case, as the plica duodeno-jejunalis. The free edge of the plica will correspond to, although it will not be identical with, the free edge of the vertical fold. It is not necessary to assume that the duodenum moves with the shifting serous membrane. Repeated illustrations are given elsewhere, to show that peritoneum can be displaced without involving equivalent displacement of the viscus that it covers.

It is significant to note that both the plica and the vertical fold are attached to the same part of the duodenum, and along precisely the same line. They are both, moreover, conspicuous by their thinness, and by the absence of visible blood-vessels.

Several instances have been reported in which, as the result, probably, of intra-uterine peritonitis, the intestines have continued to occupy in the adult the position assumed in early foetal life. In such cases, the colon is found wholly to the left of the middle line, while the small intestines occupy the right side. The bowel has not been rotated in the usual manner, and the vasa intestini tenuis still arise from the right side of the superior mesenteric artery. The duodenum is quite to the right of the spinal column. Abnormalities of this description have been noted by Sir James Simpson (*Edinburgh Medical and Surgical Journal*, 1839, p. 26), Dr. Hilton Fagge (*Guy's Hospital Reports*, vol. xiv), Mr. Lockwood (*BRITISH MEDICAL JOURNAL*, vol. ii, 1882, p. 574), Professor Chiene (*Journal of Anatomy and Physiology*, 1868, p. 15), Dr. John Reid (*Edinburgh Medical and Surgical Journal*, 1836, p. 70), and Dr. Bruce Young (*Journal of Anatomy and Physiology*, 1884, p. 98). In the description of the parts given by the last named observer in a very able paper, this significant passage occurs: "Extending downwards from the inner side and apex of this curve of the duodenum, in the vertical antero-posterior plane, was a membranous layer, about two inches broad and one inch long, which lay two inches to the right of the middle line of the abdomen, and was attached below to the upper surface of the mesentery." Dr. Bruce Young thinks that this fold was a morbid adhesion; but one cannot fail to be struck with its resemblance to the vertical fold met with in animals, and which one would expect to find in this particular abnormality of the bowel.

It has been already said that the fossa duodeno-jejunalis has been credited as the cause of retroperitoneal hernia. If such a rupture form, the pocket becomes enlarged, and lodges more and more of the small intestine. The bowel finds its way behind the peritoneum; and in a complete case, such as that described by Sir Astley Cooper, the whole of the small intestine, with the exception of the duodenum, is hidden from view, and occupies a large sac that is formed in the middle of the abdomen, and surrounded by the large intestine.

Sir Astley Cooper described, however, another form of retroperitoneal hernia, to which he gave the name of the mesocolic. In this rupture, the small intestine was contained within a sac that formed a tumour to the left of the middle line, and that had been evidently developed at the expense of the peritoneum leading to the descending colon. There are three significant facts about the form of hernia that require notice. In the first place, the orifice of the sac was some way removed from the duodenal bend; in the next place, the commencement of the jejunum was not involved in the rupture; and thirdly, a branch of the inferior mesenteric artery ran along the free anterior margin of the orifice of the sac. Now, Treitz and others have endeavoured to show that this form of hernia also is developed from the duodenal fossa. If such were the case, it is hard to understand why the orifice should become so much displaced, and why the commence-

ment of the jejunum, that is theoretically the first segment of the gut to enter the sac, should be entirely free of it.

Treitz and his followers endeavour to explain this mesocolic hernia by asserting that the duodenal plica is often turned to the right, and the jejunum to the left. To aid the explanation they describe certain remarkable arrangements of the "horns" of the plica.

With regard to these statements, which are faithfully repeated in all accounts of these retroperitoneal hernia, I can only say that I have never found the plica or its fossa turned either to the right or to the left, nor have I ever met with the remarkable arrangements of the fold that have been so carefully described as essential to the production of some varieties of this hernia.

My statement is based upon a careful examination of forty-eight plicae, of each of which a drawing was made as the parts lay exposed *in situ*. In no instance was the upper margin of the plica other than horizontal, and all the chief deviations from the typical arrangement I have already described. I venture to think that the mesocolic hernia of the type described by Cooper, may be readily explained by a reference to Fig. 5 A, which was taken from the body of a female foetus at full term. It will here be seen that a deep pouch has been formed in the parietal peritoneum, leading to the descending colon by a branch of the inferior mesenteric artery. The mouth of the pouch is directed upwards. Such a fossa could readily engage a loop of small intestine, more readily even than the duodenal fossa; and, when a retroperitoneal hernia so originating had been fully formed, then the three features that I have already alluded to would be clearly marked; that is to say, the orifice of the sac would be some way from the duodenum, the commencement of the jejunum would not be involved, and a branch of the inferior mesenteric artery would be found skirting the orifice of the sac on its anterior aspect. Folds and pouches such as this, formed by blood-vessels, would appear to be not uncommon. I have met with two such in the mesentery of two fetuses at full term. They were in both instances formed by the ileo-colic artery, and in both examples the fold of the serous membrane bounded a not inconsiderable fossa, the mouth of which was directed downwards.

THE BROWN LECTURES ON PATHOLOGY.

Delivered at the University of London, December, 1884.

By VICTOR HORSLEY, B.S., M.B., F.R.C.S.,
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LECTURE III.—TRAUMATIC FEVER: PYREXIA FOLLOWING SIMPLE FRACTURES.

ACCORDING to received notions, as far, indeed, as they are comprehensible, numerous kinds of fever, of utterly different natures, have for many years been grouped together under the term surgical or traumatic fever. Putting aside the first term, surgical fever, which has nothing in the world to recommend it, and everything to lead us to get rid of it, let us examine the second, as it has formed the subject of many monographs, to the substance of which I shall have to refer in describing my own observations.

The expression traumatic fever has invariably been employed to denote the febrile state which usually follows an injury to the body, with or without a lesion of the skin or mucous membranes. Now, if this febrile state were always the same, that is to say, if it invariably succeeded the injury at the same interval of time, and ran a course of the same character, although it might be of different degrees of severity, then we might justly say that here was a condition which might be termed traumatic fever, subject, of course, to certain considerations to be enumerated directly. But, at present, the term embraces fevers of widely different incubation-periods, and varied modes of progress and conclusion. This is easily comprehensible when we find that, in a paper written as lately as a few years ago, Lucas Championnière describes puerperal fever, pyæmic fever, that of septicaemia, and urethral fever, all under the same title, namely, traumatic fever.

The most recent scientific work on this and kindred subjects (Brisson's *Science and Art of Surgery*, 8th edition, 1884) divides the septic and urethral fevers from the pyrexia following a simple injury under the headings septic, traumatic, and urethral fevers respectively.

Now, at the outset, I venture to suggest that the term traumatic fever should be restricted solely to those instances in which it can be

Fig. 1.—Traumatic Pyrexia after Simple Fracture of Limb-bones. (Types I and II.)

The upper (dotted) line represents Type I; the lower (continuous) line Type II. Normal temperature, 98.4°. The last eighty hours' temperature is given at intervals of four hours, not hourly, as in the first eighty hours.

shown that a well marked pyrexia follows a simple injury to the body, and when such injury is absolutely uncomplicated by septic or other possible sources of fever.

A definition of the term simple injury is clearly required, if an attempt is to be made to separate one class of clinical observations from another, by using it as a basis of classification. The general notion of a simple injury is a disruptive lesion of a tissue without exposure to the exterior; and this obviously will include all possible cases in which such an accident may occur without lesion either of the skin or of the mucous membranes; indeed, one might, to borrow an illustration from embryology, assert that it was solely a solution of continuity in the mesoblast.

This mode of viewing the subject will compel us to classify all fever following injuries, whether simple or compound, under the following headings, namely:

1. *Traumatic Fever*, defined as above.
2. *Aseptic Fever*, pyrexia which follows the infliction of a severe wound, the treatment of which is carried out strictly antiseptically, and a condition in which there are none or very slightly marked constitutional symptoms.
3. *Septic Fever*, or fever following decomposition in the seat of injury. This must be subdivided under the headings of:
  - a. *Sapraemic Fever*, in which the septic poisoning is due simply to absorption of the chemical substance sepsin, the product of albumin catalysis.
  - b. *Septicæmic Fever*, in which the septic pyrexia follows infection with a poison which does not cause diffuse inflammation.
  - c. *Pyæmic Fever*, in which the poison causing it is also capable of producing foci of suppuration in different parts of the body.
4. *Neurotic Fever*, or pyrexia, apparently caused by some disturbance of the central nervous system, such disturbance being excited by direct stimulation of the nerve-centres or of the peripheral nerve-trunks.

Of all these varieties, we have, at present, only to do with the first and last, but passing reference must be made to these second, or a septic fever, since Volkmann (*Sammlung Klin. Vorträge*, No. 121) has constructed a very definite theory concerning it, and has adduced evidence to support it from the very class of cases that shall presently be laid before you. Briefly, his theory imagines that, whether by subcutaneous rupture or by antiseptic incision, a pyrogenous or fever-causing stuff is developed from the injured tissue, which, absorbed into the circulation, gives rise to the same fever. It seems to me that this theory does not fall within the category of those which rest on a basis of scientific experiment; for, apart from the fact that the state of the tissues after a simple fracture is not identical by any means with that after an amputation (which is his most striking example), we have, in the deliberate operation, an obvious complication, namely, the application to the tissues of a powerful chemical reagent, carbolic acid. Although the conflicting evidence on the toxic influence of carbolic acid appears to point to the probability of its lowering the temperature of the human being in most cases, it adds a new element for consideration, which renders comparison uncertain.

It was this uncertainty concerning the pathology of simple traumatic fever which led me to investigate the subject, keeping in view the definition before stated; and I shall now lay before you the result of my observations. As material, I have accumulated in the surgical

reports of University College Hospital, during the last two years, abstracts of the cases of simple fracture in which there was no lesion of the skin, of which cases notes were taken during the last two years under my supervision, and, for two years previously to that, under Mr. Stanley Boyd. Consequently, the evidence in support of my proposition is purely clinical.

In the tables before you, each case is very briefly abstracted, while the temperature is fully recorded, with the interval of time since admission at which it happened to be taken. From these cases, 168 in number, it has been possible to construct several composite charts of the course of the pyrexia, each of which forms a distinct type, and, consequently, might have been presupposed to possess a different clinical history, and such we shall find to be the case.

But now, grouping the cases together, out of the 168 I find that, during the years 1881-82, 91 per cent. showed distinct fever, and during the years 1880-83, 92 per cent. This agreement is remarkable, since Volkmann and Genzmer (*loc. cit.*), who only examined 11 cases, found the low percentage of 78.5 per cent., while Angerer (*Klin. und Exper. Untersuch. über die Resorp. von Blut. Extravasat.*, 1879) gives an even lower figure, namely, 70 per cent.; but he, again, only examined 10 cases. Stickler, of New York (*New York Medical Record*, February 11th, 1882), found that, of 13 cases, 12 showed a febrile state, which gives a percentage of 92, which exactly agrees with my observations. Possibly, this marked discrepancy might be explained by differences of racial characteristics, such as Chauffard believes in (*La Fièvre Traumatique*, 1873); or, more likely, that the German authors included cases of slight injury. However this may be, in the absence of fuller statistics from abroad, we must leave the cause of this difference undecided.

From the cases of which I have abstracted the details published in my reports, it has been possible to distinguish clearly between two classes of cases, namely, those in which there was a rapid rise of temperature, and little or no swelling; and those in which there was a slow rise of temperature, with a very marked swelling. It will be better, since the etiology of these two types differs, to describe the course of the pyrexia in each instance, and afterwards to discuss the pathology of the condition.

I will take the first group of cases, namely, those in which we find a rapid initial rise of the temperature, with very little swelling. On this chart (No. 1), which illustrates this group, you will see two temperature-curves figured, which are composite, and constructed from 60 cases; the upper one representing a severe degree, and the lower one a milder degree of evidently the same kind of pyrexia. The probable explanation of the differences will be given directly, but attention is drawn to the fact that we have to deal with an exceedingly sudden rise in an hour to a high point, and still continued gradually to a higher level during the next twenty hours. The temperature then very gradually falls to normal in about 160 hours, or, in other words, nearly a week.

In the second variety of this group, it will be noted that, while the fever lasts, it is more constant; but, at the same time, it is much shorter in duration, lasting, on the average, three and a half days, or half the time.

Now, etiologically, these two varieties of the same group differ somewhat. Thus, from the accompanying table, you see that those individuals in which the severer symptoms occur are the

younger, averaging below thirty years of age, while, in the second type, they average 34 years; more than twice the number of cases in Type I being below 35 years of age, while, in Type II, this condition of things is reversed, and more cases are over thirty-five than under it; so that, as far as the age of the patient goes, we find a difference that might have been suspected from *a priori* reasoning, since we are all familiar with the ease with which a rise or fall can be produced in the temperature-curve of a child or young person; and the same reason also accounts for the fact of the curve in Type I rising to twice the height of that in Type II.

FIG. 11.—Traumatic Pyrexia after Fracture of Limb-joints. (Type II.)

In the absence of further data, it is impossible to compare the homogenetic changes in these two types; but, considering that the

diet in these cases is not, as a rule, reduced at all, we may perhaps see, in the differences of the so-called febrile reaction, another instance of the diminished activity of tissue-metabolism in more advanced life.

So much for those cases in which swelling is usually conspicuous by its absence. We will now examine the pyrexia in those instances in which swelling is the most marked feature, and determines the characteristic shape of the temperature-curve.

From this chart (Fig. 11), you see that, just as in Type II, there is in Type III a rapid rise nearly to 100° within four or five hours after the accident; and no doubt this initial rise is due to the same cause in the two cases; but (reserving that point for future discussion) the curve steadily rises for two days (with slight remissions and acceleration towards the end of the second day) to 101°; and in one instance I noted a temperature of 102.4°. From this point, it more rapidly falls to the eightieth hour, when it almost reaches 99°; but this return to the normal, although constant and well marked, is soon followed by a second rise, usually above 100°, but never, or very rarely, reaching 101°. This second rise is very peculiar and constant, almost invariably occurring at the hundred and twelfth hour, nothing like it existing in the other curves. From the second acme, the temperature reaches the normal in a very variable time, but usually in two or three days more.

The total duration of this third type of pyrexia averages seven and a half days, but very frequently is prolonged to the tenth or twelfth day.

As, to the etiology of this type, as one might, perhaps, have expected, the ages show that, on the whole, it is the younger and the oldest individuals who suffer most, those in middle life escaping, the majority being below 35. One is inclined to attribute this simply to the fact that those who are most helpless will probably suffer the severest injuries; and it will, of course, be conceded that, other things being equal, the note of "considerable bruising and swelling" which marks these cases in my tables of abstracts means simply that the amount of damage inflicted is more severe than in the types previously described. And at the same time, of course, there is another factor not to be lost sight of—namely, the greater liability to damage, causing the above mentioned anatomical changes of the tissues of individuals whose age approaches either extreme of life.

With this preliminary outline of the clinical features of the different types, which I believe one is justified in constructing from the cases before you, it will be necessary to complete the description of the etiology of traumatic fever, as I understand it, before attempting to explain its pathology.

At the outset, I stated that 91 to 92 per cent. of all the cases showed a so-called febrile reaction. It will be interesting to glance for a moment at those instances in which there was absolutely no pyrexia, to see if there is any obvious explanation for this very exceptional condition. Unfortunately, although we might have expected to gain some clue as to the causation of the febrile state by studying the absence of it in such cases, I have not been able to draw from them more than the following facts.

In the first place, the average age, 39½ years (nearly forty years), is very high; in fact, there was only one case below 10 years of age out of twelve, in which there was no pyrexia. In half the cases, there was sufficient swelling to call for special attention, and the seat of fracture varied directly with the proportion of *probable* injuries; thus the femur was fractured three times (always in the lower third), the tibia three times, both bones of the legs five times, the fibula once. Of the twelve patients, all with one exception (gout) appeared to enjoy good health, and two were definitely alcoholic; so that, as far as the latter point goes, there is no evidence that the well known effect of alcohol, ether, or chloroform played any part in the prevention of pyrexia in these cases.

Considering the less degree of fever in Type II as compared with Type I, both being degrees of the same character of cases, it seems to me that the only constant factor in the etiology of these apyrexia cases is the average age; and thus, from the above explanation of diminished pyrexia, we easily understand the occurrence of apyrexia. Such an explanation can only be taken for what it is worth in the total absence of experimental fact; and the extremely interesting fact remains, that a certain proportion of cases showed no fever at all.

I now propose to discuss briefly the condition of the part which is the seat of injury, and at the same time will refer to the clinical history where it is required to supplement the description of the pathological appearances.

In the group of cases which includes my first two types, and in which you may remember there is little or no secondary swelling and

edema, we have the bone broken, and the sharp ends of the fragments irritating the torn branches of nerve and ruptured muscle. At the same time, there is more or less extravasation of blood; and we must admit theoretically the possibility in each case of more or less direct absorption of fat from the damaged marrow in the veins and lymphatics. Thus there are three possible exciting causes of a rise of temperature, namely (1) moderate stimulation of an afferent nerve; (2) absorption of extravasated blood; and (3) so-called fat-embolism. These possible causes examined in detail show us how little substantial work has been expended on this fundamental subject, and consequently how impossible it is to come to any definite conclusion.

Severe stimulation of an afferent nerve causes, as is well known, a decrease of temperature, the decrease varying with the intensity of the stimulus, and, on the other hand, gentle stimulus of an afferent nerve has been shown (Albert and Stricker, *loc. cit.*) to cause a rise of temperature in the part supplied by it.

But the question really amounts to this: are there centres in the brain and spinal cord, reflex irritation of which, by stimulation of the kind suggested, is followed by a general rise of temperature, that rise being maintained for several days? Although, as I shall have occasion to show in my fourth lecture, on Urethral Fever, there is good ground for believing in such heat-centres, I am not aware that the crucial test has been put to the above theoretical explanation, by producing a fracture in a limb which is the seat of motor and sensory paralysis. We will put aside, therefore, the idea of a neurosis, and take up the possible cause, namely, the extravasation of blood. A good deal has been written on this point, and it has been shown by Angerer (*loc. cit.*) that the artificial production of a hæmatoma is followed within four hours by a marked febrile reaction. Before further discussing Angerer's very valuable work on this point, we must confess that Albert and Stricker (Ueber Wundfieber, *Mediz. Jahrbücher*, 1871, p. 39) have proved the possibility of causing a rise of temperature of

Fig. III.—Fever after Extravasation of Blood.

The continuous line represents the temperature-curve after production of acute traumatic aneurysm in a dog (normal temperature, 101°). The dotted line represents the temperature after hypodermic injection of ox-blood in a man (normal temperature, 98°).

1° Cent. or more in a dog, by simply performing the operation of exposure of a vessel for the purpose of injecting into it. This we must regard as a combination of the first two of the possible causes of traumatic fever, namely, irritation of nerve-fibres, coupled with some absorption of extravasated blood, etc.

Further, not to have occasion to refer to it again, this is partly the notion of Volkmann and Genzmer (*loc. cit.*), that the extravasated blood and the torn tissues together supply a fever-exciting substance, which

is absorbed into the general circulation. In passing, let me draw your attention to the pyrexia produced by (1) simple transfusion, (2) the injection of leucin into a vein (Fig. IV).

With these provisos, we will now examine Angerer's methods and results. Of the latter I have drawn up a chart (Fig. III), to show the rise of temperature in his experiments. In the uppermost curve is seen the fever following the subcutaneous division in the dog of the great blood-vessels just above the elbow, with a large hæmatoma as the result; and he is inclined to attribute the rapid rise of the temperature, 1° Cent., to the absorption of the products of the extravasation. However this may be, the condition is either not comparable to the ecchymosis of a simple fracture, or that lesion is not the sole cause of traumatic fever; for, as I have demonstrated, the average length of the pyrexia is a matter of several days, usually a week. It may be admitted that the initial rise in traumatic fever is due to a combination of the two circumstances just mentioned, and that the maintenance of the pyrexia is effected by some cause or causes unknown; but it seems to me more likely that the rapid initial rise in his experiment, with a consequent gradual fall, is due to the same cause as that imagined by Albert and Stricker, namely, anything but the absorption of a pyrogenous substance.

Granting that we cannot admit it to be a neurotic fever following the disturbance produced by the laceration of nerve-filaments, there remains the possibility of the symptoms being produced by fat-embolism. Although fat-embolism is said by some German writers to occur with great frequency, and to show itself by the appearance of oil in the urine, I must confess that, with the exception of two obvious cases in which I detected fat-globules in the expectoration, and in which there were definite lung-symptoms, my investigation of the urine in simple fracture, even with the aid of osmic acid, has not yielded confirmatory evidence of these statements; consequently, one is driven back to the neurotic theory.

Fig. IV.—Traumatic Pyrexia.

Now, there is a pathological condition which may play a part in the causation of traumatic fever, through the intermediation of the nervous system, a condition which, so far as I know, has not been credited with influence in this direction, namely, tension. Every surgeon is familiar with the fact that a few drops of pus pent up in dense connective tissue, richly supplied with nerves, will cause a rise of three or four degrees above the normal. Now, in such a case, it is impossible to conceive the absorption of pyrogenous stuff to be an adequate explanation of this symptom; and, from the fact that, from the moment the tension is relieved, the fever falls, the conclusion is almost inevitable that the traumatic stimulation was the cause of the febrile rise.

Indeed, it is hard to conceive the possibility of any other explanation when the fact of so-called idiosyncrasy is brought to mind. Taking, for example, these cases of traumatic fever, I showed you that the only possible tendency towards an apyretic state which could be discovered was the influence of age on the parenchymatous tissues,

while the conditions of injury were precisely the same as those observed in the most severe cases of pyrexia, namely, large bones broken, and I may now add that, in half the instances of pyrexia, swelling was noted either as simply present, or as very considerable. And, again, the tendency of some individuals to develop a pyretic state is a fairly ascertained scientific fact.

It is useless to speculate in the absence of experiment, especially as I shall have occasion to refer to this point in my fourth lecture. I shall, therefore, proceed at once to the third type of traumatic fever; namely, that in which there is marked swelling of the part, and the type in which the temperature rises slowly, and shows a double-pointed composite curve.

I may again point out that, after the initial rise in four hours, the pathology of which I have just discussed, the highest point is reached at about the forty-eighth to the fiftieth hour. It would seem to be a justifiable assumption that, if the temperature rises in this steady way for two days, the cause producing it is developing during that time, and reaches its height a few hours before the temperature-curve attains its acme. Now, it is interesting to observe, clinically, that this is the actual behaviour of the cedema, or, as I have roughly termed it, the swelling, which sometimes follows a simple fracture, and which was a constant feature of the cases we are now considering.

I, therefore, consider I am justified in attributing the peculiar character of this type to that particular complication. In confirmation of this belief, let me show you this tracing of the temperature-curve in an experiment performed by Angerer upon his colleague, Dr. Fehleisen (the dotted line in Chart III). Under the skin of the forearm of Dr. Fehleisen was injected sufficient defibrinated perfectly fresh ox-blood to cause a tumour of the size of an apple; this gradually disappeared in a few hours, and was succeeded by a secondary swelling of the arm, together with severe constitutional symptoms of fever.

It will be seen from the chart that the temperature-curve in this case is parallel to that of my third type; and, it seems to me that the pathological process in both is practically the same, namely, the development of acute cedema, thus causing severe tension in the part. I am, of course, quite aware that this view may have to undergo considerable correction as soon as I shall have had the leisure to examine it experimentally; and, moreover, I desire that it should not be thought that in these cases, where the amount of cedema-fluid was very considerable, I deny the possibility of the symptoms being due to the absorption of pyrogenous stuff. It was only in the instance before cited that I considered it to be practically inconceivable.

Having reached this point, we must review the pathology of the condition of simple fracture coupled with cedema. From the fact of the cedema commencing around the seat of fracture, and invading the distal parts first, it is probable that two causes are at work. In the first place, the walls of the blood-vessels in the neighbourhood of the fracture must be damaged by the injury, and, so damaged, will allow the transudation of cedema-fluid; further, from the same cause, we may have, in many of the veins, thrombosis; and from the action of these two factors, coupled with the pressure of extravasated blood, the occurrence of cedema is very comprehensible. But, if we admit the first of these causes, at the same time, we practically allow that the process is inflammatory; and such a conclusion appears to me inevitable, since we now regard inflammation in a vascular tissue "as a destructive process," causing injury to the walls of the blood-vessels, with the transudation of fluid and corpuscles as a consequence. Most pathologists would admit that the temperature-curve obtained by Angerer's second experiment is an instance of so-called inflammatory fever, and yet not admit that the state of the tissues around a simple fracture is inflammatory. Is it not rather discussing terms than processes when we see that certain stages are absolutely identical, those stages being: 1, application of some irritant force; 2, dilatation of vessels, followed by active congestion; 3, cedema?

The exudation of corpuscles varying directly with intensity and extent of action of the irritant, it would seem that such exudation is merely complementary, and that we can apply the term inflammation to the early stages alone with perfect accuracy.

I have dwelt somewhat at length on this point, being perfectly conscious that it touches upon the question of the origin of callus; but, beyond admitting that responsibility, there is no time to enter further upon it.

I have endeavoured to substantiate my statement that two distinct kinds of fever follow a simple fracture, and are due to different pathological causes. I have purposely abstained from entering into a detailed examination of the theory which attributes all rises of temperature to irritation of the central nervous system; partly because I

must go at some length into that question in my fourth lecture, and partly because a great deal more work on the subject is wanting.

I would suggest, in conclusion; that I have shown cause why the term traumatic fever should be restricted to cases of simple injury, without any exposure to the exterior or other complication.

ON CERTAIN NEW METHODS IN THE TREATMENT OF DISEASES OF THE SKIN.

LUPUS: PYROGALLIC ACID: CROTON-OIL TREATMENT OF RINGWORM: LICHEN RUBER.

By GEORGE THIN, M.D.

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LUPUS.—If the number and variety of therapeutic measures advocated in the treatment of a particular disease be any indication of the futility of treatment, that of lupus must be considered as being still in a very unsatisfactory position. During recent years, a great amount of literature has been devoted to the treatment of lupus; yet, at the present time, it is probably correct to say that there is no approach to unanimity as regards the methods which can be considered as generally applicable in the management of this affection. It was only the other day that the treatment of lupus by scarification, introduced by Mr. Squire, and for a time enthusiastically adopted by the physicians of the Hôpital St. Louis, in Paris, seemed to have secured the adhesion of those who had the advantage of large experience in the treatment of the disease. M. Besnier, who himself extensively practised it at that hospital, laid down precise rules for the operation, and taught the method to a great number of French and foreign medical men; but he has already found it necessary to supersede it by another method of treatment, which is recommended with a confidence equal to that which was, a few years ago, given to scarification. He makes it a reproach to the latter method that patients must be kept continually under treatment, fresh foci of lupus must be attacked in the cicatrix, and that years pass without, in many cases, obtaining anything like a true or final cure. Not only so, but he more than once hints that it is possible, by scarification, to favour an auto-infection in the patients; to augment the number of lupus-patients who become phthisical, or to favour and render active an infection which has already taken place; that is to say, M. Besnier looks on the treatment of lupus by scarification as being practically a failure. For my part, in my own very much more limited experience, I must confess that I have been disappointed. It is no doubt true, as I have found, that a limited lupus of the face may be cured by systematic and frequently repeated scarifications; but in several cases, in which I was enabled to persevere for an unusually long period, although, with one exception, great amelioration took place, a complete cure was not attained. The foci of new growth, which continually reappear, require, as a rule, more energetic treatment than simple scarification.

The new method which M. Besnier recommends is what he calls "interstitial and discrete cauterisation" by the thermo-cautery. In a paper published in the *Annales de Dermatologie et de Syphiligraphie*, vol. iv, p. 377, he remarks that, at the present time, the galvanocautery and the thermo-cautery are the exclusive agents which he employs in the treatment of all the forms of lupus. In their application, he has recourse to delicate instruments, which are varied according to the different indications which require to be fulfilled and suited to each individual case. The advantages which he claims are smooth and pliant cicatrices, and destruction of the neoplasm, whilst the minimum of destruction of sound tissue is attained. M. Besnier figures a variety of simple and multiple needles for the galvanocautery. He lays it down as a rule that, whether the thermo-cautery or the electro-cautery be employed, it is essential that the platinum should only be heated to a dull red heat, and never to a white heat, for two reasons: in order to prevent the operation from being bloody, and also to enable the operator to see distinctly the parts which are to be cauterised. When the affected part is of small extent, as is often seen in lupus vulgaris of the cheek, or in isolated discs of lupus erythematosus, he makes, with a fine point of platinum reddened by heat, a series of punctuated applications, the points being separated from each other for about the distance of a millimetre, so that the patch is literally tattooed. If the electro-cautery be in the form of a fork, or be multiple and pointed, the operation can, of course, be executed more rapidly. M. Besnier considers that this treatment, if his instruments be used, is applicable to every kind, form, and variety of