SOME CONSIDERATIONS ON THE DISEASE RHEUMATISM

by

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SOME CONSIDERATIONS OF THE DISEASE RHEUMATISM.

The term Rheumatism is one of great antiquity, going as far back as the time of Hippocrates; and, as the name indicates, it was employed by the early authors to denote a flowing, or catarrh.

From these early times, down to the present time, the term has been used to denote many and various conditions; indeed, there is probably no word in the whole of medical nomenclature that has been so freely used and abused. The name of rheumatism, like that of gout, carries with it the impress of humoral pathology.

The words rheuma and catarrh are used by the Greek writers with similar meaning; and their etymology is also alike, for the one term was derived from ῥέω, and the other from κατάῤῥέω. The notion was that of an acrid humour generated in the brain and distributed over the body. In course of time, diseases of the mucous membranes became known as catarrhs, while the name of rheumatism was confined/

fined to painful affections of the joints, bones, or muscles.

Prior to the seventeenth Century, gout, rheumatism, and all forms of articular disease, were not differentiated, but included under the name arthritis; they were different forms or manifestations of the same disease.

In the seventeenth Century, Baillon, or Ballonius is said not only to have distinguished catarrhs from rheumatism, but also to have differentiated gout from rheumatism. Gout and Rheumatism were, neverthless, considered to possess a very close relationship.

Until within comparatively recent times, the relationship of gout and rheumatism was generally accepted. The supposed constitutionalism, the arthritic diatheses, the metastases of the one and the visceral lesions of the other, were the grounds for believing in this relationship. There are many at the present time who believe in a relationship. It is not a rare thing to hear such expressions as "gouty rheumatism", or "rheumatic gout"; although, of course, such expressions may be the outcome of difficulty in diagnosis.

The association of rheumatism with gout by no means/

means exhausts its connections; as time went on, it acquired others. The arthritis, as seen in a case of rheumatic fever, has been and is, no doubt, regarded as the chief manifestation of rheumatism. Therefore, if arthritis should unaccountably develop in diseases in which it, as a rule, has no part, and is not symptomatic of, the services of the term "rheumatism" are called in, and we get such conditions as Scarlatinal Rheumatism and Gonorrhoeal rheumatism. Then, again, "cold" has long been regarded as the chief causative factor in the production of rheumatism. So, in affections in which "cold" to all intents and purposes has been responsible for the condition, the services of the term are again requisitioned, and we get such expressions as rheumatic iritis, muscular rheumatism, rheumatic facial paralysis.

It may be further stated, so universally has this arthritic element of rheumatism been recognised, so universally has this "cold" cause of rheumatism been accepted, that any ache, pain, or swelling, which is clothed in sufficient vagueness and obscurity, is regarded as of rheumatic origin. As a result of this, it is doubtful if there is any affection in which the lay public make their own diagnosis/

nosis - not to mention their own treatment - so readily, generally, and confidently, as in the one they call "rheumatism". That the field of rheumatism has proved to be one of the richest for the charlatan and quack need occasion no surprise.

Of course, one is not unmindful of the progress made with regard to our knowledge of rheumatism proper; how the arthritis of rheumatism was differentiated from the arthritis of gout; how the shifting character of the joint affection was observed; the non-suppurative character of the inflammation; the copious sweats; how heart affections were associated with the rheumatic process, the chorea; the subcutaneous nodules; the varieties of erythema; in short, until we arrive at our present state of knowledge of the subject.

The point I am desirous of bringing out is, that during that progress of knowledge, the career of the disease rheumatism has been characterised by a mixture of the real and spurious, characterised by so much that was non-rheumatic being bound up with what was genuinely rheumatic; characterised by having such a wide, scattered, ill-defined province. It is only true to say that this mixture of true and false rheumatism, - this wide scattered, and ill-defined province of the disease, - obtains largely at the present time.

The conclusion this state of affairs inevitably leads to is, the disease rheumatism has not yet found its proper place in the domain of scientific medicine. Why has rheumatism not found its proper place? The explanation is, lack of knowledge as to the pathology and etiology of the disease.

It is a fact that, in the main, all our knowledge of rheumatism has been derived from clinical study; and, while that clinical study has ever advanced our knowledge of the disease, there has always been lacking, that pathological and etiological knowledge which is essential to place the disease on a sound and scientific basis.

As regards the pathology, it must be owned that we are - at all events, until quite recently, were - in complete ignorance as to the morbid process which is responsible for the disease rheumatism.

Failing any actual knowledge of the pathology of rheumatism, observers and investigators have been restricted to forming theories based on whatever clinical facts might be known at the time.

From time to time, various theories as to the pathology of rheumatism have been formed, one theory giving place to another as fresh facts and phenom-

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ena were discovered and had to be explained; and, by a sort of process of exclusion, that theory which was the most comprehensive in accounting for the phenomena of the disease was the one that found most favour.

The chief theories, dealing with the pathology of rheumatism, that need be mentioned are:-

(a) The nervous,

(b) The metabolic, including

1. The chemical or lactic acid.
2. The neuro-chemical.

(c) The infecive theory.

Excepting the infective theory, it may be stated that these theories were formed when rheumatic fever was regarded as representing typically and essentially the phenomena of rheumatism; and although heart affections, chorea, etc., may have been suspected to be in some way associated with the rheumatic process, they did not there occupy the place they now do, viz., as being manifestations of rheumatism.

Hence, these theories only attempt to account for the phenomena of rheumatic fever - the arthritis, perspirations, etc., - and do not attempt to explain the occurrence of heart affections, chorea, subcutaneous nodules, and other manifestations. Allowing/

ing, however, the explanation of these latter occurrences to pass, the theories have been found insufficient in accounting for the phenomena of rheumatism then known.

The Nervous theory is based on inductive reasoning, viz., special lesions having caused arthritis, therefore rheumatic arthritis is brought about through the nervous system.

Stated shortly, a chill, or exposure, acting on and irritating a considerable cutaneous surface, afferent impulses were transmitted to the medulla, oblongata or spinal cord and there acted on the trophic centres for the joints; as a result, efferent impulses were transmitted from these trophic centres to the joints, setting up arthritis.

Supporters of this theory endeavoured to explain some of the other symptoms, e.g., the proximity of the sweat centre to the trophic centres accounted for the excessive sweating.

Without going into this theory further, it is difficult to see how it can explain the shifting character of the arthritis; or, how a more or less evanescent peripheral irritation can disturb trophic centres so as to set up arthritis lasting for five or six weeks, and to do so without irreparably damaging/

damaging the joints involved. Etiologically, the "cold", or chill, is inadmissible.

The Metabolic theory, likewise, has chill, or exposure, as the causative factor with, as in the lactic acid theory, a chemical poison as the materies morbi; and, as in the neuro-chemical theory, a chemical poison acting through the nervous system as the materies morbi. The metabolic theory is one which has met with a great deal of acceptance and, possibly, even now possesses many supporters.

This theory may be said to be the outcome of the long association of rheumatism with gout, the completing of the analogy between the two diseases. Gout is a constitutional disease, arthritis is a prominent feature of the disease, heredity plays an important part in the etiology of gout. Gout is characterised by "metastases". Dietetic errors lead to faulty metabolism causing an excess of uric acid in the blood. The uric acid is regarded as the materies morbi of gout.

Rheumatism is a constitutional disease. Arthritis is a prominent feature. Heredity plays an important part in the etiology of rheumatism. The visceral lesions correspond, - indeed, they were called so by the older authors - to the metastases of gout; then, to complete the analogy, exposure and chill lead to faulty metabolism causing an excess of lactic/

lactic acid in the blood. The lactic acid is regarded as the materies morbi of rheumatism.

And so we get the lactic acid theory. Exposure to cold, or chill, acting on a considerable cutaneous surface, causes contraction of the superficial blood vessels and thereby prevents the elimination of the poison - which is the result of muscle metabolism - by the skin, and causes an accumulation of it in the blood. The poison is lactic acid. Of course, after any excessive muscular action, a chill would cause a greater accumulation. The lactic acid accounts for the acidity of the sweat. The excessive sweating is considered to be brought about by Nature endeavouring to eliminate the poison.

Apart from the fact that this theory does not account for the manifestations of the disease already mentioned, it is in itself unsatisfactory. It has not been proved that there is excess of lactic acid in the blood, while the disease rheumatism is in progress. Again, if a chill leads to faulty metabolism thereby causing an excess of lactic acid, and the skin and kidneys are as fast as possible eliminating the lactic acid, by what process is this excess of lactic acid maintained over a period of several weeks? As in the case of the nervous/

nervous theory, the shifting character of the arthritis has to be explained.

In the neuro-chemical theory - which is a development of the lactic acid theory - Latham attempts to explain the shifting character of the arthritis. The theory, however, as enunciated by him, is so elaborate and hypothetical, so much is pre-supposed, e.g., the excess of lactic acid and uric acid, that it cannot be accepted.

The cases recorded by Foster, in which symptoms resembling those found in rheumatism were produced by the administration of lactic acid, greatly strengthened the lactic acid theory. With some people, they put the question beyond all doubt. It cannot be wondered at that the dramatic unexpectedness of these cases had a somewhat dazzling effect and seemed to say the last word on the pathology of rheumatism. It was on reflection, however, bound to be confessed, because the administration of lactic acid produced certain symptoms resembling those found in rheumatic fever, therefore lactic acid was the chemical poison of the disease, was reasoning of far too facile and loose an order to be conclusive. If the administration of strychnine causes symptoms resekbling those found in tetanus, it does not/

not follow that strychnine is the materies morbi of tetanus.

Having dealt briefly with these two theories, the nervous and the metabolic, and seeing that they are objectionable, not only in so far as they do not satisfactorily account for the phenomena of rheumatic fever, but also in that they cannot in any way be made to explain the manifestations which are now known to form part of the disease rheumatism, it is clear that these theories do not assist in the solution of the pathology of the disease. They must, therefore, be abandoned as inadequate.

Without considering, at this stage, the infective theory, it would be well perhaps to review the position held by rheumatism at the time these two theories were in favour.

As has been already stated, rheumatic fever, with its polyarticular arthritis, sour-smelling sweats, fever, etc., - was looked upon as representing essentially the rheumatic process. The arthritis, of course, was the main feature of the disease.

The heart affections and any visceral lesions that arose were regarded as complications. The disease was described as a "Constitutional" one. A close/

close parallel was drawn between it and gout.

Exposures to cold, wet, etc., was regarded as the chief etiological factor. Heredity, also, was believed to play an important part etiologically. When the symptoms of rheumatic fever were not characterised by much severity, the disease was described as "Subacute".

Then there was the great field covered by what was termed Chronic Rheumatism. All the joint troubles that followed genuine attacks of rheumatic fever were included in chronic rheumatism. So important a feature of the disease was the arthritis considered to be, that practically all joint pains - with or without swelling - were regarded as forms of Chronic Rheumatism. As illustrating the prominence of the joint affections, some text-books on medicine classify the disease as one belonging to the locomotor system.

So assuredly was "cold" considered to be the chief etiological factor of rheumatism, that all pains - more or less obscure - which could be attributed to exposure, etc., were put down as rheumatic. Thus, we get that branch of chronic rheumatism called muscular rheumatism, pleurodynia, torticollis, lumbago, etc.

Then/

Then, the influence of heredity - from the etiological standpoint - must not be overlooked. If a person's parents were known to have had rheumatism, any pains that that person might have - which might not necessarily be attributed to "cold" and could not be accounted for - were put down as rheumatic.

So commonly, loosely, and freely was the expression rheumatism, or rheumatic, used, that many medical men, when examining hearts, for instance, and inquiring into the history of the cases, were satisfied with the patients' own statements as to whether or not they had had rheumatism; and, there is no doubt that many, if they could obtain any account of obscure arthritic pains, were satisfied as to the history of rheumatism. In this connection, I might instance a case of a young man who was desirous of joining a corps of police for South Africa. The young man is of exceptionally good physique; and, was thoroughly examined by myself, and found to be sound. The medical officer who examined the candidates was, for some reason best known to himself, dissatisfied with the heart sounds of this particular candidate, and asked him if he had ever suffered from rheumatism. The candidate replied/

replied that he fancied he had had some pains in early boyhood. The officer, with a prodigious shake of the head, rejected him. Knowing the candidate as I do, the whole affair borders on the ludicrous. There were, also, those conjoint rheumatic cases, cases in which the rheumatism was supposed to exist in association with other diseases. Here, again, on the one hand, the arthritic element determined the nature of the malady; and, on the other, the etiological factor - the "cold" - determined it. If arthritis accompanied gonorrhoea, scarlatina, we got gonorrhoeal rheumatism, scarlatinal rheumatism. If "cold" appeared to set up iritis, facial paralysis, we got rheumatic iritis, rheumatic facial paralysis.

From the foregoing it is seen how extensive was the province of rheumatism; so extensive that the terms of admission - if the expression may be allowed - into that province must have been of a very easy nature. It is not surprising that the true rheumatism carries in its train so much that is pseudo-rheumatism. The explanation of this is, the knowledge was such that the province of the disease could not be clearly defined.

It is not far from the truth to say that the cause/

cause of this conglomerate mass of true and false rheumatism was owing to this, - that, on the one hand, arthritis and, on the other, "cold" as a causative agent were practically regarded as being pathognomonic of rheumatism.

Having thus considered the position of rheumatism as a disease when these two theories, the nervous and the metabolic - were chiefly in vogue, we have now to consider the position of rheumatism in the light of further knowledge which has been acquired as the result of clinical and pathological study.

It has already been stated that this fresh knowledge further demonstrates the inadequacy of the two theories to explain the pathology of the disease, and that it necessitates the abandonment of them. Therefore, it almost follows as a sequence, that this discovery of fresh clinical and pathological facts has given rise to quite a new conception of the disease; that it has led to the formation of a new theory as to the etiology and pathology of the disease.

What is this new conception? Succinctly stated, it is that rheumatism is a disease produced by a single morbific agency, which is capable of giving rise to various manifestations; each manifestation/

festation, as much as another, - in its own right, so to speak, - being part of the rheumatic process. Further, no one particular manifestation is essential for the production of rheumatism; any one, or many manifestations, may be present in the production of the disease.

The disease is regarded as being peculiar to childhood as much as it is to adult life; the difference being in the nature of the manifestations; or rather, it should be said, the proclivity of certain structures to be attacked in one way at one period of life, and others to be attacked in another way at another period.

The chief manifestations of rheumatism are:- arthritis, endocarditis - or, perhaps more properly, valvulitis, myocarditis, pericarditis, chorea and nervousness, subcutaneous fibrous nodules, varieties of erythema, tonsillitis and pharyngitis.

There are other manifestations:- pleuritis, pneumonia, hyperpyrexia, neuritis, and other affections of the nervous system.

The manifestations met with chiefly in childhood are:- heart affections, chorea, nervousness, and subcutaneous nodules. Those met with chiefly in adult life are:- arthritis, heart affections, pleuritis/

pleuritis, pneumonia, hyperpyrexia, and fever (?). The erythemata and throat affections are fairly common to both.

A point of great interest in contrasting the rheumatism of childhood with that of adult life is, that the arthritis, which is so pronounced a manifestation in adult life, is in abeyance in childhood.

The rheumatism of childhood is mainly distinguished from that of adult life by the nature of the inflammatory process. In the one case, it is subacute, fibrous tissue-forming, afebrile; in the other, it is acute, hyperaemic and febrile.

It is the case that rheumatism in childhood is not characterised by much fever, while in adult life it is. It certainly is remarkable that rheumatism is not characterised by fever in children, especially as children are so susceptible to fluctuations of temperature. It seems to me that this fact assists in settling that vexed question, Is fever a part of the disease rheumatism? Is it a manifestation? There are those who say that fever is not a manifestation, but that it is due to the local lesions. Again, there are those who say that fever is a manifestation independently of the local lesions.

If fever is a manifestation one would <u>a priori</u> expect/

expect to find it as a manifestation in childhood. If the local lesions cause the fever why do they not cause fever in childhood? While not believing the fever to be a manifestation, I think it is only in a secondary sense that the local lesions cause the fever. In solving this matter, one must bear in mind that distinction between the inflammatory process of adult life and childhood; how the one is acute, hyperaemic; and the other is subacute, fibrotic. It seems to me that the fever depends on the acuteness, or hyperaemicness, of the inflammatory process. This hyperaemicness is best exemplified in the arthritis of rheumatic fever. The arthritis of rheumatic fever is always accompanied by fever. If the arthritis subsides, the fever abates. If there are fresh exacerbations of arthritis, the fever re-appears. The Salicylates, by controlling the arthritis, correspondingly control the fever.

This acute, hyperaemic inflammation is present in the other manifestations of rheumatic fever. In childhood this acute, hyperaemic inflammatory process is not, as a rule, present. Fever, therefore, is not a marked feature in the rheumatism of childhood. One would expect an affection like pericarditis in a child to cause fever. But the rheumatic pericarditis of childhood has not that acute hyperaemic inflammatory/

flammatory process. There is, therefore, not that fever one would expect to find.

The non-suppurative character of the inflammation - in so far as the joints are concerned - has long been known. As far as all the manifestations are concerned, it may be said to be non-suppurative.

The inflammation, as was hinted above, has been described as appearing in two varieties in rheumatism. The one variety is acute, being characterised by fever and hyperaemia, the latter causing an exudation of lymph but not affecting the tissue elements. On the subsidence of the congestion, there is a rapid absorption of the exudate, and the parts are as they were before.

It is considered that this variety is what obtains chiefly in the adult manifestations of the disease, viz., the arthritis, tonsillitis, the erythemata; and that the endocarditis, pericarditis, pneumonia, etc., when occurring with the other manifestations of rheumatic fever, are of this hyperaemic variety. This hyperaemic inflammation is, more or less, transitory.

The other variety obtains chiefly in childhood; it is afebrile, of longer duration, and affects the tissue/

tissue elements in that it causes proliferation of fibrous tissue - Sclerosis - and leads to permanent changes. It is instanced in the subcutaneous nodules, the afebrile but more serious endocarditis and pericarditis, the chorea, the subacute arthritis.

While some sharply distinguish between these two varieties, it appears to me they might be regarded as different degrees of one form of inflammation, describing the one as acute and the other as subacute.

In the acute inflammation, it is said that the tissue elements are not affected, that no permanent effects are produced, that the affected structures return to the state they were in before, and that in these respects the acute variety is a different form of inflammation from the other variety.

I contend that these points practically characterise the subacute inflammation, in so far as there are no permanent effects and the structures return to the state they were in before. In the case of the subcutaneous nodules and the chorea, there are no permanent effects, the affected parts return to the state they were in before. It will be said that in the endocarditis, pericarditis, and subacute arthritis, are found the permanent effects of this afebrile/

afebrile inflammation. But is there not a modifying element in these conditions which is absent in the others? Can it cause wonder that the fibrous overgrowths on the valves of the heart do not disappear, when we consider the ceaseless acting of the valves? The same applies to the fibrous formation in the pericardium when we consider the ceaseless acting of the heart. Then in the subacute arthritis, the pain is not so severe, the rest is not so absolute, the joints are moved sooner, and so we get this modifying element. What is this modifying element? It might be called want of rest; or, movement, or friction. At all events, it is responsible for the permanent effects; and were it not present, I consider the affected structures in endocarditis, pericarditis, and subacute arthritis, would return to the state they were in before.

It is this modifying element which imparts to rheumatism its dangerousness, seriousness, and gravity. It must be admitted that the endocarditis of the acute variety, which is not supposed to be characterised by fibrosis, does not always escape without permanent effects, because of this modifying element.

Thus, the inflammatory process, whether in the manifestations of adult life, or in those of childhood, may be regarded as being the same in kind, but different in degree.

It is convenient to speak of two varieties of inflammation for clinical purposes; but, as a matter of fact, there are three degrees of inflammation met with in rheumatism. There is:-

1. An acute hyperaemic degree, with exudation of clear, or bloodstained fluid, with, there may be, minute haemorrhages or emboli.

2. A less acute degree into more pronounced connective tissue swelling and a fibrinocellular exudate.

3. A non-acute, or subacute degree, with a peri-vascular fibrosis and areas of Sclerosis.

While distinguishing the rheumatism as typified in childhood from that in adult life, it must be remembered that there are all grades of the disease between these two types. We may get the manifestations peculiar to adult life in childhood, and vice versa. For example, rheumatism may be acute in childhood, subacute in adults; arthritis may be absent in adults, present in childhood; subcutaneous nodules may be present in adults, absent in childhood.

Of the manifestations, I shall only refer specially to the subcutaneous fibrous nodules, chorea, and nervousness.

The subcutaneous fibrous nodules, which are found/

found more frequently in children than in adults, are looked upon as being pathognomonic of the disease. Whereas the other manifestations may appear as morbid conditions in connection with other diseases, this manifestation appears to exist solely as the result of the rheumatic poison.

These nodules are similar in structure to the vegetations in endocarditis; and, as a rule, when there is a crop of nodules, there is a simultaneous development of endocardial vegetations. Large nodules appear to indicate the presence of grave cardiac mischief. The nodules after a varying duration disappear. The fibrous formations on the endocardium and pericardium would, doubtless, likewise disappear but for the modifying element already mentioned.

Chorea, whether as a manifestation of rheumatism or due to any other cause, is surrounded by a great deal of mystery. Morbid pathology throws very little light on the matter. In a recent fatal case, the pathologist reported that the condition found in the brain was not incompatible with a rheumatic toxaemia.

Seeing that chorea is a manifestation chiefly met with in childhood, and is of the afebrile order/

order, the hypothesis, that it is the result of proliferation of fibrous tissue in some part of the nervous system, is not an unreasonable one. It would, thereby, harmonise - consist - with the subcutaneous nodules, endocarditis, etc., in the nature of the inflammatory process. If there be an overgrowth of fibrous tissue in chorea, it is, as in the case of the subcutaneous nodules, characterised by complete resolution.

As a manifestation of rheumatism, chorea is peculiarly associated with the manifestation, endocarditis. Indeed, the association appears to be as close as that of the subcutaneous nodules to endocarditis. Maclagan holds the view that rheumatism is a disease of the motor apparatus; and to bring the chorea into line with this view, he describes it as being "essentially a disease of the motor centres," - probably because of the choreiform movements. But, is chorea essentially a disease of the motor centres? There is a psychical element in chorea which entitles one to doubt it.

Nervousness is a manifestation often met with in children, who are attacked by rheumatism. This manifestation is closely related to chorea. Indeed, it is not easy to say where nervousness, as a manifestation, ends and where slight chorea begins.

May/

May it not be possible, in a child attacked by the rheumatic poison and displaying the manifestation nervousness, for this nervousness to culminate in an attack of chorea, should the child be acted upon by such exciting causes as fright, or strong emotion.

These manifestations - the subcutaneous fibrous nodules, the chorea, and nervousness - may be said to occur only as manifestations of the afebrile and fibrous-tissue-forming order. The arthritis, endocarditis, and pericarditis, occur as manifestations in both degrees of inflammation - acute and subacute -- hyperaemic and fibrous-tissue-forming. The erythemata, tonsillitis and pharyngitis, may also be said to occur in both degrees, but they are distinguished from the arthritis and heart affections by the inflammatory process always remaining hyperaemic and not fibrotic. The pneumonia, pleurisy, cerebral manifestations, - such as hyperpyrexia - appear to occur only in the acute hyperaemic degree of inflammation.

These manifestations do, without doubt, occur as genuine expressions of rheumatism. There may be other manifestations, but classification is difficult. There is a danger of loosely applying the/

the term "rheumatic" to any event that may occur during an attack of rheumatism. For example, if nephritis, or bronchitis, should occur during an attack of acute rheumatism, there is a post hoc, etc., tendency to call them manifestations of rheumatism.

When an attack of rheumatism manifests itself, it - broadly speaking - conforms to one of two types.

1. An acute, febrile, hyperaemic inflammatory process occurring in one type.
2. A subacute, afebrile, fibrotic inflammatory process occurring in the other type.

One or more manifestations may be present in an attack; but, when present, they are of one type or the other.

While these two types are distinguished clinically, it is to be remembered that the inflammatory process is essentially the same, any difference being merely one of degree, of acuteness.

The seat of the rheumatic process is chiefly in the fibrous structures of the joints, muscles, tendons, fascia, valves of the heart, pericardium, pleurae, and some undefined portions of the nervous system. That the serous membranes in connection with these structures play an important part in the rheumatic/

rheumatic inflammation is also well recognised. The endothelium appears to play a peculiarly important part - a vital part - in combating the action of the rheumatic poison.

Having considered this modern conception of rheumatism as a disease, we must now consider the new theory as to the etiology and pathology which has resulted from further clinical and pathological study.

Stated shortly, it is that rheumatism is caused by an infective agent, and that this infective agent, or its toxines, accounts for the manifestations and phenomena of rheumatism.

Without going into such etiological points connected with rheumatism, as age, sex, occupation, injury, or other exciting cause, I shall only refer to such influences as atmospheric conditions, heredity, etc. For any information bearing on these points, we are mainly dependent on observations derived from the study of cases of rheumatic fever.

Statistics have been made with regard to season, temperature, and rainfall; but they are completely at variance. This variance, or disagreement, however, is of value in giving us some negative results. It shows that rheumatic fever, and so the disease rheumatism, is independent of, and not/

not governed by, meteorological conditions.

This strikes a severe blow at the long accepted view that exposure to cold or wet was the chief etiological factor in the production of rheumatism. Of course, no one doubts that cold and wet may, in so far as they are exciting causes, play a part in the causation of rheumatism.

One very important etiological point that is established is, that rheumatism sometimes occurs in epidemics; and that these epidemics vary in type and character. This epidemic - occurring feature of rheumatism forms a great pillary of support to the Infective theory as to the etiology and pathology of the disease.

Heredity has long been known to be an important factor in the causation of rheumatism. That it is so is practically universally accepted. There is some elasticity about the meaning of heredity. With some, it means that, if a person's parent or parents suffered from rheumatism, it is a certainty that sooner or later, that person will manifest rheumatism; with others, it means that that person has a special liability to be attacked by the disease - a predisposition for it.

The question of heredity is one of great difficulty and complexity, and it is impossible to estimate/

estimate the etiological influence of it.

Assuming that there is a percentage of cases of rheumatism showing a family history, there is also a percentage of cases that shows no such family history. It is not unreasonable to assume that many of the cases with the family history were cases of rheumatism independently of that history, cases that arose de novo.

There have been many individuals, in whose families there was a history of rheumatism, who had never suffered from the disease. It must be remembered that there was a time in the history of rheumatism, or of any other disease, for that matter, when it was impossible for heredity to be an etiological factor.

The view that a disease is hereditary is one that is very prone to be vitiated by post hoc propter hoc reasoning. No one doubts that there is a law of heredity; but there is also a law of variation - this latter, however, seems to have little count in medicine.

If there is one disease above all others, in which heredity was believed to play a prominent part, it is tuberculosis. At one time, if a person's parents were tuberculous, that person was practically/

practically doomed. The teaching now is more optimistic, gives a brighter and more hopeful outlook. It teaches, - a person born of tuberculous parents may have a predisposition for the disease, but if removed from his surroundings and the risk of direct infection, that person has no mean chance of escaping the disease. This constitutes a great change on the older teaching; it might almost be said to ignore the traditional view as to the heredity of tuberculosis.

May it not be so with rheumatism? Granting that there is a pre-disposition, what about the same conditions, surroundings, influences, etc., existing with the child, as existed with parent? May not they be responsible for the occurrence of the disease, when it is often attributed to heredity? I mean that those conditions, surroundings, influences, etc., may promote, favour, the attack of the disease, when heredity is often credited with the cause; and, that there is great likelihood of parent and offspring being subjected to similar conditions, surroundings, and influences, etc.

There is, also, what is called the "basic arthritic diathesis". These diathetic states are closely associated with heredity in disease; an undefinable/

undefinable, un-get-at-able something is inherited, which makes the individual specially liable to manifest the disease.

In tuberculosis there is a tuberculous diathesis; but here, again, there is a tendency to break away from the traditional teaching. Some hold that what is called the tuberculous diathesis indicates that the disease is actually in progress; others consider that it indicates that the tissues of the individual are not possessed of great resisting power should they be attacked by the disease - that there is a vulnerability of the tissues. This is certainly more scientific than the older belief.

In the case of rheumatism, if this diathetic state distinguishes the rheumatic constitution from the non-rheumatic; if it is transmitted from father to son; if it is essential for the production of rheumatism; how did it arise in the first instance? How did the individual, who in the history of the world, was the first to suffer from rheumatism, who could not possibly have the diathetic state or rheumatic constitution transmitted to him, acquire the disease?

Can individuals not be attacked and acquire the/

the disease to-day in the same way as that first, original sufferer from the disease? If there is a basic arthritic diathesis, there is no reason why there should not be a basic peritonitic diathesis, or basic pleuritic diathesis, etc. The idea of a vulnerability of the tissues appears to me to be more comprehensible, rational and scientific. Individuals possess a stomach, liver, lungs, etc., as well as joints; and, they are all liable to morbid processes. Whether or not, the morbid process takes place, when the individual or organ of the individual is attacked, depends on various conditions and circumstances; as one instance, it depends on the state of health of the individual - that is, on the state of his various systems, such as the nervous, circulatory, alimentary systems; the systems that regulate the power of resistance; that regulate the state of the tissues and functions of the various organs.

We must now consider the infective theory as explaining the pathology of rheumatism.

A few years ago, this was nothing but a theory, with little to support it beyond the inadequacy of preceding theories to account for the phenomena of rheumatism. Although, to-day, no micro-organism has/

has been discovered which is universally accepted as the specific cause of rheumatism, nevertheless, great progress has been made, and is being made in the bacteriology of rheumatism. It is scarcely an exaggeration to say that the infectiousness of rheumatism is established. There only remains to establish the specificity of the micro-organism.

A great many analogies and parallels have been drawn between rheumatism and other diseases to support views as to the pathology of the disease. We saw how there was believed to be a complete analogy between gout and rheumatism, which led up to the lactic acid theory.

Many analogies have been drawn between rheumatism and other diseases which are due to the introduction of a poison from without. Some are carried further than others, but sooner or later, they break down; none are complete.

The sore throat, erythematous rashes, local lesions likened rheumatism to the specific fevers. The erythemata caused it to be compared to erysipelas.

It has been likened to pneumonia because of its epidemicity, its non-communicability, and its tendency to recurrence.

Pyaemia/

Pyaemia, with its polyarticular arthritis, endo-carditis, pericarditis, pneumonia, pleuritis, shows a remarkable resemblance to the manifestations of acute rheumatism.

An analogy has been drawn between rheumatism and malaria; both being peculiar to low-lying damp localities; the fever curves, the non-communicability, non-immunity, and the specificness of the treatment.

It might be compared to tuberculosis in that certain structures have a proclivity to be attacked by the tubercle bacillus at one period of life and others at another. Thus, there is a tuberculosis of childhood, as there is a rheumatism; a tuberculosis of adult life, as there is a rheumatism.

It might, in some respects, be likened to syphilis; how a pregnant mother suffering from rheumatic fever, the offspring may soon show similar symptoms.

It is, however, surely not necessary to get a complete analogy between rheumatism and any other infectious disease to satisfy one that it is of infective origin. It seems to me they all assist in supporting the infective theory.

The manifestations of rheumatism in childhood constitute the chief difficulty in the reasoning by analogy./

analogy. The explanation of the analogies breaking down is, that the framers of them take rheumatic fever only as representing the disease rheumatism, and do not deal with rheumatism in childhood. In measles, scarlet fever, typhoid fever, pneumonia, pyaemia, malaria, there is nothing corresponding to the rheumatism of childhood. Tuberculosis, a disease due to an infective agency, affords a parallel for rheumatism in childhood. At one time, phthisis, or what was popularly known as "consumption", was regarded as a disease by itself. It was hereditary, constitutional. It might be said to correspond to rheumatic fever. It was then found that the disease phthisis could be produced directly by inoculation; and, it is interesting to note, such a conclusion was hotly contested at the time. The discovery of the tubercle bacillus was then made; and that the bacillus was the micro-organism which was responsible for, not only phthisis but also many manifestations of one disease included under the name tuberculosis. Phthisis, in short, was a manifestation of a disease; just as rheumatic fever - or acute rheumatic arthritis - is now regarded as a manifestation of rheumatism. It must, at one time, have astonished not a few physicians that tabes mesenterica, "water in the brain", and "consumption", were all caused by/

by the same infective agent, and were different manifestations of the same disease.

It is a characteristic of tuberculosis that in childhood, the tubercle bacillus more readily attacks the meninges, the lymphatic glands, - whether they be bronchial or mesenteric, - and the bones; that in early adult life, it more readily attacks the tissues of the lungs. The inflammatory process set up by the tubercle bacillus may be acute or subacute - non-fibrotic or fibrotic. It differs from the inflammatory process in rheumatism in that the non-fibrotic inflammation is peculiar to childhood, and the fibrotic to adult life.

Thus, do we not get a broader, wider analogy, which is more in keeping with the modern conception of rheumatism? The manifestations which are peculiar to adult life in each disease; those peculiar to childhood in each disease. The analogy can be even carried into the history of the two diseases. How in the one case phthisis was regarded as essentially the tuberculous process; how in the other, acute rheumatic arthritis was essentially the rheumatic process. How they were both regarded as hereditary and constitutional diseases. How each is now regarded as a manifestation among many/

many manifestations of a disease.

I am not, however, concerned in drawing an analogy between the two diseases; but simply to establish, as it were, a precedent for an infective agent causing different manifestations of a disease at different periods of life.

And so in the case of the specific fevers, we get a precedent for sore throats, rashes, and local lesions being caused by infective agents.

In pyaemia we have a precedent for an infective agent causing polyarticular arthritis, endocarditis, pericarditis, pneumonia, pleurisy.

Pneumonia may be said to establish a precedent for an infective agent causing a disease characterised by occurring in epidemics of varying types, by non-communicability, non-immunity. Cerebro-spinal meningitis and syphilis likewise might supply precedents.

It is remarkable as showing the complexity and manysidedness of rheumatism that it has phenomena which resemble phenomena in all these diseases mentioned.

I submit that this series of what I call precedents supports and strengthens the infective theory as to the pathology of rheumatism. Not only/

only so, but that it supports and strengthens the view that rheumatism is caused by a specific micro-organism.

In addition to this "precedent" or "analogous" support, the infective theory has gained direct and positive support from bacteriological investigation. So much so, that with many it is not a question whether or not, rheumatism is due to an infective agent; but, rather, what is the nature of the infective process? As this latter question has yet to be settled beyond doubt, it naturally gives rise to various views.

There is, firstly, the view that rheumatism is not a disease sin generis, but a particular reaction of the tissues to varied infections.

Secondly, that rheumatism is the result of a mixed infection of bacilli and cocci.

Thirdly, there is the view that, while rheumatism is of microbic origin, it is not due to a specific micro-organism; but, that it is a form of septicaemia which owes its origin to streptococcal and staphylococcal infection.

Fourthly, there is the view that rheumatism is due to a specific micro-organism. This view is subdivided into

(a) That it is due to a specific bacillus.

(b) That it is due to a specific diplococcus.

The third and fourth views are the most important. There is a semblance of justification for the third view. Acute rheumatism, with, it may be, arthritis, endocarditis, pericarditis, pneumonia, fever, pleurisy, etc., presents a picture not unlike that of a form of septicaemia: and so, supporters of this view consider that rheumatism is an attenuated septicaemia caused by an attenuated micrococcus. There is, however, another picture, which the disease rheumatism can present, - unlike any form of septicaemia. Subacute rheumatism, as manifested in a child with follicular tonsillitis, an eruption of erythema papulatum, a crop of subcutaneous nodules, and practically afebule, presents a picture: are we to regard this as an attenuated septicaemia, then, truly, it would owe its origin to extremely attenuated streptococci and straphylococci.

To arrive at a correct solution of this matter, one must take a broad survey of rheumatism as a disease. If one allows the manifestations of acute rheumatism to overshadow those of subacute, error is certain to ensue. The manifestations of childhood can be explained as due to an attenuated septicaemia.

The/

The fourth view, that rheumatism is due to a specific micro-organism is the one, to my mind, deserving of most favour.

The features of rheumatism appear to be sufficiently characteristic and distinct to justify it being regarded as a disease due to a specific micro-organism. The fact that it sometimes occurs in epidemics must not be lost sight of in this connection. I have already stated that the disease tuberculosis furnishes an example of a specific micro-organism causing manifestations - some peculiar to childhood, others to adult life - and setting up an inflammatory process which may vary in type; and, that in these respects a parallel can be drawn between rheumatism and tuberculosis.

I consider this parallel is not without value in supporting this fourth view.

Many investigators - in this country and on the Continent - have set to work with the object of discovering the specific micro-organism of rheumatism. In this country the most valuable work has been done by Drs Poynton and Paine. They commenced their researches with the object of confirming Achalme's claim that he had discovered an Anthrax - like bacillus which was the cause of rheumatism./

rheumatism. They at first failed to find this bacillus; when they did find it, they did not consider it to be the cause. During their search for this bacillus they, incidentally, came across a diplococcus with remarkable frequency. They have isolated and cultivated this diplococcus from at least eighteen cases which were diagnosed as rheumatic fever by the physicians in attendance. They have taken these diplococci from the valves of the heart, pericardial fluid, tonsils, subcutaneous nodules, in the human subject. They have intravenously injected them into many rabbits, and the results taken collectively may be said to have given as complete a picture as possible of the manifestations and phenomena of rheumatism.

They have produced in the rabbit - a painful polyarticular arthritis, valvulitis, pericarditis, myocarditis, pneumonia, pleurisy, subcutaneous nodules, chorea. The peritoneum showed the same peculiarity - as in the human subject - of escaping. The inflammatory process conformed to the types met with in rheumatism, e.g. it was non-suppurative, it showed the hyperaemic characteristics, as also the fibrotic. The microscopical appearance of the nodule in the rabbit conformed to that found in/

in the subcutaneous nodules of the human subject. They have taken the diplococci from the lateral ventricles, the pia mater, the fibrinous exudate in the joint of the inoculated rabbit; and, in turn, injected them into other rabbits and produced conditions similar to rheumatic manifestations.

The diplococci found in the inoculated rabbit are indistinguishable from those found in the cases of rheumatic fever: they are the same morphologically and culturally.

Drs Poynton and Paine consider this diplococcus to be the specific micro-organism which causes rheumatism.

This organism, which according to Poynton and Paine appears in pairs, seems to be identical with that isolated by Triboulet, Wassermann, and others. These latter, however, describe it as appearing in chains, and call it a streptococcus.

Drs Beaton and Ainley Walker are carrying on an investigation into the etiology of rheumatism, which primarily had as its object the testing of the observations of these above-mentioned investigators. As a result of their observations they agree that a micrococcus is present in the lesions, and is the causal agent, of rheumatism; and they believe the coccus they have isolated to be identical with that obtained by Triboulet, Wassermann, Poynton and/

and others. As this micrococcus appears both in pairs and chains, they prefer to speak of it as the "micrococcus rheumaticus".

It may be mentioned that Drs Beaton and Walker, besides having taken this micrococcus from cases of acute and subacute rheumatism and, on injection, produced in animal manifestations of rheumatism, have taken it from three cases of chorea and produced in animals manifestations of rheumatism.

Drs Beaton and Walker have gone very fully into the cultural characters of this micrococcus and they find there is nothing in these ordinary characters to distinguish it from any other streptococcus. They have, however, applied Marmorek's test: they have grown two specimens of this micrococcus in a medium of filtered streptococcus bouillon; and conclude from that that this micrococcus is specifically different from the ordinary hyogenetic streptococci of human origin.

This is a most important result; and, if it be confirmed by other workers, the specificity of this micrococcus will be established. This result promises to demolish the view that rheumatism is not a disease sui generis; as, also, the view that rheumatism is an attenuated septicaemic.

These/

These different investigations are still being carried on: when they are completed there is reasonable expectation that the etiology and pathology of rheumatism will be finally settled.

There seems to be little ground for the view, that the tonsils form the nidus for the organisms, and that the latter remain in the tonsils and pour their toxins into the system. The tonsils may be a portal for the organisms getting into the system; but, judging from the inconstancy of the faucial inflammation in cases of acute and subacute rheumatism, it seems hardly likely that the tonsils act as the toxin-factory of the organisms.

There are many who, while believing that rheumatism is due to an infective agent, consider that the infective agent merely plays the part of an excitant and that the thing essential for an attack of rheumatism is a "peculiar habit of body", or a "special soil". This "peculiar habit of body", and "special soil", seem to me to be easily recognised: are they not aliases of the "diathetic state", the rheumatic constitution? If the diplococcus isolated by Poynton and Paine, or the micrococcus rheumatics of Beaton and Ainley Walker, prove to be the specific causal agent of rheumatism, and if the effects produced/

produced on the injection of the organism in rabbits are genuinely rheumatic, I take it we must assume that these rabbits possess this "peculiar habit of body", this "special soil". I would further ask, in the experimental production of these rheumatic manifestations in rabbits, how much is to be attributed to heredity, the rheumatic diathesis, the rheumatic constitution?

In the face of the results of these bacteriological investigations to which I have referred, it seems almost like labouring the point to adduce further evidence in support of the infective theory. Yet, one ought not to disregard the indirect support gained as the result of the study of infective agents and the part they play in the production of disease.

A few years ago cases of inflammation of the peritoneum, which appeared to arise as independent forms of inflammation, were described as idiopathic. Such cases are not believed in now, but are regarded as being, in the main, caused by micro-organisms. The synovial membrane of joints is comparable - anatomically and physiologically - to the other serous membranes, e.g., the peritoneum, pleurae, pericardium, and the meninges. And so, in many cases of inflammation of the joints, infective agents/

agents - whose action was considered to be limited to structures other than the joints - are held responsible for the condition.

The study of the pneumococcus - micrococcus lancelotus of Fraenkel - shows how wide are the effects of infective agents. Originally considered to be the causal agent of acute lobar pneumonia, it is now known to cause in addition, pleurisy, pericarditis, meningitis, peritonitis, and arthritis. The infective agents of other diseases are now known to attack serous membranes, not excepting those of joints. The infectious diseases in which arthritis is known to be a manifestation are - tuberculosis, syphilis, gonorrhoea, common forms of septicaemia, pneumococcal infection, typhoid fever, scarlet fever, erysipelas, dysentery, influenze, and glanders.

Before the part played by infective agents in the production of disease was known, the arthritis, which appeared in many of these diseases mentioned, had its pathology simply explained by the use of the terms "rheumatic", or "rheumatism". For example, - gonorrhoeal rheumatism, rheumatic typhoid arthritis, scarlatinal rheumatism, dysenteric arthritis, and other obscure cases of arthritis. Thus rheumatism, which seems to have been regarded as the predominant partner/

partner in all joint diseases, was made the pathological scape-goat for forms of arthritis appearing in many diseases.

The study of the action of infective agents has resulted in divesting rheumatism proper of all this pseudo-rheumatic arthritis; it has, also resulted in defining more precisely the province of rheumatism as well as the other diseases concerned.

Seeing that each of the infective agents of the above mentioned specific diseases is capable of setting up arthritis, it would appear that the joints are very prone to be acted on by micro-organisms. That being the case, there is substantial ground for the hypothesis that the arthritis of rheumatism is caused by a micro-organism.

In short, it may be concluded that the study of the action of infective agents, and more particularly the study of infective arthritis, has resulted in clearing the ground for and giving great support to the theory that the causal agent of the disease rheumatism is an infective one.

Cases have been recorded with a view to showing that rheumatism is contagious. Thoresen, Mantle and Kellman have recorded such cases. The contagiousness of rheumatism will require a great deal more proof than these cases afford. The matter, however, is interesting in that there can be no doubt in the minds of Thoresen, Mantle and Kellman as to the infectiousness of rheumatism.

It need only be mentioned that rheumatism is regarded by some as a miasmatic disease. Dr Maclagan draws an elaborate analogy between rheumatism and malaria. His arguments are by no means convincing. He describes the manifestations of rheumatism in childhood as "anomalous" forms of rheumatism. In following his argument, one always has the impression that he holds a brief for the Salicyl bodies.

At this point, I might refer to the claim of the Salicylates to be regarded as anti-rheumatic specifics. I do not propose going into the treatment of rheumatism, any more than I do into the details of the symptoms of the various manifestations.

The introduction of the Salicylates marks a great triumph in the treatment of rheumatic fever. The manner in which they control and repress the arthritis, with its pain and fever, is known to all. Their influence, however, is not universal enough to justify them being designated anti-rheumatic specifics. Their influence on subacute articular rheumatism and the other manifestations is practically ineffective.

It is in the arthritis of acute rheumatism that their marvellous effects are produced. There is a tendency among the adherents of this claim to regard/

regard all affections as of rheumatic origin if they are relieved by the administration of the Salicylates. Many cases of febricula and influenza are relieved in a striking manner by their use.

Having considered the view that rheumatism as a disease, is a complex one with various manifestations - some peculiar to childhood, some peculiar to adult life, with, it may be said, an inflammatory process peculiar to childhood and one peculiar to adult life, - and that these manifestations, one and all, have as their causal agent, a specific micro-organism; and, believing as I do, that this view is the correct one, it is quite evident that this view will have to come to an understanding, as it were, with former views as to the nature of rheumatism.

The view that acute rheumatic arthritis, with its special signs and symptoms, was essentially the rheumatic process must be discarded. The acute arthritis may be the most apparent, palpable manifestation of the disease; but, it is, after all only a manifestation. A severe attack of rheumatism may manifest itself without the involvement of joints at all. The rheumatism of childhood is generally/

generally spoken of as abarticular, in contra-distinction to the articular rheumatism of adult life.

The view that exposure to cold, or wet, was the chief etiological factor in the production of rheumatism can no longer hold the high place it did. Exposure to cold, or wet, may, no doubt, play a part as exciting causes; they may render the individual more susceptible to the attack of the micro-organism by lowering the resisting power.

The influence of heredity as an etiological factor cannot be estimated; its influence should certainly not be over-rated.

Then this modern view of rheumatism demands the severance of the long connection with gout; the two diseases are neither related nor analogous according to this view.

However, so long and so intimate has been the association of the two diseases, that many will continue to adhere to the association in spite of this latest view of rheumatism.

The two diseases have always, as it were, been spoken of in the same breath. I have before me now an advertisement in which a medical man testifies to the value of a certain mineral water in the "gouty and rheumatic diathesis."

The/

The two diseases have always been classified together under the same heading, - it may be under Constitutional Diseases, or Certain General Diseases. Some text-books on medicine at the present time place rheumatism among the Constitutional Diseases. The word "Constitutional" ought to be abolished altogether as a most unscientific term. It is a misleading term; it appears to convey an intelligible meaning when, as a matter of fact, it is merely a magniloquent expression for ignorance. What are the chief "Constitutional" diseases? Gout, rheumatism, diabetes mellitus, diabetes insipidus, rickets, scurvy, purpura, - the diseases as to the exact nature of which we are ignorant.

Cancer, tuberculosis, and pyaemia, were at one time regarded as "constitutional" diseases. Heredity is closely allied with this constitutionalism, and between the two of them we get that indefinable <u>something</u>, which neither the anatomist nor the chemist can get hold of, and which is known as the diathetic state. In the latest editions of many text-books on medicine, the authoris - in anticipation of the demonstration of the Infective Theory - have removed rheumatism from its place among/

among the Constitutional Diseases and described or placed it among the Infectious Diseases. This marks an advance in the right direction.

In the field of chronic rheumatism, a great deal of paving, and pruning must be done to make it harmonise with this modern view of the disease.

No doubt, a great deal of what was regarded as chronic rheumatism may be genuinely chronic rheumatism. That there should be a form of rheumatism which was chronic is by no means incompatible with the Infective Theory. One might almost describe the tertiary stage of syphilis as chronic syphilis.

What is certain is that the diagnosis of chronic rheumatism demands a great deal more care, scrutiny, and inquiry, as to any previous occurrence of rheumatic manifestations, than has hitherto been the case. All obscure aches and pains - whether arthritic, muscular, neurotic, - cannot be accepted off hand as being due to the rheumatic poison. The mere fact that a person has at some time had arthritic pains, does not justify a diagnosis of rheumatism.

The term "rheumatism" has been of great service as an aid to diagnosis in many obscure conditions and affections. If there was any justification/

fication for its use as a diagnostic in the past, there is certainly none now, in the light of the modern conception of the disease. To use it in this sense nowadays is, to borrow an expression, unsportsmanlike.

Post-rheumatic affections, all those arthritic affections which are really the effects of rheumatism, - have to be distinguished from genuine chronic rheumatism. They are really no more examples of chronic rheumatism than many cases of cardiac dilatation, or cardiac incompetency with all its signs and symptoms of backward pressure.

In this new conception of rheumatism, there is no such thing as Scarlatinal Rheumatism, Gonorrhoeal Rheumatism. Either the diseases rheumatism and scarlatinal rheumatism and gonorrhoea - are both present in the individual at the same time, or, as is more likely the case, - one might say, actually the case, - the infective agent of Scarlatine, of Gonorrhoea, has set up the arthritis.

I remember attending a case of diphtheria; the injection of antitoxine was followed by a polyarticular arthritis. What was it? Was I to call it diphtheritic rheumatism, or antitoxinal rheumatism?

There are many of these hybrid diseases - such as/

as rheumatic purpura, rheumatic facial paralysis, rheumatic iritis, dysenteric rheumatism, etc., - which are now excluded from the province of rheumatism.

Thus may be gathered some idea of the profound change this newer view works on the older ones. It means that the disease is put on a new basis altogether; it is entirely re-modelled.

The modern conception necessitates the contraction and expansion of the province of the disease at one and the same time.

On the one hand, all that pseudo-rheumatism must be cast out; on the other hand, many conditions, that formerly held no place in the disease, are now included, embraced, in its province.

It cannot but be admitted that the modern view defines the province of the disease with a clearness, such as never before was the case. This view dispels all that mustiness and vagueness which are part and parcel of such expressions as "constitutional", "diathesis". It does not permit the term "rheumatism" to symbolise all forms of arthritis, and all "cold" caused affections.

A firmer mental grasp can be taken of this conception of the disease; it can be viewed as a composite whole. The conception of the disease in short, is more scientific.

This conception is the result of further knowledge of the disease obtained by clinical and pathological study.

This further knowledge combined with pathological and clinical knowledge of other diseases has resulted in the formation of the infective theory as to the etiology and pathology of rheumatism. No other theory gas yet been advanced which accounts so fully and completely for the phenomena of rheumatism. No other theory has yet been advanced which is so near to being proved.

It was remarked above that many writers of text-books had placed rheumatism among the Infectious diseases, and that it marked an advance in the right direction. There is, however, still left something to be desired in the description of the disease. With many writers, acute rheumatic arthritis, or rheumatic fever, is still represented as being typically the rheumatic process. The rheumatism of childhood, or subacute rheumatism, does not get dealt with as it ought. It cannot be insisted too strongly that subacute rheumatism is as important, if not more important, than acute rheumatism.

Some of the manifestations are mentioned chiefly as complications.

Some/

Some show a lingering regard for pseudo-rheumatic affections. Even gout is described, either immediately before, or immediately after, rheumatism, by some writers.

It must be remembered, however, that rheumatism is a disease of great antiquity; that it has long held a position firmly established in the medical mind, as well as the lay public mind; that traditions die hard; that allowance must be made for peculiar obstacles, beliefs, and prejudices.

If it be that rheumatism as a disease, is on the eve of being put in its proper place in Medicine, it can, truly, be said that the history of the progress of our knowledge of rheumatism affords one more instance of -

"Science moves, but slowly slowly,
Creeping on from point to point."