GULSTONIAN LECTURES ON THE HEAT OF THE BODY.

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LECTURE III.—*Concluded.*

THE effects of pyrexia which we have considered hitherto have been those which are discovered after death. But there are several conditions to be studied upon the living patient, which, also, we may look upon as consequences of pyrexia.

When we were going over, a short time ago, the proofs that there is an augmented oxidation of the tissues in fever, one of the proofs which we noted was the increased formation of urea. But if, indeed, fever beget fever, we should expect to find that an increased excretion of urea is not only an accompaniment of pyrexia, but also a consequence thereof. Is this really so? when the temperature of the body is raised by artificial means, is the excreted urea increased in quantity? Bartels and Naunyn have made experiments upon this question, and answer it in the affirmative. For example, Naunyn shut up a dog in a vapour-bath, and thereby raised his temperature in three hours from 102.2 deg. to 108.5 deg.; the quantity of urea excreted during that period was much greater than natural. And thus we have another argument in favour of an increased destruction of the body-tissues dependent upon pyrexia.

Next: in the tissues which are open to our inspection during life, I mean the mucous membranes and the skin, are there any changes which we may regard as due to the pyrexia?

First, of the mucous membranes. Laennec declares that pulmonary catarrh usually complicates fevers of all kinds. Now we may undoubtedly drop Laennec's qualification, pulmonary catarrh, and plainly say that catarrh in general, of the respiratory, digestive, and urinary passages, usually complicates fever. For what else than a catarrh of the mouth is that furred tongue to which we so often look for purposes of diagnosis and prognosis in febrile diseases? And, again, with regard to the lungs, Laennec observes that "one of the most interesting results which auscultation has afforded him, is the constant existence of a latent or manifest pulmonary catarrh during the whole course of continued fever. In the beginning, and often during the whole of the disease, the catarrh is latent, without cough or expectoration, and can be detected only by means of the stethoscope. In the eruptive fevers, the pulmonary catarrh is quite as constant, and mostly manifest. And in intermittent and symptomatic fevers, there is also pulmonary catarrh, usually latent." Laennec goes on to remark that the inflammatory fever of nosologists, characterised by the rosy colour of the face, the moisture and cleanness of the tongue, and the moderate heat and dampness of the skin, is of all fevers that in which the signs of catarrh are least marked. And let us note that by inflammatory fever was meant a fever from which symptoms of putridity and debility were, in like manner, wholly absent. Another fact of which Laennec speaks, and to which we can all bear witness, is a very striking illustration of the close connexion between catarrh and pyrexia. "I have often wondered," says he, "how that, in fevers which come to a perfect crisis, at that very moment all the signs of an intense and extensive catarrh vanish along with the coma, the meteorism, the frequency of the pulse, and the heat of the skin." I think that I have said enough, or, what is much better, that Laennec has said enough, to justify our looking upon catarrh as a consequence, almost necessary, of pyrexia.

Secondly, as to the skin. Except during the cold stages, an injected state of skin is so constant an accompaniment of pyrexia, that we may fairly consider the one to be an effect of the other. That is to say, the skin is injected whilst the patient is radiating more heat than natural therefrom. The exact appearance of the injected skin differs in different cases, and varies in the same case, according to conditions which we do not understand. I do not speak of specific eruptions. The present unwonted epidemic of small-pox has afforded us the means of seeing these pyretic eruptions, common to any kind of fever. I refer to the rashes which appear in variolous patients before the outbreak of the peculiar eruption. Sydenham, who must have been studied of late

more than for many years past, in our necessity of knowing something about a disease which to many of us had seemed almost extinct, refers to these eruptions where he says that small-pox breaks out sometimes like erysipelas, sometimes like measles. Sometimes like erysipelas: that is to say, the exanthem is an uniform bluish like that of belladonna-poisoning—a roseola, in fact, roseola variolosa as it has been called. Sometimes like measles: this, apparently, is not at all uncommon, for the shotty papulæ of variola to be preceded by a measly eruption. And to these two forms of rash in small-pox, others may be added. The precedent eruption sometimes possesses all the characters of a scarlet-fever rash, minute scarlet points thickly set. Sometimes the eruption is identical with that which is commonly called the subcuticular mottling of typhus. In a patient at Saint Bartholomew's, the eruption simulated, first, the rash of measles, then that of scarlet fever, before the appearance of the true variolous eruption upon the fifth day. There is nothing peculiar to small-pox in these exanthemata. The scarlatiniform, roseolous, and typhus-like eruptions are very common in pyrexia. I have seen perfect subcuticular mottling in sunstroke and in acute tuberculosis; hence the difficulties of diagnosis, with which we are all too familiar. These eruptions have been called sudoral by Trousseau, when the fluxion upon the skin results in sweating.

I must now ask your attention to another subject.

The temperature of the healthy body is a mean struck between a very variable amount of heat produced, and a corresponding variable amount of heat lost; and the body has the power of balancing this gain and loss very nicely within certain limits; it has the power of regulating its temperature independently of voluntary means. It is said that great muscular exercise may increase the activity of the body's combustion to such an extent that the quantity of carbonic acid excreted shall be augmented nine-fold. Now, not more than one-fifth of the force set free becomes mechanical work, the rest passes into heat; yet the excessive amount of heat generated in the case supposed does not produce pyrexia, but it is got rid of in some manner; the regulating power of the body maintains its authority. In fever, likewise, there is an increased production of heat and an increased loss of heat, yet the temperature is not maintained at the normal standard, but at an abnormally high standard. How is this state of things to be explained? An adequate answer to this question would obviously carry us to the very starting place of fever, to its immediate cause. Let us prepare ourselves for the discussion of this great subject, by recapitulating the facts which have been learned concerning the influence of the nervous system upon the heat of the body. We are not yet compelled to content ourselves with hypotheses; and therefore I will pass by the more or less happy guesses of early physiologists, and come at once to the Croonian Lectures which Brodie read before the Royal Society in 1810. He showed that, when a dog was pithed and artificial respiration was kept up, the temperature fell from 100 deg. to 78 deg. in two hours and a half; in fact, the animal cooled more rapidly under artificial respiration than he would have done had he been simply left alone after having been pithed. From experiments of this kind Brodie drew the following conclusions: that the heat of the body is not due to changes produced in the blood by respiration; that, when the air respired is colder than the natural temperature of the body, the effect of respiration is not only not to generate animal heat but actually to diminish it; and that, when the influence of the brain is cut off, no heat is generated. We will not stay to criticise Brodie's conclusions, but merely observe the fruitful field of experiment which he thus opened. In 1836, he returned to the topic, with reference to a case of injury to the spinal cord between the fifth and sixth cervical vertebrae, the temperature of the body rising to 111 deg. Since Brodie's time, the same accident of injury to the spinal marrow, and the same consequence of great elevation of temperature, have been observed in five cases; namely, three by Billroth, by Simon, and by Frerichs, and two cases which were recorded by Dr. Hermann Weber in the first volume of the *Transactions of the Clinical Society*.

Next came Bernard's experiments upon the sympathetic nerve, of which Marey's theory of fever, which we previously discussed, was the direct offspring. When the sympathetic is divided, the part which is thrown thereby into a state of active congestion, with increased afflux of blood, is raised in temperature: there is an approach towards equalisation of the temperatures of the internal and of the superficial parts of the body. But the body generally, and the deeply seated parts in particular, really fall in temperature, because of the increased loss of heat from the actively congested patch of skin. When this loss of heat is prevented, by preventing radiation from the skin, the internal temperature does not fall. Budge went a step further, by showing that the vaso-motor nerves are paralysed not only by section of the sympathetic, but also by section of the corresponding portion of the spinal cord.

At this point Tscheschichin took up the subject. He found that division of the spinal cord was followed, not as in Brodie's case by ele-

vation of temperature, but contrariwise by depression of temperature. For example: a rabbit's spinal cord was divided between the third and fourth cervical vertebrae: the temperature of the rectum fell to 79 degs. in four hours: the animal lived sixteen hours after the operation. Tscheschichin explains this fall of temperature in the following manner. First, the production of heat is diminished as a result of weakening the force of the heart's contractions, and also of impeding the respiratory movements; and, secondly, the loss of heat is increased for the reason of which we spoke with reference to the sympathetic nerve, namely, the dilatation of the cutaneous vessels. The loss of heat which ensues upon varnishing the skin of animals, and which causes their death, was, you will remember, found to be due to the same condition. Now varnished animals can be kept alive if the loss of heat by radiation from the surface be prevented; therefore it becomes of interest to know whether the depression of temperature consequent upon section of the cord can be obviated in the same manner. Tscheschichin says it cannot; he divided the spinal cord of a rabbit between the fifth and sixth cervical vertebrae, and immediately wrapped the animal in cotton-wool. Nevertheless, the temperature fell, although not nearly so quickly as in the other experiment: moreover, life was prolonged. The day after the operation, the temperature was 89.75 degs.; the rabbit did not die until the third day. Thus this experiment seemed to prove that increased loss of heat will not account for all the depression of temperature in the cases under consideration. If this be so, there must be diminished production of heat also, probably consequent, so Tscheschichin supposes, upon interference with the functions of the heart and lungs. And if this be so, we should expect that division of the par vagum would depress the temperature. Well, as a matter of fact, section of the vagi produces no very definite effect upon the body-heat; at first, certainly, the temperature does fall a little, the respiration is slackened, and the contractions of the heart become very frequent; but the temperature is soon raised by the inflammatory engorgement of the lungs which rapidly ensues. So far then, section of the vagi hardly bears out Tscheschichin's opinion that the generation of heat is diminished.

The higher up in the cord the section is made, the greater the depression of temperature. But so soon as the uppermost part of the medullary oblongata is reached, so soon as the cord is severed from the pons Varolii, a great change is found to occur in the symptoms. The temperature, instead of falling, greatly rises. The animal may live for several hours even after this extremely severe lesion. A rabbit operated upon in this way by Tscheschichin rose in temperature to 108.75 deg. before it died, about two hours after the operation. He regards this pyrexia to be indicative of an inhibitory power residing above the medulla oblongata, and controlling the independent activity of the cord; to divide the brain from the cord unchains the spinal animal functions, and suffers the combustion of the body to go on without restraint. Pyrexia, then, is a paralysis of this inhibitory centre; a paralysis which can be induced in sundry ways; for instance, by injuriously active substances present in the blood, septic and other matters; or by an external irritation which acts powerfully upon the psychical and sensible sphere of the organism, and affects the moderator centre reflectively.

Such is Tscheschichin's theory; but his experiments, as we have already seen, are at variance with the clinical experience of Brodie and others. In order to clear up the cause of this discrepancy, the vivisections have been repeated by Naunyn and Quincke. These observers note, in the first place, that two of the cases of accidental injury to the cord, followed by high pyrexia, are stated to have occurred in the summer time; in Brodie's, Billroth's, and Dr. Weber's cases, there are no particulars upon this point, so that the question is again suggested: Was the depressed temperature, in Tscheschichin's experiments, really due, after all, to loss of heat? He endeavoured to obviate this objection, we saw, by wrapping the animals in wadding, and the temperature certainly fell much less rapidly than when no such precaution was taken. Naunyn and Quincke regard this method of preventing loss of heat as imperfect. Instead of using cotton-wool, they put the large dogs they operated upon into a box maintained at a heat between 78 deg. and 86 deg. And, instead of cutting the spinal cord with a sharp instrument, they crushed it, and thereby sought to imitate accidental injuries more closely, and also to prevent loss of blood. A dog, at a temperature of 103.5 deg. was operated upon in this way, on a level with the seventh cervical vertebrae. In twenty-four hours the temperature fell to 74.5 deg., and then the animal was put into the warm box; his temperature rose in five hours to 104.5, and then he died. Even when the dog was put into the box, immediately after the vivisection, there was a slight fall of body-heat at first. A dog at 98.75 deg. was put into the box immediately after his cord had been crushed; his temperature fell to 96.25 deg. in an hour and a half, and then began to rise, reaching 105.5 deg. at the time of death, six hours and a half after the operation. With regard to this slight fall in temperature, we

must not forget that the box was not heated above 86 deg., that is, twelve degrees below the heat of the body. These experiments seem to explain the cause of the incongruity between the experience of Tscheschichin's and that of the clinical observers whom I mentioned. It is very probable that nerve-fibres run down the cord which convey an influence controlling the combustion-processes of the body; when these fibres are destroyed, there is an excessive production of heat in the parts concerned. For the production of heat is really increased, and not, as Tscheschichin supposed, diminished. And the higher the injury to the cord, the greater the tract of body set free from control, and the greater the production of heat. More concerning these fibres is not known; whether they be identical with the so-called trophic nerves, or with the vaso-motor nerves, all is uncertain.

Let us now pass to another topic. When we discussed the subject of the natural temperature of the body, we also inquired into the influence which external circumstances exert upon that temperature, and especially the influence of external heat and cold. Supposing the temperature of the body to be raised above the standard of health; supposing, that is, a state of pyrexia; what is the influence of external heat and cold upon the temperature? is the effect similar to that produced in health, or diverse from it?

We will first put the case of a high external temperature. The data upon this part of the subject are not numerous. Wunderlich noticed that, during the hot summer of 1865, the temperature of his fever-patients was much higher than usual. In four weeks of July, twenty-five of his patients died, and fourteen of these died at a body heat of 104 deg. or higher; eight, or one-third, dying at 107.5 or higher. The danger which ensues to febrile patients from external heat is hereby well illustrated.

Upon the subject of external cold applied to fever-patients, there is much more to say. Any concession made to the doctrine that much of the danger in a given case of fever is due to the pyrexia, involves the obvious therapeutical indication, to lessen the heat of the body. How is this to be done? By cold applied externally so as to abstract heat directly? But what we have seen concerning the action of external cold upon the healthy body, certainly does not look promising for such a method of treating pyrexia. I refer to the elevation of the internal temperature which is consequent upon moderate cold applied to the surface. It becomes then very important to ascertain by direct experiment whether in pyrexia there is a similar reaction against abstraction of heat; whether a moderately increased loss of heat from the skin induces, not a depression, but an elevation of the temperature of the internal parts. Experiment proves that there is such a reaction and elevation of temperature in pyretic patients. Again, we saw that this elevation of temperature in healthy persons was due to an increased production of heat; and the observations of Liebermeister seem to prove that the same holds good of fever-patients. So far, then, even experiment is against the use of external cold as a means of diminishing pyrexia. And yet, on the other hand, we have the testimony of three-quarters of a century to the good effects of cold applied externally in fevers. The practice of Wright and Currie has been confirmed and extended in our own day by the experiments of Brand, Liebermeister, and many others. How is this beneficial effect of cold in fever to be explained? Or rather, in what manner can the augmented generation of heat in pyrexia be diminished by cold baths? Two explanations offer themselves—probably both are valid at the same time. First, the fall of temperature which follows after moderate abstraction of heat in a healthy person occurs in fever-patients also. The temperature rises at first during the bath, but a lowering of the body-heat soon follows, and the depression of temperature lasts longer than the elevation; yet, in severe pyrexia, the depression soon passes off, so that the baths must be frequently repeated in order to produce a decided cooling effect. Secondly, the temperature rises during the bath for a time certainly, but fever-patients lose more heat during this period than healthy persons do. Moreover, the power to counteract cooling by spontaneous augmentation of heat generated—a power which is limited in health—in fever is still more limited; that is to say, under great abstraction of heat, less heat is produced in fever than in health. Hence, not only is the frigorific action of the bath greater, but its calorific action is less in fever-patients than in healthy people; more heat can be abstracted from the pyretic, and their power of maintaining a standard temperature is more easily overcome.

Schröder has made some observations upon the quantity of carbonic acid expired by fever-patients when treated by the cold bath, which confirm the conclusions at which we have just arrived. His experiments go to prove that the formation of carbonic acid—in other words, the consumption of the body, is really diminished by cold baths when employed in typhus fever. Immediately after the bath, the quantity of carbonic acid is found to be greater than before the bath; the muscular

exertion of getting out of the water, and the quickened respiration, possibly, have something to do with this. But, in from twenty to fifty minutes after the bath, the quantity of carbonic acid expired is much diminished, the temperature of the body being lowered at the same time. Schröder also found that less urea was excreted by typhus patients on the days when cold baths were given than on the days when they were not given.

To sum up, the cold water treatment does not shorten the duration of a specific fever, but only lessens the severity of its most important symptom, the pyrexia; and, at the same time, the disposition to lethargy, frenzy, putridity and debility, is lessened, whereby support is given to the doctrine that these typhoid symptoms are, partly, at least, due to the height and length of the pyrexia, and not altogether to the specific character of the fever.

Before we quit the subject of pyrexia, allow me to recapitulate a few results which we have obtained concerning it. The healthy body possesses a power of keeping up a mean temperature of about 99 deg.; to this end, the amount of heat generated, and the amount dispersed, are both under control. In pyrexia, this regulating function is disturbed in two ways: first, the standard of mean temperature which the body strives to maintain is pitched higher; next, the powers by which the body keeps up this standard are weakened. At the onset of pyrexia, in the cold stage, when the standard of mean temperature rises rapidly, the vessels of the skin contract, so as to prevent any loss of heat equivalent to the augmented production; in the healthy state, the cutaneous vessels would, of course, relax under such circumstances. When the actual temperature has reached the ideal febrile standard set up, the generation and the loss of heat become adjusted so as to maintain this morbid temperature; this is the hot stage. Lastly, as soon as the standard falls, a sweat breaks out, whereby to bring the febrile body-heat down to the normal as quickly as possible.

Now we have reached the utmost limits of our knowledge; before us lies a wide wilderness of the unknown. Yet men have not ceased to be busy with this outlying region. Ignorance and incuriosity do, indeed, make up a soft, easy, and wholesome pillow for a well-contrived head to rest upon; but it is not in man to be content with that philosophical repose. All pretences to pry further into the nature of fever have either been mere dreams of a shadow of smoke, or have fallen far short of the mark. In this way, fever may be well explained to be an increase of the calidum innatum; to be an effort of nature, or of the archæus, or of the soul; to cast out something hurtful from the body; or to be the result of irritation. But the theories of fever, which are not open to the charge of explaining the little known by the less known, do yet, for the most part, err by narrowing the meaning of the word fever down to the measure of our knowledge. The theories of fever put forth are either comprehensive enough and altogether groundless, or are founded on fact but altogether unable to explain more than a part of the subject. Among the latter class of theories, I may mention the Boerhaavian theory that fever is due to increased action of the heart and increased resistance of the vessels; Liebig's theory of an increased combustion of the structures of the body setting free more force than the natural functions require; Marey's theory of equalisation of the external and internal temperatures; C. O. Weber's theory of a poisoning of the blood by the products of the destruction of the tissues, calling forth an increased metamorphosis of tissue. All these theories include a portion, but a portion only, of the truth. They all mark a discovery in physiology. Theories which are less exclusive become, at the same time, more vague. Such are Virchow's declaration that fever is not mere elevation of temperature, but elevation of temperature due to a peculiar cause, which, to all appearances, can exist nowhere else than in the nervous system; and, next, Tschschichin's theory that fever is a morbid augmentation of the activity of the spinal centres, in consequence of an affection (weakening, paralysis) of the moderating parts of the brain, whereby a series of chemical processes rise to a height such as is never reached in natural brain-function.

But although we may admit the truth of Mayer's words, that, the human body even in the state of health being to us the riddle of the sphinx, when we pass on to consider morbid processes, we light upon a sea of question and wonder; yet I think that we can forbode something of what we want for a true theory of fever. We want to know what are the antecedents of that disturbance in the heat-regulating function of the body which commonly manifests itself as pyrexia. We want to know what are the other consequences of those antecedents; what, in fact, are the concomitants of pyrexia. And we want to know what are the necessary consequences of pyrexia and of its concomitants. When knowledge is obtained on this behalf, we shall be able to define exactly and comprehensively what we mean by fever, and not until then. For, although pyrexia is the most obvious, most important, and most constant symptom of fever, yet I think we should not deem the words

pyrexia and fever to be convertible terms. Pyrexia is simply a word to signify an elevation of the body-heat above the natural standard; but surely fever has a much fuller meaning than this. Fever-heat, as Virchow says, is not mere elevation of temperature, but elevation of temperature of peculiar origin. By external heat, by exercise, a healthy body may be raised to a heat much above the standard of health, but as soon as these non-naturals cease to operate, the body is able to reduce its temperature to the normal. This is just what the febrile body does not do; the standard is pitched higher than in health; and we no more know why a febrile person maintains his pyretic temperature than why a healthy person maintains his 99 deg.

More than this: elevation of temperature does not even constitute a constant sign of fever; that is to say, a fever-patient may be not pyretic. The cause of this sometimes seems to lie in the fact that febrile persons are more strongly influenced by circumstances than healthy persons are. We have already seen that this is true with respect to external cold. Let us consider the effect of muscular exertion.

"With the same amount of chemical combination, the more work there is produced, so much the less heat is directly generated; just as, reversely, the formation of heat is at the expense of work." (Mayer.) In fever, the muscular debility is great, and the amount of work produced proportionately small; in fact, the degree of diminution in the mechanical power of the body is commonly taken as a measure of the severity of the illness: nearly all the force set free by the febrile patient manifests itself as heat. But sometimes fever-patients are compelled to put forth much muscular effort in spite of themselves and their feelings. This is, perhaps, most often seen in the case of the poor admitted into hospitals after a long journey. And what do we often find to be the condition of fever-patients under these circumstances? Their temperature is not raised above the natural standard, and may be even depressed below it; so that several hours' rest in bed is required before the pyrexia is restored. Yet we cannot refuse to the exhausted patient the name of a fever-patient, because it happens that just at one time his temperature is not elevated. And here I may allude to the facility with which many fever-patients sweat under small exertion—a sure sign of debility; "faint and unnerved, they run into a sweat."

But muscular exertion and external cold will not always explain a depressed temperature (algidity) in patients suffering from a disease which is usually attended by pyrexia. Tubercular meningitis is a disease in point. The temperature is sometimes extremely high, especially towards the end of life, but occasionally falls very low, lower than in the algide stage of cholera. I speak of the temperature high up in the rectum. Yet we do not hesitate to class tubercular meningitis amongst febrile diseases, in spite of its being occasionally attended by prolonged algidity such as I have described. And the regulating function being at fault in pyrexia, perhaps if we knew in what manner, and by what means, this function is disturbed, we might be able to explain why fever should sometimes be accompanied, not by an elevated, but by a depressed temperature.

TRACHEOTOMY IN INFANTS.

I SEE from a short note by my friend and former teacher, Dr. Joseph Bell, in last week's JOURNAL, that he is by no means disposed to allow of the existence of any hard and fast line for the age at which tracheotomy ought to be performed. I can very well remember the case to which he refers, and I can also remember some others where successful issues were obtained for the operation in very young infants. I know that my late lamented friend, Dr. Frederick Steele, had some successful cases in very young infants, and I think Mr. Annandale had one or two also. Some years ago, in Edinburgh, where the operation of tracheotomy for croup seems to me more common than elsewhere in this country, the young surgeons seemed to vie with each other in its performance; and it was something to have had the "youngest case" of recovery. I did the operation thrice in patients under twelve months old, and in an infant aged seven months it was successful. In another, aged nine months, I believe that the mother, unmarried, deliberately destroyed the child by obstructing the tube three weeks after the operation.

My own impression is that we ought to give patients the chance of recovery that tracheotomy affords, even at these early ages. The chief objection to the operation is the extreme difficulty of its performance. I know no operation in surgery more difficult than tracheotomy in an infant that has previously undergone the usual routine of treatment for croup, complicated as it is by the smallness of the trachea, the difficulty of feeling it, and the oedema induced by the usual blistering and poulticing. The facility of the operation and the chance of success increase rapidly in proportion to the age of the patient.

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