concerned in specialised, co-ordinated movements, in emotions, and also in intellectual processes. The fact that some patients in these attacks fall suddenly, and remain still for a few moments before the co-ordinated movements commence, has been regarded by Dr. Hughlings Jackson (adopting a theory of the late Dr. Dickson) as evidence that the "co-ordinated" phenomena are always due to the release of lower centres from the control of the higher by the temporary discharge of the latter. He has urged that an actual discharge of grey matter can only give rise to inco-ordinate "brutal" effects. That co-ordinated convulsive action may succeed attacks of true epilepsy is beyond all question; but it is also, I think, beyond question that the co-ordinated phenomena are not always preceded by epileptic symptoms. In some cases which I have watched, the attacks commenced with the co-ordinated movements which characterised them throughout. The tonic rigidity, which initiates the attacks in other cases, cannot be regarded as a true epileptic symptom; it is rather part of the hystero-epileptic attack, and may recur during its course. Thus I think that there is in many cases no evidence of true epileptic symptoms, that the co-ordinated convulsion, which constitutes the chief symptom of the hystero-epileptic attack, may exist alone.

Even for the cases in which this symptom succeeds a true epileptic seizure, it may be doubted whether the hypothesis of "loss of control" affords an entirely adequate explanation. We can discern no difference between epileptiform attacks which are, and those which are not, followed by such symptoms. This suggests that this occurrence depends upon some other condition, a condition pertaining to the nervous organisation of the individual and not to the form of attack. This is supported by the fact that cases in which epileptic attacks are succeeded by "co-ordinated convulsions" present (as we saw in the first lecture) distinct characteristics in age, sex, causation, conditions under which the fit occurs. In these respects, they resemble the patients in whom such attacks of hysteroid character occur without any preceding epileptoid symptoms. These facts make it probable that there is in these epileptics, in addition to the instability of nerve-tissue which causes epilepsy, also the instability, whatever it may be, which gives rise to hysteroid attacks. "Loss of control" may be the immediate mechanism by which this instability is thrown into operation, but will not alone account for its existence, and is no necessary condition for its manifestation. Nevertheless, in some patients the hysteroid tendency may be so slight that it never manifests itself spontaneously—never except during the post-epileptic state. Hence I think that the old conclusion is the true one; that all patients who present these co-ordinated convulsions after epileptic attacks are really the subjects of both epilepsy and hysteria. The existence of the epileptic symptoms may be obvious, or may be most difficult to detect; initial simple tonic spasm cannot be regarded as evidence of their existence, and it may be practically impossible to say in many cases, without the rare opportunity of witnessing the onset of the attacks, whether there is or is not an initial truly epileptiform seizure. This is the explanation of a point in the method of these lectures which may have occasioned surprise; while endeavouring to distinguish the two diseases, I have not ventured to separate them. Such a separation I believe to be practically impossible; to attempt it would entail an error, the amount of which would be unknown, and certainly large. I have therefore considered each fact with regard to the entire series; endeavouring to ascertain, however, the extent to which it is true of the cases with and those without hystero-epileptic or co-ordinated convulsion. The former term, "hystero-epileptic", has been employed in its popular meaning, without reference to theory, as a designation for the severer forms of hysteroid attack, in which their violence, severity, and recurrence bring them practically nearer to the disease which we call epilepsy than to that which we designate hysteria. I cannot but think that it would be a gain if we were to discard the dubious word hysteria, even in its modification, as a designation for such attacks, and call them simply "co-ordinated convulsions".

Such forms of attack, in which the convulsive phenomena are violent in degree and disorderly in sequence, are in the lowest grade of co-ordinated phenomena; while the highest are those forms of involuntary action which we call automatic. In these, complex actions are performed, sometimes with considerable skill, of which the patient is subsequently entirely unconscious. Dr. Hughlings Jackson has long maintained that these are really post-epileptic phenomena. That they are commonly such, all must, I think, agree. The older view was that these were true epileptic events, the result of such changes in the brain as might in another degree or part constitute an ordinary epileptic attack. While recognising that they are commonly post-epileptic, I doubt whether the older view must be entirely discarded. Many of the phenomena mentioned in the first lecture may make us hesitate to assent to the conclusion that the discharge of epilepsy must always cause inco-ordinate symptoms. What, for instance, is the vision of an old woman

making ugly faces, which one patient had sometimes as the aura of a fit, sometimes as an attack of *petit mal*, but an automatic action of a co-ordinated discharge in the centre for visual ideas? The remarkable aura which I detailed at length suggests the same explanation, and so do many others that were alluded to. I have seen a patient suddenly, while being watched, without the slightest pallor, hesitation, or any symptom to indicate a discharge in the highest centre, proceed to empty his pockets, take off his coat, or to perform some action which in him characterised, and I think constituted, his attacks of minor epilepsy. With regard to the attacks of post-epileptic automatism, the same difficulties face us as in the case of the co-ordinated convulsion. We have the facts that in one individual attacks may always be followed by automatic action, and in another similar attacks may never be so followed. This suggests that there is, underlying the phenomenon, a condition of the centres on which it depends. Were it not for this condition, the epileptic attack would not lead to it; and this condition may, in rare cases, cause its primary manifestation.

[To be concluded.]

THE CROONIAN LECTURES ON SOME POINTS IN THE PATHOLOGY AND TREATMENT OF TYPHOID FEVER.

Delivered before the Royal College of Physicians of London.

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LECTURE II.

WHEN we proceed to consider the clinical and pathological phenomena of typhoid fever, and to compare them with those of allied diseases, it is evident that we may divide them into two classes—1. Those which are peculiar to this form of fever; 2. Those which are common to it with many other febrile diseases;—the first being the characteristics of the species, the second those of the genus. To the former class belong the etiology and mode of dissemination, the period of incubation, the intestinal lesions, the roseola, and the type and duration of the fever; to the latter, the high temperature, the febrile consumption and wasting, the albuminous infiltration and softening of many organs and tissues, and various disturbances of the principal functions of the body.

I propose first of all to consider the pathology of some of these secondary phenomena, which are indeed of primary importance in the treatment; for our control over the former is so restricted that, when once the system has become infected with the typhoid poison, it is out of our power to prevent it from producing the typical lesions. If we examine carefully the organs of a fatal case of typhoid fever, or of any of the infective fevers, or even of other febrile diseases which are not due to an infective process—though here the changes are less constant and less marked—we shall find some or all of certain morbid conditions.

The first is a granular or albuminous infiltration of the cells of most of the glandular organs, especially the liver, the kidneys, the salivary glands, the pancreas, the gastric glands; and also a similar change in the striped muscular fibres, both of the heart and of the voluntary muscles. The change consists in the appearance in the protoplasm of the cells and fibres of extremely minute granules, soluble in acetic acid, giving a clouded appearance which has been compared to that of ground glass; this obscures the nuclei, and causes, when advanced, some enlargement and irregularity of form. In extreme cases, the cells thus affected disintegrate into a detritus, this being usually, though perhaps not necessarily, associated with fatty degeneration. Unless the change is very extensive, the affected organs do not show any marked alteration to the naked eye; they may be somewhat enlarged, of diminished consistency, and, unless congested, paler than usual, with their normal shiny appearance somewhat dulled. Virchow, as is well known, regarded this granular infiltration as an inflammatory parenchymatous exudation; and in many cases these changes are associated with processes which are unquestionably inflammatory, but they may occur independently, and must probably be looked upon as essentially of a degenerative nature; often, indeed, they are but the first stage towards fatty degeneration. A change, which is probably of the same nature, occurs

in the nerve-cells of the central nervous system; they become granular, with an increased deposit of granular pigment.

It can hardly be doubted that the result of a large number, if not of the majority, of fatal cases of typhoid fever is due to these changes. If we exclude those deaths which are directly caused by the intestinal lesions, as perforation and hæmorrhage, which after all are the minority, the remainder may for the most part be ascribed to failure of the heart and central nervous system. And of these, failure of the heart, with its concomitant congestion of the lungs, plays the most important part.

The affection of the heart, then, is of primary importance, and its pathology and symptoms have been most carefully investigated by many observers. Very often the heart presents no obvious changes to the naked eye; in other cases, it is more or less softened and altered in colour; but, without a microscopical examination, it is impossible to pronounce with any certainty on its condition. The changes may affect both the muscular fibres, the interfibrillar connective tissue, and the vessels. The fibres present to a greater or less degree the granular changes which I have described; the striæ are more or less obscured, and frequently with the albuminous granules oil-globules are present. The muscle-nuclei often show indications of proliferation, and a small-celled infiltration is frequently visible between the fibres, due either to an exudation or to proliferation of the connective tissue. M. Hayem also describes a proliferation of the cells of the tunica intima of the small arteries, which he ascribes to an endo-arteritis. Not unfrequently there is a diffused sanguineous infiltration of the intermuscular connective tissue. Side by side with the altered fibres, we commonly find others which are quite normal. These changes are frequently scattered in patches through the walls of the heart, and are often well marked in the musculi papillares of the mitral valve. We have, then, here evident signs of an irritative lesion as well as of degeneration, and M. Hayem regards the process as essentially a myocarditis.

The symptoms of this degeneration of the heart are well known, and had attracted the attention of all the great writers on fever before the intimate pathology of the affection was studied. They consist in feebleness of the cardiac impulse, which sometimes becomes almost imperceptible; an alteration in the character of the first sound, which becomes short and faint; and to this must be added the soft systolic apex-murmur which is frequently present in severe cases of typhoid, and which disappears again during convalescence, and is probably due to want of tone in the musculi papillares of the mitral valve allowing some regurgitation. This systolic murmur is often present before the other signs are very pronounced.

Corresponding changes occur in the pulse; the usual febrile dirotism becomes exaggerated, and, although the pulse may remain for a time tolerably full, it becomes extremely compressible, undulating, and shows more or less irregularity, not so much of rhythm as of force.

When death is caused by this condition of the heart, it is usually preceded by a gradually increasing hypostatic congestion of the lungs, and takes place slowly; but occasionally it is quite sudden, from syncope. Usually the symptoms of this degeneration of the heart in typhoid do not show themselves till the later stages of the disease. But there are reasons for believing that the affection may begin quite early, and run a very rapid course. Last year, a woman in the London Fever Hospital died quite suddenly from syncope about the twelfth or fourteenth day of the disease, before any intestinal sloughing or ulceration had taken place; and the heart was found in an advanced stage of granular infiltration. These cardiac changes are by no means peculiar to typhoid fever; they are met with in typhus, small-pox, erysipelas, scarlatina, diphtheria—where they are usually extremely well marked; and in this disease, as is well known, sudden death by syncope is by no means uncommon.

The next change which is common to typhoid and many other fevers, is the so-called vitreous or waxy degeneration of the striped muscles, described by Zenker. The fibres swell, lose their striation, and the tube of sarcolemma becomes filled with a semitransparent, colourless, homogeneous material, which has been compared to wax in appearance. These altered fibres present transverse cracks and fissures, and ultimately break up into irregular fragments, which become smaller and smaller, and at last disappear, leaving the sarcolemma empty. Then, if the patient recover, the fibre is regenerated by a cell-growth within the sarcolemma. Most probably the process consists primarily in a coagulation of the contractile muscle-substance analogous to that which takes place in the rigor mortis. This change is common to many fevers, as typhus, small-pox, scarlatina, tuberculosis. In typhoid, it is often very extensive, and may cause considerable temporary destruction of muscle. It may affect all the striped muscles, but is most frequently met with in the muscles of the abdominal wall, the adductors of the thigh, the diaphragm, and the tongue. Though not very often present in the heart, it does occasionally occur here also.

Another change which is common to all febrile diseases is the diminution of the red blood-cells, probably due to increased disintegration on the one hand, and to diminished formation on the other.

A fourth change is the wasting of the body. This is usually proportionate to the intensity and duration of the fever; and in typhoid, the serious lesions of the digestive canal tend still further to increase it. Leyden reckons the average daily loss of weight in high continued fever at about 7 per 1000 of the total weight of the body, which is about half that which takes place in total deprivation of food; and Chossat has shown that death takes place from inanition when the body has lost 40 per cent. According to this calculation, it would take eight weeks of high continued fever to reduce the weight 40 per cent. and cause death from inanition; but in chronic fever, a much greater loss can be supported, in consequence of the febrile process maintaining the temperature, and so preventing death from cold—inability to maintain the temperature being one of the causes of death in inanition. It rarely happens that death takes place in typhoid merely from the febrile wasting, though this is often very great, and exceeds the daily average given by Leyden. I have recently had under my care a case where the weight was reduced in about four weeks from eleven stone to eight stone—a loss of nearly 30 per cent. Supposing the fever to have continued for eight weeks, this would have produced a loss of nearly 60 per cent. It would, however, seem that the amount of wasting is greater in persons who are muscular and corpulent than in those who are spare and lean: the latter class, it is well known, bear an attack of fever better than the former. The causes of this wasting of the body in fever are twofold. The first is simple inanition, due to diminished ingestion and assimilation of food, the result partly of failure in the appetite, partly of impaired digestive and assimilating power, and partly of restriction of the diet in consequence of certain theories as to the injurious effects of food in fever. The influence of this last cause varies, of course, with the mode of treatment which happens to be in vogue. In this country, in consequence of the teachings of Graves, Stokes, Todd, and other authorities, fever-patients are for the most part given as much food as they can assimilate; but there have been times, especially on the Continent, when the routine fever-diet would barely suffice to ward off death from inanition. Under any circumstances, it is probable that the amount of food assimilated in fever does not amount to half that taken in health.

This wasting from inanition takes place chiefly at the expense of the adipose tissue and of the store-albumen, *i.e.*, the albumen of the blood, lymph, and interstitial fluids; to a less degree, and chiefly in the later stages, at the expense of the tissue-albumen.

The second cause of the wasting in fever is the increased consumption due to the febrile process. This in man affects both the fat and the albumen, though in the artificial septic fever of dogs it appears that the consumption of the hydrocarbons is not increased. The consumption of albumen is measured by the amount of the urea discharged, which in fever very much exceeds that which could be produced by the metabolism of the nitrogenous food, and, notwithstanding the restricted diet, often exceeds the amount passed in health on a full diet. This great increase in the discharge of urea is always most marked in the earlier stages of the fever, and in protracted fevers, as typhoid, becomes much diminished in the later stages. There are strong reasons for believing that this increased formation of urea takes place at the expense of the tissue-albumen, and not of the store-albumen; for it is found that it is accompanied by an increase of the salts of potash in the urine, while the salts of soda are commonly diminished: hence it is probable that the urea is derived from albumen which is associated with potash, and not with soda. Now, this is the case with most of the tissues, especially the muscles and red blood-cells; while, in the liquor sanguinis and lymph, the salts of soda predominate: hence in all probability the former, and not the latter, are the sources of the urea produced by the febrile consumption. The colouring matter of the urine appears also to be increased in fever, and this must necessarily be derived from the coloured tissues, especially the red blood-cells, which we have seen to undergo great diminution; and, as extensive degenerative changes are commonly found in the muscles, it is probable that these tissues especially suffer in the febrile process.

The consumption of hydrocarbons, as shown by the increase of carbonic acid in the expired air, does not admit of so exact a determination—the proportion in each expiration is always diminished, but, owing to the increased frequency of the respirations, the total amount expired in twenty-four hours is always increased. Senator reckons this increase at 37 per cent.—a far larger quantity than could be afforded by the carbon set free by the increased disintegration of the albumen.

We have now to consider what is perhaps the most important phenomenon of fever: the temperature. The maintenance of the temperature of the body depends, of course, on a balance being kept up between the production and dispersion of heat, this balance being maintained by a re-

gulating apparatus; in consequence of which, in a state of health, the production of heat on the one hand, or its dispersion on the other, may be very greatly modified without producing more than slight and temporary alterations of temperature. There can be no doubt that this regulation is effected through the nervous system, though the exact mode of operation is still somewhat obscure.

When the influence of the intracranial part of the nervous system is removed, as by the crushing or division of the spinal cord below the origin of the phrenics, the temperature of the body, as is well known, cannot be maintained; it has become *labile* instead of *constant*. Sometimes a great rise takes place, sometimes a fall. In man, the former is the more common effect, though several cases have been recorded where the temperature has fallen to extremely low degrees. In small animals, the effect seems to depend very greatly on external conditions. Under the influence of external warmth, the temperature will rise to extreme heights; if the external conditions favour cooling, the temperature will sink to such a degree that the animal dies of collapse. These alterations of temperature, due to injury of some part of the nervous system, are probably produced by alterations in the dispersion of heat, which is effected chiefly by the lungs and skin, and consequently regulated by the respiratory and vaso-motor centres, together with the special nerves (if such there be) which control the sweat-glands.

What part the nerve-centres play in the regulation of the production of heat is a question of great difficulty, and one about which physiologists are by no means agreed. There can be no doubt that the chemical processes by which heat is produced in the animal body are independent of the nerve-centres. This is shown by the fact that heat continues to be produced after the complete destruction of the spinal cord; nay, the production goes on for some time after death has not only destroyed all nervous influence, but has brought the circulation itself to a standstill. It has been proved by Valentin that the *post mortem* production of heat is a constant phenomenon; and, in consequence of the diminished dispersion from arrest of the cutaneous circulation and respiration, we often see an actual rise of temperature. Generally, however, this *post mortem* formation of heat is only indicated by a retardation of the cooling of the body. Nevertheless, it can hardly be doubted that the production as well as the dispersion of heat can be greatly modified by nervous influence, though we are not, perhaps, justified in assuming the existence of a special heat-exciting and heat-depressing centre. The phenomena are most probably vaso-motor. It is evident that if more or less blood flow through the more active heat-producing organs, more or less heat will be produced.

When we proceed to consider the cause of the high temperature in typhoid fever, where it often remains above normal for several weeks, we can hardly doubt that there is some disturbance of the regulating function; for no mere alteration of the conditions, either of the production or dispersion, will cause more than slight and temporary changes in the temperature; but the nature of this disturbance is still very obscure. That the vaso-motor system is rendered more unstable in typhoid is shown by the phenomena of the so-called *tâche cérébrale*, which are almost always well marked. Now, in a disease like typhoid, in which the temperature usually runs such a typical course, with regular diurnal variations, corresponding, though in an exaggerated manner, to the diurnal fluctuations in health—with so steady a rise to the fastigium, and decline to the defervescence, so that from a mere inspection of the chart we can generally diagnose the disease—it is quite certain that the regulating apparatus has not ceased to act. The view has been put forward, and widely accepted, that in typhoid fever the regulating point has been simply raised and adapted, as it were, to a temperature, say five degrees above the normal. If, however, we compare the behaviour of the temperature in this disease and in health, this view becomes untenable. In the first place, the temperature has become more labile; it can easily be raised and depressed by causes which would have no effect in health. Thus, changes in diet, slight exertion, mental emotion, will often cause great temporary alterations. The same point is shown by the effect of remedies. A dose of thirty or forty grains of quinine will hardly affect the temperature in health, but in typhoid fever will generally reduce it three or four degrees, and keep it down for several hours.

Secondly, we miss in fever that compensatory reaction which Jürgensen has shown to occur in health when the temperature is artificially reduced. According to his observations, if a healthy man be placed in a cold bath, the temperature may be reduced many degrees, in proportion to the coldness and duration of the bath; and it continues to sink for some time after removal. Soon, however, it again rises, and continues to do so till it has gone above the normal point. If the bath be repeated, the same phenomena show themselves; but, after a certain number of repetitions, the reactive rise counteracts the chilling effect of the bath, so that further immersion no longer reduces the temperature of the rectum. In typhoid fever, this compensatory rise does not take place,

or at any rate to a much less degree; and, after fifty or even a hundred and fifty repetitions, the last bath reduces the temperature as effectually as the first. Hence, in typhoid fever, we regard the regulating influence as weakened, or at any rate rendered insufficient for the new conditions caused by the fever, but neither abolished nor set at a higher point.

We, however, not unfrequently meet with cases where the temperature appears to follow no law, but where the regulating function seems to be entirely suspended: where sometimes the temperature will rise to hyperpyretic heights, far exceeding those that are ordinarily met with in fever, and incompatible with the continuance of life; at other times it will fall far below the normal, and the patient will sink into a state of collapse.

There can, I think, be little doubt that these hyperpyretic temperatures are due to a paralysis of the heat-regulating functions of the nervous system, and are mostly an indication of a general failure or paralysis of the nerve-centres. Usually, therefore, their occurrence is a certain sign of approaching death; and in this form hyperpyrexia is very often seen at the termination of all febrile diseases, including typhoid. In these cases, the patient does not die from hyperpyrexia, but he has hyperpyrexia because he is dying; and no lowering of the temperature by the abstraction of heat, as by a cold bath, has any effect in retarding the fatal event. Indeed, where there is complete paralysis of the regulating centres, it is not possible to lower the temperature except quite temporarily; it rises again immediately the abstraction of heat is desisted from. In other cases, however, hyperpyrexia may occur without any general or irremediable failure of the functions of the nervous system, though the high temperature itself will, if not reduced, soon lead to fatal coma. In these latter cases, if we can succeed not only in lowering the temperature, but also in stimulating the nerve-centres to a resumption of their functions, the patient will recover. Such cases are most frequently seen in acute rheumatism. In the zymotic fevers—as typhoid—extreme hyperpyretic temperatures, as 108 to 110° Fahr., commonly indicate a general paralysis of the nervous centres and impending dissolution. The regulating function being thus weakened relatively or absolutely, and the temperature rendered more labile, we have now to consider what is the factor which causes the temperature to maintain itself above the normal point, mere lability being as likely to cause depression as exaltation. The lability, indeed, often continues longer than the fever; and very commonly, for some time after complete defervescence has become established, extremely slight causes, which would have no effect whatever in health, will send up the temperature many degrees.

The opinion generally entertained is, that the cause of the fever-heat is increased combustion of the tissues, which we have seen to be the most characteristic phenomenon of fever. But, although in fever the combustion is excessive in relation to the amount of fuel supplied by the food, it is not necessarily in excess of that which takes place in health, where the supply of food is abundant and the muscles are well exercised; and it has not been found possible to establish any definite relation between the discharge of urea and carbonic acid on the one hand, and the temperature on the other. Attempts have been made to clear up this point by estimating the amount of the dispersion of heat in fever: if this be lessened, the temperature may of course rise, though the production of heat be not increased, or even be diminished; if, on the other hand, the dispersion of heat be increased, and at the same time the temperature rise, it is evident that the production must be increased in a still greater degree. The results obtained by different experimenters are conflicting, but on the whole there can be little doubt that for the most part the dissipation of heat is increased in fever. During the initial rigors, indeed, it is most probable that the dissipation of heat is much diminished from contraction of the cutaneous capillaries; and in all cases there is reason for believing that the dissipation as well as the production of heat is subject to very great fluctuations. Senator considers that periodic contractions and dilatations of the cutaneous capillaries take place, favouring the retention or dissipation. It is, however, quite conceivable that the balance may be disturbed in the other direction; the combustion, though increased relatively to the amount of food taken, may be actually below that in health, the dissipation may not be diminished in proportion, the labile temperature may yield readily to external conditions which favour cooling, and so we may have the paradox of an afebrile fever.

In the artificial septic fever produced by the injection of pyrogenetic substances, if small animals, as rabbits and guinea-pigs, be experimented on, and if the temperature of the air be below 75°, and means be not taken to prevent the dissipation of heat, the temperature will fall below normal; if, on the other hand, the air be above 75°, or the animal be wrapped up in wadding, the temperature will rise to febrile heights. In larger animals, as dogs, the influence of the surrounding conditions is less marked, and under ordinary circumstances their tem-

perature always rises; but if they be shaved and exposed to a cold atmosphere, then here also a fall takes place. Similar phenomena occur in the human subject, and many cases and even epidemics of typhoid have been met with in which the temperature has been subnormal throughout the whole course of the disease.

One such epidemic has been reported by Dr. Strube. During the siege of Paris by the Germans in 1870, an epidemic of typhoid fever broke out among the troops, beginning to show itself during the march to Paris, and attaining its greatest height in October. In November, a decline took place, which was followed by a fresh outbreak in December. These two outbreaks differed greatly in their characters: the later one resembled in all respects the ordinary form of typhoid; the earlier one presented very different features. In many of the cases, the temperature throughout was subnormal, and in others never exceeded the normal point. The roseola was unusually profuse; the nerve-symptoms were of marked severity, and were in inverse ratio to the temperature, consisting of violent delirium alternating with stupor; the duration of the fever was very short, defervescence usually taking place at the end of a fortnight. Of the twenty-three fatal cases, in twenty death took place during the first fourteen days. The abdominal symptoms were slight, but the characteristic lesions were found on *post mortem* examination. All the cases were characterised by great prostration. These cases presented some features which were probably due to this peculiarity of the temperature; thus, the pulse was but little accelerated, seldom exceeding a hundred; the tongue did not become dry and brown, and the enlargement of the spleen was either absent or much less marked than usual. Dr. Strube attributed the peculiar features of this epidemic to the depressed condition of the troops; they had been exposed to great hardships on the way to Paris, over-fatigued by forced marches, and very insufficiently supplied with food, and the supply continued deficient for some time after their arrival, owing to difficulties of transport. In the later outbreak, these conditions were no longer present.

It now becomes an interesting subject of inquiry, whether the mode of production of heat is the same in fever as in health, *i.e.*, whether it is produced in the same organs and by the same kind of combustion.

The source of the animal heat is, of course, the combustion of the food and the tissues; wherever metabolism of food or protoplasm is going on, *i.e.*, wherever there is living tissue, heat is being set free. Hence every living cell of the body contributes to the production of heat; but heat is set free more abundantly in some organs than in others, and, taking the body as a whole, the principal sources of heat are the muscles and abdominal viscera, especially the liver, the temperature of the blood in the hepatic vein being the highest in the body. The heat set free by the muscles depends largely on their functional activity; when in exercise it is very great; when they are at rest it is small. The production of heat by the abdominal viscera is probably much more constant, but is necessarily dependent on the assimilation and metabolism of the food.

Now, in fever, the production of heat by the abdominal viscera, so far as it is due to the metabolism of the food, must necessarily be diminished in consequence of the restricted diet, and the production by muscular contraction is also much less than in health, as the muscles are for the most part kept at rest. As to how far the heat-producing functions of the liver and other abdominal viscera are modified in fever, little is known. It is possible that an increased production takes place in the spleen, which is so constantly found enlarged and softened, and where it is probable great disintegration of red blood-cells takes place. It is remarkable that, in the cases recorded of pyretic typhoid, this enlargement of the spleen was either absent or less marked than usual. With regard to the muscles, however, we have evidence that, notwithstanding the diminution of functional activity, the production of heat in them, is very active. It has been found by direct measurements that, while in health, when the muscles are at rest, their temperature is below that of the blood in the left ventricle of the heart; in fever it is higher, and both clinical and pathological observations show that the metabolism of the muscular tissue is greatly increased. Thus there is the great muscular weakness so characteristic of fever, the increased discharge of the salts of potash and of creatine and creatinine by the urine, and the various forms of degeneration found after death in fatal cases. We have, therefore, strong reasons for believing that the muscles in fever, though for the most part in a state of rest, are an important source of the febrile heat, and that this production of heat takes place at the expense of the contractile tissue, and not, as in muscular contraction, from oxidation of hydrocarbons. In fever also, though the voluntary muscles are kept at rest, it must be remembered that the heart and respiratory muscles are more actively exercised.

[To be continued.]

ABSTRACT REPORT OF LECTURES ON THE COMPARATIVE ANATOMY OF MAN.

Delivered at the Royal College of Surgeons of England.

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LECTURE I.

THE term "Anthropology" is now applied to the natural history of man, and is based chiefly on the sciences of anatomy, physiology, and on history, reports of travellers, and the remains of works of art of nations now extinct. The great scope and interest of the subject, as well as its most convenient subdivisions, are well illustrated by the prospectus of the teaching at the Anthropological Institute of Paris—an institution founded about eight years ago, in which there are at present no fewer than six chairs, all held by distinguished professors: 1. Comparative Anatomy, in relation to Anthropology, by P. Broca; 2. Biological Anthropology, or the application of Anatomy and Physiology to Anthropology, by Topinard; 3. Ethnology, or the Study of the Races of Men, by Dally; in this department, the differences in language, customs, and habits of the various races of mankind are described, and the mode in which these have originated is discussed; 4. Linguistic Anthropology, by Hovelacque; 5. Palaeontological and Prehistoric Anthropology, by Mortillet; and 6. Demography, which includes what we commonly call social and vital statistics, and Medical Anthropology, by Bertillon. These subjects are publicly taught in a school, supplied with all the necessary appliances, founded partly by private munificence, but also liberally endowed by the municipality of Paris and the Department of the Seine. There is also in Paris a complete course of General Anthropology, given yearly by M. de Quatrefages, in connection with the Jardin des Plantes. To these institutions we have nothing comparable in Britain; and neither at our universities nor elsewhere is any branch of anthropological science systematically taught. The present lectures only embrace a small portion of one of the six subdivisions enumerated above, namely, that of Biological Anthropology. This science is one of observation purely; and, in proportion as the materials upon which our observations are founded are multiplied, so will the value of the observations be increased. These materials are collected in museums, which at present in this country are not so complete as might be desired, and as should be, considering the extent of our possessions and the amount of our intercourse with foreign races. Till lately, the British Museum entirely ignored anatomical anthropology; and it is only by the Royal College of Surgeons that anything has been done to forward the science. Our collection, which is considerably the largest in this country, was founded by John Hunter, who, simultaneously with Blumenbach in Göttingen, was the first collector of anthropological material. The collection of John Hunter has been increased at various times till it now contains about twelve hundred crania of different races. The largest private collection in this country is that of Dr. Barnard Davis of Shelton, in Staffordshire. It considerably exceeds that of the College, both in number and in variety of specimens. Happily, this collection is about to be united to that of the College; and, under the care of the Council of the College will be made accessible for all who may wish to pursue the study of anatomical anthropology. The officers of the army and navy have also made very good, though much smaller, collections for their respective hospitals at Netley and Haslar, but owing to those being wide apart and not situated in easily accessible positions, they have not been made so much use of as might have been the case had circumstances been different.

One of the most important additions which has been made to the museum since the last course of lectures, besides Dr. Barnard Davis's collection, only a small portion of which has arrived, is a series of skulls collected in the Fiji Islands, in 1876, by Baron Anatole von Hügel, forming part of a donation made by Mr. Erasmus Wilson. They consist of sixteen crania of the *Kai Colos* or mountaineers of the interior of the western portion of Viti Levu, and five crania from the western coast and small islands adjacent. The inhabitants of the Fiji group are generally described by ethnologists as a mixed race compounded of Melanesians and of brown Polynesians, as the islands are situated on the confines of the territories inhabited by these two races;