

# Clinical Lecture

ON

## THREE CASES OF SEVERE INJURY TO THE HEAD.

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GENTLEMEN,—The first case I bring under your notice to-day is the man I now show you, who has been under observation for some time in the wards. His history is as follows. He was admitted on October 16th—that is, fully three weeks ago. The account given of him was that when drunk he fell down a stone staircase, lighting on the back of his head with considerable force. He was brought to the hospital, and his condition was found to be this. On the back of the head there was a contused wound of the scalp running crossways and about one and a half to two inches in length. The bleeding was considerable. The wound extended in depth to the bone, the pericranium being laid bare. On examining with the finger there was no slit or depression in the bone. Blood was issuing in moderate quantity from the nose and left ear. Generally, the patient was insensible and lay on his back with the muscles of the extremities relaxed. The face was pale, the skin generally blanched, with the surfaces cold (temperature lowered). The pupils were regular, rather dilated, acting slowly to light. The breathing was shallow and quiet, with occasional sighing. The pulse was 60, small, empty, and uneven. The treatment adopted was simply application of warmth to the surface of the body, and attention to the wound, which was washed with a solution of carbolic acid and dressed with antiseptic dressings.

October 17th: Pallor on left face and skin; patient beginning to be restless; insensibility not so deep; can be more easily roused; is irritable when disturbed, especially if his eyes, which are kept firmly closed, be opened; speaks incoherent when roused. Retention of urine, catheter had to be used. Pulse 100, fuller, quicker, and more even; temperature 101°. Wound doing well.—18th: Patient still restless and irritable; lies on his side with legs and arms flexed; face flushed and head hot. Pulse 102; temperature 102°. Cold applied to head. Wound doing well.—19th: Patient still restless, irritable, confused, and incoherent. Pulse 108; temperature rose in the evening to 103.5°. Patient had an enema of house medicine and soap. Use of catheter no longer necessary. The high temperature continued for several days, and gradually subsided; the patient gradually regained consciousness and became rational. Pain in the head was complained of and deafness. He is now as you see him, almost well. He complains occasionally of pain in the head, and feels dizzy when he stands up. His memory is somewhat defective, and his manner generally is peculiar. Temperature and pulse are normal. On the left ear he is very deaf. On examination with the speculum, there is a raw line extending across the membrana tympani, pointing to a rent having been present.

The second case I shall relate is a most interesting one. About two years ago I was called to see a youth, aged seventeen, who had sustained a severe injury to the head. The history I got was that the lad was standing with his hands in his pockets near some companions who were throwing the hammer. His back happened to be to the thrower. The hammer swerved from a straight course, and made directly for the boy's head. He was called to, ducked his head, but only so far as to bring it exactly athwart the parabolic course of the weapon, which felled him bleeding to the ground. When I saw him he was lying on the floor of a house into which he had been carried. On the upper and back part of the head to the left side there was a wound surrounded by a considerable swelling of a soft, doughy nature with hard edges. Generally, he was insensible, or could be roused slightly when spoken to very loudly; the face was pale, and the surface of the body cold; the

breathing was shallow, quick, and tolerably regular with occasional sighing; the pupils were unaffected, sensitive, though somewhat slowly, to light. On consultation with Dr. Rodger it was resolved to enlarge the existing wound, and evacuate the blood effused into or below the scalp. This was done in order to examine the state of the bone. On doing so, and getting rid of a large quantity of effused blood, there was found a large, deep, circular depression of the skull, with a diameter of at least three inches, and corresponding to the globular hammer which struck it. The outer table of the skull was shattered, several large fragments were removed, and these I now show you. The inner table was depressed but regular—that is to say, there appeared to be no spiculae projecting into the brain likely to give rise to irritation. As the symptoms were solely those of concussion, and no signs whatever of compression manifested themselves, the edges of the wound were brought together, except at the centre, where a sufficient opening for the escape of any discharge was left. Cold water dressings were applied. Some time after this the patient vomited. Now, gentlemen, it would but weary you to detail the course of this case. Suffice it to say that it ran uninterruptedly towards recovery. Under the influence of warmth to the surface of the body the symptoms of collapse gradually wore off. The mental symptoms disappeared, and consciousness was slowly recovered. There was no great reaction. The temperature at no time rose much above 100°. From first to last there was no unequivocal sign of compression. The wound healed slowly by granulation. The youth is now in perfect health, bodily and mental—bereft, it is true, of a large piece of the outer table of his skull, and having a permanent depression of great depth and size of the inner table.

The third case is that of a dyer, aged fifty-three, who was admitted into Jacob's Ward on the forenoon of Saturday the 22nd of May last. The account was that when drunk he had, that forenoon about 10 o'clock, fallen down a stone staircase and alighted on the top of his head. At the visit at 12 o'clock his condition was as follows. He was lying on his back, insensible but not profoundly so, for he could be roused when spoken to loudly, but only roused. His face was pale, surface of body cold. The pulse was about 80, small and empty. The breathing, though slow, was shallow. The pupils were slightly contracted but regular. On the top of the head, on the right parietal and frontal bones, there was a slight swelling, but no wound or depression. That was at 12 o'clock. In about an hour and a quarter—that is, about three hours and a quarter after the receipt of the injury, matters had very much changed. The insensibility had increased and developed into profound coma. The pupils were now irregular, the left was much dilated and right still contracted. The breathing was slow, deep, stertorous, with puffing of the cheeks. The pulse was fuller and slower. The left arm and leg were rigid as compared with the right. The urine was retained and contained albumen. The swelling on the top of the head was much more distinct. Rapid intracranial effusion of blood was diagnosed. A consultation was held to consider the propriety of an operation. It was concluded not to operate on the grounds that the patient appeared to be moribund, and it was impossible to say in what part of the brain the bleeding was taking place, whether on the surface or at the base. This decision I afterwards had reason to regret. Meanwhile the symptoms progressed. The rigid muscles of the left side became paralysed. The face became flushed, and perspiration poured from it. The pulse was now full and very slow, but immediately before death small and quick. The temperature rose, and immediately before death was 104°. The patient died thirty-eight hours after the receipt of the injury. At the post-mortem examination there was extravasation of blood in the scalp over the right frontal and parietal bones. There was simple fissure of the skull, at the anterior part being separation of the inter-parietal suture, and, as it extended backwards, diverging into the right parietal bone. There was no depression. The upper surface of the brain was lacerated, and a large quantity of blood effused below the dura mater. The kidneys were granular.

Injuries to the head have always had and always will have a peculiar interest to the surgeon. In the literature of surgery affections of the head and brain occupy a very prominent place. Since the classical writings of Pott and Abernethy much has been written on these, as they have received the most careful attention from all surgeons of

note. For this there are many obvious reasons. Extremely liable to injuries of various degrees of severity, from slight cut or bruise of the scalp to severe compound fracture of the skull and laceration of the brain, it falls to the lot of every surgeon, nay, every practitioner, to treat many of these, and on the judicious or other management of even the apparently most trivial cut may depend to the patient consequences of the gravest kind. In these the possible dangers immediate or remote are very many, and in these more than in any other affections that I know of is the unexpected wont to take place. A patient comes to you with a slight cut on the head the result of a fall or blow. You, thinking the matter trivial, assure your patient that the injury is of no moment, and dismiss him after a two minutes' consultation, occupied mainly with general remarks. You hear nothing of him for a day or two, when you are sent for to see him and find him in bed. A rigor and it may be sickness have seized him. The wound looks angry, a suspicious redness surrounds its edges. Great general depression characterises your patient, and despite all your precautions your patient, especially if he be an elderly and debilitated individual, is dead within a week of receiving his injury from erysipelas of the most malignant type. On the other hand, you are called to a case where very severe injury has been received. You find severe contusion to the scalp and soft parts, unmistakable fracture of some part of the skull, and with it very great depression, while the general appearance of the patient is alarming in the extreme. You rapidly form and express a prognosis of the worst kind. Notwithstanding this the patient may take a turn, reaction set in, and complete recovery take place. Now these are no fancy pictures, they occur in practice every day, and the fact that they do teaches these two lessons. In the first place think not too lightly of any injury to the head, however slight it may at first sight appear; warn your patient of the possibility of serious mischief accruing if the wound be neglected, and if serious consequences follow—as they may do in any case—then you are commended for your foresight. On the other hand, however desperate the case may appear, do not too rashly volunteer a bad or fatal prognosis, as you may find yourself very unexpectedly in the wrong. Hippocrates it is, think, who, with his usual sagacity, has a remark to this effect, that no wound of the head is too trivial to be neglected, and no injury too severe to be beyond hope; and Hippocrates undoubtedly is right.

Now, with these general remarks, let us look a little more closely at the cases I have brought under your notice. These are very fair examples of the conditions known as "cerebral irritation," "concussion," or commotion of French writers, and "compression." Now, the first remark I would make is that one of the difficulties we, as clinical teachers, have to contend with is that students straight from systematic lectures, or from reading books, are apt to expect to find cases much more typical, so to speak, than they usually are. For example, take "concussion" and "cerebral irritation," two conditions, each presenting a certain series of symptoms, with which you are familiar in your systematic lectures. Now, you find, and this is only a necessary condition of the systematic exposition of a subject such as the one under consideration, certain symptoms given as characterising the one, and certain symptoms as belonging to the other of these states. From this you would expect to find in actual practice each example of the one condition or of the other sharply defined, so that you would be enabled to say categorically this is "cerebral irritation," or this is "concussion," as the case may be. This is far from what you will really experience. The science of clinical surgery is of the most concrete kind, each case forming a problem to be solved in itself, generalisations being only to a certain extent applicable.

Of the cases I have cited two recovered and one proved fatal. Look particularly at the symptoms of the early stage, and you will be struck with the similarity of these in all three cases. In all three had been a severe blow to the head. In all three there was immediate insensibility; in all three there were paleness of face and pallor of body; in all three there was shallow breathing; in all three there was small, rather slow, empty pulse; in all three the pupils were neither dilated nor contracted, but regular and acting slowly to light. These lasted for a longer or shorter time in all three. Now, what do these symptoms point to? Well, they are just the symptoms of "concussion"—that is to say, they are the symptoms which you find after a severe blow to the head, when either recovery may take place, or death may quickly

follow, and the post-mortem examination may show neither depression of the skull, nor laceration of the brain, nor effusion of blood. In other words, there was in all three, disregarding meanwhile the termination of the cases, a first stage of "concussion." This is what, I believe, happens in the great majority, if not in all, cases of severe injury to the head; indeed it is difficult to conceive of the possibility of force sufficient to cause fracture or laceration of the brain and hæmorrhage being applied to the skull without causing this "concussion," especially when it is remembered that a mere blow from the fist often suffices to cause stunning, which is no other than slight concussion with temporary effects. From this statement it follows that it is, strictly speaking, incorrect to compare or contrast "concussion" with "cerebral irritation" and "compression;" for, as we shall afterwards see, these belong to different stages altogether of the effects of a blow.

Now, what is this "concussion"? What is this obscure, mysterious condition which is accompanied by symptoms so severe, and which may be so transient? Much has been written on this subject, and the older writers were greatly in the dark about it. They were therefore left to assumptions which recent investigations have clearly proved to be untenable. Before referring to the explanations which have been offered, I should like to point out that the one outstanding symptom common to these cases is insensibility of greater or less profundity and of longer or shorter duration. This points to a suspension of the functions of the cerebrum, and the question is, "How does a blow, how can a blow effect this?" The answer to this resolves itself into an account of what pathology and physiology have taught us on the matter. It must be remarked that the pathological changes in the brain are often remarkably slight to appearance and may be overlooked. This it was that misled Pott and writers of his time, and drove them to the first assumption in regard to the condition of the brain—viz., that as a result of the blow vibrations occurred; these reverberated from the side of the skull opposite to that receiving the blow; and thus, by a series of reverberating vibrations, there was caused molecular displacement of the minute elements of the brain. Now, gentlemen, consider for one moment, and do you think it at all probable, having regard to the extremely fine constitution of the brain, that all this shaking can take place without producing laceration and consequent hæmorrhage? But apart from this, is there reason to believe that a blow, however severe, can, in the conditions in which the brain is placed—viz., in a cavity with unyielding walls, and completely filling that cavity—I say is there reason to believe that a blow can produce such a thorough and through shaking and misplacement of molecules as this theory supposes? Experiments of a very interesting kind come to help us here, and, to my mind, settle the question in the negative. Alquié and Gama took a glass vessel, filled it with material of the consistence of brain, suspended in it a number of fine dark threads, and then concussed the vessel. No motion of the threads whatever was observed, showing that, although there may have been motion of the whole mass, the individual particles did not move. Similarly skulls filled with sand, in which an opening covered with a membrane had been made, and into which a long needle with paper on the end had been sunk, gave entirely negative results. There is, in fact, no evidence that such molecular changes as supposed by Pott to take place occur, and his theory consequently falls to the ground.

Another theory is that based on the discovery of Rokitsanski and Nélaton—viz., a number of minute extravasations of blood in the brain. The theory was that the pathology of concussion was just a contusion of the brain with small extravasations. Unfortunately for this theory, it is an undoubted fact that cases occur where these apoplexies are entirely absent; and all, therefore, that can be inferred from their presence is that concussion and contusion occasionally co-exist.

I come now to the third and by far the most feasible theory of concussion—viz., that propounded by Fischer of Breslau.<sup>1</sup> It is shortly stated thus: The blow to the head produces reflex paralysis of the vessels of the brain. Serious interference with the nutrition of the cerebral ganglia is produced, and this it is which gives rise to the symptoms of concussion. In this connexion it is necessary to state that the one constant condition found post-mortem in fatal cases

<sup>1</sup> For a full discussion of this theory see a very interesting clinical lecture, entitled *Ueber die Commotio Cerebri*, by Fischer, forming No. 27 of Volkmann's *Sammlung Klinischer Vorträge*. 1871.

of concussion is an empty state of the arteries and a congested state of the veins. This is the pathology of concussion. Looking at the question from a clinical point of view, and without going minutely into the matter, which would be impossible now, it will suffice to ask, and if possible answer, two questions—viz., 1. Can a blow applied to the head produce this paralysed state of the vessels? 2. Given this condition of the vessels, does it account for the pathological appearances found after death and the symptoms during life? In regard to the first question, there is evidence that a blow can produce such a condition. It is well known that irritation applied to the skin may produce a marked reflex effect on the vessels of the brain and elsewhere. Nothnagel irritated by electricity the skin in the neighbourhood of the crural nerve in rabbits, and thus produced reflex contraction of the vessels of the pia mater. This contraction, however, was always of very short duration, and, as Fischer points out, does not explain the duration of the symptoms of concussion. Other experiments, however, are more to the point. Goltz has shown, in his well-known experiment of giving a blow to the belly of a frog, that paralysis of the heart and vessels can be produced, and that symptoms very similar to those of concussion accompany it, while Koch and Filehne, by concussing the skulls of dogs and rabbits by a series of rapid blows with a hammer, produced the same results. These experiments certainly go very far to answer in the affirmative the first question. In regard to the second question it is sufficient to say that an empty state of the arteries and a congested state of the veins is the only condition which is found constantly to accompany the symptoms which clinical observation discovers to be those of concussion, and that this condition is that which results from paralysis of the vessels and, it may be, a partly paralysed state of the heart. So much for what I have called the first stage of all the three cases, and its explanation. In all three the symptoms were identical; the cases differed only in the duration of this stage.

On following the cases further, they are now found to diverge. What is called reaction sets in. The paralysed condition of the vessels and heart begins to wear off. The tide of stronger circulation sets in. In the first case the symptoms I have described manifested themselves. They were—(1) Patient extremely irritable; (2) patient lying on side, with legs drawn up; (3) eyelids firmly closed; (4) quick pulse and fever, temperature reaching 103°; (5) mental symptoms lasting two or three weeks. Now, what do these symptoms indicate? They are fairly marked symptoms of a condition which has been called "cerebral irritation," and what is that? I believe it to be no other than a variety of the stage of reaction, or more properly perhaps a degree of reaction. It is probably due to a hyperæmic state of the brain, more particularly of the meninges, as evidenced by considerable rise of temperature and febrile symptoms generally. The symptoms of this condition have always appeared to me to be very similar to those found in non-traumatic cases where inflammation of the membranes of the brain is believed to exist. But what of laceration of the surface of the brain? Is that not the pathology of cerebral irritation? There is no positive evidence to show that it is. Experiments on animals prove that the cerebrum can be cut or torn to a considerable extent without giving rise to marked symptoms. Clinical experience points to the same conclusion. In a remarkable case which happened in this hospital, under the charge of the late Dr. Kerr, where a man bending forward in front of a circular saw in motion had his forehead ripped open, and the brain so lacerated or torn that a considerable quantity of brain substance escaped, recovery took place without any marked cerebral symptom whatever. It is extremely doubtful if laceration *per se*, and apart from hæmorrhage and secondary changes, does give rise to any symptoms other than those that would result from destruction of the part of the brain lacerated. It is certainly not rational to ascribe the symptoms of cerebral irritation to superficial laceration. Be the exact pathology of cerebral irritation what it may—and our knowledge of matters cerebral is anything but complete—what I wish to point out is that, looked at clinically, it belongs to the second reactive stage of concussion, and it is therefore unscientific, as inconsistent with observation and fact, to contrast it with this as if it were a distinct condition *ab initio*. One other symptom in this case calls for remark—viz., bleeding from the ear. What did that indicate? In this case probably only rupture of the membrana

tympani. There was no escape of clear fluid, and therefore nothing to point to fracture of the base of the skull.

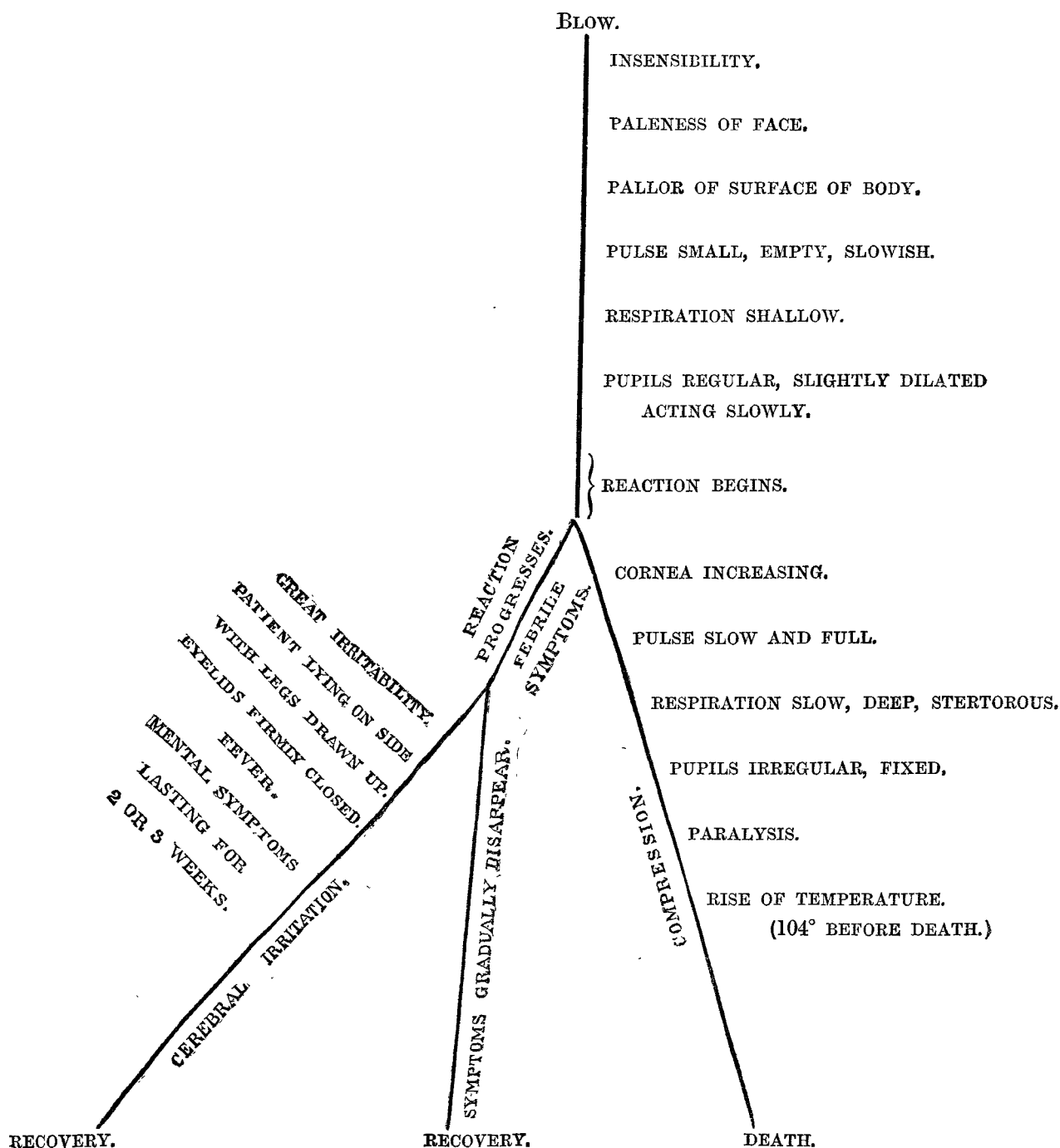
The second case was a more typical one of concussion, where the symptoms gradually subsided, the general paleness disappeared, the pulse recovered, and consciousness gradually returned. Vomiting took place as reaction began. The reactive febrile symptoms were moderate. Perfect recovery took place. The interest attaching to this case was the depression of the skull. In this case there was about as much depression as well could be, and yet from first to last there was not a single symptom of compression. Now, this raises the important question, Can depression of a fragment of bone, acting as it does on only a comparatively small part of the brain, and apart from any secondary changes which may make place as the result of the blow and depression, give rise to symptoms of compression? If this case teaches anything at all it indicates that it is exceedingly doubtful if it can. Now, gentlemen, I do not wish to speak dogmatically or infer too much from one case, but a case such as this, admitting the possibility of such a thing, forms a very staggering exception, and, I think, has great value attaching to it when weighing facts and evidence *à propos* of this question. When it is remembered that previously symptoms were ascribed to compression which belong to concussion simply because depression was present, and when the fact is taken into account that experiments on animals performed by Pagenstecher and others (which I need not detail) show that distinct symptoms of compression come on only after a large quantity of fluid is forced into the skull and produces great pressure on the brain, then it is extremely difficult to see how a depressed piece of bone, exerting at most a comparatively slight degree of pressure and acting only on a very limited part of the brain, can determine symptoms so marked as those of real compression.

If, then, depression of bone does not produce symptoms of compression, what does? Our third case, I think, goes far to answer this question. Here we had a first stage of concussion, and here, too, reaction set in, but with a very different result. There being considerable laceration of the brain and rupture of vessels, with the recovering circulation hæmorrhage took place. There was found post mortem a very large effusion of blood. Now, apart from an effusion or abscess formation that may occur later in the history of any given case of injury to the brain, it is highly probable that this is the constant cause of real compression occurring early. What, then, are the symptoms of these conditions? Our case gives us them. They are:—(1) Coma increasing in profundity; (2) pulse becoming slow and full; (3) respiration becoming slow and stertorous; (4) pupils uneven; (5) paralysis; (6) temperature rising as symptoms increase. These are unmistakable symptoms, and they come on after symptoms of concussion; and, like cerebral irritation, compression is an outcome of concussion. Instead, then, of putting the symptoms of these conditions, as is usually done, in parallel columns, I have arranged the symptoms in diagrammatic form (see next page), showing at a glance the course any given case of injury to the head may take. Of course this table has not reference to any secondary complication that may arise, such as abscess formation, and it should be remembered that all the stages vary much in duration in any given case. If death take place from concussion *simpliciter*, as it may do, then it is likely to occur very early. So much for the symptoms. As to *diagnosis*, our cases did not present any great difficulty. In the third case, at one stage the question "Drunk or dying?" might have occurred; but the very rapid course of the symptoms very soon made the real state of matters clear.

Now, gentlemen, what do these cases teach us as to the *treatment* of severe injury to the skull? In other words, suppose you were called to see a case similar to any of these I have described; what would you do? and why? In the first stage the indications would be to restore the enfeebled circulation. This is effected by applying warmth to the surface of the body by means of hot bottles, &c. If the collapse be profound friction to the surface of the body, or the application of an irritant, such as mustard, will do good. It is no use being too officious at this stage, as time must be allowed. During the reactive stage, if there be symptoms such as those we have in the first case—viz., those of so-called cerebral irritation, then cold to the head, shaving the hair, darkening the room to obviate the irritating effects of light, will be beneficial. If there be insomnia bromide of potassium, alone or combined with chloral, will be found useful. In all cases attend to the bladder and bowels,

keeping the latter tolerably freely open. In cases such as the second the indications for treatment are few beyond attention to the bowels. But this case and the third case suggest very interesting questions as to local treatment, and more particularly as to when and what operative procedure should be employed. What are the conditions that render early trephining necessary? This is the great question that has divided surgeons. Pott laid down the law very strongly that this operation should be done in all cases of fracture of the skull with depression, and, after describing the operation and various measures to be employed, he complacently

fracture with depression is not an indication for operating. This is just exactly what would be expected from what I have already said when discussing the symptoms as to depression being a cause *per se* of compression. Depression of a part of the skull, it was argued, cannot give rise to symptoms of compression. Elevation, therefore, is no use. But what of our third case? In this case there were indications for trephining; and I regretted that the operation was not done, although the reasons for not doing it were quite valid. There were distinct indications of compression, although without depression, and it would have been interesting to have



indicates that the surgeon who has adopted these may say to himself, in the words of Pope,—

“Thus far was right; the rest we leave to heaven.”

Heaven, however, was not in many cases too propitious. This dictum of Pott's was followed by surgeons at the time blindly. Thus it has been too often in the history of surgery. Many a *hereditas damnosa* has been handed down in this way. Abernethy, however, put a check on it; and from the observation of cases where he found that fracture with depression did not of itself in every case, or in the majority of cases, prove fatal, advised waiting for symptoms. He simply used his own judgment, and he was right. What do our cases teach as to this point? If there be anything that our second case proves at all, it is this, that fracture of the skull with marked depression is not an indication for trephining. But it teaches more: it shows that compound

seen if these would have been relieved by opening the skull. Here, again, we have corroborated what we have previously said as to the cause of symptoms of depression coming on early—viz., that they are probably due in all cases to rapid intra-cranial hæmorrhage. What, then, are the indications for trephining early in severe injury to the skull? They may be, I think, arranged under the two following heads:—

1. When there is reason to believe that there is rapid intra-cranial hæmorrhage going on with or without depression. This fact will generally be indicated by such symptoms as I have described in the third case, but a presumption may be afforded in favour of bleeding from the exact situation of the injury. For instance, if there be a fracture at the anterior inferior angle of the parietal bone, it is quite possible that the middle meningeal artery may be injured. This does occasionally happen, and should be kept in view.

2. When a foreign body has penetrated the skull and is



lodged in the brain, and cannot be removed without enlarging the opening. These are the indications for early trephining. But what, you will say, of stellate fractures, do these not present absolute indications for operating? In answer to this I would say that it is extremely questionable if they do. But granting that they do they come under this second head, for in these the presumption is that the inner table of the skull being shattered the fragments are driven in upon the membranes and are thus for all practical purposes foreign bodies which sooner or later will by their irritation cause inflammation of the membranes of the brain.

Gentlemen, I have touched upon various points of great interest in connexion with injuries to the head. It has not been possible to discuss these fully within the limits of one lecture. I have endeavoured to explain the more salient symptoms of the cases I have described to you and to draw what practical conclusions the facts fully warrant. I trust that what I have said will in some measure stimulate you to the study of these all-important affections.

## ABSTRACTS OF

## Lectures

ON

## DIGESTION.

*Delivered before the Royal Institution of Great Britain,*

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### LECTURE IV.

THE acid chyme, when it leaves the stomach, contains a large quantity of undissolved proteids, and of fat totally unacted upon; it also includes the starch that has remained unchanged by the action of the saliva, and undigested cellulose. It becomes mixed with the bile, which is to be regarded as an excretion to a great extent, and as a by-product of important actions going on in the liver. By its alkalinity it helps to neutralise the acid chyme, and thus to establish conditions favourable to pancreatic digestion; it usually contains no unformed ferment.

The principal remaining digestive juice is that afforded by the pancreas, which is one of the compound racemose glands, with a general resemblance to the salivary glands. Its tubular acini contain cells which in the fasting condition have very granular contents, together with an outer clear zone. When the gland has been in action for some time, after food has been taken, the contents of the cells are much less granular, and the clear zone increases in extent. The pancreatic juice is a secretion variable in quantity according as food has been ingested recently or not. It is almost *nil* during fasting, and becomes considerable almost as soon as food has entered the stomach. It attains a maximum about two hours after this time, being secreted in smaller quantities until about the sixteenth hour after food has been taken. It is markedly alkaline, the alkalinity being due to sodic carbonate, which is almost as important an ingredient here as hydrochloric acid in the gastric juice.

Pancreatic juice contains three if not four distinct ferments—pancreatic diastase, trypsin (a proteolytic ferment), a fat-decomposing ferment, often termed “emulsive” ferment, and also a milk-curdling ferment. The action of the diastatic ferment upon starch is exactly the same as that of salivary diastase, the ultimate product being dextrins and sugars (especially maltose.) This power is most important, seeing that in ourselves much starch arrives at the stomach unchanged, and in the herbivora the saliva is not diastatic at all.

Of trypsin, the proteolytic ferment, although it has not yet been prepared in a state of chemical purity, it may be said that it is not so exigent as pepsin in the conditions under which it acts. It will exert its transforming powers in a neutral, a very feebly acid, or in a definitely though not very intensely alkaline liquid. Trypsin seems to cause more profound changes even than pepsin, for after the peptones are formed, some of them appear to split up further into leucine,

$C_6H_{13}NO_2$ , tyrosine,  $C_9H_{11}NO_3$ ; the body indol,  $C_8H_7N$ , possesses a foul smell, which appears in the course of prolonged pancreatic digestion to be connected in its formation with the development of bacteria.

The administration of food which has already been partially digested by pancreatic ferments proves most valuable in certain cases, and relieves much suffering. This is to be noted in favour of pure scientific investigation, for when the experimental researches by which our knowledge of the action of the pancreas has been obtained were commenced, it was not at all obvious that they would lead to the relief of human suffering. The recent wide use of peptonised foods is not a little due to the researches in this direction of Dr. William Roberts, F.R.S., of Manchester.

To Claude Bernard is due the establishment of the fact that pancreatic juice emulsionizes and partially decomposes fats. Both pancreatic juice and minced pancreatic tissue are capable of emulsionising olive oil, which remains in the emulsified condition after standing many hours. The fats, as acted upon by pancreatic juice, are able to enter the lacteals of the small intestine. In an animal killed two or three hours after a mixed meal, the lacteals are found filled with a white liquid consisting very largely of emulsified fat. An argument drawn from comparative anatomy is of interest here. In the rabbit the pancreatic duct is not closely connected with the bile duct, as in many animals, but opens into the small intestine thirteen or fourteen inches below the bile duct; and, in correspondence with the function assigned to the pancreatic secretion, a rabbit killed some hours after a meal has milky chyle only in the lacteals of that part of the intestine below the entrance of the pancreatic duct.

Pancreatic juice further has a saponifying action upon neutral fats, decomposing them into glycerine and a fatty acid. This has been denied by some observers, but the failure to verify this action depends upon the readiness with which the fat-decomposing ferment of the pancreas is itself decomposed by acids. Therefore special precautions are requisite to prevent the destruction of this ferment in making experiments. The addition of a little carbonate of soda, to preserve the alkalinity, is necessary to retain the ferment in activity; otherwise it is killed or prevented from acting by the very acid which it forms. But under conditions which preserve the alkalinity of the digesting mixture, the saponification of fats under the action of pancreatic juice can be most satisfactorily observed.

There is, no doubt, a further ferment in the pancreatic juice, which has, like the gastric juice, the power of curdling milk or precipitating its casein. Thus, if to a neutral or even faintly alkaline emulsion of oil of sweet almonds and gum arabic there be added a sufficient quantity of a blue solution of litmus, and a few drops of a feebly alkaline, freshly prepared glycerine extract of pancreas, and the mixture be rapidly raised to the temperature of the mammalian body, the reaction changes almost instantly from alkaline to acid, as shown by the transformation of the blue colour of the mixture to red. This acidification of neutral fats by the pancreatic juice, which on many grounds is to be connected with an unformed ferment, is doubtless associated with the emulsifying powers of the pancreatic juice, though it is doubtful whether the ferment directly influences the emulsionising process as is implied by the term given to it by Claude Bernard of “ferment emulsif.”

As to the absorption of the products of digestion, that subject does not formally enter into the present course; but it may be said, briefly, that water, soluble salts, peptones, and sugars are absorbed directly into the blood for the most part, while the fats enter almost exclusively into the lymphatics.

The lectures were illustrated not only by diagrams, but also by slides exhibited with the aid of the electric light, and by numerous typical and instructive chemical experiments.

At the second diet of a Sheriff and Jury Court held at Dumfries last week a student of medicine was charged with culpable homicide, in so far as, on April 9th last, having been called to attend the wife of a hostler who was suffering from cramp in the stomach, he administered to her, by hypodermic injection, from twelve to fifteen minims of solution of morphia, whereby death resulted in about fourteen hours afterwards. A plea of guilty was tendered, but it was pleaded that the fatal consequences resulted from an unfortunate mistake. A fine of £20 was imposed, the alternative being a month's imprisonment.