CHAPTER VIII.

THE NATURE OF MALARIA.

Of the nature of malaria we have no certain knowledge. Its existence is known to us only by its effects. The diseases which constitute the most common and most striking of these effects, are the various forms of intermittent and remittent fever.

These maladies prevail chiefly in marshy localities, in which there is much decomposing vegetable matter. Whatever puts a stop to this decomposition, seems to arrest the development of the miasmatic poison. Cold weather, the complete drying up of the marsh, its drainage, its submergence under water during a very wet season, seem to have the effect of checking the spread of malarial diseases. These are exactly the circumstances which would check vegetable decomposition.

Marshes situated near the sea, and liable to be occasionally inundated by it, are more malarious than those which have no admixture of salt water. The reason given for this is that the salt water kills the fresh-water plants. The marsh thus contains more decaying vegetable matter than it would contain if it were always fresh.

Malarial diseases are most common in autumn, when the vegetable matter formed during the summer begins to decay, and while the heat necessary to its decomposition still prevails.

The association of malarial disease with vegetable decay is undoubted. What is the nature of the association? and how is it to be explained?

Finding this association all but invariable, the conclusion was not unnaturally drawn that the malarial poison was a product of vegetable decomposition, and various gaseous products of such decomposition have been suggested as possibly constituting the toxic agent. It has been found, however, that not one of these produces malarial disease.

Many marshes, too, contain much decomposing vegetable matter, and present all the conditions which have been indicated as necessary

for the development of malaria, without being a source of malarial disease. Where malaria prevails, vegetable growth and decomposition are generally abundant and active. But the converse does not hold: for vegetable growth and decomposition may be abundant and active in a locality which is not malarious.

No product of vegetable decomposition has been proved to be competent to cause intermittent or remittent fever; and no causal connection has been established between vegetable decay and the occurrence of these maladies.

But there is another view of the matter—another possible explanation of the association of vegetable decay with malarial development.

We have seen that intermittent and remittent fevers are most common in autumn, when vegetable decay is most abundant. This is the reason why a causal relation has been supposed to exist between the two. But decay is a mere sequence of death; and death simply the cessation of vital activity. Autumn marks the time at which the annually recurring active growth of plants naturally comes to an end. The increased prevalence of malarial disease in autumn, is thus associated with the cessation of the growth of living, as much as with the onset of the decay of dead, vegetable matter: and it is quite possible that the association may be with the former, rather than with the latter —that the arrest of the processes which constitute vegetable activity, may have more to do with the development of malarial diseases, than has the occurrence of the changes incident to vegetable decay.

The action of salt water may also be thus explained: in killing fresh-water plants, it puts a stop to their vital activity, and to their action on their environment.

Malaria exists in the ground, emanates from it, and infects the atmosphere in its neighborhood. Of this there is ample evidence.

1. The diseases to which it gives rise are associated with certain conditions of the soil.

2. Such disturbance of the ground as cultivation implies, often leads to their occurrence.

3. The poison does not operate at more than a certain height above the sea-level.

4. People who sleep near the ground are more apt to suffer than those who are more elevated.

A poison which is in the ground, and which infects only that part of the atmosphere which is near the ground, has its seat in what may be called the vegetable tract. The chief area of malarial infection, is the area of vegetable influence. The ground in which the roots are, and the atmosphere in which the stem and leaves are, represent the chief seats of the malarial poison.

Now, supposing this poison to be something which may be taken up and absorbed by plants during their growth (and, be it gaseous, liquid, or solid, such a supposition is quite admissible), it is evident that less of the poison would be spread abroad, and be available for the production of disease, during spring and summer, when vegetation is active, than in autumn, when the cessation of growth would leave much of it free. While the cold of winter might temporarily check, or even destroy it. Thus the autumnal development of malarial fever may be casually associated with the autumnal cessation of active vegetable growth.

Evidence in support of this view is found in the purifying effect of vegetation on a malarial atmosphere, as testified to by some facts noted in connection with the natural history of intermittent and remittent fevers.

The marsh poison is carried about in air currents, and may thus give rise to malarial disease at some distance from its home. These malaria-laden breezes may be robbed of much of their deleterious properties, and rendered comparatively innocuous, by having to pass through a belt of trees. On the other hand, the cutting down of trees has been followed by the development of intermittent and remittent fevers in localities which have thus been opened up, and exposed to direct currents from a malarious district.

It is claimed, too, for some trees and plants, and that not without some show of reason, that they possess the property of removing malaria and rendering a district more healthy. The one quality common to all the trees and plants which have been so commended, is rapidity of growth, and, therefore, great activity of the nutritive processes.

We know that malaria may be taken into the system of man, either through the lungs, or, in drinking water, through the digestive organs. There is no reason why it should not be absorbed by plants, either in water through their roots, or from the atmosphere through their leaves,

or by both channels. This is probably the mode in which trees exercise their purifying effect on malarious atmospheres. The cessation of their growth in autumn is the probable reason why malarial fever is most prevalent at that season. The malarial poison may be as abundant in summer; but the active state of vegetation at that season, leads to the absorption of much of it by the leaves and roots of plants, and to its consequent destruction as a morbific agent. In other words, malaria seems to be developed during the hot season; but chiefly at the latter part of that season, when ordinary vegetation ceases to be active, does it have full scope for its disease-producing powers.

Again, malarial atmospheres are more deadly during night than during day. The literature of the subject abounds with instances in which the members of a ship's crew who went ashore in a malarial district only during the day, escaped, while those who remained ashore all night, suffered from malarial fever. This deleterious effect of night air is well known to dwellers in malarial districts, who, during the day, go freely and with impunity into places which they would not think of visiting during the night. No satisfactory explanation has been given of this. It has been supposed to be due to chilling of the body by the night air; but the night may be oppressively hot, and the result be the same as if it were cold. It has been attributed to lowering of vitality during sleep; but sleep may be indulged in during the day with impunity; and being awake at night, does not protect from danger.

Dr. Mitchell,[1] who strongly and ably advocated the view that malarial poisons were allied in nature to the fungi, thought that, like fungi, these poisons were developed only during the night. "When we observe the extraordinary tendency of fungous vegetables to develop their power only at night, we detect *another analogy between malaria and the fungi*. In vain do we search in the latter part of a day for young mushrooms. A field which at evening exhibited not a single plant, is often whitened by their little umbrellas in the morning. . . . . Supposing the minutest fungi to possess the general properties of the class to which they belong, we may readily perceive what prodigious influence must be exerted on them by the damp rich air of a swamp."

[1] On the Cryptogamous Origin of Malarious and Epidemic Fevers, by J. K. Mitchell, A.M., M.D., Professor of Practical Medicine in the Jefferson Medical College of Philadelphia. Lea and Blanchard (1849).

Another explanation of the deleterious effects of night air is, that vapors which are dissipated abroad during the day, are again condensed near the ground at night; and that such vapors are apt to retain the malarial poison in them.

Both these views may contain an element of truth. But they do not explain the purifying effect of vegetation on a malarial atmosphere. Another factor is almost certainly at work.

If the view which has been advanced as to the mode in which trees act as purifying agents on a malarial atmosphere be correct, it necessarily follows that this action must be less during night than during day; for night is the time at which leaves cease to perform their absorbent function; and at which, therefore, they cease to exercise a purifying influence on a malarial atmosphere.

The facts with which we have to deal are—

1. That malarial fevers increase in prevalence when vegetable growth ceases to be active.

2. That malarial atmospheres are most deadly at night.

3. That growing trees exercise a purifying effect on such atmospheres.

It seems to me that the view which best fits into, and explains, these facts, is that which regards the marsh poison as something which may be taken up by the leaves and roots of plants, in the ordinary performance of their nutritive function. Such a view does not exclude from consideration the operation of the other agencies to which reference has been made.

But still the question remains, What is the nature of this poison?

The old view that it was a gaseous product of vegetable decay cannot be accepted; for such products have been shown to be incompetent to cause malarial fevers. Vegetable decay is abundant in many places where malaria is unknown; and malaria abounds where there is little or no decaying vegetable matter.

The opinion most generally entertained now-a-days is that the malarial poison consists of minute organisms. "I have no hesitation," says Niemeyer, "in saying decidedly that marsh miasm—malaria—must consist of low vegetable organisms." Though the evidence is scarcely sufficient to warrant so strong a statement, there can be no doubt that this is the view which best accords with the

phenomena noted in connection with the origin and spread of malarial disease.

The usual objection to this view is that, if such were the nature of the poison, its existence could be demonstrated by the microscope. But that by no means follows. The microscope can demonstrate the existence of very minute organisms; but beyond a certain point it cannot go. And it has to be borne in mind that the limit to microscopic demonstration of such minute objects, is not so much the mechanical power of the microscope, as the visual power of our eyes. An object may be magnified 30,000 times and be visible; and yet disappear from the field of our vision when magnified 60,000 times. The object is there, but its image is so attenuated by the increased power of the microscope, that our eyes can no longer detect it. Professor Tyndal has demonstrated that the atmosphere habitually teems with particles so minute that they cannot be detected by the highest powers of the microscope, and that many of these particles are organized. With the knowledge that organized particles so minute do exist, we cannot fail to see that our inability to demonstrate that malaria is particulate and organized, is no proof that such may not be its nature.

Certain it is, that this view is the one which best explains the phenomena with which we have to deal. Adopting it, we can at once see (1) why a damp locality favors the development of malarial fever; for moisture is favorable to the development of organized life: (2) why the drying up of the soil, and the onset of cold weather lead to an opposite result; for such conditions check the growth of organisms: (3) why complete flooding of a marsh has the same effect; for such an event puts a stop to direct communication between the soil and the atmosphere. (4) This view gives also an adequate explanation of the purifying effect of growing plants and trees on a malarial atmosphere. If malaria is organized, the organisms of which it consists are so excessively minute that they have hitherto escaped individual detection; they are, therefore, probably not more than $\frac{1}{30000}$th of an inch in diameter. To the absorption of such minute particles by the roots and leaves of plants, there is no obstacle.

The researches of Lanzi and Terrigi,[1] and the more recent ones of Professor Klebs and Signor Tommasi-Crudeli,[2] made in the malarial

[1] Centralblatt, f. Med. Wiss. 1875. [2] Allg. Wien. Med. Zeit. 1879.

district of the Agro-Romano near Rome, point to the conclusion that the malarial poison is an organism which may be obtained from the soil, and may be cultivated in the bodies of animals. This organism, say the last two observers, belongs to the genus bacillus, and exists in the soil of malarial districts in the form of shining ovoid spores; to it they propose to give the name of *bacillus malariæ*. By inoculating rabbits with liquids taken directly from malarial soil, and containing this bacillus, there was produced fever, often of an intermittent type. Another result of such inoculation was enlargement of the spleen.

It is apparent that the balance of evidence and of authority favors the view that malarial poisons are minute organisms. Such being the case, we shall assume that they are so; and shall proceed to investigate the probable mode of action of such organisms on the system. For if this view of their nature be correct, the phenomena to which they give rise in the system must be such as an organism would produce; and a detailed consideration of these phenomena will tend to throw light on the true nature of the poison which causes them.

CHAPTER IX.

THE MODE OF ACTION OF MALARIA.

MALARIA may act like ordinary medicinal and poisonous agencies; it may act after the manner of a contagium, and have its action intimately connected with organic development; or its mode of action may be altogether peculiar.

To say that malaria acts like ordinary medicinal and poisonous agencies, is not to explain its mode of action; for of the manner in which many of these produce their effects, we know little or nothing. We know that opium contracts, and belladonna dilates, the pupil; that ergot stimulates the uterus; that cantharides acts on the bladder, and arsenic on the stomach and rectum. But why each of these agencies has its own special action: why belladonna never contracts the pupil; why the action of cantharides is specially on the bladder, and that of arsenic on the stomach and rectum, we do not know. Under these circumstances we cannot expect to learn much of the mode of action of malaria from a study of the effects of ordinary medicinal agencies.

Malaria differs, too, so widely from such agencies, both in its nature, and in the effects to which it gives rise, that it is likely also to differ from them in its mode of action.

That contagia are organisms, and that their morbific action is intimately associated with, and dependent on, their organic growth, are propositions which I have elsewhere[1] considered and maintained in some detail. The evidence in support of this view may be briefly summed up as follows:—

1. The effects produced in the system by a given contagium, bear no relation to the quantity administered—a small dose acting, if it act at all, as vigorously as a large one.

2. During its action the poison is largely reproduced in the system.

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[1] The Germ Theory applied to the Explanation of the Phenomena of Disease, by T. J. Maclagan, M.D. (Macmillan & Co., 1876).

3. The quantity eliminated from the system is always greatly in excess of that received into it.

4. The locality in which its action is most marked—the seat of the local lesion in the eruptive fevers, for instance,—is also that in which the poison exists in largest quantity.

5. The action of the poison ceases, while the system contains a much larger quantity of it than sufficed to cause the disease to which it gives rise.

6. The maladies produced by the contagia are communicable from the sick to the healthy.

The existence of such peculiarities, creates a broad line of demarcation between contagia and ordinary medicinal agencies.

They also separate them from malaria: for in malarial disease there is not the same evidence of organic reproduction; there is no evidence of elimination of the poison; and malarial fevers are not communicable from the sick to the healthy.

The question of the mode of action of malaria is, indeed, beset with peculiar difficulty; for (unless, possibly, in the experiments of Klebs and Tommasi-Crudeli already referred to) the poison has never been separated, and experimented with.

In the case of ordinary medicinal agencies we have the substance itself, the agent whose action we are investigating, in our hands, and under our eyes. We can administer it in any way, in any quantity, and under any circumstances. With some of the contagia, too, we can separate the poison, regulate the dose, and study the effects under various circumstances; and from what is observed in these, may be drawn inferences applicable to the whole of the contagia.

If, with all these advantages, we are still lacking in information regarding the manner in which medicinal agencies and contagia produce their effects,—if with the poisons at our disposal, and ready to our hand for experimental investigation and research, we can come to no satisfactory conclusion regarding the manner in which they act,—can it be wondered at that we should know nothing as to the mode of action of malarial poisons, which have never been separated, and have never had their existence recognized, except through the maladies to which they give rise?

If the malarial poison is an organism—and the evidence all favors the view that it is so,—it is more likely in its mode of action to resemble contagia than ordinary medicinal agencies. It is to be noted, as evidence in support of such a view, that the phenomena to which it gives rise in the system, are more analogous to those resulting from the action of contagia, than to any effects produced by ordinary poisons and medicines.

Contagia and malaria are both intangible agencies introduced into the system from without. Both are associated with bad hygienic conditions. Both have for the most striking of their effects on the system, the occurrence of idiopathic fever. In each the fever is specific in nature; in each it has a more or less distinctive course.

But still further analogies may be traced. The continued fevers to which the contagia give rise, are divided into two classes, the eruptive and the non-eruptive. The former are represented by typhus, typhoid, scarlet fever, measles, small-pox: the latter by relapsing fever.

Malarial fevers may also be divided into two similar classes, the eruptive and the non-eruptive. The latter are exemplified in intermittent and remittent fevers: the former in rheumatic fever.

The distinction between an eruptive and a non-eruptive fever is, that the latter consists simply of fever: the former of fever *plus* a local lesion. The local lesion of rheumatic fever is the joint affection.

The existence of such analogies constitutes a reasonable foundation for the view, not only that malaria is allied in nature to contagium, but that it is also likely to resemble it in its mode of action.

If it act after the manner of a contagium, and owe its effects to its organic development within the system, we shall find evidence in support of this view in a detailed consideration of the phenomena to which it gives rise. In other words, if the poisons which produce the different forms of malarial fever, are organisms whose morbific effects are due to their organic development within the system, the symptoms of malarial fever will be such as would result from the propagation of an organism in the system.

The essential characteristics of the form of fever which is universally regarded as of malarial origin, are its intermitting or irregular course, and its occurrence independently of a local inflammatory cause.

The question which we have to consider is the competence of an organism to produce such a result.

That the reproduction of an organism in the system is capable of producing the essential phenomena of the febrile state, we have, in the case of the continued fevers, shown in some detail.[1]

An organism has for its most distinctive characteristics, the power of organic reproduction and development, and a certain action on its environment. This latter is dependent on the former, and consists mainly in the consumption of nitrogen and water. But nitrogen and water are the very materials required by the tissues of the body. The propagation in the system of millions of organisms having such wants, must lead to an enormous increase in the consumption of these materials. This means simply the development of fever; for such an increase must give rise to quickening of the circulation, increased tissue waste, increased consumption of water, rise of temperature, and increased elimination of urea. This aggregate of phenomena constitutes the febrile state. The propagation in the system of such organisms as we believe malarial poisons to be, is, therefore, competent to the production of the most prominent and essential feature of the maladies to which these poisons give rise—idiopathic fever.

But though there are many analogies between continued and malarial fevers, there are also great and important differences. These, equally with the analogies, call for consideration. They are as follows:—

1. There is ample evidence that the poisons of the continued fevers are reproduced in, and given off from, the system in large quantity during the course of the maladies to which they give rise. There is not such evidence in the case of the malarial fevers.

2. The continued fevers are communicable from the sick to the healthy. Malarial fevers are not.

3. The continued fevers have a continuous, regular course, and a definite period of duration. The course of malarial fevers is intermitting and irregular, and their period of duration indefinite.

4. One attack of the continued fevers confers, as a rule, immunity from a second. There is no such immunity in malarial fever.

Let us consider each of these points separately.

[1] Maclagan, op. cit., p. 34 et seq.

1. *Contagia are reproduced in and given off from the system. Malaria is not.*

The evidence of the reproduction of the poisons of the continued fevers, is their elimination in increased quantity, and in an active form. And the evidence is sufficient. A poison cannot be received into the system in small quantity, and be given off from it in large, without having been reproduced there. But though increased elimination is a proof of reproduction, its absence is not a proof to the contrary. An organized poison might be reproduced in, and exercise its action on, the system without being eliminated in an active form. Its life history might be completed in one cycle of growth within the system, and itself come to an end in the course of the morbid action which it had set agoing. Or it might be destroyed and disintegrated in the system, and thrown off by the eliminating organs. In either case it would pass from the system in a form different from that in which it entered in.

It is certain that in malarial fevers the poison is introduced from without. It is probable that this poison is an organism. If it be so, it is capable of organic growth and reproduction; and its action is likely to have a relation to these processes.

The phenomena of these fevers are indeed such as can scarcely be explained in any other view. A short residence, even one night, in a malarial district, may give rise to a fever of some weeks' duration. If, during one night, the patient inhaled enough poison to cause so prolonged an attack of fever—if the whole of the poison requisite for the production of such an illness, existed in the system at the commencement of the attack, he surely should be killed right off by a dose competent to produce such an illness. Again, if the dose taken suffice to cause the fever, why does it not go on acting? Why are there intermissions and remissions? Why is the patient one day in a raging fever, and the next day free from it? And why, in the absence of treatment, does this alternation of pyrexia and apyrexia go on indefinitely?

In the case of rheumatic fever the same difficulty presents itself. If the whole of the poison necessary to produce an attack of a month's duration exist in the system at the commencement of the seizure, how is it that all the joints do not suffer at once? Why does the local inflammation present intermissions and remissions? Why does the inflammation quit a joint, and, after a time, come back to it? And why, in the

absence of appropriate treatment, does the disease have so prolonged a duration?

If the whole of the poison necessary to the production of all the phenomena of the malarial fevers existed in the system at the commencement of the illness, they would lose their intermittent and remittent character, the full effects of the poison would be produced at once, and their phenomena would be concentrated into one violent attack of fever, which could scarcely be recovered from.

If the whole of the poison of rheumatic fever existed in the system at the onset of an attack of that disease, the symptoms would be developed, not gradually as is the case, but all at once—the joints, instead of being affected by twos and threes, would all suffer at the same time, and the disease would lose some of its characteristic features.

It is impossible to explain the phenomena of the malarial fevers on the supposition that the whole of the poison necessary to the production and full development of each, exists in the system at the time of its onset. Their varying course, their often-increasing severity, their prolonged duration, and their small mortality, can be accounted for only on the view that their poisons are reproduced during the course of the maladies to which they give rise—that that which produces the symptoms of to-day has to-morrow ceased to be active; but has given rise to an offspring which keeps up the action which its parent set agoing; and in its turn hands down to its offspring the same morbific properties which itself received.

Such reproduction is peculiar to organized structures.

That the poisons of malarial fevers are somehow destroyed in the system there can be no reasonable doubt. They enter it from without; they are almost certainly reproduced therein; and they are not eliminated in an active form. They must, therefore, be destroyed and disintegrated. The only alternative view is that they remain permanently in the system; and that is an untenable position.

It seems to me that we must accept the view that malarial poisons are destroyed, probably in some of the eliminating organs, and that the products of their destruction are eliminated with the ordinary excreta. The presence of these products in the system may partly explain the unusually copious urinary deposits which are noted in connection with intermittent, remittent, and rheumatic fevers.

The fact that malarial poisons are not eliminated from the system in an active form, does not prove that they are not reproduced in it.

2. *Malarial fevers are not communicable from the sick to the healthy.*

Communication of a disease means the passage of its poison from the bodies of the sick to those of the healthy. It, therefore, presupposes the elimination of that poison in an active state. Malarial fevers are not communicable, because their poisons are not so eliminated. Non-communicability is at once the proof, and the necessary consequence, of non-elimination in an active state.

3. *Malarial fevers have an intermitting indefinite course, and an irregular period of duration.*

The poisons of the continued fevers and of malarial fevers, if organisms, are also parasites. As such they require for their development something more than the bare materials requisite to organic growth—something which they find in their nidus, and which is as necessary to their reproduction as are nitrogen and water to their organic growth. The materials requisite to organic growth exist all over the body, and are practically unlimited. The special material which the poisons find in their nidus (and which for convenience sake we call the second factor) is not so. It exists only in the nidus, in definite and limited quantity, and may, therefore, be readily exhausted. In typhoid fever, for instance, it is limited to the intestinal glands—in small-pox to the skin. So long as any of this second factor exists, the poison continues to be propagated, and the febrile symptoms are kept up. When it is exhausted, the system no longer possesses the necessary nidus—no longer presents to the poison the conditions requisite to its continued action—its propagation ceases, and the fever comes to an end.

The quantity of the second factor is not the same in all persons. It has an average, however; and the period requisite for using up this average, represents the mean duration of the malady. If the quantity be large, the contagium finds a rich field for its propagation and development, and the resulting attack is prolonged and severe: if small, the field is a poor one, and the resulting attack is short and mild.

The course of the continued fevers is continuous and regular, because the quantity of the second factor in the nidus does not fluctuate; and because, therefore, the contagium goes on being reproduced

steadily, continuously, and without break or intermission, until the nidus is exhausted.

Their period of duration is definite, because the second factors necessary to the propagation of their poisons, exist in limited and definite quantity, are exhausted in a given time, and when exhausted, are reproduced tardily or not at all.

In typhoid fever, for instance, the second factor necessary to the propagation of the poison of that disease, has its seat in the intestinal glands. In the absence of these glands (as in infancy, in old age, and after their destruction during an attack of typhoid) the typhoid poison has no action on the system; it is as impotent for evil as is the poison of small-pox to one who has already suffered from that disease.

At the commencement of an attack of typhoid fever, these glands contain a certain quantity of the second factor. It is evident that this quantity must be both definite and limited. It is because its quantity is definite, and not liable to vary during the course of the disease, that the fever is continuous. It is because it is limited, and is not apt to be reproduced, that the fever has a fixed period of duration.

If there existed a parasitic organism whose second factor, after having been exhausted, was readily and quickly reproduced, before the first factor, the organism, was all eliminated from the system, the fever resulting from the propagation and growth of this organism would consist, not of one continued attack, but of alternations of pyrexia and apyrexia; and as the exhaustion and reproduction of the second factor might go on indefinitely, so also might this alternation. In this way there would be developed a disease having all the characteristics of intermittent fever.

If the second factor were still more rapidly reproduced, it might never be thoroughly exhausted, its quantity might only be considerably reduced, and continue to rise and fall, and rise and fall, for some time, without being quite used up. In this way there would be produced a disease having all the characteristics of remittent fever.

If the second factor, instead of being localized, as it is in the eruptive fevers, existed in the blood, such rapid reproduction would be very likely to occur; for the blood is an everchanging fluid, containing nothing but what, when removed, is likely to be replaced.

That the second factor may exist in the blood, and may be so

quickly reproduced as to give rise to alternations of pyrexia and apyrexia, is evidenced by what we see in relapsing fever.

This is the only one of the continued fevers in which an organism has been detected in the blood. It is also the only one in which the course of the fever is not continuous, and in which there is no characteristic local lesion. With reference to the spirilla which is found in the blood, it has been proved that it exists during the pyrexia, and is absent during the apyrexia, and there can be no reasonable doubt that the spirilla is the poison whose propagation causes the disease.

"The characteristic feature of relapsing fever is the relapse. There is a febrile attack of six or seven days' duration, then a period of freedom from fever of a week's duration, then another febrile attack of shorter duration than the first, and then another period of freedom from fever, which is generally permanent; but there may be as many as four or five relapses. During each period of pyrexia the spirilla is found in the blood: during each period of apyrexia it is absent. The pyrexia we attribute to the growth and propagation of the organism—the apyrexia to its absence. If we can explain the cessation of its propagation, we shall have accounted for the distinctive course of relapsing fever.

"The absence of the spirilla during the apyrexia may be due either to some peculiarity of the contagium, or to some peculiarity of the second factor.

"If the contagium were an organism which naturally went through a series of changes involving alternate periods of activity and repose; and if the phenomena of the febrile state were the result of such changes as occurred only during the period of activity, it is evident that the propagation of such an organism in the system would give rise to a malady characterized by alternations of pyrexia and apyrexia. The spirilla might thus give rise to relapsing fever.

"But if such were the case—if each febrile attack corresponded to the advent of another period of active growth of the parasite, we should probably find some change in its external appearance, some evidence of a further development of the organism. We should probably find, too, that it was present to some extent during the apyrexia. But such is not the case. The spirilla is found only during the pyrexia, and

presents in the second, third and fourth seizures, exactly the same appearance which it presented in the first.

"Again, if such were the explanation of the distinctive course of relapsing fever, we should almost certainly find the course of the malady the same in each case. If the contagium had certain normal stages of development to go through, these stages would always be the same; and the course of the symptoms to which they gave rise would be the same also. But this is not in accordance with fact. The number of pyrexial attacks is generally two; but there may be only one, or there may be three, four, five, or even six.

"For these reasons we conclude that the cause which gives rise to the distinctive features of relapsing fever, is not to be found solely in some peculiarity of its contagium. The only other possible cause is some peculiarity of its second factor.

"The peculiarity of the second factor of relapsing fever is its general distribution in the circulating fluid. Could such a peculiarity induce the phenomenon now before us—a re-accession of fever?

"In the eruptive fevers we attribute the decline of the febrile symptoms to the exhaustion of the second factor. In relapsing fever we attribute their decline to the same cause.

"The permanent duration of convalescence in the eruptive fevers we attribute to the fact that this exhaustion is permanent—that the second factor is not reproduced. The absence of such permanency in relapsing fever we attribute to the opposite cause—the second factor *is* reproduced. The occurrence of the characteristic second seizure of relapsing fever is due to the circumstance that the second factor is reproduced in the blood before the first is thoroughly eliminated from it; its early reproduction leading to the renewed development of such germs as remain, and a consequent second pyrexial attack.

"If such be the explanation of the relapse, it is evident that cases might occur in which, either from more rapid elimination of the first factor, or from more tardy reproduction of the second, the former might be thoroughly eliminated before the latter was reproduced. The consequence would be the absence of the usual characteristic of the disease—there would be no relapse, the attack being completed in one seizure. That such cases do occur is an established fact in the history of relapsing fever. Of 2425 cases collected by Murchison, 724 had no

relapse. Of 100 consecutive cases under Murchison's own care, 4 were completed by one pyrexial attack. Of 400 recorded by Litten, 6 had no relapse.

"Again, if the second factor be so frequently and so quickly renewed, it is evident that the process might be repeated more than once, and that a third seizure might be caused in the same way as the second, and a fourth in the same way as the third; the sole requisite to the production of a pyrexial attack, being the reproduction of the second factor, prior to the complete elimination of the first. Such cases are observed in every epidemic. Of 1500 cases collected by Murchison, a second relapse, *i.e.*, a third pyrexial attack, occurred in 109, or in 1 in 14; a third relapse in 9, or in 1 out of 166; and a fourth relapse in 1 of the 1500. Of Litten's 400 cases 35½ per cent. had a second relapse, while 7 of them had three, and 3 had four relapses. The mode of production of each seizure is the same; the second factor is reproduced before the first is eliminated."[1]

From what we find occur in relapsing fever, we know that the period of pyrexia corresponds to the period of abundant propagation of the organism—the period of apyrexia to its absence.

From what we believe regarding the nature of the poisons of malarial fevers, from a consideration of the phenomena to which they give rise, and from a comparative study of what is observed in the somewhat analogous case of relapsing fever, we regard it as in the highest degree probable, first, that the poisons of intermittent and remittent fevers find the second factors necessary to their propagation in the blood; second, that this second factor is at no time very abundant; third, that it is, therefore, quickly used up during the active propagation of the poison; and fourth, that when used up it is quickly reproduced. The rapid using up and speedy reproduction of the second factors, has much to do with the production, not only of the characteristic intermissions and remissions of these fevers, but also with their equally characteristic indefinite period of duration. Their course is intermitting and irregular, because the quantity of the second factor essential to the reproduction and action of their poisons, fluctuates and varies during the course of this action; and because, therefore, the quantity of

[1] Maclagan, op. cit., page 34 et seq.

poison reproduced varies from day to day. Their period of duration is indefinite, because the tendency to the speedy reproduction of the second factor is not limited to a definite period of time.

In rheumatic fever the second factor exists, not in the blood, but in the fibrous textures of the motor apparatus.

The functional activity of these textures cannot be impaired without embarrassment, for their duties are important, and cannot be delegated; they, and they alone, can do the work which falls to them. Any change in their composition is, therefore, likely to be temporary.

In the irregular distribution of this second factor, in its tendency to speedy reproduction, and in the consequent alternations of diminution and increase in quantity, we find an adequate explanation of the characteristic irregular course and duration of rheumatic fever.

In remittent rheumatic fever, as in common remittent fever, the second factor is so speedily reproduced that there is no time for more than a partial declension of fever and pain. They remit, but do not intermit.

In the intermittent form, as in ague, the second factor is reproduced so tardily that the symptoms of one seizure have time to decline, and the temperature to become normal, before the onset of the next. Its reproduction is delayed long enough to permit of the declension of the rheumatic symptoms, but not long enough to permit of the destruction of the whole of the rheumatic poison prior to its renewal. The disease is intermittent for the same reason that it is subacute. The rheumatic constitution is not marked. There is not enough of the second factor to give rise to an acute attack; and not a sufficiently marked tendency to its speedy reproduction to make the disease remittent.

In the malarial fevers, as in relapsing fever, a time comes when the renewal of the second factor is so retarded, that the poison is all got rid of before that event takes place. The advent of this time marks the onset of convalescence.

4. *One attack of malarial fever confers no immunity from a second.*

The insusceptibility to the action of the poisons of the eruptive fevers, enjoyed by those who have once suffered from them, constitutes one of the most striking characteristics of these maladies.

As the decline of the fever is due to the exhaustion of the second

factor, so immunity from a second attack is due to its non-reproduction. If, after having been exhausted, the second factor be not reproduced, the system no longer presents the conditions requisite to the propagation of the contagium, and it does not act. That such is the explanation of the immunity from second attacks enjoyed by those who have once suffered from these diseases, is evidenced by the fact that, as a rule, no amount of exposure to their poisons, not even their direct introduction into the system, serves to produce a second seizure.

It is to be noted that this immunity is peculiar to the eruptive fevers; that is, to those forms of continued fever in which the second factor is localized in some particular organ: and that relapsing fever, the only one in which the second factor is not thus localized, is also the only one in which one attack confers no immunity from a second. In relapsing fever the second factor exists in the blood.

Now it is evident that a lasting impression may be more readily made on a formed and solid organ, than on a constantly changing fluid like the blood. An organism which finds its second factor in the former is, therefore, more likely to produce a permanent impression than one which finds it in the latter. Moreover, it is to be observed that some of the tissues which are the seat of the local lesions of the eruptive fevers, are apt to undergo permanent change in the ordinary course of nature. I would specially instance the tonsils, which are affected in scarlatina; and the intestinal glands, which are involved in typhoid fever; both of which dwindle away, and ultimately disappear, as years advance.

In intermittent and remittent fevers, the second factor also exists in the blood; and is, therefore, likely to be readily reproduced.

The same tendency to the reproduction of their second factors, which serves to account for the varying course and indefinite duration of these maladies, serves also to explain their tendency to recur again and again in the same individual. The second factor is reproduced; and with its reproduction there is a renewed susceptibility to the action of the first.

In rheumatic fever the second factor exists in tissues whose functions are so important, and so peculiar, that a permanent change is not likely to take place in them during their period of functional activity. The same natural tendency which originally led to the existence of this

second factor in these tissues, leads also to its reproduction, after it has been exhausted during a rheumatic attack.

It is generally said that one attack of malarial fever (intermittent, remittent, or rheumatic) produces a greater susceptibility to its subsequent recurrence. The truth is, that the constitutional peculiarity which led to the first, leads equally to subsequent attacks; and the later as well as the earlier attacks are merely the evidence of the existence of this peculiarity.

We have now given the reasons for our belief, first, that the rheumatic poison is malarial in nature, and therefore allied to the poisons of intermittent and remittent fever; second, that malarial poisons are organisms, and therefore allied to the contagia; third, that they act after the manner of these, and owe their morbific action to their organic development within the system. We have further endeavored to explain the differences noted between the effects produced by these two different classes of poison. In doing all this we have laid a reasonable foundation for a miasmatic theory of rheumatism.

Hitherto we have dealt with malarial fevers generally. It was necessary to the elucidation of our special subject that we should do so. Regarding intermittent and remittent fevers we have said only what was necessary, on the one hand, to explain the etiological relationship which exists between these maladies and rheumatic fever; and on the other, to exemplify the similarity which obtains between the mode of action of their poisons, and those of the continued fevers. In doing so, we have necessarily dealt with phenomena which are common to all forms of malarial fever.

But if the miasmatic theory of rheumatism be correct, and if it be the case that the poison of that disease is an organism whose morbific action is intimately associated with its organic development—we should be able to explain by it, not only the features which rheumatic fever presents in common with other forms of malarial fever, but likewise those which are peculiar to, and distinctive of, it. We should, in short, on this theory be able to explain the whole of the phenomena of the disease.

Those which have hitherto come under our notice are:—

1. The occurrence of fever.

2. The non-elimination of the poison.
3. The non-communicability of the disease.
4. Its varying course and indefinite duration.
5. The liability to repeated attacks.

These are the general phenomena of the malarial fevers, which are noted in rheumatic as in the other forms; and the explanation of which we have endeavored to give.

It remains for us to consider the special features of rheumatic fever, and to explain, on the miasmatic theory, the essential and characteristic phenomena of that disease.

We have seen that rheumatism is essentially a disease of the motor system; and that this motor system consists of two parts, a loco-motor and a vasculo-motor apparatus.

Though no pathological distinction can be drawn between rheumatism affecting the loco-motor and rheumatism affecting the vasculo-motor apparatus; and though neither can be satisfactorily considered without reference to the other; it is, nevertheless, convenient for clinical purposes that a distinction should be drawn between them, and that each should be separately dealt with. Indeed, rheumatic inflammation of the vasculo-motor apparatus is so formidable and so serious a disease, that it demands special and separate consideration.

What we have to say on the general subject of rheumatism shall, therefore, be considered under the two heads of *rheumatism of the loco-motor apparatus* and *rheumatism of the vasculo-motor apparatus.*

CHAPTER X.

RHEUMATISM OF THE LOCO-MOTOR APPARATUS.

ACUTE rheumatism we regard as a form of malarial fever. What we have now to do is to apply this miasmatic theory to the elucidation and explanation of its distinctive phenomena.

These phenomena are:—

1. The occurrence, along with the general febrile disturbance, of a local inflammatory lesion.

2. The almost entire limitation of this lesion to such parts of the motor apparatus of the body as are habitually subject to active movement and strain.

3. The tendency of the disease to attack those of a particular age.

4. Its hereditary transmission.

5. The presence in the blood of an excess of fibrine.

6. The presence in the blood of an excess of lactic aid.

7. The occurrence of profuse perspirations.

8. The shifting character of the joint affection.

These we shall consider in the order enumerated.

1. *The occurrence, along with the general febrile disturbance, of a local inflammatory lesion.*

When dealing with malarial fevers generally, we saw that the course of their febrile phenomena was such as could be explained only on the view that the poison which gave rise to them was reproduced in the system. What was then said on the general subject of malarial fever, applies also to the special subject of rheumatic fever. The special peculiarity with which we have now to deal, is the association with this febrile disturbance of a local inflammatory lesion.

This association occurs under two different circumstances: (1) The inflammation may precede the fever; or (2) the fever may precede the inflammation. In the former case, the fever is the result of the inflammation, and is said to be symptomatic; in the latter, its onset precedes the evidence of local inflammation, and it is said to be idiopathic.

An instance of the former we have in pleuritis or synovitis following cold or injury: an instance of the latter we have in the bowel lesion of typhoid fever, and in the sore throat of scarlatina.

The first evidence of the existence of acute inflammation of fibrous or serous tissue is pain. The first evidence of febrile disturbance is a feeling of cold and *malaise.*

In purely local inflammations, such as those already instanced, pain is the first symptom. Shivering, *malaise,* and general febrile disturbance may quickly follow, or may even be contemporaneous with the pain; but they do not precede it.

In the local lesions of the specific fevers the case is different. Before their existence can be determined, there is evidence of constitutional disturbance. In the one case, the local symptoms precede the constitutional: in the other, the constitutional precede the local. Which first show themselves in rheumatic fever?

There is much variety in the mode of onset of this disease. Sometimes the initial symptom is a distinct rigor, followed by general febrile disturbance, and the speedy development of the characteristic joint affection. At others, the patient complains at first only of chilliness and general *malaise,* accompanied by aching of limbs. Soon fever is decided, and the pains are localized in the joints. In milder and subacute attacks, the local joint affection is often the first and only thing complained of; but careful inquiry nearly always elicits the fact that the patient had been "out of sorts," or had "had cold" for some days before the joint affection declared itself. In whatever way the disease commences—suddenly by a distinct rigor, or so gradually that the exact time of its onset can scarcely be determined—the local inflammation is preceded by constitutional disturbance.

In rheumatic fever, then, the constitutional disturbance precedes the local inflammation of the joints, and cannot be regarded as altogether consequent on, or symptomatic of, it.

But though this is true, it is equally the case that the full development of the fever is contemporaneous with the height of the joint affection. When once the disease is fully established, the local inflammation and the constitutional disturbance go hand in hand—they rise and fall *pari passu,*—and there can be no question that the febrile disturbance, though antecedent to, is much increased by the local inflammation.

In this respect the joint inflammation of rheumatic fever resembles the local lesions of the eruptive fevers, more than it does common inflammation due to a local cause.

How is the inflammation of the fibrous and serous tissues induced? That it results from the action of the rheumatic miasm there can be no doubt. The question is, "How does the miasm act?"

It may act in one of two ways: either as a direct irritant to these tissues, causing in them the same kind of excitation that cantharides produces in the bladder, and arsenic in the stomach and rectum; or it may act after the manner of a contagium, and owe its action on the fibrous and serous tissues to its propagation in them.

We have already seen that the mode of onset, and general as well as local symptoms of acute rheumatism, are such as can be explained only on the view that its poison is reproduced in the system during the course of the disease. The gradual onset of the rheumatic inflammation, its shifting character, its uncertain course, its occasionally prolonged duration, and its gradual decline, can be explained only on the view that the poison is reproduced in the system during the course of the malady, and that a fresh supply of poison is thus being constantly brought into play. On this view, the occurrence of the local inflammatory lesion is readily explained.

If such be the mode of action of the rheumatic poison—if it owe its morbific effects to its reproduction within the system—the characteristic inflammation of the fibrous and serous tissues will bear, to rheumatic fever, the same relation that the local lesions of the eruptive fevers bear to these diseases. In other words, the seat of the rheumatic inflammation is, on this view, the nidus of the parasitic organism which produces the disease.

The action which takes place in the nidus, is that which implies the fecundation and propagation of the organism, as distinguished from its organic growth. What does such a special action imply? In the absence of any definite knowledge on the subject, it would be natural to suppose that the fecundation of an ovum or seed required a greater expenditure of force than the mere maintenance of its vitality—that a greater degree of energy was required to start those forces which the existence of life implies, than was requisite to keep them up after they had been set agoing. And we know from observation that such is the

case. The immediate consequence of the fecundation of the ovum of one of the higher animals, is an increased flow of blood to it, and increased vascular excitement of the maternal organs concerned in the process. During the period of germination and flowering of many plants, the increased action which accompanies the process may be so great as to cause a decided rise of the temperature of the part in which the action occurs. So great is this rise in some cases, especially in some of the araceæ, that the heat produced may be felt by the naked hand.

"Now, I do not for a moment mean to say that the process which takes place during the fecundation of organized poisons, is the same as that which occurs in the impregnation of ova, and in the germination of seeds. I merely say that it bears to it sufficient analogy to warrant us in founding an argument thereon. Contact with its special nidus, is as essential to the development of the germ of a parasite, as is contact with the seed of the male to the ova of the higher animals, or contact with the pollen to the pistils of plants.

"The contagia, as parasites, find in their nidus something which is essential to their fecundation, and without which they cannot be reproduced. The poison of any of the eruptive fevers may be introduced into the system, but unless it reach its second factor in its nidus, it is not propagated.

"There certainly takes place in the nidus an action which takes place nowhere else, and which must be accompanied by some evolution of force. From what is observed in the case of higher organisms, we may conclude that this evolution of force must be accompanied by increased activity in, and increased vascularity of, the part in which the action occurs. Increased vascularity is the primary and essential condition of the local lesion of each of the eruptive fevers. The part so affected we regard as the nidus in which the contagium finds its second factor; and the localized active hyperæmia which constitutes the local lesion, we regard as the necessary result of the action which accompanies the fecundation of the contagium particles. Active hyperæmia implies hyperaction. Such hyperaction as the fecundation of thousands, or even millions, of germs implies, is adequate to produce the hyperæmia, and even the marked inflammatory mischief, which constitutes the local lesion." (Maclagan, op. cit., p. 181.)

What is here said regarding the eruptive fevers, equally applies to

rheumatic fever. The poison of that disease finds its nidus in the fibrous tissues of the motor apparatus of the body. The inflammation of these tissues, which constitutes the essential and characteristic feature of rheumatic fever, is the necessary accompaniment of the hyperaction which the fecundation of an organism in them implies. The joint inflammation of rheumatic fever, thus bears to the general febrile disturbance of that disease, the same relation that the local lesions of the eruptive fevers bear to their general symptoms.

Just as in the eruptive fevers we find the distribution of this nidus vary both in situation and in extent, so also do we find similar variations in rheumatic fever. In scarlatina the skin and throat are the common seats of the second factor; but it is not always equally distributed between the two. When in excess in the skin, there is an abundant eruption and little sore throat; when in excess in the throat, there is bad sore throat and little or no eruption. So in measles, the second factor has two seats, the skin and the mucous membrane of the respiratory tract. If concentrated in the former, there will be an abundant eruption, and no chest complications; if in the latter, the eruption will be scanty, and the chest complications serious.

It is the same in rheumatic fever; the second factor may be widely distributed over all the serous and fibrous tissues of the motor apparatus; or it may exist only in those of one or two joints, say the knees and ankles. In each case the severity of the attack will be directly as the amount of the second factor. In the former, its wide distribution will lead to a correspondingly extensive local lesion; the heart and all the large joints may be inflamed, and the illness be a severe one. In the latter, the attack will be mild, and the local inflammation confined to the knees and ankles.

The heart complications of acute rheumatism bear to that disease the same relation that throat complications bear to scarlatina, and chest complications to measles. Though by no means a necessary part of the disease, they are normal to, and symptomatic of, it. The cases in which they occur are those in which the rheumatic poison finds its second factor in the vasculo-motor, as well as in the loco-motor, system. Exceptional cases there are, too, in which this second factor exists only in the vasculo-motor system; and in which, therefore, the rheumatic poison manifests its action only on the heart. Such cases are the analo-

gies of those rare instances in which the poisons of the eruptive fevers produce febrile disturbance without the usual eruption. We may have a rheumatic affection of the heart, without contemporaneous joint affection, just as we may have scarlatinal sore throat without other evidence of the disease, and just as we may have typhus sine eruptione, morbilli sine morbillis, and variola sine variolis.

2. *The local inflammation is limited almost entirely to such fibrous and serous textures as are habitually subjected to strain and active movement.*

A highly developed joint, such as suffers in rheumatism, consists of a sac of synovial membrane investing the cartilaginous ends of the bones, and reflected over the fibrous capsule and ligaments. External to this purely articular arrangement are the tendons of the muscles which move the joint, and without which the joint structures proper would be useless. Many of these tendons run in grooves or sheaths, having a lubricating surface similar to that which exists in the joint.

All the structures here enumerated are necessary to the formation and functional completeness of a freely mobile joint. But they do not all suffer when that joint is the seat of rheumatic inflammation. It is only the fibrous and serous textures—the ligaments, tendons, and the lining membranes of the joints and tendinous sheaths—on which the action of the rheumatic poison is habitually exercised.

But when we come to examine the subject more minutely, we find that the serous membranes very often escape, and that the only structures which invariably suffer, are those fibrous textures which are subject to habitual strain.

The symptoms of rheumatic inflammation of a joint are pain, swelling, and redness. Of these, pain is the only one which is invariably present. It is the earliest and most essential symptom of rheumatic disturbance of a joint; without it rheumatism cannot be said to exist. But rheumatic disturbance of a joint frequently occurs without any swelling or redness. In chronic rheumatism, and in mild attacks of the subacute form of the disease, there is seldom any swelling, and never any redness; there is, in short, nothing in the external appearance of the joint by which the existence of the ailment could be diagnosed; there exists only the subjective symptom of pain.

Pain exists in every case of rheumatism of the loco-motor appara-

tus, and is *the* essential symptom of the disease. It depends on, and is the evidence of, inflammation of the fibrous textures. Such inflammation is the primary and essential feature of rheumatism.

Swelling is frequent, but exists only in acute and subacute attacks—that is, in the severer forms of the malady. It is a result of inflammation of, and consequent effusion into, the sac of the synovial membrane. Inflammation of that membrane is not an essential feature of rheumatism. It occurs in all acute, and most subacute attacks, but not in the mild and more chronic forms of the disease. In other words, it occurs only in cases in which the inflammation of the fibrous textures is marked; but not in those in which these textures are only slightly inflamed. Cases in which the joints swell are attended with more pain—the evidence of inflammation of the fibrous textures—than those in which there is no swelling. The greater pain is not due to the synovial inflammation and effusion; for it is severe before there is evidence of the synovial membrane being involved. When we contrast the agony of acute rheumatic inflammation of a joint, with the less severe pain of a more decided synovitis resulting from injury, we get an idea of how much of the pain of the former is due to inflammation of the fibrous textures, and how little of it to inflammation of the synovial membrane.

Swelling is always secondary to pain. Even in the most acute cases, in which the swelling is greatest, and comes on soonest, pain exists for a time before the joint begins to swell. We know, from what is observed in injuries to joints, that inflammation of the synovial membrane very quickly gives rise to effusion and swelling.

Redness of the surface occurs only in cases in which the inflammation is sufficiently severe to lead to the extension of the hyperæmia to the vessels of the skin. In many cases this symptom does not exist. The joints may be very painful and considerably swollen without being red. A slighter amount of inflammation is necessary to its production in joints situated near the surface, such as the wrist and knee, than in those which are more thickly covered, such as the hip and shoulder. In subacute attacks of rheumatism involving several joints, the wrist is often the only one which shows any redness of the surface. This is no doubt due to the fact that in that joint many of the fibrous and tendinous structures are situate almost immediately under the skin. The hyperæmia which accompanies this inflammation is more likely, therefore, to extend to the surface.

What is the meaning of these facts? What their bearing on the pathology of the joint affection?

We find that the fibrous textures are inflamed in *every* case of rheumatism; but that the serous tissues suffer only in those in which the inflammation of the fibrous textures is accompanied by decided febrile disturbance, and is sufficiently marked to render the case more or less acute. We further find that in no case—not even the most acute—is inflammation of the serous textures ever primary: there is always, and in all cases, a period at the commencement of the disease in which the inflammation is confined to the fibrous textures and in which pain is the only local symptom; and only after these textures have been inflamed for at least some hours, is there evidence of inflammation of the synovial membrane.

As the inflammation always commences in the fibrous textures, and as it is only in cases in which the inflammation of these textures is more or less acute, that the synovial membrane suffers, the question naturally presents itself to our minds, whether the inflammation of the synovial lining may not result from the direct extension to that membrane of a prior inflammation of the fibrous ligaments and tendons. In other words, a careful consideration of the facts with which we have to deal, raises a grave doubt whether the rheumatic poison has any direct action on the serous tissues, and whether the inflammation which undoubtedly takes place in them may not be due to the extension of the inflammatory process to them from the contiguous fibrous textures, rather than to any direct action on them of that poison.

The evidence favors the view that such is the case, and that the fibrous structures of the joints are the primary seat of rheumatism. We know that synovial membrane is very susceptible to inflammatory action, and that such action is very liable to spread to it from contiguous structures. Injury to any one point of such a membrane is very apt to be quickly followed by a general inflammation of its whole surface. We know, too, that such inflammation is developed very rapidly, and is speedily followed by effusion into the joint.

Recognizing this peculiarity of synovial membrane, we cannot fail to see that acute inflammation of the fibrous structures over which it is reflected, and with which it is in immediate contact, is very likely to extend from them to it. The more acute the inflammation, the more lifely is this to happen.

This is quite in accordance with what is observed in the joint inflammation of rheumatism. It is in acute cases that the synovial membrane is most affected, and that the joint swelling is most marked. It is in such cases that the inflammation of the fibrous textures is most severe, and most liable to spread to surrounding textures.

We shall by-and-by see reason to believe that inflammation of the lining and investing membranes of the heart, is also secondary to prior inflammation of the subjacent fibrous textures of that organ.

3. *The action of the rheumatic poison is confined almost entirely to those of a particular age.*

The age of liability to rheumatic fever is from fifteen to fifty.

This immunity of the very young and very old, can be accounted for only in one or two ways: either the rheumatic poison does not enter their systems; or, having entered, it does not act.

The former is a position which cannot be maintained. There is no reason why an agency which may gain entrance to the system at twenty or fifty years of age, may not equally gain entrance at ten or sixty. The portals of the system are as free and as open at the one age as at the other.

The second is a position which may reasonably be maintained: for there is distinct evidence that some poisons, to which that of rheumatism bears analogy, may, under certain circumstances, be introduced into the system without producing effect. We know, for instance, that a person who has not been vaccinated, and has not had small-pox, cannot be exposed to the poison of that disease without almost certainly taking it. But we also know that, having once suffered, he may be constantly exposed, and may even have the poison directly introduced into his system, without again suffering from its action. So with each of the eruptive fevers—one attack confers, as a rule, immunity from the future action of its poison. Here we have adequate proof that a poison, and a very potent one too, may gain entrance to the system without producing any effect on it. The circumstances which give rise to this immunity we have already referred to. We now note the fact in connection with the acknowledged immunity from rheumatic fever of the young and old, and in exemplication of the view that such immunity is due, not to absence of the rheumatic poison, but to insusceptibility to its action.

We have seen that the rheumatic poison acts almost solely on those fibrous and serous tissues which enjoy a high degree of functional activity, and are subject to a great amount of movement and strain. But these tissues are not equally active at all periods of life. In infancy and early life, the child has not the physical strength and stamina necessary for active movement, and there is no strain thrown on his fibrous tissues. After the age of fourteen or fifteen, matters change. The child has now reached an age at which work and vigorous exercise begin to form part of his daily life; and when adult life is reached, hard work and active exercise are of constant occurrence. This implies vigorous and free movements of the larger joints, and increased force and activity of the heart's action. It necessitates, also, a state of preparedness for such action. At any time he may be called upon to make efforts necessitating such movement and action; and the requisite facilitating and restraining forces must be there to prevent mishap. The period of life at which efforts implying strain on the fibrous textures are made, is from fifteen to fifty, or thereabouts. This, therefore, represents the period of highest functional activity of those serous and fibrous tissues whose function it is to facilitate and restrain movement. It also represents the period of greatest liability to the action of the rheumatic poison.

The period of liability to the action of the rheumatic poison is the period of functional activity of the tissues specially involved in that disease. And in the fact that from fifteen to fifty is the period of such activity, we have the explanation of the special tendency of rheumatic fever to affect people of that age.

The same thing is noted in connection with some of the eruptive fevers. It is specially marked in the case of typhoid. The intestinal glands whose inflammation constitutes the characteristic feature of that disease, exist in infancy in but a rudimentary state. After two or three years, they begin to increase in size and functional activity, and go on increasing till adult life is reached. From that time till middle age they are prominent objects in the intestinal wall. They then begin to diminish in size and functional activity, and go on diminishing as age advances, till in old age they are practically non-existent, and have ceased to exercise any function. The liability to the action of the typhoid poison, is directly as the size and functional activity of these

glands. In infancy and old age the disease is all but unknown. The period of greatest liability to it is from fifteen to thirty-five. All this is inadequately explained on the view that these glands are the nidus in which the typhoid poison finds the second factor necessary to its propagation.

On exactly the same view is to be explained the special tendency of the rheumatic poison to affect people between the ages of fifteen and fifty. This is the period of functional activity of those tissues whose inflammation constitutes the local lesion of the disease, and which bear to the rheumatic poison the same relation that the intestinal glands bear to that of typhoid fever. These tissues form a suitable nidus for the propagation of the rheumatic poison only during their period of functional activity. Before and after this period, they do not present to that poison the conditions requisite to its development and action. Hence, before and after that period, there is insusceptibility to the action of the rheumatic poison.

4. *Hereditary transmission.*

Many diseases are said to run in families—to be transmitted from father to son. And the facts warrant the statement. A gouty parentage gives a liability to gout. The children of phthisical parents are in turn apt to die of the same disease. Rheumatism is also thus inherited.

But when we say that a man inherits a disease from his father, we do not mean to say that he comes into the world suffering from it, or with its seeds already in him. In the case of gout, he may enjoy perfect immunity from it during the greater part of his life, and begin to suffer only when forty or fifty years of age. In the case of phthisis, perfect health may be enjoyed for twenty years, and then the fatal inheritance declare itself. In the case of rheumatism, this inheritance seldom declares itself before fifteen, and is generally lost again after fifty.

The son may be born before the father has himself suffered from the malady which he is believed to have transmitted to his offspring. Or the father may even not suffer at all: he may be simply the medium of transmission to his son of a malady from which his forefathers had suffered. Evidently it is not the disease itself, but only the family tendency to it, which is transmitted. To transmit an actual disease, the father must have its poison in his system when his son is begotten; in which case the child will be born with the malady already developed.

This is well instanced in the case of syphilis. But that is a very different thing from what occurs in the case of rheumatism. What is there transmitted, is not the disease, but a tendency to it—a greater or less liability to contract it.

This tendency is generally referred to as a constitutional predisposition. But to give it a name is to indicate, not to explain, its existence. What is a constitutional predisposition to rheumatism? Wherein does it consist? And what do we mean when we say that a man has inherited rheumatism from his father?

Acute rheumatism consists in inflammation of the white fibrous and serous tissues of the motor apparatus. Its poison is a miasmatic organism, which is propagated in the system, and finds the nidus requisite to this propagation, in those tissues whose inflammation constitutes the specific lesion of the disease. For the production of acute rheumatism, therefore, two factors are necessary—first, the poison introduced from without; second, that peculiar condition of the tissues of the motor apparatus which imparts to them their special fitness to act as a nidus for this poison. Which of these factors is it that is transmitted? or is it both? One or both it must be, if we recognize transmission at all.

It is certainly not both, for their co-existence in the system at birth would give rise to the disease in the infant. As certainly it is not the first, for a miasmatic poison is essentially one which is received into the system from without, and which gives rise to a disease which is not communicable. It can only be the second: it can only be that peculiar condition of the tissues of the motor apparatus which renders them a fitting nidus for the propagation of the rheumatic poison.

The difference between a rheumatic and a non-rheumatic subject is, that the motor apparatus of the former contains that special ingredient which is requisite to the propagation and action of the rheumatic poison; while that of the latter does not. Between the fibrous tissues of the two men there is no difference that can be detected either by the anatomist or the chemist; but in the one, these tissues afford a nidus for the propagation of the rheumatic poison—in the other, they do not. In the one the tissues of the motor apparatus contain something which is wanting in those of the other. It is the presence of this something, which constitutes the peculiarity of the rheumatic constitution. It is the tendency to the development of this peculiarity, which is trans-

mitted from father to son, and makes each generation susceptible to the action of the rheumatic poison.

That such a peculiarity should be inherited, consists with all that we know of hereditary transmission. There is no reason why internal peculiarities should not be transmitted, as well as external—why a peculiar condition of the brain, of the stomach, of the liver, should not be handed down from father to son, as well as a special cast of features, a particular color of hair, or a peculiar shape of the limbs. And we know as a fact that certain peculiarities of internal organs are transmitted.

Furthermore, there is no reason why peculiarities of individual structures should not descend from generation to generation, as well as peculiarities of individual organs: indeed, peculiarity of an entire organ presupposes peculiarity of its individual parts.

It consists with all reason, that peculiarities of the motor system should be inherited, as well as peculiarities of the nervous, digestive, osseous, and other systems of the body.

That some peculiar condition of the motor system is handed down in rheumatism, we know. That this condition declares itself by a special susceptibility of the tissues of the motor apparatus to the action of the rheumatic poison, we also know.

But more than this we cannot say; for in this, as in all other cases of hereditary transmission, we can only indicate, not explain, the fact. So far as the bearing of this fact on the miasmatic theory of rheumatism is concerned, we can only say that that theory perfectly consists with it.

5. *The presence in the blood of an excess of fibrine.*

In many forms of inflammation, the blood contains an increased quantity of fibrine. In acute rheumatism this excess is specially marked.

Fibrine was at one time believed to be an important nutrient constituent of the blood. It is now known that such is not the case. There is ample evidence that it is a product of tissue waste—an excrementitious, rather than a nutrient, compound. The evidence of this is, that it accumulates during fasting, and during many ailments accompanied by increased waste. It exists also in increased quantity in the blood after fatiguing exercise; and Brown-Sequard has shown that

the more a muscle is exercised by galvanism, the more fibrine does the blood issuing from it contain. There can be no reasonable doubt that fibrine is a product of the retrograde metamorphosis of nitrogenous tissue.

We know that inflammation causes increased metamorphosis of the tissue in which it occurs. We know that rheumatism consists in inflammation of the tissues of the motor apparatus. We know that these tissues are the chief source of the fibrine of the blood. We know, therefore, that rheumatic inflammation must be accompanied by increased formation of fibrine. The presence in the blood of an excess of fibrine during the course of acute rheumatism, is thus a necessary result of the morbid action which constitutes the characteristic feature of the disease. It results from increased metamorphosis of the nitrogenous elements of muscle and fibrous tissue.

6. *The presence in the blood of an excess of lactic acid.*

This, one of the most characteristic phenomena of acute rheumatism, has already been considered.

It has been shown that lactic acid is a normal product of the retrograde metamorphosis of muscular tissue; that its formation in excess during muscular exercise results from increased wear and tear of the tissues of the motor apparatus; and that its excessive formation in acute rheumatism is due to the same cause, only differently induced. In the one case, the increased metamorphosis results from exaltation of a natural function; in the other, it is a consequence of a pathological process, viz., inflammation of the tissues of the motor apparatus, induced by the action of the rheumatic poison, and leading to excessive formation of lactic acid, in the manner already explained.

The argument may be briefly summed up as follows:—

Rheumatism is essentially a disease of the motor apparatus of the body. Acute rheumatism essentially consists in acute inflammation of the parts of that apparatus which are most subject to strain. The tissues chiefly involved are the white fibrous. But these are so intimately and inseparably connected both anatomically and physiologically with the muscles, that vascular disturbance of the one cannot fail to be participated in by the other. The inflammation of the fibrous textures which constitutes the local lesion of acute rheumatism, must therefore be accompanied by increased metamorphosis of the highly vascular

muscles which, with them, form the motor apparatus of the affected joints. As a result of the action of the rheumatic poison, we thus have increased retrograde metamorphosis of the whole motor apparatus, muscular as well as fibrous. Fibrous tissue contains only nitrogenous material; muscular tissue contains both nitrogenous and non-nitrogenous. Its non-nitrogenous material is glucose. We know that glucose is the natural source of lactic acid in health. It is almost certainly also its source in disease.

The difficulty has been to account for the increased metamorphosis of the glucose. On the view which has been advanced as to the nature of the rheumatic process, this difficulty is satisfactorily disposed of.

According to the miasmatic theory, the increased formation of lactic acid in acute rheumatism, results from increased metamorphosis of glucose, the *non-nitrogenous* element of the tissues of the motor apparatus, just as increased formation of fibrine and of urea result from increased metamorphosis of the *nitrogenous* elements of the same tissues. Each is a necessary result of the action of the rheumatic poison on the tissues of the motor apparatus.

To another possible source of lactic acid in acute rheumatism a brief reference must be made.

The rheumatic poison is introduced from without, is reproduced in the system, but is not eliminated in an active form. What, then, becomes of it? It must be destroyed in the system. In the absence of any definite knowledge of the composition of the poison, we do not know the exact nature of the changes which such an event would imply. There is, however, nothing in the nature and composition of an organism, such as we believe the rheumatic poison to be, and nothing in the nature and composition of lactic acid, to prevent us from regarding the latter as a possible product of the destructive disintegration of the former. The first decided evidence of an excess of lactic acid is its excretion by the skin. By the time that this occurs, the rheumatic poison has produced other symptoms characteristic of its action. It has been reproduced, and is undergoing disintegration as well as reproduction. Lactic acid is a possible product of such disintegration. Its presence in excess might, therefore, result from the destructive disintegration of the organism which produces the rheumatism.

Whichever view we take—whether we regard the excessive formation of lactic acid as a result of increased disintegration of muscle, as a result of the destructive disintegration of the rheumatic poison, or as a result of both processes,—it is evident that we must look upon the acid as one of the results of the action of the rheumatic poison and in no case as in itself constituting that poison.

The view that the acid is a consequence of increased metamorphosis of muscular tissue, is clearly that which most commends itself to our reason. The other is a mere supposition, to which it is right that a reference should be made, but which has little to commend itself to us. It consists with either view that the more acute the case—that is, the greater the reproduction of the rheumatic poison—the larger the quantity of lactic acid formed.

7. *The occurrence of profuse perspirations.*

Abnormally free action of the skin is a characteristic of acute rheumatism. As a rule, the more acute the case, and the more intense the joint inflammation, the more free is this action. The perspiration has an acid reaction, and continues during the whole course of the acute symptoms.

By some these sweats have been regarded as exhausting and debilitating, and, therefore, as injurious. By others they have been looked upon as salutary. When two such antagonistic opinions are held, we may safely infer that neither expresses the whole truth.

In some circumstances, profuse perspiration is both evidence of weakness, and a cause of increasing debility. In others, it is unquestionably salutary, and seems to be the means by which certain disturbances of the system are brought to an end. In rheumatic fever it cannot be said to have either effect.

There is no evidence that it causes such debility as results from the night sweats of phthisis; but those who study it in connection with these sweats, will naturally conclude that it is weakening. There is no evidence that it produces the salutary effects which are noted in connection with the critical perspirations of pneumonia, and other acute febrile ailments; but those who study it in connection with these critical sweats, will naturally conclude that it is salutary.

All cases of phthisis do not have night sweats: they are among the unfavorable symptoms of some cases.

All cases of pneumonia do not have a critical perspiration: it is one of the favorable symptoms of some cases.

But all cases of acute rheumatism have acid sweats; and have them, not at one period only, but continuously through the whole course of the fully developed disease.

There is no evidence that they exercise a favorable influence on the course of the ailment: there is no evidence that they influence it unfavorably. The acid sweats of acute rheumatism are altogether peculiar—altogether different from those noted in connection with exhausting maladies, and the crisis of acute febrile ailments. They constitute one of the ordinary characteristics of the disease—one of the common results of the action of the rheumatic poison. How are they produced?

We have seen that the blood in acute rheumatism contains an excess of lactic acid. This it is which gives to the perspiration its acid reaction and odor. As this acid has been regarded by many as the cause of the rheumatism, its excretion by the skin has naturally been looked upon as a thing to be desired; and this is the foundation for the belief that the profuse perspiration of acute rheumatism is salutary. For so, on this view of the matter, it ought to be. The probability is a hypothetical one, however, which is not supported by fact; for practically we find that the most profuse perspiration affords no relief to the pain. In fact the pain is, as a rule, most severe in cases in which the sweating is most free.

When considering the lactic acid theory we saw that one of the most common and constant effects of the internal administration of that acid, was increased action of the skin. The dry and branny skin of cases of diabetes becomes, under its influence, moist and perspiring. If such be the effect of a comparatively small quantity given by the mouth, in a disease in which there is great difficulty in getting the skin to act at all, what is likely to be the effect of its formation in large quantity in the system during the course of a malady in which there is no difficulty in getting the skin to act? Clearly profuse perspiration. That lactic acid is formed in very large quantity in the course of acute rheumatism, is evidenced by the quantity eliminated by the skin. Normally it is converted in the system into carbonic acid and water, and in that form

is thrown off by the lungs and skin. The elimination of the unchanged acid indicates that there has been formed a larger quantity that can be so converted.

There can be no reasonable doubt that the acid perspirations of acute rheumatism are due, not to an effort of nature to eliminate the rheumatic poison, but to the stimulant action on the skin of the excess of lactic acid formed during the increased metamorphosis of the tissues of the motor apparatus. These perspirations are to be regarded as neither prejudical nor beneficial; but as simply one of the necessary symptoms of the disease during whose course they occur. They occupy in its symptomatology the same position as increased elimination of urea, and bear to the local lesion of rheumatic fever the same relation. The excess of acid is the result of increased metamorphosis of the non-nitrogenous, the excess of urea the result of increased metamorphosis of the nitrogenous, elements of muscle.

8. *The shifting character of the joint affection.*

This is one of the most striking peculiarities of a rheumatic attack. In the history of all forms of the disease, acute, subacute, and chronic, it occupies a prominent place; and no theory of rheumatism can be regarded as satisfactory, which does not recognize and account for it.

It is impossible to explain it on any theory which recognizes only the existence of a poison equally distributed through the blood, acting like an ordinary medicinal or poisonous agency, and acting, therefore, equally and continuously so long as it exists in adequate quantity.

The miasmatic theory, which regards the rheumatic poison as a parasitic organism, requiring for its development and action a second factor which is localized in fibrous and serous tissues, which exists in varying amount in different parts of these tissues, and which may be exhausted and renewed over and over again, satisfactorily explains this, as all the other peculiarities of the local lesion of acute rheumatism.

The second factor may exist in larger quantity in one joint than in another. In that which contains most of it, the inflammation will be most marked and of longest duration. But the second factor may be exhausted and renewed again. Its exhaustion implies decline of inflammation, its renewal a re-accession of it. The tendency to its renewal may be so great that it may take place in the fibrous textures of

a given joint more than once during the course of a single attack of the disease. Such a joint would, therefore, suffer more than once. What applies to one joint, applies to all.

The exhaustion of the second factor in one joint, may coincide in point of time with its renewal in another. In that case the decline of the inflammation in the former, will coincide with its onset in the latter. In this we have the explanation of the apparent occurrence of metastasis. The inflammation seems to leave one joint and go to another; in reality it is a mere coincidence. The decline of the inflammation in the one, has nothing to do with its appearance in the other; by mere accident the second factor is exhausted in the one, at the same time that it is renewed in the other. And it would be wonderful indeed if such a coincidence did not sometimes occur. Occasionally the metastasis seems to be to the heart: but here, too, it is a mere coincidence; for, in its tendency to become the seat of rheumatic inflammation, the heart is in exactly the same position as a joint.

During the course of even the most continuous and prolonged attack of acute rheumatism there are noted from time to time partial remissions, and subsequent exacerbations, both of fever and of pain. The fever is not a continued one; and the joints first affected do not suffer continuously, and with equal severity, during the whole course of the attack. There are many ups and downs, and shiftings and changes, in the course of both the general disturbance and the local inflammation, before the time is reached at which both begin permanently to decline. During the course of the attack, all the large joints of the body may suffer, but seldom, if ever, all at the same time, or all in the same degree.

As already stated, such prolonged acute attacks are to be regarded as a succession of short ones, which follow each other so closely, that the one overlaps and runs into the other. There is no distinct interval to separate them from each other, but only a partial remission to mark their individual existence. This view quite explains the shifting character of the joint affection.

Another peculiarity of rheumatic inflammation may here be referred to. It tends to affect the same joints on both sides of the body. If one knee or elbow is inflamed, the probability is that the other will also suffer: if the right wrist is affected, the left is not unlikely to be involved, either at the same time, or shortly afterwards.

Of this peculiarity the miasmatic theory affords a reasonable explanation. That which determines the seat of the action of the rheumatic poison, is the presence of the second factor, the irregular and uncertain distribution of which has already been referred to. If this factor exist only in the fibrous textures of the knees and ankles, only the knees and ankles will afford a suitable nidus to the rheumatic poison, and only the knees and ankles will become inflamed during its action. So with all the other joints: they are, or they are not affected during a rheumatic attack, according as their fibrous textures are, or are not, a suitable nidus for the rheumatic poison. If the second factor, whose presence makes the fibrous textures of a joint a suitable nidus, exist in those of the right wrist, the probability is that if it exist anywhere else it will be in the left wrist: the same individual peculiarity which led to its existence in a given joint of one side, leading also to its development in the corresponding joint of the other.

Hitherto we have dealt with the action of the rheumatic poison on the *loco-motor* apparatus. We pass now to the consideration of its action on the *vasculo-motor*.

CHAPTER XI.

RHEUMATISM OF THE VASCULO-MOTOR APPARATUS.

THE vasculo-motor apparatus is made up of the following textures:—

1. The hollow muscular substance of the heart, which, by its contraction, initiates the movement of the blood.

2. Rings of white fibrous tissue, which surround and form the basis of the arterial and ariculo-ventricular openings; and to which the muscular fibres are attached.

3. Fibrinous valves whose structure is continuous with that of the rings, and whose function it is to close the various openings of the heart, and keep the circulating fluid in the proper channel.

4. A membrane which lines the interior of the heart's cavities, and is reflected over the fibrous structure of the valves; and whose smooth surface facilitates the onward flow of the blood.

5. An investing membrane which covers the heart externally, and whose peculiar formation and smooth, glistening surface facilitate the free action of that organ.

All these structures, except the endocardium, find their analogues in those which go to form a complete and perfect joint.

The function of cardiac muscle, like that of voluntary muscle, is to initiate movement.

The function of the fibrous structure of the rings and valves, like that of the fibrous tendons and ligaments, is to afford attachment to muscle, to regulate normal, and to resist abnormal, movement.

The function of the pericardium, like that of the synovial membrane of the joints, is to facilitate free movement.

The endocardium alone has no analogue in the loco-motor apparatus.

When considering the action of the rheumatic poison on that apparatus, we saw that the structures on which it acts primarily and chiefly, are those fibrous textures which are habitually the seat of strain, the ligaments and tendons.

It is the same in the vasculo-motor apparatus. The parts which

suffer most, are the fibrous rings and valves—the structures, that is, whose function is essentially one of resistance—the former in consequence of the attachment to them of the muscular fibres; the latter in consequence of the pressure of the blood column.

It is its tendency to affect the heart that imparts to rheumatism its most serious features. In the majority of fatal cases, death is attributable to some form of cardiac inflammation.

Bouillaud was the first who insisted on the essential nature of the connection between rheumatism and this inflammation. Its frequent occurrence in the course of that disease had indeed been pointed out by others before him, notably by Pringle.[1] But it is to this distinguished French physician that we must accord the credit of having first insisted on the frequency, and true nature, of the heart affection. Before his time it was looked upon as a sort of metastasis, or retrocession of the inflammation from the joints to the heart. He regarded it as "one of the elements of the disease;" and as early as 1835 advocated the view, which has since been generally accepted, that pericarditis and endocarditis are of frequent occurrence in the course of acute rheumatism, and are to be regarded as produced in the same way as the joint inflammation.

"La péricardite existe chez la moitié environ des individus affectés d'un violent rhumatisme articulaire aigu. Sous ce point de vue la péricardite n'est, en quelque sort, qu'un des éléments de la maladie dite rhumatisme articulaire aigu, laquelle, considerée d'une manière plus large et plus exacte qu'on ne l'a fait jusqu'ici, constitute une inflammation de tous les tissus séro-fibreux en général, développée sous une influence spéciale. Or, le péricarde étant de nature séro-fibreuse, comme le tissu où reside le rhumatisme articulaire proprement dit, il n'est pas étonnant que la péricardite cöincide si souvent avec ce dernier: que le rhumatisme du péricarde, en un mot, ait lieu dans les circonstances qui produisent un rhumatisme des synoviales articulaires et des tissus fibreux sur lesquels elles se déploient, lequel n'est, pour ainsi dire, qu'une péricardite articulaire.

"L'endocardite, à l'instar de la péricardite, se manifeste sous les mêmes influences que le rhumatisme articulaire aigu: et bien que cette

[1] Observations on Diseases of the Army, by Sir John Pringle. 1761.

phlegmasie puisse éclater quelquefois pendant le cours d'un grand rhumatisme articulaire aigu et d'une manière purement *métastatique*, suivant l'expression de certains pathologistes, il n'en est pas moins vrai que, le plus souvent, le tissu séro-fibreux interne du cœur se prend en même temps que celui des articulations: c'est aussi ce que nous avons vu pour le tissu séro-fibreux externe du cœur. Sous le point de vue de leur structure et de leurs fonctions, les parties du cœur qui s'enflamment par l'influence des causes productrices du rhumatisme articulaire ont la plus grande analogie avec les parties des articulations qui sont le siège de ce dernier. Les cavités du péricarde et de l'endocarde représentant, sous le rapport qui nous occupe, des espèces de cavités articulaires, il n'est pas étonnant que leurs phlegmasies cöexistent si souvent avec celles des cavités articulaires proprement dites."[1]

In all forms of rheumatism, acute, subacute, and chronic, the heart is apt to suffer. In the acute, the cardiac inflammation partakes of the generally acute character of the attack, and the symptoms are well marked from the commencement. In the subacute, the symptoms are less marked, and the immediate result of the cardiac mischief less a source of anxiety. In the chronic, its onset is so gradual that the cardiac affection seldom attracts attention until it is so far advanced as to cause serious disturbance of the heart's action, and the general symptoms of cardiac disease.

The exact proportion of cases of acute and subacute rheumatism during whose course recent cardiac inflammation occurs, is differently stated by different observers. And the nature of the question is such that discrepancies must exist.

In acute attacks, the heart is more apt to suffer than in subacute; and young patients are more liable to this complication than old ones. The acuteness of the attack, and the age of the sufferer, are thus important elements in determining whether or not cardiac complications are likely to occur. An observer, the majority of whose cases are subacute, and the average age of whose patients is thirty, will have a smaller

[1] Traité Clinique des Maladies du Cœur, 1835: and Traité Clinique du Rhumatisme Articulaire, 1840. Par J. Bouillaud, Professeur de Clinique Médicale à la Faculté de Médecine de Paris; Membre de l'Académie Royale de Médecine, etc.

percentage of cardiac complications than one who happens to have a larger number of acute cases, and the average age of whose patients is twen

Fuller and others have endeavored, in studying this question, to distinguish between acute and subacute rheumatism. But independently of the difficulty, nay the impossibility, of drawing a distinct line of demarcation between them, there is no pathological reason for doing so. Acute and subacute rheumatism are merely different degrees of severity of the same disease. That the heart is more apt to suffer in acute cases, is no doubt true; but this may be partly due to the more frequent occurrence of such cases in young people: and we sufficiently express the fact when we say that the more acute the attack, the more likely is the heart to suffer during its course.

A study of the statistics bearing on this point, leads to the conclusion that we are very near the truth when we say that, in the course of acute and subacute rheumatism, recent cardiac inflammation occurs in about thirty per cent., or in nearly one case in three. But such a general statement of the fact, is bald and misleading, without the additional statement that, as years advance, the tendency to such complications diminishes.

Here we are brought face to face with two facts which are specially prominent in the history of cardiac rheumatism:—

1. Rheumatic inflammation of the heart is most common in young people.

2. It is more apt to occur in the acute than the subacute form of the disease.

If the miasmatic theory be correct, it should afford a reasonable explanation of these, as of all the other phenomena of rheumatism.

1. *The heart is specially apt to suffer in young people.*

This is a fact which has been observed and commented on by most writers on the subject. One of the most recent of these, Dr. Peacock,[1] gives it as the result of his observations on 233 cases of acute and subacute rheumatism, that of those under twenty-one years of age, 33.3

[1] St. Thomas's Hospital Reports, Vol. VI., 1875; and Vol. X., 1879.

per cent. suffered from recent cardiac disease; while of those over forty, only 16.6 so suffered; "showing that the occurrence of cardiac complication is much more to be apprehended in young people than at more advanced ages." This accords with general experience. What is the explanation of it?

We have seen that rheumatism of the loco-motor apparatus is essentially a disease of adolescence and early manhood; and that the textures which suffer most, are the fibro-serous tissues of the large joints. The general explanation of these facts is, that the tendency of a given portion of fibrous or serous tissue to be affected by the rheumatic poison, is directly as its functional activity. Adolescence and early manhood are the periods of life at which the functional activity of the loco-motor apparatus is at its height. The large joints are the parts of that apparatus which have most work to do. Hence rheumatism is most common in young people, and in the large joints.

Applying the same reasoning to the case of the heart, we find an adequate explanation of the fact which we are now considering—that that organ is more apt to be the seat of recent rheumatic inflammation in young people, than in those of more mature years.

Muscular exertion increases the force and frequency of the heart's action. The more work the loco-motor apparatus is called upon to do, the greater is the demand for blood in its textures, and the greater the force and frequency of the heart's action.

In other words, the loco-motor and the vasculo-motor apparatus work hand in hand; functional activity of the former necessitates functional activity of the latter. In this physiological fact we have the explanation of the pathological one which we are now considering. Rheumatism of the loco-motor apparatus is most common in youth, because youth is the time at which the tissues involved in that disease enjoy the highest degree of functional activity. Rheumatism of the vasculo-motor is most common at the same age, and for the same reason. Sudden and rapid movements, involving correspondingly sudden and rapid increase in the force and frequency of the heart's action, are more frequent before than after the age of forty. Before that age the heart's structures are, therefore, subjected to greater and more frequent strain than they are called upon to bear in more mature

years. With the decreasing functional activity of advancing years, comes also diminished susceptibility to the action of the rheumatic poison.

It is a *clinical* fact that the age of susceptibility to the action of the rheumatic poison is from fifteen to fifty.

It is a *physiological* fact that the tissues on which that poison acts have a higher degree of functional activity during the earlier, than during the later, years of that period.

It is a *pathological* fact that the tendency of a given portion of tissue to be affected by the rheumatic poison is directly as its functional activity.

It follows that the textures of the heart must be more subject to rheumatic inflammation in youth than they are in more mature years. And all observation shows that they are so.

2. *Inflammation of the vasculo-motor apparatus is more common in acute than in subacute attacks of rheumatism.*

What is an acute, and what a subacute, attack?

An *acute* attack is one in which the rheumatic inflammation is both extensive and severe; affecting several joints, and affecting them smartly. In other words, it is a rheumatic attack occurring in one in whom the rheumatic constitution is very marked; in the fibrous tissues of whose motor apparatus the second factor requisite to the action of the rheumatic poison, is abundantly and widely distributed; and on whom, therefore, that poison exercises a very decided action. The more abundant and wide the distribution of the second factor, the more likely is it to exist in the vasculo-motor as well as in the loco-motor apparatus, and the more likely is the heart to be affected.

As a result of its abundance, the inflammation of the individual joints is severe: as a result of its wide distribution, many joints suffer.

We have seen that there are fifteen common seats of rheumatic inflammation—fourteen in the loco-motor, and one in the vasculo-motor system. The wider the distribution of the second factor, the larger the number of these seats likely to suffer at one time. In this respect, the heart is in the same position as a joint; so that the wider and more abundant the distribution of the second factor, the more likely is that organ to suffer.

Thus the same circumstances which make a rheumatic attack acute and severe, tend also to give rise to heart complications: and thus is explained the fact that such complications are most apt to occur in acute cases. The heart suffers in such cases for the same reason that a large number of joints do.

A *subacute* attack is one in which the rheumatic inflammation is neither extensive nor severe; affecting few joints, and affecting them in a comparatively mild manner. In other words, it is a rheumatic attack occurring in one in whom the rheumatic constitution is not marked; in the fibrous tissues of whose motor apparatus the second factor requisite to the action of the rheumatic poison, is but sparsely distributed; and on whom, therefore, that poison exercises a comparatively slight action.

The more scanty the quantity, and sparse the distribution, of the second factor, the less severe the inflammation of individual joints, the fewer the number likely to suffer, and the less the likelihood of the heart being affected.

The same circumstance which renders the attack mild, diminishes the tendency to heart complications.

Given a severe and acute rheumatic attack, during whose course ten of the fifteen common rheumatic centres are affected—the heart is more likely to be among the ten which suffer than among the five which escape: the chances are two to one against it. Given a mild and subacute attack, during whose course only five centres are affected—the heart is more likely to be among the ten which escape than among the five which suffer: the chances are two to one in its favor.

The effect of age in increasing and diminishing the danger to the heart we have already considered.

The influence of these two agencies, the age of the sufferer, and the severity of the attack, ought to be considered conjointly; for severe attacks of acute rheumatism seldom occur except in youth; and nearly, if not quite, always in youth for the first time. It is probable that the severity of the attack has, in the manner just explained, as much to do with the production of the heart affection, as has the youth of the sufferer.

The rheumatic constitution is not acquired, but natural—maybe

inherited. A man who has it, can scarcely reach the age of forty in a temperate climate, without suffering from rheumatism: and he is more likely to suffer for the first time between the ages of twenty and thirty, than between thirty and forty—and that simply because the former decade comes first.

If his constitution be a markedly rheumatic one, he will suffer severely, and the majority of his rheumatic centres will be affected. The heart is more likely to be in the majority which suffer, than in the minority which escape. That organ suffers, therefore, not so much because the man is young, as because his constitution is a markedly rheumatic one; and because exposure to the rheumatic poison is too common to permit of the likelihood of his getting beyond youth without suffering from its action.

That the rheumatic constitution is less marked after middle age, has already been seen. This involves diminished susceptibility to the action of the rheumatic poison: and this is common to the whole motor system —the heart as well as the joints.

Rheumatic inflammation of the heart is generally described as occurring under the three forms of endocarditis, pericarditis, and myocarditis, — inflammation of the lining membrane, inflammation of the investing membrane, and inflammation of the muscular substance.

That each of these different structures is frequently inflamed, there can be no doubt. And yet this classification of the various forms of cardiac inflammation is, from a pathological point of view, inaccurate and misleading. Its fault and its weakness consist in its making no mention of the fibrous structures of the heart. We shall presently see that there is good reason to regard these structures as the chief seat of the action of the rheumatic poison; and to look upon inflammation of the investing membrane as often, and inflammation of the lining membrane as always, secondary to prior inflammation of the subjacent fibrous structures.

But as this classification of the different forms of inflammation about the heart, is simple from an anatomical, and not positively erroneous from a pathological, point of view; and as it, moreover, possesses the advantages pertaining to convenience and long usage, we shall continue

to employ it, rather than try to introduce the complication of a fresh nomenclature. The neglect of the fibrous structures which forms its fault, we shall endeavor to rectify by grouping these structures with the lining membrane, and including under the term endocarditis, not only inflammation of the lining membrane proper, but also inflammation of the fibrous texture of the rings and valves.

CHAPTER XII.

ENDOCARDITIS.

By the endocardium the anatomist means the thin transparent membrane which lines the interior of all the cavities of the heart; is reflected over its valves and muscular folds; is continuous, on the left side, with the lining membranes of the aorta and of the pulmonary veins; and, on the right, with those of the pulmonary artery and systematic veins.

The pathologist must attach to the term a wider meaning, and include under it, all the structures which lie inside the heart—the fibrous rings and valves, as well as the lining membrane.

It is of importance, in connection with our present subject, that this distinction should be borne in mind; for we shall presently see that the endocardium of the anatomist, the lining membrane of the heart properly so called, is probably never the primary seat of rheumatic inflammation.

We now use the term in its wider pathological sense.

By endocarditis we mean inflammation of any or all of the non-muscular structures situated *inside* the heart—the fibrous texture of the rings and valves, as well as the lining membrane.

In the natural history of endocarditis two facts stand out prominently: first, it rarely affects the right side of the heart; second, it is confined to the region of the fibrous rings and valves. It is incumbent upon us to consider and, if possible, explain these two peculiarities.

1. *Endocarditis rarely affects the right side of the heart.*

Rheumatic endocarditis is produced in the same way as rheumatic arthritis, and is due to the operation of the same agency.

In considering the action of the rheumatic poison on the loco-motor apparatus, we saw that the parts of that apparatus which suffer most,

are those which enjoy the highest degree of functional activity, and are habitually subject to free movement and active strain—the large joints. We further saw that the particular structures in these joints which are the seat of rheumatic inflammation, are those on which the strain falls —the fibrous ligaments and tendons.

Applying this to the case of the heart, we find in it a sufficient explanation of the tendency of the rheumatic poison to affect the left, rather than the right, side of that organ. For it is with the vasculo-motor as with the loco-motor apparatus—with the heart as with the joints—the rheumatic poison acts chiefly on the fibrous textures which enjoy the highest degree of functional activity, and are most subject to strain. The valves of the heart consist mainly of white fibrous or tendinous material, similar to that which forms the ligaments of the joints and the tendons of voluntary muscles. Those of the right side have the same structure as those of the left—only they are thinner and contain less of the tendinous material.

They are thinner and weaker because they have much less work to do—much less strain to bear.

The difference between the two sides of the heart in this respect, is evidenced by a reference to the contractile force of each ventricle. The walls of the left ventricle are much stronger and thicker than those of the right, "the proportion between them in this respect being as three to one."[1] It follows from this that the left ventricle acts with three times the force of the right, and that the valve which closes the left auriculo-ventricular orifice is subject to three times the strain which is thrown upon that which closes the corresponding orifice of the right side. "The work done by the right ventricle may be set down as one-third of that of the left."[2]

The fibrous textures of the right side of the heart thus bear to those of the left, the same relation that the fibrous ligaments of the small joints bear to those of the large: they are not subject to the same strain; they have not the same degree of functional activity; and they do not suffer in the same way from the action of the rheumatic poison. The right side of the heart escapes for the same reason that the joints of the toes do.

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[1] Quain's Anatomy. [2] McKendrick's Physiology.

2. *Endocardial inflammation is limited to the fibrous rings and valves.*

Inflammation never affects the whole surface of the lining membrane of the heart. It is almost entirely limited to the part which is reflected over the valves; and when other parts suffer, the mischief is almost invariably attributable to mechanical injury, produced by the rubbing on the affected portion of the endocardial surface, of an already damaged valvular segment. Moreover, the inflammation does not affect the whole valvular surface: only one side of a segment suffers—that, namely, which comes in contact with another segment in the act of closure,—in the aortic valve, its convex surface, and in the mitral, its auricular. The damage is further limited in its early stage to the line at which the segments come into contact. This is well shown in Figs. 1 and 2.

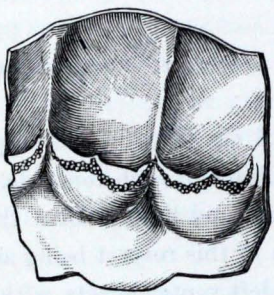

FIG. 1.

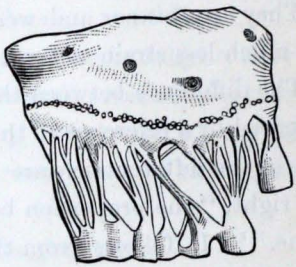

FIG. 2.

FIG. 1.—Inflammation of Aortic Valves.—The earlier stage of the process. Showing the situation of the inflammatory granulations.

FIG. 2.—Inflammation of Mitral Valve.—The earlier stage of process. Valve seen from the auricular surface. Showing the situation of the inflammatory granulations.

The limitation of the disease to this particular part of the valve, has been attributed to the fact that it is the part which is most exposed to friction. "In its earliest stages it always occurs near the edges of a valve in the formation of a line of little elevations along the contact line of its segments, where the friction is greatest."[1]

"It is those portions of the valve which come into contact in the act of closure, and are thus most exposed to friction, which are especially involved, and in which the changes usually commence."[2]

[1] Lectures on Pathological Anatomy, by Samuel Wilks, M.D., F.R.S., and Walter Moxon, M.D., F.R.C.P.

[2] An Introduction to Pathology and Morbid Anatomy, by T. Henry Green, M.D., F.R.C.P.

Of the important part played by friction in the production of inflammation of the lining membrane of the heart, there can be no doubt. Occurring in other than its valvular portion, it is almost always due to the rubbing on its surface of a damaged valve. Knowing this to be the case,—and finding that inflammation of its valvular portion commences at the line of contact of the different segments of the valves, and therefore at the point at which, if anywhere, they are apt to rub,—we cannot fail to see that there is good reason for the belief that friction plays an important part in the production of inflammation of the lining membrane of the heart's cavities.

But the question arises—Why is there friction? Why do the segments of the valves rub against each other? When a valve is already damaged or roughened, it is easy to see how further damage may be done; but in a smooth uninjured valve how is the mischief set agoing? It cannot be that the valves constantly and naturally rub against each other—for in that case, if friction produced endocarditis, no one would be free from it; rheumatic and non-rheumatic subjects would equally suffer; and a smooth healthy valve would be the exception.

For the segments of the valves to come in contact is natural; but contact does not imply friction. Healthy valves normally come into firm and close contact, without in any way rubbing against and irritating each other; and this all the valves of the heart do 70 times every minute—100,800 times every day,—and yet there is no evidence of friction or irritation, until suddenly some day—after this smooth action has gone on uninterruptedly at this rate for maybe twenty-five years, and after the segments of each set of valves have, without injury and without rubbing, come into close and direct contact more than nine hundred millions of times—suddenly some day, the rheumatic poison gains entrance to the system, and the smooth working of the valves comes to an end—they begin to rub; the friction gives rise to irritation and inflammation of the surface of the valve, and the symptoms and signs of endocarditis are developed. How is this? How can the rheumatic poison cause the segments of the valves to rub against and irritate each other? That they do rub is undoubted. That the rheumatic poison is the cause of the morbid change is equally undoubted. The question which we have to consider is how the friction is produced.

If the inflammation of the endocardial covering of the valve be the

result of friction—and there is no reason to doubt that it is so; and if this friction be a result of the action of the rheumatic poison on the valve—and there is no reason to doubt that such is the case,—it is evident that there must be a stage of the valvular lesion which precedes the inflammation of the endocardial covering. If the morbid change which takes place in that covering is produced by friction, there must be some prior change in the valve, interfering with the naturally smooth and innocuous contact of its segments, and causing them to rub against each other; there must, in short, be a stage of the endocardial change which precedes the friction.

Wherein does this stage consist?

Structurally, a valve consists of two folds of the endocardium, enclosing between them the fibrous or tendinous material which imparts to the valve its strength, its capacity to resist strain, and to perform the function which it is intended to fulfil.

If the inflammation does not commence in the former, it must begin in the latter; for there is no other structure to be affected. And there is every reason to believe that the primary seat of rheumatic endocarditis is the fibrous structure of the rings and valves, and not their endocardial covering.

We have seen that the rheumatic poison acts chiefly on those fibrous structures whose function it is to resist strain; and that the greater the strain the greater the liability to the action of that poison. In this we found the explanation of the tendency of that poison to affect the valves of the left, rather than those of the right, side of the heart.

The part of the valve whose function it is to resist strain, is not its external endocardial covering, but the fibrous structure which that encloses. The fibrous structure of the valve is, therefore, the part on which we should expect the rheumatic poison to act primarily and chiefly. And so, in fact, it is found to be.

The stage of rheumatic endocarditis which not only precedes the evidence of inflammation of the endocardial covering of the valves, but also precedes and gives rise to the valvular friction which induces that inflammation, is the stage of inflammatory thickening of their subjacent fibrous texture.

Rheumatic endocarditis consists primarily and essentially in inflammation of the fibrous texture of the interior of the heart.

Were the morbid change limited to this, the results, though serious enough, would be much less serious than they are. Unfortunately it is not so limited.

The inflammatory process which takes place in the valvular fibrous tissue, gives rise to multiplication of its cellular elements, and consequent thickening of the valve. Normally the valve is so perfectly adapted to the size of the orifice which it is intended to close, that, in the act of closing, its segments come into perfect and firm contact without any friction. They are firmly pressed against each other, but they do not rub.

But if the segments of the valve be thickened, it is evident that they must come in contact sooner than they ought. The size of the orifice to be closed being unchanged, and the force which closes it remaining the same, these thickened segments must come in contact before the closing force is expended. The continued operation of that force after the segments of the valve are in full contact, must lead to further movement of the segments. Normally their movement is completed at the moment of perfect and close contact; but here it is continued for an appreciable time after that event—with the necessary result of causing the valvular segments to rub against each other at the point of contact.

To put it otherwise. The swelling of the subjacent fibrous textures of the valves, necessarily elevates, and makes more prominent, the superficial covering of the swollen part. As the segments of the valve close, the unnaturally prominent endocardial covering of each comes into abnormally early contact with the opposing segment. The continuance of the act of closure, and therefore of valvular movement, after such contact is complete, necessarily causes these elevated surfaces to rub against each other. And thus is produced the friction which gives rise to inflammatory change in the external endocardial coat of the valve. The rubbing against each other of the abnormally elevated coats of the segments of a valve, produces friction and inflammatory change in the valvular endocardium, just as the rubbing of a valvular vegetation against the endocardial lining of one of the heart's cavities, produces friction and inflammatory change at the point at which it comes in contact with that membrane.

When rheumatic inflammation attacks the fibrous rings of the cardiac orifices, it produces there the same thickening as when it has its

seat in the valve; but thickening of the fibrous texture of the rings produces no such rubbing as takes place when the segments of the valves are thickened: there is not, therefore, the same evidence of inflammation of the lining membrane. The sole result is thickening of the fibrous ring, and consequent narrowing of the orifice—more or less stenosis—causing some obstruction to the onward flow of the blood.

The primary action of the rheumatic poison may be on the valves, on the fibrous rings, or on both. It is more commonly on the valves than on the rings, probably because the former are subject to greater strain than the latter. The most important fact for us to bear in mind is that *rheumatic endocarditis is primarily and essentially a disease of the fibrous structures of the heart.* It is, therefore, limited to that portion of the endocardial contents in which these structures exist. The small portion of the lining membrane of the heart which covers the fibrous valves, is very frequently inflamed: the much more extensive portion which lines the heart's cavities, suffers rarely, if ever. The sole difference between the two is, that the one is, and the other is not, in direct contact with the fibrous textures.

The lining membrane of the heart differs in structure, in nature, and in function, from that which invests it externally. Error and misconception have arisen from not recognizing this; and from regarding endocarditis as bearing to the endocardial lining membrane, the same relation that pericarditis bears to the pericardial investment.

There is little or no analogy, either physiological or pathological, between the two membranes. In acute rheumatism, extensive and general inflammation of the outer investing membrane of the heart is common: such inflammation never occurs in the lining membrane. Injury to it is secondary to change originating in the fibrous textures, and is limited to those parts of it which are in immediate contact with them.

The function of the endocardial lining is, by presenting a smooth surface to the liquid blood, to facilitate its onward flow. It has exactly the same part to perform as the lining membrane of the arteries, with which it is structurally continuous. Neither in the heart nor in the arteries does this membrane show the least tendency to take on inflammatory action: and when inflammation does occur in it, it shows no tendency to spread. The irritation produced by the rubbing of a valvular vegetation against the lining membrane of the ventricle, may be

so great as to cause ulceration at the point of contact; but the mischief is limited to this point, and shows no tendency to extend beyond it. There is no such disease as acute general inflammation of the endocardium. The membrane has no vessels, and inflammation cannot spread over its surface, as it does over that of the vascular pericardium.

In further exemplification of the view that rheumatic endocarditis is primarily a disease of the fibrous textures of the heart, it is to be noted that, in the aortic valves, early evidence of inflammatory mischief is found, not all over the seat of contact of the segments, but at one particular part of it. That part is the line of fibrous tissue on which the greatest strain falls.

A segment of the aortic valve consists of a duplicature of the endocardial lining membrane, enclosing within it the fibrous structure. At the centre of its free border is an elevated fibro-cartilaginous nodule—the *corpus Arantii*. Bands of fibrous tissue stretch across the valve to this nodule, from the border of valvular attachment to the aortic ring. Some of these run along its free surface. Others spread out over the body of the valve, and come to a point, as it were, at the corpus Arantii. Between the fibres of the free margin, and those of the body of the valve, there is, on each side of the nodule, a small space over which no fibres run. This space, called the *lunula*, consists simply of a duplicature of the endocardial lining. It is the thinnest part of the valve. In the act of closure, the three segments of the valve are thrown together into the middle of the aortic outlet, and the three corpora Arantii come into contact at its centre. The three lunulæ also come into contact; and are firmly pressed against each other by the aortic column of blood. They are all equally pressed down by this column; but the pressure thus exercised on each individual segment, is counterbalanced by the counter-pressure on its other side of the two remaining segments. Thus this thin portion of the valve is freed from strain; for the greater the pressure of the blood column, the more perfect the contact of the lunulæ, and the greater the counter-support which they give each other.

The part on which the strain falls, is the thicker fibrous portion of the valve which bounds the lunula below. The strain begins where the counter-pressure of the opposing segments ceases. "The force of the

reflux is sustained by the stouter and more tendinous part of the valve."[1]

This stouter and more tendinous part it is which suffers in acute rheumatism. The lunula is not affected. The granulations which are formed on the surface of the endocardial lining in the earlier stage of inflammation of that membrane, are limited to the convex surface of the line of fibrous tissue which passes from the attached border of the valve to the corpus Arantii, and which bounds the lunula below. (Fig. 1.) This band is at once the lowest part of the seat of valvular contact, and the highest part of the seat of valvular strain. Above it, except perhaps at the fibrous free edge of the valve, there is no strain: below it, there is no contact. It is the seat of strain; therefore its fibrous tissue is the seat of rheumatic inflammation. It is the lowest line of the seat of contact; therefore its endocardial covering is the seat of friction, when rendered unduly prominent by thickening of the subjacent fibrous tissue.

In the mitral valve there is no lunula; the whole structure is equally strained; and no one part is weaker than the rest. A weak point would be very liable to give way, for the strain on this valve is both greater, and more equally diffused over its surface, than is the strain on the aortic. In this latter valve there is only the blood pressure to be resisted: but in the case of the mitral there is both the blood pressure and the strain of the tightened chordæ tendineæ.

The whole valve and the fibrous ring may be the seat of inflammatory thickening: but here, as in the aortic valve, the evidence of inflammatory change in the endocardial lining membrane is limited in its early stage to the auricular surface, and to the line of contact of the valvular segments. (Fig. 2.) In other words, in the mitral as in the aortic valve, the granulations which are formed in the earlier stage of inflammation of the valvular endocardium, are formed at that line which is at once the seat of strain and the point of contact. It is the seat of strain; therefore its fibrous tissue is the seat of rheumatic inflammation. It is the point of contact; therefore its endocardial covering is the seat of friction, when rendered unduly prominent by inflammatory thickening of the subjacent fibrous tissue.

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[1] Quain, op. cit.

Microscopic examination of the affected portion of valve, confirms this view of the nature and seat of the morbid change. On making a section of one of these granular nodules, and submitting it to such examination, it is found that the seat of swelling is the deep layer of fibrous tissue, and that the superficial lining of the valve is merely raised up by the multiplication of the cellular elements of the subjacent fibrous texture. "If a valve with these nodules be cut for the microscope across the plain of its curtain, so as to show a section down through one of the small nodules, this will be found to be composed of a simple cloudy swelling of the tissue of the valve through a multiplication of the cellular elements in its fibrous structure, which here and there by its excess raises the surface into a little hillock. If the hillock takes the form of a distinct projecting grain, you will always find on the top of it a cap of fibrine separated from its substance by a line which the microscope defines very clearly. This cap of fibrine differs in composition from the hillock itself, though the difference is more easily seen than described, for the organization in both is very low; but the fibrine is almost structureless, while the hillock of swollen valve substance shows the regularly placed nuclei of fibrous tissue." [1] (Fig. 3.)

FIG. 3.—Acute Endocarditis.—A granulation from the mitral valve, showing a fibrinous coagulum upon the surface of the granulation. × 10. (Rindfleisch).

The cap of fibrine here described, is fibrine which is deposited directly from the blood on the roughened surface of the endocardium, just as it would be deposited around any foreign body. The true seat of the inflammation, is the deeper fibrous tissue of the valve.

The symptoms and signs of rheumatic endocarditis vary with the seat of the disease, and the severity of the attack. Occurring, as it does, in the course of acute rheumatism, any febrile disturbance to which it may give rise is apt to be lost in that attributable to the joint inflammation. That endocarditis slightly raises the temperature, there

[1] Wilks and Moxon, op. cit.

can be no doubt; but the invasion of fresh joints has a more marked effect in that way; and as the cardiac complication generally occurs at a stage of the illness when fresh joints are apt to suffer, it is seldom possible to say how much of the abnormal rise is due to the heart affection. Inflammation of the endocardium may cause less general disturbance than inflammation of a joint; while its local effects may be so slight as to be imperceptible to the patient.

The symptoms special to it are subjective and objective. The subjective are not commensurate with the serious nature of the ailment. They vary with the severity of the attack. Frequently they are altogether absent. In very acute cases there may be pain in the cardiac region; the heart's action may be disturbed and rapid; the breathing accelerated, oppressed, or even labored; and the patient very anxious. If both aortic and mitral orifices and valves are acutely inflamed, such are apt to be the symptoms. But in such acute cases the muscular substance of the heart is generally more or less involved in the inflammatory mischief, and it is probable that some of the symptoms are due to this, as much as to the endocardial inflammation. Moreover, the pericardium is also liable to be inflamed in such severe cases.

When the inflammation is less severe and extensive, and limited to the endocardium, there may be no more than a sense of uneasiness in the region of the heart, with little or no disturbance of the breathing, or of the cardiac action. In many cases, indeed in the majority, subjective symptoms are entirely absent; and the sole indication of the existence of the endocarditis, is the altered character of the heart's sounds—the alteration generally consisting in the development of one or more murmurs.

The absence of subjective symptoms makes it necessary that the heart should be frequently examined. At every visit this should be done. If in a case of acute rheumatism, in which we are sure that no endocardial murmur existed at our last visit, we find one developed at our next, we may be certain that it is due to a recent endocarditis.

It has been said that such murmurs may be of anæmic origin. I do not think that nowadays they ever are so. They may sometimes have been so in the old days of free bleeding; but in the absence of such treatment, it is impossible for an anæmic murmur to be developed in the short time that suffices for the production of those which we are

now considering. In acute rheumatism an anæmic murmur could scarcely be developed till late in the case; those now under consideration generally appear in the early stage of the disease. Moreover, anæmic murmurs are basic; those due to rheumatic change in the endocardium are more often mitral; and when basic, are all but invariably limited to the aorta.

The physical signs are the only certain diagnostic indications of the existence of endocarditis.

In the aortic valve we have seen that the morbid change consists in thickening of its segments, and in roughening of their convex surfaces. (Fig. 1.) During the systole of the heart these segments are thrown against the walls of the aortic outlet. Thickening of their fibrous texture must, therefore, cause a diminution of the calibre of this outlet—a diminution which is directly as the extent of the morbid change; while the deposition of fibrine on the surface of the valve, not only further diminishes the aortic outlet, but, by presenting to the blood a roughened surface, interferes with the naturally smooth and easy passage of that fluid from the heart into the great vessels. The result, and the physical evidence, of this state of matters, is the development of a systolic murmur, loudest at the base, and transmitted into the aorta.

If one or more of these segments should be so altered as to render due closure of the valve impossible, some of the blood regurgitates back into the ventricle from the aorta, and there is developed a diastolic basic murmur, transmitted downwards into the heart, and most distinct at mid-sternal region. But such change, when it occurs, is usually one of the later after effects of the valvular lesion.

In the mitral valve the morbid change consists in thickening of its structure, and in roughening of its auricular surface at the line of contact of its segments. (Fig. 2.) But these changes in the mitral valve do not produce on the cardiac circulation the same effect as similar changes in the aortic. In the latter the thickened valve is thrown against the wall of the ventricular outlet, so as to form part of its circumference; and its roughened surface is thus presented to the direct current of the blood as it passes through that outlet. In the former the valve is suspended, as it were, in the ventricular cavity, away from the

auricular outlet while the blood is flowing through it. It may have its fibrous textures thickened, and its endocardial covering roughened, without causing any change in the condition of the orifice which it is intended to close, and without producing any obstacle to the onward flow of blood, such as results from inflammatory thickening and roughening of the aortic valve.

But such morbid change in the mitral valve produces more serious disturbance of the valvular function than does a corresponding change in the aortic. For in the latter the thickening is partial; the lunulæ are not affected; and so long as they come well into contact the valve continues to perform its function.

But in the mitral valve there is no lunula; the whole segment is thickened. Here thickening means loss of mobility, loss of pliability, and consequent loss of adaptability—a condition which is exaggerated by the roughening of the surface of the valvular segments at their line of contact. The result is, that the two segments of the valve either do not act with sufficient rapidity, or do not come into close and perfect contact; a certain quantity of blood regurgitates back into the auricle; and there is developed a mitral systolic murmur.

Though the most common, this is not the only murmur which may be developed at the mitral orifice during a rheumatic attack. A præsystolic murmur, the evidence of narrowing of the mitral orifice, may also be noted with, or without, a coexistent systolic one. The narrowing of the auricular outlet which gives rise to this murmur, results from inflammatory thickening of the fibrous textures around that outlet. This thickening is produced by rheumatic inflammation of the fibrous texture of the auriculo-ventricular ring, just as thickening of the valve is produced by similar inflammation of *its* fibrous elements. Inflammation of both the fibrous rings and valves, indicates a more decided action of the rheumatic poison on the endocardium, and a more severe attack of endocarditis, than inflammation of only one of them. Cases in which there is this general affection of the fibrous textures of the endocardium, are more apt to be accompanied by disturbed and irregular action of the heart, and by increased frequency of respiration, than are those in which the valves alone suffer. But this general affection of the fibrous textures of the endocardium may exist, and all these objective indications of its existence may present themselves, without any

pain or uneasiness about the heart, or any subjective symptom directly referable to that organ.

This absence of pain in rheumatic inflammation of the fibrous textures of the heart, is one of the chief points of distinction between it and similar inflammation of the fibrous textures of the joints.

Another distinction, and one of much more serious import, is that the results of rheumatic inflammation of the fibrous textures of a joint, are generally transient, and perfectly recovered from; while those of like inflammation in the fibrous textures of the heart, are apt to be permanent, and never recovered from. Why is this? The poison which produces the inflammation is the same in both. The tissue on which it acts has in each the same structure, and a similar function. Why, then, should the results be so different?

The temporary character of the injury to the loco-motor fibrous textures, has been ascribed to the absorbent effects of the pressure exercised on the effused products by the surrounding solid structures. The permanence of the damage to the vasculo-motor fibrous textures has been ascribed to the absence of such pressure. "The permanence of the injury in the case of endocarditis is simply due to the want of counter-pressure. In the joints the swollen membranes are pressed against the other solid structures as soon as the liquid effusion is removed. This pressure causes absorption of all the new products, whereas in the heart there is no direct pressure of solids against the inflamed valves, which stand freely in fluid blood, so that the new products persist."[1]

It seems to me that a more probable and adequate explanation of this unfortunate difference, is to be found in the fact that when the fibrous textures of a joint are inflamed, they get perfect rest, and are thus placed in circumstances favorable to complete recovery: while the fibrous textures of the heart not only get no rest, but, from the greater frequency of the heart's action, are called upon to do an increased amount of work. They are thus placed in circumstances which make complete recovery all but impossible.

Acute cases of rheumatism, in which the fibrous textures of the joints are smartly inflamed; in which there is a considerable amount of

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[1] Wilks and Moxon, op. cit.

thickening and effusion; and in which complete rest is given to the inflamed textures, are also those in which these textures are most fully and speedily restored to their natural state.

In chronic cases in which the inflammation is slight, in which the patient continues to go about, and in which, therefore, the inflamed ligaments and tendons do not get rest, these textures are more apt to be permanently thickened.

If pressure were the agency which removed the effusion, it ought to disappear more speedily in these chronic cases than in the acute; for the act of locomotion supplies this factor, and would lead to speedy absorption of the effused products, and early restoration of the fibrous textures to their normal state.

The long duration of the thickening and stiffness of the joints in such cases, is to be explained in the same way as the persistent nature of the cardiac damage. Absence of rest leads to imperfect recovery and permanent injury.

It is extremely doubtful if a heart whose fibrous textures have once been thickened by inflammation, and whose endocardial lining has once been roughened, ever recovers its normal condition. For the fibrous textures continue to be strained, and the roughened surfaces of the segments go on rubbing. Irritation is thus kept up after the primary inflammation has disappeared; the thickening of the fibrous textures becomes chronic and permanent; and fresh rheumatic attacks add to the mischief.

With the advance of time the morbid change becomes more marked: the valvular segments become contracted and misshapen; the cardiac circulation is more disturbed; the muscular walls of the heart hypertrophy; its cavities dilate; and the sufferer enters on a prolonged course of misery, whose only termination is death.