms were masked by the more urgent cerebral disturbance. It is to this case that the chart, exhibiting the variations of temperature in rigor, refers.

The opportunities which the surgeon has of studying the effects of lesions of particular parts of the brain are unfortunately few; not because cases in which many such lesions occur are rare, but because the shock and concussion consequent on the violence inflicted, and the complicating sequences of the injury, deprive the lesion of those simple diagnostic characteristics, which enable the physician more frequently to identify and localise symptoms in disease. Such, at least, is the result of my own experience and observation; and although one-sided paralysis or convulsion point to the opposite hemisphere of the cerebrum as the seat of mischief, I am unacquainted with any diagnostic signs by which to determine whether it is the upper or under surface of the hemispheres which is the seat of laceration or of pressure from extravasation of blood; unless we except affections of the special nerves of sense.

I have pointed out and exemplified the fact that even extensive lesions of the cerebral hemispheres do not give rise immediately to paralysis; but that this condition is a common sequence of such lesions, after an interval of three or four days, more or less, when inflammatory changes have extended to the corpus striatum or optic thalamus. When paralysis occurs as an early symptom, these bodies are, one or both of them, the seat of lesion, or are pressed upon indirectly or by blood extravasated in their immediate neighbourhood. In the hemiplegic condition resulting from lesion of the optic thalamus or corpus striatum, the face is only slightly affected, especially about the ala nasi and angle of the mouth; the patient can voluntarily close his eye. But where the trunk of the facial nerve is diseased, the facial paralysis is universal and more strongly characterised. The former condition alluded to is exemplified in an interesting way, in a case, the record of which I have in my Dresser's report. The patient, a young man, was the subject of an injury to the head, by which the left parietal bone was extensively fractured, the coronal suture separated, the dura mater torn open, and the brain extruded. This patient survived till the fourth day, and was sensible for a considerable part of the time. The first symptom of paralysis was a slight loss of expression on the left side of the mouth, about twelve hours after the accident. This was more marked on the second day, when he began also to complain of some tingling and formication of the left hand, with loss of power; his articulation also became indistinct. The hemiplegic condition became rapidly developed in the leg and arm, and was complete before he lost consciousness, but the facial paralysis did not proceed beyond a marked loss of expression on the left side. The autopsy revealed, among other lesions, "softening of the cerebral substance, with effusion of blood extending to the *right* corpus striatum and optic thalamus which were both involved, the structure of these bodies being distinctly softened, and presenting a blood-stained colour."

Of aphasia as associated with right hemiplegia I have no particular record. In several instances, both of fatal and recovered injuries of the head, I have noticed both difficulty in articulation, and apparently in the selection of words to express ideas; but this condition existed without hemiplegia, or with hemiplegia indifferently of either side. In one particular case, in which the patient at first talked thickly, and was subsequently unable to speak, though evidently intelligent when roused, there was found considerable contusion, with extravasation of blood in the upper and anterior part, as well as on the posterior and inferior surface of the left anterior lobe of the cerebrum. But there was no paralysis in this case; and the patient survived only eight hours. He had been struck on the occiput, and his condition and manner at first led to the impression that he was intoxicated.

Traumatic lesions of the cerebellum, pons, and crura cerebri are, comparatively, infrequent; and, when met with, they usually coexist with other injuries, and thus the special characteristics associated with these lesions are masked; and even in disease they are not constant. In one case under my care, in which more than half of the right cerebellar hemisphere was occupied by an abscess, severe headache, flashes of light, singing in the ears, were the chief symptoms. In another case of severe laceration of the cerebellum, shock and other injuries hastened a fatal termination; but in neither of these cases was there convulsion; and in the former no evidence of want of co-ordinating power. When this last named defect is present, together with dilated pupils and pain in the head, and involuntary periodic cries are uttered, without interference with the intellectual functions, we may predicate pretty certainly the ex-

istence of cerebellar disease. When hemiplegia exists, it is on the side opposite to the lesion.

The co-existence of hemiplegia of one side, with paralysis of the third nerve of the opposite side, is indicative of lesion of the crus cerebri on the side on which the third nerve is paralysed.

ON PUNCTIFORM ECCHYMOSES IN THE INTERIOR OF THE BODY AS A PROOF OF DEATH BY SUFFOCATION.

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THE accompanying table has been constructed with the view of throwing some additional light on the important points in dispute between the two celebrated French and German experts, Tardieu and Lijman (*Annales d'Hygiène et de Médecine*, tom. i, 2 s., No. 8, p. 371, No. 56, pp. 365 and 388, No. 57, p. 104; *Vierteljahrsschrift für gerichtliche und öffentliche Medizin*, Caspar u. Horn, i, 50, vii, 84, viii, 187 and 278, xix, 73), as to the diagnostic value of those minute effusions of blood on certain internal surfaces, known as punctiform or capillary ecchymoses; or from their respective seats, as subpleural, subpericardial, and subpericranial ecchymoses.

Of the 59 cases of sudden death here tabulated, all of them the subjects of medico-legal investigation, 13 were published in April last, in another connexion. (Vide *British and Foreign Medico-Chirurgical Review*, No. 82, p. 454.)

The spots or effusions in question, with the few and partial exceptions noticed in the table, were found to vary in size from a pin-point to a large pin-head, and in number from three or four to thirty or forty, in the respective localities.

In 52 of the cases, the blood-spots occurred on the surface of the lungs; in 31, on the heart; in 22, on the inner face of the scalp; in 18, on the pericranium; in 14, on the thymus gland; in 9, on the interior of the pericardium; in 8, on the root of the aorta; in 5, on the liver; in 2, on the skin; and in 4, singly, on the spleen, diaphragm, pulmonary artery, and pleura costalis.

In most of the instances, the minute rounded spots were in greatest numbers on the thymus gland, the lungs, and the heart; their numbers, though still considerable, being, as a rule, fewer on the pericardium and scalp, while they were only met with in threes and fours on the liver, spleen, diaphragm, pulmonary and aortic arteries, and costal pleura. In one of the skin-cases, the ecchymoses appeared in a cluster over the cartilages of the fourth and fifth right ribs; in the other, they were massed in large numbers on the fronts of the neck and chest.

The ecchymoses on the scalp and pericranium differed from the blood-clots not frequently met with on turning down the scalp in infants perishing from determination of blood to the head in difficult labours; these last being of irregular forms, and of different sizes, up to half an inch and more in breadth.

The marked preponderance of the instances of these ecchymoses in early life will be seen from the table.

The *post mortem* inspections pointed more or less clearly to the *proximate* cause of death in each of the first 26 cases. Thus, in Cases 1 to 26 inclusive, we find 11 presenting distinct and obvious traces of fatal violence; 4, more or less distinctly deaths by pneumonia; 2, by pulmonary oedema; 2, set down at the time as deaths from cold; 2, deaths by drowning; and the remaining 5 respectively, as deaths by hanging, pulmonary apoplexy, cerebral apoplexy, scarlatina, and heart-disease.*

The evidence pointing to the proximate cause of death in the 6 cases which follow was less satisfactory. It has been put down hesitatingly as bronchitis (?), from the observed amount of mucous fluid in the air-passages and air-cells of the lungs.

Cases 33 to 38 were obviously deaths from suffocation, as this term is usually understood; and, in 4 of them, in the form of it known as smothering.

Passing from these first 38 cases in the table to the last 21: in the absence of any of the usual morbid appearances which indicate the mode of death, we have nothing to guide us as to its cause, beyond the presence in these of the capillary ecchymoses. The light mucous froth, usually white, sometimes red, on which Tardieu is disposed to rely as a corroborative sign, was certainly present in 17 of the cases; as were also the dilated air-cells in 1, and the submucous and subcutaneous contused appearances around the lips in 1—signs which this authority likewise regards as characteristic. But these appearances were equally met with

in others of the cases which could not be set down as pure instances of suffocation.

It will be observed that 15 of the last set of cases were new-born infants; and the others, infants of from twelve days to fifteen months, averaging under two months.

The close resemblance of the last set of cases to the 6 set down as *presumably* deaths by bronchitis, deserves also to be noticed; but, for the amount and the extension to the air-cells of the lungs, both sets of cases must have occupied the same place in the table.

It likewise deserves notice here, that although punctiform ecchymoses are noted as present in the cases where suffocation alone could not be justly predicated, it is when we come down to the lowest set of cases in the table, that the blood-spots become the most numerously diffused on the different surfaces.

But, besides the marked preponderance of these appearances, both as to frequency and variety of seat, in the last 20, as contrasted with the cases standing above them in the table, I think it important to state that, as far as my own observation extends, the cases at the top of the list require to be received as rare and exceptional instances of such appearances in the forms of death there noted. For instance, the 2 cases set down were the only instances of ecchymoses encountered in upwards of 200 drowned bodies; the 1 case the single instance in betwixt 40 and 50 hanged persons; while I have never met with ecchymoses after death by strangulation. On the other hand, it is but equally fair to state that I have sought in vain for the blood-spots in nine cases of undoubted deaths by suffocation; all, however, adults.

Without going so far, then, as to deny the occasional though rare occurrence of punctiform ecchymoses after deaths from other causes, or insisting on the necessary presence of these minute effusions of blood in every instance of death by suffocation, I am disposed to conclude that the discovery of such spots in the dead body, in considerable numbers, in well-defined shapes, and limited in size, on the heart, lungs, and thymus gland, especially in infants, either with or without other corroborative tests, and in the absence of other obvious morbid conditions, will afford the strongest presumptive evidence, if not absolute proof, of this special form of sudden death.

CLINICAL MEMORANDA.

[Under this head, we shall publish from time to time, as materials accumulate, short records of remarkable cases in practice which are sufficiently rare, interesting, or instructive, to deserve record, but do not call for lengthened statement or comment. Brevity and point should be the valuable characteristics of cases forwarded for this column.]

HYDATID OF THE LIVER DISAPPEARING UNDER THE ADMINISTRATION OF IODIDE OF POTASSIUM.

(Reported by N. HECKFORD, Esq.)

ABOUT two years ago a young woman, aged 22, came under treatment for what was diagnosed to be a hydatid of the liver. The tumour was undoubtedly connected with this organ, and occupied the whole of the epigastrium and part of the left hypochondrium. It was smooth, globular, dull on percussion, semi-elastic, and indistinctly fluctuating. The parasite was evidently growing rapidly, as the patient had not noticed the enlargement until the previous fortnight. As is usual, there was little constitutional disturbance, some pain and dyspepsia being the only attendant symptoms. She was put under a course of iodide of potassium—thirty grains a day—and at first as a mere matter of form. To the surprise of all, however, the tumour began to diminish, and by the end of five weeks had entirely disappeared. The measurement round the waist decreased to the extent of four inches and a-half during this time.

Here arose another interesting point. The girl, judging from her physiognomy, was, without doubt, the subject of inherited syphilis. Was the case, then, possibly one of syphilitic infiltration of the liver? Unhappily, two months afterwards its true nature was definitely ascertained. The patient, whilst apparently in good health, was seized with acute hepatitis, which terminated fatally. She had frequently recurring rigors, bilious vomiting, local pain, diarrhoea, sweating, and rapid emaciation.

After death, several abscesses were found in the right lobe. With the exception of a small portion of its free edge, the left lobe was wanting, its place being occupied by a shrunken hydatid cyst, which contained some clear yellowish fluid. There was no decomposition of the cyst-wall or its contents. I am of opinion, therefore, that the abscesses were not the result of septic poisoning, and that so far as the hydatid went, she was cured. There were no evidences of pyæmia in the other organs.

* The blood-spots in No. 16 were probably, and in No. 15 possibly, due to *spanæmia* (purpura). There were no vibices, however, on the skin in either.