of its special effects. Its degree of diffusibility will enable it to come in contact with nervous tissue sooner than the vegeto-alkalies, and not so soon as the anæsthetics; while in its action it holds an intermediate position between these agents. The degree of diffusibility also explains why alcohol affects the higher brain-centres first. The delicate structure of these centres, which contain comparatively little dense tissue, is soon permeated by the drug; and hence the intellect and moral nature suffer first. But I must refer the reader to the able remarks made by Mr. Herbert Spencer on this point in the appendix to the first volume of the second edition of his *Psychology*.

THE GERM-THEORY OF DISEASE APPLIED TO THE EXPLANATION OF THE PHENOMENA OF IDIOPATHIC FEVER.*

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IDIOPATHIC fever occurs in its most typical and frequent form in the common epidemic diseases of this country. These diseases are contagious, and are due to the entrance into the system, from without, of the poison which gives rise to them.

That contagion is particulate may be accepted as proved. That it is organised is equally certain. The facts of infection, and the fact that the contagium-particles are reproduced to an enormous extent within the system, leave no room for doubt on that point. Being organised, it must be animal or vegetable.

The study of the nature and mode of action of contagion is an old one. Hitherto the subject has been looked at chiefly from an etiological point of view, and much valuable knowledge has thereby been obtained regarding both the nature of contagion and the causation of disease. It seems to me that the subject may also be studied with advantage from the pathological side, and that a consideration of the results likely to follow the propagation in the system of millions of minute organisms may throw some light on the mode of production of those phenomena which are common to the contagious fevers. A careful study of the question from this point of view has led me to the conclusion, that there is not a little fallacy in the modern views of the causation of idiopathic fever, and that the phenomena which constitute that condition are not so much the result of deranged action of the tissues, as they are the necessary consequence of the propagation in the system of minute animal organisms.

An organism which is too minute for ocular examination has its place in nature defined by its action on its environment. The action of an animal organism on its environment consists in the consumption of oxygen, nitrogen, and water, and the giving off of carbonic acid. It shall now be my endeavour to show that the propagation in the system of such an organism is competent to the production of all the phenomena of idiopathic fever. The essential phenomena of this condition are:—1. Wasting of the tissues, especially the nitrogenous. 2. Increased consumption of water. 3. Premature heat. 4. Increased frequency of the cardiac action. 5. Increased frequency of respiration.

It is not my intention to criticise the views at present held as to the mode of production of these phenomena; I wish only to explain my own belief that each of them is a necessary result of the mere propagation in the system of an animal organism.

1. *Wasting of the Nitrogenous Tissues.* This must be due to one of two causes—diminished nutrition, or increased disintegration. In fever, it is believed to be due to the latter cause; and on this belief is founded the generally accepted theory of Virchow. That there is increased metamorphosis of the tissues is unquestionable (as will be explained afterwards), but that this increase is not adequate to the production of all the phenomena of fever is equally certain. The chief agency in the loss of bulk which takes place in fever I believe to be diminished nutrition, a defective supply of the materials necessary to the formation of tissue. How is this produced? The one cause to which, directly or indirectly, are to be ascribed all the phenomena of idiopathic fever, is the contagium. This we believe to be an animal organism; as such, it consumes nitrogen. Whence does it get it? Let us trace the nitrogen from its entrance into, to its exit from, the body; note the various changes through which it goes, and see at what point it would most readily be appropriated by the contagium-particles. The nitrogen is taken with the food, and first appears in the circulation in the form of what Voit calls circulating or store albumen; in this form it passes to the tissues, which, in the performance of their usual nutritive action,

lay hold of the nitrogen thus conveyed to them, and incorporate it into their substance; the nitrogen thus passes into the form of what Voit has called fixed or organ-albumen; in this form it remains so long as required, and then again passes into the circulation as circulating albumen, in which state it is conveyed to the liver and other glands, where it is transformed into urea, and, as such, is eliminated by the kidneys. Now, at what point in this changing course is the contagium most likely to lay hold of the nitrogen? Clearly at that at which it would most readily be given up to it. There are four conditions in which the nitrogen has entered into such definite combinations that it is not likely to quit them at the call of the contagium; (a) when it exists in the form of what I shall call constructive circulating albumen; (b) when it is fixed in the tissues; (c) when it is in the form of retrogressive circulating albumen; (d) and when it has been formed into urea. There are three points at which it is in a transition state, passing from one combination to another, and is in no sense fixed; (a) when passing from the circulating to the organ albumen; (b) when passing back from this form to that of retrogressive albumen; and (c) when it is being transformed into urea. It is at one of these three points, when the nitrogen is in a transition state and is free to enter into a new combination, that it is most likely to be appropriated by the contagium. At which of the three does such appropriation take place? So far as the facilities for obtaining the nitrogen are concerned, there is not much to be said in favour of any one of them; the nitrogen is equally unstable in all. Much, it seems to me, must depend on the actual condition in which the nitrogen is presented to the contagium. It is evident that the state of the nitrogen, as it passes from the constructive to the organ albumen, must be very different from that in which it passes back from the organ albumen to the circulation; and, also, from that in which it passes from the condition of retrogressive store albumen to the form of urea. In the first case, it presents itself in a form suitable for the formation of tissue; in the two others, it is unfit for such a purpose, and is destined only for excretion. The only time, then, at which the nitrogen presents itself in the system in a form at once unstable and fit for forming tissue, is when it is undergoing the transition which results in the formation of organ albumen from the constructive store albumen. Now, it needs no elaborate argument to show that the action which results in the formation of the protoplasm of the contagium-particles, bears a much closer analogy to those constructive changes which result in the formation of the tissues, than it does to the retrograde changes which accompany their disintegration and result in the formation of urea. The formation of the tissues, and the formation of the protoplasm of the contagium particles are both constructive steps; both result in the formation of *animal* matter; and it is but reasonable to suppose that the only source whence the tissues can get the nitrogen which they require in a suitable form, is also the most likely source of that which the contagium-particles require for the formation of their protoplasm. For these reasons, I believe that it is when the constructive store albumen is undergoing the changes necessary to the formation from it of the organ albumen, that the nitrogen is appropriated by the contagium-particles. Such appropriation is the main cause of the wasting of the nitrogenous tissues. They waste because they are deprived of the nitrogen necessary to their formation. But here an apparent difficulty presents itself. Observation has shown that the amount of nitrogen eliminated from the system bears a direct relation to the quantity consumed, and is little, if at all, influenced by any other agency. Now, if the tissues are deprived of nitrogen by the contagium-particles in the manner indicated, how comes it that the amount of urea eliminated is, as a rule, much above the normal standard? It happens thus. The contagium-particles pervade the whole body; they take their nitrogen from the constructive albumen at the moment at which it is being yielded up to the tissues; it is in the tissues, therefore, that their protoplasm is formed. Now, the action which leads to the formation of this is identical with that which results in the formation of the normal tissues, and produces exactly the same effect as would be produced by a corresponding increase in the normal demands of these tissues. The effect of the propagation of the contagium-particles in the tissues, therefore, is the same as that which would result from excessive tissue-action. But tissue-action consists of a retrograde as well as a constructive change; these actions go on simultaneously, and neither can be said to precede the other, any more than the passage of a saline fluid through a membrane precedes the passage of an albuminous fluid situate on the other side. The moment at which the constructive albumen is undergoing the change which normally results in the formation of organ-albumen, is that at which its nitrogen is taken up by the contagium-particles. The simultaneous action by which the worn-out organ-albumen is transformed into retrogressive is equally far advanced. The contagium-particles step in to deprive the tissues of much of what they require for the form-

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ation of organ-albumen; no such agency, however, intervenes to prevent the completion of the contemporaneous action which results in the formation of retrogressive albumen. This action, therefore, continues as usual. The constructive changes result in the formation of contagium as well as of tissue; the retrogressive result only in the formation of retrogressive store-albumen, from which the urea is formed. With the blood circulating so rapidly as it does in fever, there must, therefore, be increased formation of retrogressive albumen, and consequent increased formation of urea.

2. *Increased Consumption of Water.*—That the enormous quantity of water taken into the system during the course of fever is not retained as water is certain; that it is not eliminated is equally sure. The dry skin, the scanty urine, and the constipated bowels sufficiently show that. What, then, becomes of it? The system requires no such quantity, and is incapable of utilising it. The only abnormal agency of which we know is the contagium. Can it give rise to this excessive demand for water? It can. In virtue of the properties common to all organisms, contagium-particles require water for their growth and propagation. Their demands for this lead to the consumption of an enormous quantity in the system. They take up that which the tissues require for their own use, and continue to consume the water as soon as it enters into the circulation. It is this consumption of water by the contagium-particles which leads to the thirst, dry skin, parched tongue, scanty urine, and diminished gastric and intestinal secretions which are more or less common to all fevers.

3. *Preternatural Heat.*—For an explanation of this, the most striking and characteristic phenomenon of idiopathic fever, we turn again to the contagium. This is a minute animal organism which is reproduced to an enormous extent in the system, and which in its formation consumes oxygen, nitrogen, and water, and gives off carbonic acid in the same way as all animals do. Now, there is no reason why the changes which such consumption and elimination imply should not, in the case of the contagium-particles, be accompanied by the same evolution of heat which, we know, results from a like action in other animal structures. Admitting this (and I see no reason on which we can refuse to do so), it follows that much of the preternatural heat of idiopathic fever is due to the propagation of the contagium. There is a great increase in the amount of oxygen, nitrogen, and water consumed, and in the quantity of carbonic acid formed; and, so far as the heat-producing effect of these changes is concerned, it is quite immaterial whether they take place during the formation of the tissues and excreta, or during the formation of the protoplasm of the contagium particles. In the combination of these two causes—that is to say, in the continuance to a greater or less extent of the normal tissue-changes, and in the superaddition to these of those which necessarily accompany the growth and propagation of the contagium—we have the true and sufficient cause of the high temperature of fever.

4. *Increased Frequency of the Cardiac Action.*—The object of the heart's action is to send to the various tissues and organs of the body the blood requisite to enable each to perform its function aright. But the heart is not the sole motive power: the tissues themselves are possessed of a native force, in the exercise of which they draw towards them the nutriment which they require. We accordingly find that any local excitement to the process of nutrition produces an increased and more rapid flow of blood to the excited organ. Supposing such excitement to exist all over the body at the same time, there would necessarily result an increased rapidity of the general circulation. This is what occurs in idiopathic fever. The propagation of the contagium in the tissues leads to an increased demand for blood, just in the same way as excessive tissue-action would lead to a similar result. The wants of the tissues and of the contagium-particles are the same. By the propagation of the latter in the former, the general demand for nutriment is increased, and yet the amount supplied to the tissues is much below what they require; the oxygen, nitrogen, and water are seized hold of by the contagium-particles—and these are the very materials that the tissues call for. This action of the contagium, and the efforts of the tissues to get for themselves the nourishment which they require necessarily lead to an increased *vis a fronte*, and increased supply of that fluid whose duty it is to furnish the necessary material. The result is everywhere an increased demand for blood, and consequent acceleration of its flow through the capillaries. This, of course, leads to increased flow through the veins, and necessarily to increased frequency of the heart's action. The ultimate cause of the accelerated cardiac action is, therefore, the propagation of the contagium-particles.

5. *Increased Frequency of Respiration.*—This may partly be due to acceleration of the blood-flow; but there is another and more potent agency at work. There are two views as to the mode of production of the respiratory movements: one is, that these are due to the stimulating action of carbonic acid on the extremities of the vagus, or on the me-

dulla oblongata; the other is, that the exciting cause is deficiency of oxygen. The observations of Pflüger and others support the latter view, and tend to show that the depth and frequency of the respirations increase in proportion as the supply of oxygen is diminished, whilst they become more and more feeble as the supply of that gas is increased, until, if it rise above a certain quantity, they cease altogether. Which-ever view we adopt, whether we regard deficiency of oxygen, or excess of carbonic acid, or the combination of these two conditions, as the cause of the increased frequency of the respiratory act in idiopathic fever, it is evident that we have, in the reproduction of the contagium particles, a sufficient reason for its existence. As animal organisms, these particles consume oxygen and give off carbonic acid, thereby necessarily producing a condition of the blood which directly tends to increased force and frequency of breathing—the abnormal increase bearing a direct relation to the extent to which the contagium is produced.

Such is the theory which I venture to advance as to the causation of the essential phenomena of idiopathic fever. Did time permit, I would explain by it also the less constant symptoms which are observed in the course of febrile ailments. Meantime, I would simply give expression to my conviction that the typhoid symptoms, the head-symptoms, the convulsions, and, in short, all the symptoms that are usually attributed to the retention in the blood of excretory products, are really due to defective supply of nutrient material to the various organs, and especially to the nervous centres; they are all of anæmic rather than of uræmic origin.

A single word as to the bearing of this theory on treatment. Water *ad libitum*, fresh air, and good ventilation; as much beef tea and milk as the patient can digest; such is the treatment which clinical experience has taught to be the most successful. My theory establishes for this treatment a sound pathological basis. Once the contagium gains admission to a susceptible system, nothing will prevent its appropriating the elements necessary for its growth. Our endeavour must be to supply enough oxygen, nitrogen, and water to enable the tissues to carry on till the contagium is exhausted; therefore, water, well-ventilated rooms, and easily digested nitrogenous food, constitute the armamentarium by which the physician must combat the evil effects resulting from the propagation of the contagium in the system. "The fever must be fed," and oxygen, nitrogen, and water supplied, if possible, in such quantities that, whilst the imperative demands of the contagium-particles are met, enough may still be left to enable the tissues to carry on till the crisis comes.

THERAPEUTIC MEMORANDA.

SYPHILITIC ALOPECIA.

THAT form of syphilitic alopecia which is independent of any eruption affecting the scalp, which accompanies the so-called secondary syphilides, and which is characterised simply by an extensive loss of hair, so that the greater portion of the scalp is denuded absolutely of hair, and not the scalp only, but also the eyebrows and eyelids (of eyelashes) as well, is often a persistent affection, and in my experience is only—very tardily, indeed—remediable by general (mercurial) treatment. I refer to the condition described above, as distinguished from the syphilitic alopecia, resulting commonly (in tertiary syphilis) from the limited and "discrete" loss of hair resulting from the formation of cicatrices consequent on (tertiary) syphilitic ulceration of the scalp. This kind of alopecia, which has by some eminent French writers been assumed to be identical with *linea decalvans* (*la teigne pelade*), but which is to be distinguished from any, even the most, "diffused" forms of the latter disease by its vague want of definite limitation of margin, is, as I have found, readily (within a month or so) curable by the following topical remedies.

For the scalp: Hydrargyri iodidi rubri gr. v; attar. rosæ ℥ij; olei amygdalæ ℥x; unguenti simplicis ʒj.

For the eyebrows (where the skin is more tender), three grains of the mercurial iodide are used. The prescription is otherwise the same as before.

For the eyelids, which are more tender still, five grains of the yellow oxide of mercury, made by the recent method, are substituted for the iodide. The prescription is otherwise as above.

The French writers referred to regard secondary syphilis as merely a predisposing cause of *linea decalvans*. I, however, regard the "secondary" syphilitic alopecia as a distinct disease.

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