Rheumatism, gout, and some allied disorders / by Morris Longstreth, M.D.

RHEUMATISM, GOUT,

AND

SOME ALLIED DISORDERS

BY

MORRIS LONGSTRETH, M.D.

ONE OF THE ATTENDING PHYSICIANS OF THE PENNSYLVANIA HOSPITAL; LECTURER ON PATHOLOGICAL ANATOMY AT THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA, PA.

London:

SAMPSON LOW, MARSTON, SEARLE, & RIVINGTON,

CROWN BUILDINGS, 188, FLEET STREET.

1883.

CONTENTS.

CHAPTER V.

DESCRIPTION AND COURSE OF THE DISEASE.

CHAPTER VI.

CONSIDERATIONS OF THE INDIVIDUAL SYMPTOMS OF ACUTE RHEUMATISM AND THEIR PECULIARITIES.

CHAPTER VII.

CONSIDERATIONS OF THE INDIVIDUAL SYMPTOMS OF ACUTE RHEUMATISM AND THEIR PECULIARITIES.—*Continued.*

CHAPTER VIII.

CONDITION OF THE SKIN AND ITS FUNCTIONS.

CHAPTER IX.

GENITO-URINARY APPARATUS.

CHAPTER X.

THE URINE.

CHAPTER XI.

COMPLICATIONS, OR EXTRA-ARTICULAR LOCALIZATIONS.

CHAPTER XII.

COMPLICATIONS, OR EXTRA-ARTICULAR LOCALIZATIONS.—*Continued.*

CHAPTER XIII.

NERVOUS COMPLICATIONS.

CHAPTER XIV.

MORBID ANATOMY.

CHAPTER XV.

DIAGNOSIS AND PROGNOSIS.

CHAPTER XVI.

TREATMENT.

CHAPTER XVII.

CHRONIC ARTICULAR RHEUMATISM.

CHAPTER XVIII.

GONORRHŒAL RHEUMATISM.

CHAPTER XIX.

GOUT.

RHEUMATISM, GOUT,

AND

SOME ALLIED DISORDERS.

CHAPTER I.

INTRODUCTORY.

Rheumatism—Some Considerations Concerning its History, Nosology, and Etiology—Synonyms—Definition.

The term Rheumatism, in the sense in which it is now used, dates from about three centuries ago. Previous to this time writers comprehended under the term arthritis this disease with gout and all other inflammations of the joints, ligaments, and muscles. Anciently the words *rheuma* and *rheumatism* denoted all those diseases in which it was supposed that the blood became impregnated with acrid humors, phlegm, bile, or other noxious materials. Hippocrates states that the bile mingles with the blood in the veins and articulations, causing swelling of the joints, or extending to the whole body, producing acute pain. In this view, with some modifications, Hippocrates was followed by most of the later writers. From this group, these writers separated the diseases of the mucous membranes under the general head of catarrhs; all the other members of the group retained the designation of *rheumata*. In 1610, William de Baillou published his thesis on rheumatism, in which he showed the distinction between this disease and gout. Sydenham, who lived fifty years later than Baillou, even more distinctly divided these two diseases. Cullen, Stoll, and Van Swieten in their teachings promoted these views and framed their treatment accordingly. The humoral pathology of that day

contributed greatly to the enforcement of these theories of the nature of gout and rheumatism. Although the separation of these two maladies seemed fully confirmed, nevertheless the meteorological theories of the next generation of medical writers, assisted by the Nosologists, who attempted to force all knowledge of medical science into such bounds as they were able to give a name to, contributed to overturn the sound basis which Sydenham had given to the subject, so that early in the present century we find the two diseases grouped under the same heading, and looked upon by many as diverse manifestations of the same humor mingled with the blood.

Some Considerations Concerning its History, Nosology, and Etiology.

In early times, but especially after the separation of rheumatism from gout, it seems that the terms "rheumatism" and "rheumatic" were applied to external diseases, or, rather, to maladies which affected the exterior parts of the body; however, it must be admitted that with rheumatism were confounded many ailments which were of wholly dissimilar nature; for example, phlebitis, pyæmia, neuralgia, coxalgia, certain scorbutic and scrofulous diseases, and even some of the puerperal conditions. From this, so to speak, external nature given to rheumatism, its cause was looked upon as of external origin, and the principal cause was considered to be cold and such atmospheric variations as were connected with changes of temperature. This generally accepted theory of the causation of rheumatism led to the inclusion among the heterogeneous components of this malady other diseases, which formerly were not considered as belonging to the same group, but which were due to the same supposed cause, viz., cold. Thus, all diseases, especially those accompanied with pain, were counted as rheumatic, if not as being "rheumatism" itself. Many of these newly included diseases were affections of internal organs. Rheumatism was, therefore, no longer a disease of the external parts alone, but also a visceral affection. The brain, the lung and its appendages, the heart, the kidneys, etc., were all supposed to be the seat of rheumatism, and not only the morbid changes which we now recognize in these organs as the results of rheumatic disease, but many others were ascribed to this humoral poison. A rheumatic apoplexy, a rheumatism of the intestines resulting in diarrhœa and dysentery, and many others were added to this list. Through the advancement made in

morbid anatomy, aided by greater accuracy in clinical studies, some of these diseases have been confirmed in their position as the results of the rheumatism poison, others have been thrust away, and still others, hitherto unknown, have been brought to light and arranged in a proper relation to this morbid cause.

The position which rheumatism now occupies, as a nosological entity, is that of a group of symptoms, not necessarily and essentially connected, having a common or supposed common cause, without, however, any, as yet discovered, anatomical basis. That this should be the position of rheumatism, is the necessary result of varied opinions held concerning it from the earliest times until the present. At first the word was used like catarrh, to signify a "defluxion of an acrid humor, generated in the brain, into various parts of the body," and later, after the catarrhal affections of the mucous membrane had been separated into a distinct group, denoted all diseases due to the circulation of acrid humors. Later still it was employed to denote the vague pains of the external parts, and afterward, when Ballonius distinguished it from gout, rheumatism was applied to the same group of symptoms to which it now appertains. It is no wonder, therefore, that the term, as handed down to us, has as vague a significance as some of the pain to which it has been and is at present applied.

While at the present time we may hold more definitely in mind what we comprehend under the heading of rheumatism than was done formerly, when unquestionably neuralgia, pyæmia, coxalgia, and certain syphilitic affections were included in this group, still it must be admitted that the acute and chronic forms of rheumatic articular disease and muscular rheumatism form a class which, if not pathologically dissimilar, are nevertheless joined together by only one common symptom, viz., pain, and one common cause, viz., cold. It is self-evident that these two bases are not sufficient grounds for founding either a pathological or an etiological group of disease.

The cause of this heterogeneous classification, so evidently the result of a survival from ancient times, is to be explained partly by the failure of post-mortem investigations to disclose any adequate explanation of the symptoms, and also by the infrequency of the opportunities to conduct such investigations, owing to the rarely fatal character of a vast majority of the cases belonging to this group of diseases. Although it is true that many patients die from rheumatism, the fatality occurs mostly from the after-results of the acute disease, and, therefore, at a period when the special manifestations and characteristic lesions have subsided and can no

longer be seen in the condition in which they present themselves during the acute process. But the failure from this cause to study the special lesions of rheumatism does not seem to cover the whole ground, or to explain our great ignorance of the special pathology of this affection, either in its acute or chronic form, or its manifestations as a muscular rheumatism. Judging from the results obtained from the careful post-mortem examination of bodies dying at the period of the disease most favorable for such studies, and the entire inadequacy of the lesions in explaining the morbid symptoms, it is not probable that more frequent opportunities for such examinations would contribute very greatly to our knowledge of its pathology. The true direction, it would seem, for us to seek enlightenment is from a study of the altered condition of the blood. Already some experimental studies have been made, and very many theories have been put forward in relation to the noxious ingredients present, but as yet nothing definite has been determined with sufficient positiveness to command the universal assent of investigators and the profession in general. I cannot but think that if, at the present time, it were customary as formerly to bleed freely every patient suffering from acute rheumatism the amount of material thus so abundantly presented for examination would call up some one fully fitted to determine the problem. It is blood in the living state that is needed; blood, post-mortem, is not in a suitable condition for such an investigation. Even blood freshly drawn is so rapid in its alterations that the study of it is one of the most difficult tasks in the whole field of pathological investigation. Consequently to-day, our knowledge of the fluid, both in its normal and pathological conditions, is far behind that which we possess in regard to nearly every other organ of the body. Quite recently, however, more attention has been given to the study of the pathological states of the blood, especially in connection with infective disease, and this work has yielded abundant results, which are still to be held *sub judice*. In connection with syphilis and some of the exanthematous fevers, peculiar corpuscular elements of low forms of animal or vegetable life have been shown to be present with tolerably constant persistency. How far the presence of these morbid elements in the blood can be looked upon as the essential cause of the diseases with which they have been found remains to be proved, and is, in fact, the essence of their pathological signification. In connection with malaria, a disease to which rheumatism bears a closer resemblance than to almost any other, except gout, in the whole nosological catalogue, a newly discovered fungoid

growth[1] has been shown to be present in the blood both of patients and infected animals, and a similar minute organism has been found in the soil and air of malarious districts.

It yet remains for us to find some agent or property which, newly formed in the blood under the influence of sufficiently exciting causes, leads to the outburst of that train of symptoms which we at present include under the head of rheumatism; and also to show whether it is the same agent or product acting within the system that gives rise to both the acute and the chronic forms of the disease, as well as to the form included under muscular rheumatism.

The presence in excess of such an agent as lactic acid in the blood, which was at one time supposed to be the materies morbi in rheumatism, by no means proves that this material, or any other which may be discovered, is the essential cause giving rise to the disease. The presence of the morbid material may be due to the same cause as the disease itself—may, in fact, be only a symptom, may be a result and not a cause.

Failing to find any efficient element or basis in the blood itself which may be regarded as the cause of the disease, the solid tissue remains as yet unsearched with the degree of care which can be called at all thorough. So far, we have only had the general statement made that the articular and fibrous tissues, which are specially implicated in the disease, show no evidences, or only very slight ones, of inflammatory or other changes. It is not improbable that this field of research, like so many other unsearched fields, may yield the most profitable return.

Finally, the results both of physiological and of pathological experiments have been as yet untried. As yet no one has suggested a clue to the line in which such examinations should be conducted; but until the trial has been made in these various directions, the search for the pathological relationships of rheumatism must not be abandoned.

Rheumatism, in all its varieties, as at present classed, is a group of maladies founded on an etiological basis. Away from it have been separated various diseases as rapidly as a firm pathological foundation has been discovered for them, and it is an old block from which many pieces have been hewn away. How far the remainder is a homogeneous mass can only be known when rheumatism itself has been shown to be a consistent pathological entity, and its pathogeny is discovered. The etiolo-

---

[1] *Bacillus malariæ.* Klebs and Tommasi-Crudeli, 1879.

gical foundation on which it rests at present is a chill, or some atmospheric influence of cold and moisture. Such influences may be a good and sufficient *cause* for the disease, but it is not a valid reason for classing together such a variety of affections, having merely one factor in common, viz., pain. The older nomenclature, from which the present one is a derivative, or, in truth, the remains, or a survival, was more nearly accurate, and certainly more consistent, for it included all the other diseases acknowledging the influence of cold as their cause, and likewise having the symptom, pain, as at least one of their principal phenomena. The separation of these various diseases and their classification under different pathological headings has not been altogether logical, and, so far as rheumatism is concerned, has not advantaged us. The class rheuma might well stand to-day with its subdivisions: pleuritis, pneumonitis, mucosæ or catarrhalis, and the other "rheumata." All the latter of these diseases, as fast and so far as their pathologico-anatomical characteristics were known, have been put in proper and consistent groups, and by these discoveries our knowledge has been advanced; but rheumatism still remains unknown and unclassified, as the residuum of former ignorance.

While, as has been shown, it is impossible to regard all the diseases now described as rheumatism as identical either in their causation or pathology, it by no means follows that the term is to be rejected from our nomenclature. In fact, any attempt to effect its displacement would be attended with failure. The efforts which have more recently been made by German authors, as well as others in our own language, to designate the disease by other titles, have met with but little success, and have not tended to advance our clearness of conception or knowledge of the essential factors of the malady. Clearly it has not been the change of name which has led up to the essential pathology of any of the diseases which have been separated from rheumatism. The steps in the process have been just the reverse, and a knowledge of their pathology has shown many of them to be so diverse from rheumatism, that a change of name became imperative. A name so familiar as rheumatism would be difficult, indeed, to displace from our language, even were its pathology most fully known. The word itself implies no pathological signification, and, therefore, like many terms similarly applied, may be usefully retained. The name, cirrhosis, as applied to the liver, formerly meant merely a tawny color; now, however, it signifies the changes which lead in part to the production of that coloration, and is applied not only to the liver, but to

various organs—the kidney, lung, etc. The same may be said of apoplexy. And while in respect to both apoplexy and cirrhosis, since their morbid anatomy has become fully known, so, in respect to rheumatism, the word still lingers in the language; and, in spite of all discoveries in respect to its nature, will continue to remain, just as these other words have so long done. Even in the new German nomenclature, the authors have been compelled to retain the word as an adjective; thus, rheumatic polyarthritis or rheumarthritis, chronic rheumatic arthritis, and rheumatic myopathy or myalgia but partially displace the term commonly in use. This use of the word in its adjective form has, in their view, much to recommend it; and whatever in the future may be discovered in relation to rheumatism and its pathology, or however much the term itself may be discarded, still the word "rheumatic" could always be maintained with advantage, or even extended in its use, as signifying that great group of diseases having certain etiological features in common, and recognized as dependent in their origin on certain external causes, as cold or atmospheric influences, until, at least, the other various causal factors have become known.

Synonyms.

The following names have been applied, by various authors, to rheumatism. Thus we find it called arthritis, rheumatismus, acute rheumatism, articular rheumatism, rheumatic fever, rheumatic polyarthritis, rheumarthritis, polyarthritis synovialis, febrile rheumatism of the joints.

Definition.

A definition does not add much to our knowledge of a disease, but it seems to be demanded, serving as a row of pins on which to attach our ideas.

Rheumatism is a constitutional disease, attended with febrile disturbance, and with inflammation of structures in and around the joint, and often of other organs of the body, especially of the connective-tissue group. The disease is not infectious. The inflammation does not lead to deposit of urate of soda, does not lead to suppuration, and is erratic or shifting in character; by many is regarded as specific or peculiar, but its peculiarity is evidenced only by so violent an action leaving so little trace behind it.

CHAPTER II.

FORMS AND VARIETIES OF RHEUMATISM.

Its Varieties and their Classification—Its Nomenclature—Reasons for a Change of Nomenclature.

Forms of Rheumatism.

It is obvious, from what has been said of the history of the disease, that the distinctions between the various forms of the disease and the significance attached to the various symptoms have been estimated very differently at different times. Among the older writers very many conditions were held to be rheumatism, or a catarrh, a "defluxion of an acrid humor, generated in the brain, into various parts of the body." They were not speaking, however, as we do, of a distinct disease, and the forms of rheumatism enumerated by them must be considered as a multitude of diseases classed under one head.

The teachings of humoral pathology, with its acrid humors, even later still confused a variety of disorders under the general term *rheumata*, and it is not until still more recent times that one cause or one humor became identified with one disease which included only such manifestations as were held to be rheumatic.

When the humoral view of the origin of the rheumatic symptoms became less common or was abandoned, their cause was attributed to a dyscrasia of the blood and especially of the tissue involved in the diseased process. Both of these views required and received a special classification, the one according to the kind of humor which was supposed to be present as the cause of the disease, the other according to phenomena which the peculiar dyscrasia entailed and the effects which it produced, or, speaking in the language of modern morbid anatomy, of its pathological changes.

Every disease in which the influence of cold or "catching cold" could be shown or assumed to have preceded the painful symptoms, was classed as a rheumatism or rheumatic, and an endless confusion in nomenclature

resulted. It will be perceived that this is a very different assumption from that of the older school of humoral pathology. At both times and by both sets of writers very many different diseases, having a very varied pathology, were confused under one heading, but by the older writers they were all regarded as one disease dependent on the same humoral vice, while by later writers they were seen to be different diseases characterized by numerous unlike changes, but produced by the same causes.

This may be termed the "causal classification" and the only things which rheumatism of varied forms had in common were an identical cause, viz., cold and the general symptom of pain. The humor which on the one hand was developed by cold, and on the other hand produced the pain symptom, might, so far as their teachings go, be single or multiple. The effect the dyscrasic or solidism pathology had on the teachings of the causal school, was a demand for the classification of the forms of rheumatism, according to the tissue or organ affected. Following the writers who advanced these views, rheumatism formed a family of diseases, separated into forms or species according to the tissue or organ involved, and required a different means of treatment accordingly.

The various views of inflammation have exercised their influence in the classification of rheumatism, and when all diseases were regarded as inflammatory in their nature, the many forms of rheumatic disease were grouped according to the grade of the inflammatory process. Thus we find hyper- and hypo-sthenic and asthenic rheumatism. Since the time of Scudamore, the forms of rheumatism which he proposed and which probably have nearly the same significance as those just given, have to a great degree held their position until the present time. Scudamore included several painful affections of the nerves among rheumatic diseases, which are no longer recognized as of this nature, and although he speaks of the muscular form of the disease, did not separate it into a class by itself, as has been more recently done.

As has already been said, Ballonius first used the word rheumatism, in the sense now intended, for pains in the joints and muscles, and distinguished them from gout, which had previously been confounded with them as a general arthritic affection. However, since then even until now many vague pains, as well as some of another character unquestionably, have been called rheumatic, and while the settlement of the question of the more characteristic forms of rheumatic disease and their differentiation from diseases having symptoms of a more or less similar character

is comparatively an easy matter, even supposing the cause of the malady to remain unknown. But as we depart from this group of forms, the distinguishment of many vague pains and set of symptoms truly rheumatic from others of a different order becomes, almost progressively, more and more difficult—so difficult and uncertain that many recent observers, swaying as far away from the probable truth as did the older writers in the other direction by including every pain as rheumatic, are inclined to limit the bounds of rheumatism to the acute and chronic articular affections and include everything which has heretofore been joined to this class. The disposition to effect the separation is shown as much by this as by any direct statement, that names are applied to these other affections from which the word rheumatic is omitted.

Its Varieties and their Classification.

There has been, both in earlier times and recently, a tendency exhibited to change the names and classification of the well-recognized forms of rheumatism, in accordance with the prevailing view of the nature and supposed cause of the malady. At one time it was regarded as a general or infectious disease, at another as a fever, and again merely as a peculiar inflammation of the joints and other structures.

Scudamore divided rheumatism into three forms: acute, chronic, and subacute, the latter intermediate in its symptoms "between the high constitutional fever of the one and the absence of fever of the chronic." Chomel pointed out the order—the muscular, the articular, and the visceral, with numerous sub-orders, according to the locality and the activity of the symptoms. Fuller proposes four divisions of the disease, viz., acute rheumatism, rheumatic gout (differing from both rheumatism and gout), chronic rheumatism, and neuralgic rheumatism. What may be accepted as the latest German classification, as given by Senator, in "Ziemssen's Cyclopedia," is that of rheumatic polyarthritis or rheumarthritis, essentially a constitutional disease; chronic rheumatic arthritis and rheumatic myopathy or myalgia, affections of a local nature. Hueter calls rheumatism polyarthritis synovialis, and divides it into acute and chronic, while the other arthritic affections, though included in this title, are designated by names indicating their cause, for example, pyæmic, gonorrhœal, etc. In this country, as well as in England, rheumatic fever has been a common designation for acute rheumatism, but has not found general acceptance;

the objection which has been offered to this term is that it implies an infectious disease similar to the general class of fevers, while on the other hand this has been contended to be the more correct designation, because in this form of the disease, the pyrexia is the chief factor as distinguished from the joint trouble; in other words, that the fever produces the joint affection rather than joint involvement the fever.

Senator contends for the employment of the adjective "rheumatic," as more logical and consistent with our deficient knowledge of the pathology of the disease, and that "rheumatism" cannot be maintained in a nosological category and is "not merely superfluous but positively detrimental."

Its Nomenclature.

While all writers are not agreed on, and some do not believe in, any one underlying pathological entity as the cause of the various forms of this disease, still, so long as no certain basis of causation is discoverable, I do not believe it is possible to alter the nomenclature of the rheumatic affections and to obtain common concurrence to a new division of its forms. Neither do I think that the attachment of new names to the old forms is of any great advantage, unless perhaps it can be shown that such a change will alter the current of medical thought and investigation. If a change of name would suffice to disabuse the mind of the crude notions of the ancient humoral pathology, or lead us to think of rheumatism as something that is not merely synonymous with "cold," every one would gladly consent to the removal of the old landmark according to which our thoughts have been for ages directed. The confusion necessarily attendant upon such changes, while temporarily inconvenient, is willingly and gladly submitted to whenever the exact nature or cause of a malady, formerly confounded with something else of a similar character, is pointed out. Our ideas cluster in new groups around another set of phenomena and their cause, and thus the advancement of scientific medicine is promoted. Whether this will be the result of the efforts of the German observers, who propose new names for the chief of the rheumatic diseases—as has already happened for various ones of the lesser grades of this disease, for example, neuralgia, some forms of sciatica, and rheumatoid arthritis—remains to be seen.

Still, I do not think that even the most radical change of names yet

proposed is likely to effect any marked results. Hueter's proposed divisions of these diseases on an anatomico-pathological basis, while it is by far more accurate, and perhaps more scientific than any previous classification, is certainly not sufficiently comprehensive to include all the phenomena which we think of as rheumatism; neither does it have the merit which, as already said, is the only one which any change of names can possess—save the one founded on the yet undiscovered exact cause—of directing our thoughts into a new pathological channel. To speak of rheumatism as a synovitis is, it would seem, a step backward rather than forward in our general notions of its pathology, and it is also difficult to conceive wherein there is any gain, except from a purely morbid anatomical aspect, in classing gout, rheumatoid arthritis or arthritis deformans, gonorrhœal and syphilitic rheumatism, as well as chronic and acute rheumatism, under one heading. Polyarthritis synovialis acuta and chronica, and those occurring in the venereal subject, poly-panarthritis, and panarthritis urica, are not any more distinctive as names than those, so long familiar, for which they have been substituted. We do not wish to see gout classed again with rheumatism, from which it has been separated. Rheumatoid arthritis has too lately been placed on a separate basis for us to think of its re-alliance with chronic rheumatism with satisfaction. Gonorrhœal affections of the joints, too, have been shown to have probably a septicæmic origin, and the name of rheumatism, with which they have nothing in common except locality, ought never again to be joined to them, or the two diseases to form parts of one class.

Reasons for a Change of Nomenclature.

Until there is some improvement offered, it would seem best to adhere to the classification already in use and so generally accepted.

Acute rheumatism forms a thoroughly well-understood class of symptoms, of which, perhaps, it may be claimed that the nature and cause, or causes, are not definitely known, yet it immediately calls to mind a distinctive picture of disease. The distinctions of acute and chronic imply two things, viz., the duration of the malady, and the activity of the symptoms or of the morbid process. The acute form is always characterized by greater or less activity, and usually, though not always, this activity is of short duration; while, on the other hand, the chronic form is slow in development and changes, and long in duration, though not always so.

By many writers the word *articular* is added to the name of the disease, yet, I think, without advantage. All who believe that rheumatism is an articular disease, do so quite rightly; but such a designation would seem to detract from its general or constitutional characters. If it is intended merely to call attention to the common feature of the disease or the special phenomena of individual cases, the title is beyond criticism. It must be remembered, that among both acute and chronic cases of rheumatism there are, especially of the latter, instances in which the articular symptoms do not bear emphasizing, and appear as subordinate phenomena of the attack. The number of such cases may not be numerous; still, in this disease, which is so pre-eminently, nay, almost universally, a joint disease, they do occur, and should receive due attention in the nomenclature of the malady. Much less proper is the name—more recently applied by German authors—of polyarthritis; the number of the joints affected is not an essential feature of the disease, and, while it is exceedingly rare to see a case, especially of acute rheumatism, in which a single joint is attacked, chronic rheumatism affords very numerous examples of monarthritis. Senator, in his classification, pays deference to this difference, and distinguishes acute rheumatism as a polyarthritis, and chronic rheumatism as simply a chronic rheumatic arthritis, but Hueter applies polyarthritis to both forms.

Acute rheumatism is a constitutional disease, attended with fever, pain in various parts of the body, and almost invariably in several joints, in and around which evidences of inflammation and exudation are commonly present. The joint symptoms are often peculiar, fugitive, and erratic, but never result in suppuration or the deposit of sodium urate. The internal organs are affected often by inflammatory changes, especially the endocardium and the serous membrane.

Subacute rheumatism is a milder form of the disease, but is essentially the same affection; there is little or no fever, usually fewer joints are affected, and the symptoms in connection with them are less intense; the general vague pains of the muscles are usually present in this form, and are more marked, or, at least, more noticed, than in the acute form. The division into the acute and subacute forms is made for the sake of convenience of distinction, and does not indicate any essential differences between the two, except as to the grade of severity.

By some writers, subacute rheumatism, as I have defined it, has been confounded with the chronic form of the disease. To do this seems to

me unjustifiable; subacute rheumatism, in respect to the age and class of the patients, the circumstances which seem to produce it, as well as its course, although its duration may be more prolonged, exhibits a great similarity of type to that of the acute form. As has already been said, chronic rheumatism is not only of long duration, but its symptoms are of an inactive character throughout; it is conspicuously attended by changes in and about the joints, which are of a progressively increasing tendency and of a permanent character. Subacute rheumatism may originate an involvement of internal organs in organic changes; chronic rheumatism probably never develops them.

We may define chronic rheumatism—a disease so unlike in type the other forms of rheumatism, that many are inclined to regard it as a different morbid process—as a collection of symptoms, having, so far as known, almost only one phenomenon in common with the other forms, viz., the pain about the joints. It is unattended with fever, or this symptom is so slight that the increase of bodily heat is probably due to the pains present in the aggravated cases. There is no evidence of acute inflammation of the joints or their annexa, or of the other locomotory apparatus; but there is more or less constant pain of these parts, aggravated, or, perhaps, only produced by motion, and there is a very marked tendency to the production of various anatomical alterations of these parts, progressively increased the longer the disease lasts. It is a disease of the decline of life, frequently is consecutive to acute rheumatism, but very frequently appears without relation to the acute disease, is nearly perennial, its symptoms or effects are everlasting in the person attacked, and some of its phenomena are usually more or less accentuated by changes of weather or barometric pressure.

Some writers describe the chronic variety as composed of cases without active symptoms—*i.e.*, the milder cases of subacute rheumatism—and others combine with these nearly all the muscular and nerve pains (excluding the neuralgias) as well as those here defined. I think it is important to exclude these forms so far as possible, and consider only such cases as are chronic in both respects, viz., as to duration and as to activity of symptoms. It would be very desirable, for the purposes of classification merely, if it were possible, to form a class of chronic rheumatism cases in which the main feature is the joint changes, although we rarely find such cases unattended with pretty constant muscular pains. These cases, then, would, as a class, form a parallel with the main bulk of rheu-

matic affections, viz., the articular rheumatisms, the one being chronic, the other acute.

For the purpose of naming or classifying the rheumatic affections, a large number of authors have formed a group for muscular rheumatism. As has already just been said, many writers class this form of the disease with chronic rheumatism; there are cases, however, of muscular rheumatism which, I think, belong to acute rheumatism, and are as truly instances of rheumatic fever as the articular cases. This point has already been alluded to in objecting to the name, acute articular rheumatism as the heading for the bulk of the cases of rheumatic fever or acute rheumatism. Many, if not all, of the cases of so-called neuralgic rheumatism, I think, should be included among the muscular rheumatism. This variety is also called rheumatic myopathy or myalgia, and authors have described under it lumbago, as well as pains of this character occurring in other muscle-groups under special names.

It will be seen at once that, as soon as we pass beyond the confines of the group in which the rheumatic disease is attended with fever and a few special symptoms and complications, the classification becomes one of merest choice or whim, and is solely dictated by convenience of naming or pointing out special phenomena. The line of demarkation between the forms of the disease in all the remaining groups is a very faint one, and is at one time overstepped, by one symptom, again by another, which has been held to as defining the group to which the individual cases belong. Thus, a case of chronic, or even muscular, rheumatism, by a slight aggravation of its symptoms, or the development of new ones, may be looked upon as a subacute case.

It is unquestionably true that many cases regarded as rheumatic, especially those of chronic rheumatism, are of an entirely different nature. So long, too, as we are uncertain, or for the most part wholly ignorant of the nature of the acute form of the disease, it is manifestly unfair to attempt to be positive concerning what now are looked upon and described as its less marked manifestations. And in speaking of forms and varieties of the disease, we should disabuse our minds of the idea that the same essential malady is implied by these terms. The names and classification are useful, and even necessary, for the purposes of studying the phenomena of disease; but as rapidly as true pathological entities become known, they are gladly removed to a separate and appropriate class.

CHAPTER III.

CAUSES OF RHEUMATISM.

Causes: Hereditary Predisposition, True and False Heredity; Age, its Effects on the Various Forms; Sex; Connection with General Health and other Diseases; Climate, Country, Race, and Change of Habitation; Seasons, Atmospheric and Barometric Influences; Occupation and Manner of Life; Temperament and Constitution; Injuries; Sleep; Former Attacks and their Effects; Drugs.

Causes.

The wide-spread occurrence and the varied forms of rheumatism indicate that the *cause* of the disease must be a very generally prevailing one, and that the conditions under which it is produced are equally varied. The uncertainty of its cause, in addition to the defective knowledge of its true pathological condition, is one of the reasons why so many, and probably, dissimilar morbid processes have been included under the term rheumatism. It does not necessarily result that because the forms of the disease are varied a different cause is acting to produce them; the same cause under different circumstances, both internal and external to the individual, can result in varied phenomena. The conclusion that the cause is a general one because of the frequent occurrence of a single form, as well as all forms of the disease, cannot, however, be escaped. That the cause or the causes which produce the multifarious train of symptoms included under the head of rheumatism are varied ones, must, I think, be concluded, partly from the very variety in the symptoms and partly from the different circumstances under which the disease occurs.

Hereditary Predisposition, True and False Heredity.—One of the many circumstances included and generally spoken of under the causation of rheumatism is the *hereditary predisposition.* Naturally, statistical methods have been brought into use to substantiate this condition and relationship, but, as is self-evident, so long as both the cause and the pathological condition of the disease remain unknown, such methods of proof remain un-

fruitful. The most that statistics are capable of showing is that the disease recurs in families in a certain percentage of the members. No amount of statistics would make us believe that malaria is an hereditary complaint, or that small-pox in the family of a pest-hospital steward is transmitted from father to son. On the other hand, disease may be distinctly hereditary in its nature, yet the number of cases resulting from this cause may form a small proportion of the whole. Disease is transmitted by heredity with varied intensity in different maladies, and under dissimilar circumstances in the same malady. Similarities in the mode of life and the surrounding circumstances may frequently so impress themselves on the individual that a false likeness to hereditary transmission is given. The notion is common, not infrequently, in some secluded communities, that fevers, especially typhoid, are prevalent and fatal in certain families, whereas the cause of their occurrence, now so well known for this type of fever, is to be traced to defective sanitary conditions. Before the knowledge of the mode of propagation of typhoid fever, a prevalent disease of rural life, became diffused, we were in the same relation to the question of hereditary transmission of this disease in which we now are in respect to rheumatism.

In ancient times, when the rheumatic affections, so called, presented a confused jumbling of many dissimilar and distinct diseases, the question of their hereditary transmission was one little adverted to or discussed. Later the existence of hereditary structure was assumed as an occasional predisposing cause of rheumatism, although the authors of that period regarded its occurrence from this cause as an inference to be drawn only from general reasoning, and not from a positive demonstration of the fact. Similar circumstances of climate, local situation, and mode of life were considered more powerful in their action in the production of it in the members of the same family than its transmission by blood relationship. It was pointed out that individuals of a family subject to rheumatism escaped the supposed family taint by removal of their place of abode or by changes of habit.

It is only quite recently—since the introduction of the statistical methods of study, often so vicious in their results and rarely conclusive by themselves—that a strong predisposing hereditary tendency has been claimed. This belief can, I think, be traced in its origin to tradition, handed down as a common saying rather than to any accurate observations; at least, this is true in a great measure of the origin of the belief. It probably

started in earlier times, when a clear distinction and separation was not made between rheumatism and gout. Chomel was among the first to develop strongly the fact of its supposed hereditary nature, in his "Clinique Médicale" (1837), although he does not mention this predisposition in his earlier "Essai sur le Rhumatisme." Apparently it was not until he came to study gout with rheumatism that this relationship of the disease was developed by him. Fuller[1] fully develops his belief by the statistical methods of the hereditary nature of the disease. Among the patients admitted to St. George's Hospital he found an inherited predisposition in twenty-nine per cent. of the cases; among those in the Hospital for Consumptives, twenty-four per cent.; among the insane, thirteen per cent. Chomel states the number at one-half the cases; and Garrod at one-fourth. Many others who unquestionably confounded gout with rheumatism, repeat the statement of Chomel and are firmly convinced of its hereditary nature.

All observers, however, who not merely quoting the opinions of others, have investigated the question for themselves, have not clearly distinguished the essentials of hereditary transmission or the nature of heredity. There is a physiological heredity, the full expression of whose nature is seen exemplified in the congenital forms of syphilis, and from this strongly marked and high grade of development we find the hereditary taint in other affections exhibiting a less and less influence until its effects are scarcely traceable. And there is a statistical heredity, the only proof of whose existence consists in figures. If the proportion of cases of the disease occurring first in the parent and recurring in the offspring is nearly equal to the whole number of cases, the likelihood—nay, almost certainty—of its hereditary nature becomes nearly positive, and if, in addition, we know the character of the malady is such as not to render it improbable, or is, in the first instance, of an infectious quality, the matter becomes one almost of demonstration. The further the facts and conditions depart from this standard the less certain must become our assurance of the hereditary nature of any disease. That a rheumatic parent's child suffers from rheumatism, by no means confirms absolutely the hereditary character of the malady; neither does the escape of the offspring in any given number of instances disprove with any certainty the possibility of its hereditary transmission.

The determination of the hereditary nature of a disease, especially the proof of the negative proposition, is a matter of great difficulty, but I

---

[1] On Rheumatism, Rheumatic Gout, and Sciatica, 3d ed., 1860, p. 31 et seq.

think it will be found that a very much larger number of people whose parents suffered from it escape rheumatic affections than become rheumatic themselves, and I believe the converse of the proposition is also true. Thus, so far as the numerical facts of the case are concerned, the showing is uncertain and indefinite, and, until the nature of the malady is better known, must remain so. To know that a certain proportion of the offspring of rheumatic parents become themselves rheumatic—at least so long as the total of the one falls short of the whole number—is not an item of information of any special value, either in the prognosis, diagnosis, pathology, or treatment of the disease, and it certainly does not go to the settlement of the question of the hereditary, the true physiologically hereditary nature of the complaint.

On the question of the greater or less strength of the supposed hereditary tendency of rheumatism, the unsupported opinion of most writers is that there exists a strong hereditary tendency in its development. The popular notion is likewise to the same effect. And these two concurrent opinions can be called in question only on two grounds: first, a lack of evidence or an insufficiency in the number of cases in which the supposed tendency is exhibited, by which it appears that, at most, if such a tendency exists, it shows itself in but a low degree; second, a failure from any cause to apprehend correctly the nature of an hereditary taint or transmission.

Now I have no records of my own with which to substantiate or to disprove the correctness of the law of transmission. In my earlier notes of cases the matter was carefully inquired into, but later the inquiry has been abandoned, partly from my alteration of views in relation to the hereditary transmission of diseases in general; partly from it becoming obvious to me that, at least among hospital and out-patient cases, the replies were incorrect or confused, and partly because I found that inquiries as to hereditary diseases in general developed the fact that rheumatism in the parent was quite as often absent in the offspring as present.

That the disease is not strongly hereditary, or at least physiologically hereditary, in the same sense as syphilis and tubercle, in its narrower sense, is, I think, strongly indicated from the facts which will be discussed later, viz.: the age at which the malady develops itself with greatest frequency. Tubercle and syphilis, traced to an hereditary or congenital origin, do not lie dormant in the child; if they do not manifest themselves at birth, a very few months, at the most, serve to give rise to their most

frightful phenomena. Cases of tubercle occurring in early adult life, in children of tuberculous parents, are open to more than a suspicion of having originated *de novo*, rather than tracing their causation to heredity, for we know of so many causes which are capable of grafting a tubercular disease on a perfectly healthy stock; much more then is this likely to happen in a child whose vitality is vitiated by the ill-health of its parents. No one, of course, argues that a young man, previously healthy, who exhibits manifestations of syphilis at the age of eighteen years, inherits syphilis, because his father suffered from syphilitic disease at a similar age, or even if he was tainted at the time of the child's procreation. And yet we are called upon to believe in the hereditary nature of rheumatism on very similar evidence.

The reason of our disbelief in the hereditary nature of such a case of syphilis is that we know both the cause and the nature of the venereal infinitely more closely than the rheumatic disease. We do not need an array of figures to substantiate the proposition, and even if statistics showed that every son had syphilis at this age—and consequently every father, and it approaches lamentably close to it—no one with the sense of reason properly trained could be made to believe that syphilis, under these circumstances, was hereditarily transmitted, although nothing can be more certain, unfortunately, than the facility of its hereditary transmission. The same may be true of rheumatism, and yet, so far as our knowledge of its cause and its nature affords light on the subject, we are not in a position to either affirm or deny to it this tendency.

The histories of families, even many instances within my own knowledge, afford as numerous examples of the failure in transmission as the reverse, and it would be just as unfair to argue its non-transmissibility from this fact as to attempt to affirm its opposite character from similar facts. There exists a much greater difficulty in an attempt to prove a negative, and there dwells a strong tendency in the human mind to conclude an affirmative from a purely numerical basis. We constantly jump at conclusions from a ratio, whilst the true basis for the decision of the question rests on the ground of cause and effect, which, in respect to rheumatism, is as yet unknown.

The question needs to be studied by methods as yet unused for its decision. Hitherto its hereditary tendency has been concluded by collecting the answers of a set of people who, as a rule, are ignorant, unobserving, and perhaps prejudiced in favor of the one view of the question, and

who, to increase the magnitude of their suffering, are capable of wilfully distorting facts. It is not surprising, therefore, that sufficient evidence has been collected to produce a strong impression in favor of the hereditary character of rheumatism. I believe there would be a very different showing produced, supposing that the statistical method of proof has any considerable value in deciding the question, if we were to ascertain how many descendants of rheumatic patients escaped the disease, or determined how many of those patients, now classed as rheumatic by heredity, were born prior to their parents suffering from the disease. And while I believe that such inquiries would show a different result, or would change our opinions from what are at present held, I do not think we should be any more certain of the true characteristic of rheumatism in this respect than we are at present. Such an inquiry involves an immense amount of labor, and from the lack of knowledge of a large number of patients, could only be made to cover the ground but very partially.

Without spreading our labor over a large field, likely, at best, to yield a barren result, another method, much more accurate and definite, presents itself for trial. Let there be a careful inquiry made of the life history of offspring procreated at or shortly after the time either parent suffered from rheumatism, and before the rheumatic poison or taint can be supposed to be lost ; if such records can be compared with brothers or sisters born previous to or long subsequent to a single outbreak of rheumatic disease in the parent, great value will be added to the etiological history. A few such instances are of more value than a century's record of thousands of hospital patients. Great force would be given by every case in which the child was reared in circumstances as unlike those of the parents, both in respect to climatic influences, locality, and mode of life, providing, of course, that these circumstances were not more favoring to what we now look upon as productive of the disease. Such observations, to acquire a positive value, would require two or three generations for their completion, and vague or general recollections cannot be accepted as of any value, for the whole question depends on time, and time limited within a few months with great precision, for which no memory possesses the requisite accuracy.

The number of instances of such a concurrence of circumstances and of disease, in the experience of any one observer, must necessarily be very limited. The limitation results from many causes, two of which are especially controlling in their influence : firstly, a rheumatic attack of such

force as can carry with it an undoubted power of infection, whatever its nature may be, is of short duration ; and secondly, the concomitant phenomena and symptoms of rheumatism are distinctly unfavorable to the consummation of the procreative act. In both of these respects, therefore, rheumatism differs in a striking manner from both tubercle and syphilis, which, while they may be no more chronic in their duration, yet during their chronicity exhibit such active and, in this relation, therefore, forceful phenomena, sufficient for us to presuppose a more potent influence for their transmission than in rheumatism during its chronic stages. A few instances of the transmission of a rheumatic taint under the circumstances here supposed would not only serve to confirm, in the strongest manner, its heredity, but would afford a flood of light as to its nature—a much stronger light than the present vague notions now held have furnished to the consciousness of a careful and thoughtful observer.

Age: its Effects on the Various Forms.—It is generally stated that age is an important circumstance in the occurrence or causation of rheumatism, and the limitation at which this factor begins or ceases to act are variously stated by different observers. In respect to the occurrence of rheumatic fever or acute rheumatism (polyarthritis), the limits within which first attacks are most frequently seen have been pretty accurately studied ; and in this form of the disease the first half of life exhibits the most frequent instances. To the chronic form of the disease the last span of life is especially liable and is the favorite period.

Only one case of acute rheumatism has been recorded under the age of one month, and instances of the form of the disease in children less than four years of age are so rare and unexpected that their diagnosis is one of difficulty, and they are to be viewed with suspicion and carefully to be differentiated from other forms of joint disease, as well as from other diseases in which joint affections may arise as a complication. At the other extreme of life, the instances of first attacks of acute rheumatism become equally rare, although the uncertainty in respect to their nature, when they do occur, is much less than in early childhood. Among the older authors the years within which acute articular rheumatism was stated to occur were extended much beyond the period in which they are now recognized, and while it is probable this difference in the observations is due to some extent to an altered mode of life in more recent times which have lessened the liabilities to such attacks, yet much more is to be attributed to a more perfect recognition of the disease.

Many diseases have latterly been recognized to have a different character and have been placed in separate categories ; this is especially true of certain diseases occurring in the early period of life. Syphilitic and pyæmic joint and bone affections and also many instances of gout have become known in their true characters and have been partitioned off from the rheumatic diseases. This recognition has removed many instances of other diseases from this class, not only at the two extreme periods of life, but likewise from among those coming during more favored periods for the occurrence of the disease.

From five years to seventy was formerly stated as the period within which acute articular rheumatism occurred ; but certainly the instances of first attacks, or indeed any acute attack, after the age of fifty must be extremely rare. The period from puberty to thirty or thirty-five years shows by far the largest number of cases.

How far age, as directly expressive of bodily states and the state of the tissues and functions of the organs, is a true cause of acute rheumatism must certainly remain in doubt from the uncertain nature of the actual cause which occasions and the pathological change which accompanies this disease. In childhood, there are certain maladies which are directly to be attributed to the condition of the tissues and their functional activity, or at least to the short elapse of time since their fœtal condition showed them to be in another state. Thus, the epithelium of the lung alveoli are directly disposed to catarrhal inflammation in early childhood for this reason, and here age is a direct cause of the catarrhal pneumonia of children ; yet by far the larger number of cases of catarrhal inflammation are found in adult life, at a period at least sufficiently removed from the developmental stage of the lung tissue, so that the latter must be supposed to have lost completely its embryonic tendency. The catarrhal inflammation of adult life is due, then, not so much to the age of the subject, so far as tissue is directly influenced by its age or period of development, as to incidental causes connected with the mode of life, occupation, and natural exposure of the person.

Further, as has already been expressed in speaking of the hereditary tendency of this disease, while we not unfrequently see mothers suffering from rheumatism shortly before the period of conception, and therefore especially liable or able to convey to their offspring a rheumatic tendency, we do not find that rheumatism is frequent in the early years of life. It is not until later, after the habits of the child have changed to the more

severe and exposing surroundings of adult life, that rheumatism becomes a frequent disease. Therefore, acute rheumatism is in all probability not only not hereditary, as has been said, but not directly caused by age as such, but to the incidental facts of a different period of life. It becomes a question whether acute rheumatism would not be of equally frequent occurrence in early childhood as it is in early adult life, if children lived as their older brothers live. I do not wish to be understood as referring merely to the exposure incident to adult life as productive of a greater frequency of the disease ; it is necessary to include with the difference in clothing, outdoor life, and exposure, the equally important factors of harder work, different food, mental or other nervous strain, as well as other differences of function present at the different periods.

If children, and old people as well, invariably live and act differently from young adults, certainly in one sense age may be looked upon as a cause of acute rheumatism, and the determination of the efficacy of age in its production might become a matter of importance in a diagnostic point of view, if not also in our methods of prevention, as so frequently occurs in other diseases ; but certainly the diagnosis is clear enough in these respects without this aid.

Neither can age as the cause of the production or the reverse be brought in question here as in reference to many other diseases—for example, the exanthemata of childhood. The rheumatic diseases are not absent from any period of life because one attack has lessened the susceptibility to a return of the affection ; in fact, quite the reverse is the case. And neither is the disease ever the result of contagion. Scarlet fever, measles, etc., have their favorite period in childhood to an almost universal degree, because no one can well escape contact with the infection or contagion, rather than because childhood is a cause of the disease. Isolated communities of adults never exposed to these diseases show but little lessened susceptibility to the influence of their contagion when brought in contact with it. Individual cases of freedom from their effects may be found under all circumstances and at any age, but their bearing on the causation of the disease shows almost nothing, while the general rule for all individuals has an important effect on the question of age as a cause.

Age seems directly to influence very greatly the form in which the disease manifests itself ; in the early period of life the acute articular form is by far the most common, while, on the other hand, the chronic form is

very unusual; in the latter half of life rheumatic fever is, as we have seen, a rare occurrence, while the chronic, either succeeding an earlier acute attack or attacks, or having originally manifested itself in this manner, is peculiarly prevalent. Fuller's statistics, collected from his own work and the observations of others, show very clearly the period of life in which acute rheumatism occurs most frequently; they do not show the number of cases of the two forms of rheumatism occurring in the two respective periods of life, but he argues, partly from the general medical observation, but chiefly from the nature of the poison or materies morbi of the disease, and the manner or cause of the origin of this material, that the chronic form of the disease is more common in older subjects than in youth. We may accept the statement with very great certainty of its truth, that advanced age is more liable to rheumatism, though not in its acute form, and also that this fact is directly traceable to changes in the vitality of the tissues and their functional activity. It is probable, although the numerical proof is very difficult of attainment, that there are a greater proportion of people advanced in life who suffer from rheumatism in its chronic and subacute form, and, perhaps, suffer more or less continuously from it, than the whole number who, in youth and early age, are attacked with rheumatism in any of its forms. The proof is almost beyond our reach, for the vast majority of cases of the lesser grades of the disease, in both periods, do not come under observation; but especially is this the case among old people, because it is generally concluded that such pains and stiffening of the limbs are but the natural concomitant of age, which it cannot be hoped to escape, and for which but little relief is to be obtained. That age may exert a strong influence in modifying the form of the disease, and that this is directly due to difference of tissues, incidental to the period of life, is readily to be accepted; that, however, it offers a proof of the nature of the disease or of the actual presence of a materies morbi, and the mode of its production must be a matter of pure speculation. The latter part of the subject must be discussed later on.

We must conclude, therefore, that in early life rheumatism in any form is a rare affection; that from puberty to the age of thirty or thirty-five years acute rheumatism is a very common disease, from thirty to sixty a much less number of persons suffer from acute attacks, but still this form is not rare, and that many more suffer from the subacute or chronic form than in the preceding period. During the middle period very many die from heart disease incident to previous acute attacks. After this period a very much

larger number are affected by chronic rheumatism, in a greater or less degree, and many fewer from the acute form. Taking the whole number of patients, those who have had acute attacks in the earlier period, those who have heart disease from this cause compatible with continuous existence, as well as those who have escaped chronic cardiac complications, and also those who acquire the disease merely in its chronic form, the total suffering from rheumatic affections is much greater in the latter part of life than at any other period. Past seventy years those who have never suffered from rheumatism very rarely acquire the disease, but remain exempt from its inflictions through the rest of their days. Many, however, who have had rheumatism in one or other forms, are still found having the chronic malady. In thus summing up the forms of the disease, and the ages at which it occurs with greatest frequency, nothing has been said of the probability or improbability of the various forms owning and being produced by different and unlike causes. These forms of this disease now classed together are very unlike each other, and have so few phenomena in common, except pain and the place of its occurrence, that many are willing to do for them the office, viz., reclassification, already performed for so many other maladies, which for these reasons, and these reasons only, were previously grouped as rheumatic. This question will be noticed under the separate forms of the disease.

By some observers it is contended that acute attacks develop a tendency to the occurrence of the chronic form of the disease, while others view the acute paroxysms as an effort of the system to rid itself of a noxious material, and that if the paroxysm is fully developed, the effort is usually successful. It will be easy to recognize under these opposing conclusions the bias which the theory of the causation of rheumatism has had in influencing the deductions made from observations. It is to be feared the upholders of the theory of diathesis have been warped to believe that the diathesis once developed—and having expressed itself, as it most usually does, according to them, in an acute attack—thereafter there is entailed on the person a tendency to the continuation, perhaps in a modified form, of the malady and all the dreary evils which the word *diathesis* implies. According to these observers, the greater frequency of rheumatism in advanced life, and especially its chronic form, is readily accounted for, and is the result of an hereditary or a developed or engrafted diathesis. Other observers, who contend for the cause resting on a materies morbi—and these, unfortunately for their argument, hold most strongly to its heredi-

tary nature—point out that the accumulating poison expressing itself in an acute paroxysm frees the system thereby from the morbid matter and remains less liable to attack—that no rheumatic symptoms present themselves until a reaccumulation takes place in the blood. They hold that the paroxysm which is supposed by them to be the means of expelling the poison, is little likely in itself to perpetuate a diathesis, but rather to counteract it. The freedom from rheumatic symptoms immediately succeeding a single attack strongly makes in favor of the purging of the system of a noxious matter and as strongly in opposition to the theory of a diathesis. If the diathesis were markedly enough pronounced to produce a "well developed attack," "the victim of the disease would inevitably be a martyr to rheumatism for the remainder of life." Facts can be accumulated without end, in the multiplicity of the various phenomena of rheumatism in its many forms, to support the one or the other theory, but so long as the cause of the disease remains problematical, the theories must remain unsubstantiated.

Sex.—It is interesting to know whether men or women suffer more from rheumatism—the one sex or the other. Very various opinions have been expressed with regard to the preponderance of cases of the disease in the one sex over the other, and the majority of writers think that men are more liable to its attack than women. Among 7,230 cases of rheumatism in which the sex of the patient is indicated, 4,293 were men and 2,937 women. These numbers include all varieties of rheumatism, and have been collected from many sources—in fact, are the summation of nearly all the statistics which I have been able to find. They are mostly taken from hospital records, and merely show the number of cases, which, from their gravity, lack of home care, or other causes, sought public treatment, and do not indicate the relative proportion which exists in the whole population. There are always, in every community, and especially in the largest cities, more men who seek hospital care than women, and this is found to be the case even when in the general population the number of women exceed that of the men.

There are a number of points to be noted in relation to the statistics of sex and the various opinions expressed by authors on the relative liability of men and women to rheumatic disease. In those countries, especially the rural sections, where the women, by the nature of their occupation, expose themselves equally with the men, the proportion of cases, on a review of the figures given above, are found to approach an equality of

the sexes. Again, in manufacturing cities the same relation is found. A comparison of the opinions in relation to the frequency of the disease in the two sexes shows that many writers are unconsciously biased in their judgment by their views of its causation; those who hold that rheumatism is a diathesis, inherited or acquired, and to which every one is liable who leads a certain peculiar mode of life, naturally disregard the question of sex as in any respect influencing the liability to attacks. Or if the subject is alluded to, they explain the preponderance of one sex over the other by the habitual mode of life which may favor the development of the disease; some, on the one hand, that men expose themselves more to vicissitudes of weather, or work harder; others say that the sedentary life of women is equally cogent. Those who believe that rheumatism is in great part the result of, and is developed by, external agencies, producing, of course, peculiar functional derangements, point with telling effect to its great frequency in men who are more exposed by habit to such influences, thereby at one and the same time finding a cause and showing its effects in producing the disease. Finally, those who hold that a peculiar materies morbi is the efficient cause of rheumatic affections, express the belief that, irrespective of sex, only those, and, of course, all those, who through want, irregularities in living, and neglect of health, tend to develop this poison in their animal economies, are most liable to the disease; such exciting causes as vicissitudes of weather, occupation, etc., of course contributing their share.

The conclusion, therefore, to be drawn, is that probably more cases of rheumatism occur among men than women; we cannot disregard the showing of statistics of this fact, however we may criticise the liability to errors or incompleteness. The statistics do not compel us to believe that sex merely in itself directly influences the frequency of the actual occurrence. It is thought that in early life more men are attacked than women with acute and subacute rheumatism, especially from the greater frequency of this form of the disease among the young; and that after the catamenial period the number of those suffering from the manifestations of the disease become more nearly equal among the sexes. Statistics are neither sufficiently abundant nor accurate to determine these ratios.

Connection with General Health and other Diseases.—Opinions vary very greatly in regard to the influence which the general state of the health has in rendering people liable to, or in directly developing rheumatic affections. It would seem that, in a general way, taking in

view the difference in type, the character and circumstances surrounding the patients, the acute and chronic forms of the disease were not influenced alike by the state of health of the individual. The periods of life at which the two forms respectively are wont to appear with the greatest frequency lend probability to the truth of this opinion. Acute rheumatism is most common at an age when life is most active and health usually the most robust, while the chronic form is found most frequently in conjunction with a decline of bodily function. Of course, many exceptions to this general statement are met with, and too much weight in the decision of the question must not be given to these circumstances, unless we are, in the first place, prepared to show that individuals furnishing the instances of the disease are, on the one hand, in the cases of acute rheumatism, in the enjoyment of good health previously to or at the time of the attack; and, on the other hand, that the elderly patients are likewise enfeebled by age. Age and state of health must be rigidly separated and considered apart, and the one must not be held as implying the other.

So frequent, however, is the coincidence between the occurrence of a rheumatic fever or the development of chronic rheumatism and certain morbid conditions, of which it is unjust or misleading to speak as disease, that I think we cannot escape the conclusion that the state of health does have an influence in producing, or, at least, rendering the individual liable to rheumatic affections. It may be questioned with great surety whether a person in perfect health ever develops rheumatism. In health, the young resist cold, exposure, wettings, or dampness—whatever, in short, are looked upon by some as causes of rheumatism. Two young people who have each spent the evening dancing sleep in damp beds; one may develop rheumatism, while the other escapes; and the reason—at least, one of the reasons for the variation of the result—must be placed to the differences in their condition of previous health. In old persons, a feebleness of health, even in the absence of any of the chronic forms of disease which tend to occur with special frequency as life is prolonged, seems to favor in a high degree the occurrence of rheumatism.

It is especially to be noted that any directly acting source of weakness which may be spoken of as accidental in its nature as distinguished from the general one just alluded to, tends strongly to favor the occurrence of this disease. All such conditions as make a strong drain on the vital powers—for example, nursing or suckling of infants, apart from any local exposures, losses of blood, etc., are certainly not uncommon coincident

circumstances attending its development. Insufficient or improper food is spoken of as one cause of its more frequent occurrence among the poor than the richer classes of society.

The not infrequent occurrence of the various types of attacks during or shortly subsequent to certain diseases, indicates either a causal connection between the two maladies, or else similar or coexisting influences for the two diseases respectively. The older authors especially allude to the connection between diarrhœa, mucous discharges, and the sudden suppression of these morbid discharges as well as normal ones. In more recent times—and not a few instances are probably within the experience of any one—it has been pointed out that after both dysentery and scarlet fever rheumatism occurs with exceptional frequency. This is true in respect to these diseases not only as they occur in their epidemic form, but after more or less isolated cases. How far atmospheric or epidemic causes influence the relation is undetermined, although it is possible to suppose that the two are connected. Much more likely is it, however, that the precedent disease either develops in the system the cause of the rheumatism or leaves the body in a condition especially prone to be acted upon by other causes or favoring external circumstances. It is probable that the so-called *epidemics* of rheumatism occasionally reported by early writers have occurred as a sequence of some prevailing disease, for a true epidemic of rheumatism is not at present recognized.

Rheumatism, usually of an acute type, following scarlet fever, generally comes on before desquamation is fully completed, and it would, therefore, seem to owe its origin to the changed condition of the skin, by which either its function is altered or rendered more sensitive to external influences. In dysentery, convalescence is usually nearly completed before the rheumatic attack begins, and in this case it has been thought to owe its origin to impaired digestive function. Of the actual connection of either of these diseases with rheumatism nothing is known positively, and, on the whole, their conjunctions occur so rarely that we are provided with but very brief data for the study of the question. The puerperal state, as well as the complications or accidents which arise out of it, furnishes a not inconsiderable number of cases of rheumatism in one or other of its forms. Whether there is some special influence connected directly with the condition of pregnancy and the results immediately following this condition, or whether it is merely that the conjoined accidents, such as already alluded to, reduce the powers of the organism, is an unsettled question.

That it is the latter, rather than the former, is probable, from the small number of actual rheumatic cases occurring during the puerperal state, in comparison with the whole number of deliveries. Chomel regarded the sudden suppression of the lochial discharges as the cause of the appearance of the rheumatic attack. It is certain that not a few of the cases occurring during the puerperal state develop subsequently to the natural cessation of this discharge, and, therefore, are unconnected with it. While it is a familiar fact that disease is produced by the suppression of the menses, yet it is much more probable to suppose in nearly all cases that the morbid condition leads to the stoppage rather than the reverse; that, in other words, the suppression is the effect rather than the cause of the disease, and that the morbid state, if it is one of true rheumatism, is the cause of stoppage rather than a phenomenon developed by the retention in the uterus of materials which naturally are voided. Our present knowledge of puerperal fever and pyæmic joint disease leads us to a just explanation of the arthritic and other symptoms which unquestionably are of frequent occurrence, and has contributed to lessen our estimation of the number of instances of rheumatic disease occurring in connection with the parturient period in comparison with that formerly held.

In this connection it is proper to allude to the views held by Dr. Todd of the probability of the development of rheumatism from the absorption of the unhealthy secretion of the diseased unimpregnated uterus. He makes the comparison of these cases to the puerperal form of rheumatism, and also to those occurring after gonorrhœa, and, therefore, this view of their origin is open to much the same criticism as that just now made. The gonorrhœal rheumatism is at present separated as a distinct disease, and it is probable, if it is at all possible or desirable to erect the uterine cases into a separate class, that this form of rheumatism is likewise of a septicæmic nature.

In considering this form, as well as the puerperal form of rheumatism, authors who hold variously the diathesic and the blood-poison views, or the view of its origin from cold, exposure, etc., become at great variance with each other, and not infrequently also with their own theories, in explaining the action of these diseases in producing rheumatic symptoms. So that it would seem that such diseases, as well as others of quite unlike nature, while occasionally followed by rheumatism, bear to its occurrence, not a specific relation, but merely one of accident, and are like numerous conditions which depress the tone of the organism and render it susceptible to the development of this illness.

Some of the infectious maladies, either at their inception or subsequent to their attacks, are attended with the phenomena which closely resemble rheumatism; of such may be mentioned syphilis and typhoid fever.

Climate, Country, Race, and Change of Habitation.—Rheumatism reigns as a most frequent disease pre-eminently in temperate climates, and we may almost speak of it as a disease limited by geographical latitudes. This fact is more especially true of acute rheumatism or rheumatic fever, for it would seem that the joints and muscles of the old and feeble ache and stiffen the world over. So striking is this difference in the geographical distribution and occurrence of the two forms of the malady, that it has been made use of as one of the arguments for separating the two forms into different diseases.

The older authors all speak of the variableness of the climate as a productive cause, and of this condition as having more influence than either extremes of heat or cold; the more rapid the change the more noticeable is the effect. Latitude is not necessarily controlling in its influence; high situations within the temperate zone are liable, according to situation and exposure, to more or less unfavorable variability than the lower levels adjacent to them. Chomel states that rheumatism is rare either at the poles or the equator, but is common throughout the temperate zone, and in countries which are humid and marshy. Fuller shows that among the soldiers of the British army admitted into the military hospitals of the various colonies, the disease is "not so prevalent in the cold climate of Nova Scotia and New Brunswick as at the Cape of Good Hope," and by the reports from India, "in its acute form, at least, the disease is very rare within the tropics, and as we draw toward the poles the symptoms of acute rheumatism are almost unknown." Different portions of the same country appear to vary in the number of cases which prevail; in England it is found that in the eastern counties the number of acute cases exceed those in the western, owing, it is thought, to their exposure to the damp and colder northeast winds. In many parts of the European Continent, in fact, parts in which climate and exposure in nowise differ, or are even apparently more favoring, the inhabitants are rarely affected with the disease or entirely escape.

It seems apparent, therefore, that other influences connected with the situation besides those just alluded to concur in shielding or in inflicting the population with the malady. One of these influences to which, not only in this disease but in many others, no mention has been made here-

tofore, or, at most, but slight study has been given, is the relative difference in soil. We know that, for example, in dysentery, no more potent factor exists in determining its endemic prevalence than the character of the soil. This rule is, perhaps, more true, or, at least, noticeable in the temperate zone, where, except under certain extraordinary circumstances, the disease occurs sporadically, rather than generally, as in hotter latitudes. It would seem that the prevalent diseases are influenced more by the deep or subsoil conditions than by the superficial, although many modifications may be brought about through changes in the surface-conditions, due to length of occupation of the soil by an abundant population, the drainage, as well as other causes; and this is more especially the case in reference to certain infectious diseases, as dysentery, etc., than for rheumatism and other maladies belonging to its class. To illustrate the matter, let us take a situation which, from the character or arrangement of underlying geological formation, would be favorable from its dryness, etc., to lessening the conditions that tend to develop rheumatic disease. Such a situation would be made favorable to the increase of the disease by having overlying it a surface-covering of impermeable clay; and this would be further increased if, from increase of the inhabitants, their drainage and water supply, the clay soil retained a large amount of water. At the present time our knowledge of the influence of soils on the atmospheric conditions, and, consequently, on its inhabitants, is so limited that we are not prepared to speak definitely of such influences; but it is probable mutual relations between the soil and climate and its prevalent disease will be found to exercise a great influence on the health—probably much greater than at present supposed. It is believed by many observers that the soil exercises as much influence—nay, more, on the health of its dwellers than the prevailing winds which blow over its surface. I know of many places in this country where such influences are to be traced in the clearest manner.

In considering the question of climate and situation as influencing the prevalence of rheumatic affection, it is important that we should not omit speaking of race peculiarities. It cannot be doubted that some races show a greater susceptibility to certain diseases than others, although the accurate determination of such a question is one of the greatest difficulty, since races of men seem so closely wedded to climate and situation that the two are not separable, and we never learn to know them apart. Races are usually spoken of as hardy or otherwise, owing to the climate to

which they are habituated, although some nations of people do undoubtedly possess and maintain certain peculiarities apart from, and in spite of, changes of situation, or any other influence which may, in the course of time, be brought to bear upon them.* If, therefore, some races or nations, under changed conditions, evolute and become possessed of other qualities, while others resist such influences, we can fairly argue that, in respect to a certain disease, or it may be all diseases, a similar evolution may take place, and that nations may thus retain or increase their susceptibilities to certain noxious influences, or they may lose them, and at the same time some diseases may vanish, while others make their appearance.

In this country, with its mixture of so many races and nations derived from every source, who permanently dwell here, the study of this question can be made under specially favorable circumstances for its solution, as the climate differs so radically from that of countries from which our new people are derived, and as the individuals become so permanently incorporated, and remain subject both to new modes of living and to the changed climatic influences. Unfortunately, we are defective under any existing regulations in obtaining reliable statistics of the prevailing diseases; of the deaths only do our reports give us information. However, it is in hospitals where such questions can be studied with the greatest accuracy, and it is to the public institutions for medical relief that our new population naturally resort in greater numbers than those of native origin. From a large observation, during many years, of this class of patients, the conclusion I have drawn is that our foreign population are much more subject to rheumatic disease than those native born; and, in respect to the Irish nation, which form the largest part of this class, judging both from such statistics as are available and from their own belief in the matter, they, as a nation, are more liable to rheumatism, especially in its acute form, in their new than in their old home. It must be remembered, however, that with us, speaking generally, they are, as a class, engaged in occupations which are supposed to especially favor this disease, and, of course, this fact would counterbalance the influence coming from mere change of climate, supposing, of course, that such change actually has a real material effect in increasing their susceptibility. To these inadequate grounds for a conclusion must be added the changed type of the disease and the variable denomination given to the word rheumatism by different observers in different countries.

Seasons, Atmospheric and Barometric Influences.—Seasons of the year

have an equally striking influence in determining the prevalence of rheumatic disease. All statistics show that the common opinion is correct in assigning to the summer or warmer portions of the year a fewer number of cases than the inclement season. From May to September there occurs a very great diminution, while during the remaining three-fourths of the year it is prevalent. Following the same tendency as in changeable climates, the most variable months of the year furnish the largest number of cases—the spring months more than winter, and these latter, with their steadier cold, more than the autumn. The month of May, both in this country and England, during which winter often meets summer, is the most unfavorable, and a larger number of acute rheumatic attacks occur. Variations in the number of rheumatic cases, as influenced by the seasons, show great differences from year to year, and from season to season we constantly witness favorable or inclement autumns, winters, and springs.

In respect to variability of climate and seasons, I wish to call attention to certain atmospheric conditions, of which I have not seen any definite mention made. Among the older writers frequent reference is made to subtle and unknown atmospheric conditions and influences, of which no explanation is given. Later, however, the causes of rheumatic disease are mostly placed to the account of changes of temperature, moisture and dryness, and no notice is taken of atmospheric causes other than these. It has seemed probable to me that these subtle atmospheric influences to which the older writers allude have a reality to which we have not given full credit. It is true the influences which I mention were unknown in their true characters, and were regarded by them as mysterious. Perhaps their full appreciation has only become possible in this age of weather signal service bureaus, when every morning and evening the heretofore subtle condition of the atmosphere is forced upon our attention by the barometric record spread before us in the daily paper. That the pressure of the atmosphere does influence our bodily condition needs but to be stated to carry with it a full belief in its reality, and that, under diseased conditions, the rapid variations, as shown on the barometric scale, should exercise a stronger influence, is even more evident and rational. Every one, if at all observant, or whose attention has once been called to it, has often noticed unfavorable changes in patients suffering from typhoid or other continued fevers, pneumonia, etc., and in many chronic maladies—for example, valvular heart disease—and that a sudden fall in the atmos-

pheric pressure is likewise capable of accidentally determining the time of their fatal issue. Among patients who have suffered a long time from rheumatism, no more common observation is current than that changes of weather are foreshadowed by an increase of their "rheumatic" pains and stiffness. It is also true that very frequently the rheumatic pains prove to be false prophets, and that no rain follows the prediction.

My own careful and somewhat extensive observations and comparisons on the relations between rheumatic pains and changes of the weather have led me to the following conclusions. The painful symptoms do not predict a rain, but are an indication that the barometric pressure is lessening; and as we know the oncoming of a storm is usually preceded by a falling barometer, the pain and the subsequent rain become phenomena closely correlated in time, but not as effect and cause. The hygrometric relations have little direct connection with the occurrence of pain, and this symptom is often developed prior to an increase, either actual or relative, of the amount of moisture in the air, so that it is not the dampness which causes the pain. It is not a merely low barometer with which this symptom corresponds, but it is a rapid change in its indications, either falling or rising, which seems to be the efficient cause. A rapid rise is sometimes, though less constantly, productive of the rheumatic phenomena. The period of the actual storm, during which the barometer is stationary or is higher, is usually free from pains, although they may persist coincidently with a greater dampness of air or other causes. That pain should occur from this cause without rain following is as evident as the fact that sudden rises or falls of pressure occur unconnected with storms; that a rising barometer is less frequently attended or preceded by pain than the falling, is to be accounted for by the fact that depressions are usually suddenly developed, while elevations come more gradually and promote, even in the healthy organism, which is less affected by these variations than the diseased, a more robust and bracing condition. Why this result should obtain is unknown with certainty, though many speculative ideas or glittering generalities could be advanced for its explanation.

I have also observed that acute rheumatism, except during the critical period, follows, though I think to a less extent, the rule in other fevers, and is little influenced by the barometer, especially with reference to the pain, while in chronic rheumatism the phenomena are seen with striking regularity.

Occupation and Manner of Life.—Irrespective of any consideration of the theories of rheumatism, whether a diathesis or a materies morbi, or whether its causation be cold, dampness, etc., all of which have influenced opinions as to these circumstances, it is unquestionable that certain occupations and modes of life render individuals prone to rheumatic disease.

The other conditions we have already considered favor or nullify the effects of these two causes, but even the lightest work, the most sheltered occupation, and the most careful mode of life are not free from the effects of this disease, although all of these circumstances in the highest degree lessen the liability to it.

Severe physical exertion is held by many to directly increase the tendency to rheumatism, and if this is performed habitually, under exposure to the weather, or to sudden changes of temperature, the conditions for its development are much favored. Insufficient protection of the body, especially after the cessation of labor or exercise, greatly promotes the chances of acquiring the disease or at least of developing its acute attacks. Cold, applied by immersion in water, or by rain, furnishes abundant examples. Rapid changes of the surrounding air, either in its amount of warmth or its movement by winds and draughts, specially increase the liability of those exposed. Clothing rendered damp by rain or insufficient drying and allowed to remain on the person, especially if affected by rapid currents of cool air, and also even by warmth in the sun or in rooms artificially heated, is productive of a similar result. Sleeping in damp beds and living in damp houses are to be enumerated in the same class of productive causes of the disease. If to these are added a manner of life, unhealthy from excesses or defective in comforts, insufficiency of food or its poor quality, and oftentimes, too, simply the irregularities in the hours of eating, sleeping, and working, it would seem almost that the only reason for escape from rheumatic disease was a power of resistance of the organism too strong to succumb to the evil, and even this bar is broken down in time and finally such causes produce their usual effects.

In my own experience, the largest number of cases, in which the occupation at the time of the inception of the symptoms was definitely ascertained, furnished by a single class, were of firemen, including both those engaged as stokers on ocean-going steamers, where the circumstances are particularly exposing, and those working in firing steam boilers on land. In this labor the variations from intense heat to cold draughts are prob-

ably more frequent and constant than in any other occupation. Next in numerical order come the maid-servant, laundress, and maid-of-all-work—all three are classed together, as liable to the same kinds of exposure and under similar unfavorable circumstances. Neither class, nor both combined, formed a majority of my cases.

Temperament and Constitution.—Temperament and constitution are generally spoken of by the older writers as exercising an influence in the causation or development of rheumatism, but in recent years so much less attention has been given to their influence in diseased conditions that these subjects now receive little or no discussion. In these days of the stethoscope, thermometer, microscope, æsthesiometer, and ophthalmoscope no methods of measuring disease and its effects are very much regarded which are not effected by exact instruments, subject to record and exact comparison. One does not know where to turn to obtain any recent information of such questions as temperament and constitution, except, of course, in a few diseases. How much has been lost or gained by their passing away from medical discussion, where formerly they held so prominent a position, the temper of the present age is not fitted to determine.

Injuries.—Pains, so frequently spoken of as rheumatic, consequent on injuries, do undoubtedly exist and are familiar to every one; but whether injuries in themselves, as was formerly the opinion, are productive of rheumatism in any of its forms depends largely on the limitations given to this disease.

Sleep.—The conditions of sleep and waking, when we trace the histories of rheumatic attacks, are found frequently to influence the receptivity of the organism to cold, and to lead thus to the development of the disease. We are all familiar with the effects not infrequently resulting from lying asleep on the damp grass while the body is overheated and perspiring, or of sleeping under an open window. I have met with a number of patients who, sleeping by the open window of a railroad car in motion, have been attacked with rheumatism.

It is supposed that during the condition of sleep the organism is less capable of resisting the influences which develop the rheumatic condition, or at the least is more subject to impressions of cold. Of course this development after exposure to cold during sleep does not directly prove that the organism while in this condition is more susceptible to such influences from this cause. It is known that in sleep the condition of the circulation is altered, but this does not prove an increased susceptibility. It

must be remembered that the fact of sleeping implies a long exposure to such influences—longer, at least, than if the person were awake, and unless there can be shown a greater liability in sleep than results from an equally long exposure during waking, the influence of sleep is not clearly shown.

Although rheumatism is looked upon at present as occasional and sporadic, by the older writers not infrequent mention is made of epidemics of the disease. That favoring combinations of weather, season, and climate may vastly increase the number of cases, or that subsequent to other prevalent diseases, as already alluded to after dysentery, it may be unusually prevalent, seems unquestioned, but in all other respects the rheumatic type of disease is deficient in the characters of an epidemic malady.

This disease is said to be endemic in some countries and certain localities, and in a certain sense this may be true, as already spoken of in discussing the influences of soil, weather, and situation, as well perhaps as from other conditions, such as occupation and mode of life. But to apply the words epidemic and endemic to rheumatism is certainly a misapplication of terms, and deprives them of the implied meaning, universally connected with them, of contagiousness or infectiousness.

Former Attacks and their Effects.—In respect to the predisposition of rheumatic patients to a return of the disease, some misunderstanding of the question has arisen among writers from confusing a liability to subsequent attacks with the tendency of the disease to relapse, even before convalescence is established or shortly afterward. These two phenomena should be rigidly kept apart for separate consideration, and yet, from a careful comparison of many statements of the question, it is evident that some opinions have been misconstrued and the one result been accepted for the other. It is important also to distinguish between the various forms of rheumatism in forming an opinion on this matter.

Here, as elsewhere in dealing with the debatable question of causation, the influence of the theory of the nature of the rheumatic process has had its influence in moulding opinion. However, I do not think it can be doubted that one attack renders a person peculiarly liable to a second or even more, and in this opinion most authors concur. It is unquestionably necessary to define accurately, at least to ourselves, whether we mean that a chronic form of the affection predisposes a person to acute attacks, and also whether we hold that one invasion of rheumatic fever is liable to be followed by others, or merely that the chronic form is induced by such attacks. To say that chronic rheumatism induces a chronic

rheumatism is certainly not asserting very much, for the natural history of the disease, as well as our conception of it as implied by the name, bring up the picture of a perennial, almost perpetual malady. Whereas, to believe that it renders its subjects liable to acute attacks, cannot be substantiated and is not warranted by the facts. Rheumatic fever is a disease of youth, chronic rheumatism the disease of age. If it can be shown that the comparatively rare cases of chronic rheumatism of youth were especially liable to be succeeded by acute attacks in subsequent years, it would go very far in proving the question and, moreover, also the unity of these forms as a pathological process. The question of the liability of one attack of rheumatic fever to be followed by subsequent ones, or by a chronic trouble, must be settled by statistics. I do not find that any statistics exist sufficiently extensive to prove unequivocally such a question. Many writers show from their records of cases a striking frequency of such results, but in some instances these calculations are vitiated by inaccuracies or by uncertainty of the character of the former disease.

It must be borne in mind that the essential facts of the case may be obscured by several factors. In the first place, a person who has suffered an attack of rheumatic fever rather late in life may wear out the susceptibility to a second by simply advancing in years, while if the attack had occurred earlier in life one or more subsequent attacks might have supervened before old age arrived. And then, too, rheumatism, not in itself frequently fatal, is often complicated with valvular disease of the heart, which ends life before the usual susceptibility to the acute disease is worn out. This factor likewise must materially reduce the actual number of persons living at any one time who have suffered from a second attack, although by some observers the cardiac disease is held to increase the liability. On the other hand, there is a factor which unquestionably tends to diminish the number of second attacks of acute rheumatism, whatever may be its influence on the chronic form of the disease, and that is, that once a person has suffered from rheumatic fever he naturally is more careful in respect to exposure and shuns the conditions, so far as able to do so, which, to say the least, favor the development of the disease. Many are compelled to seek light work or different occupation owing to the occurrence of cardiac disease, and thus may escape second or subsequent attacks.

How far statistics are competent to prove the peculiar liability which one attack induces for others, may, I think be questioned, or at least

should be scanned very closely. The record of cases of rheumatic fever show that very varying periods of time elapse between the attacks. I know of none, and have not been able to find a record where the period was less than one year; usually "from three to five, sometimes even ten years" elapse. It would naturally be supposed, I think, that if one attack was an essential factor in predisposing to another, the paroxysms should follow closely upon one another. It is difficult to find a reason why an attack of rheumatic fever renders a person "peculiarly liable" to another ten years subsequently. Todd believes that the full establishment of the diathesis renders the patient "very liable to similar attacks," and Fuller says that "a paroxysm is evidently but an effort of the constitution, and generally for the time an effectual effort, to get rid of the poison," and seems to imply also that if the effort is thorough enough no more attacks would occur, provided sufficient care were exercised to avoid exposure and proper medical treatment instituted to restore the system to complete health.

This question is certainly a difficult one to decide, as are so many others of a similar nature, where the proof is in so large a degree purely numerical, without our first possessing a pretty clear — certainly very much clearer than we at present have—notion of the essential cause of the disease.

Drugs.—The discussion of the effects of drugs, cold, and general functions of the organism and the influence they have in producing or favoring the development of the disease, if they have such power in any material degree, we shall leave until the pathology of the disease has been considered.

CHAPTER IV.

PATHOLOGY.

The Various Theories: The Local or Inflammatory; the Vascular; the Nervous, comprising the Electric, the Spinal, and the Vaso-motor; the Specific Poison, or Lactic Acid Theory; the Physiological and Chemical Origin and Nature of the Lactic Acids; the Defects of this Theory; the Infection Theory.

The Various Theories.

The nature of the proximate cause of rheumatism remains to-day among the most unsettled questions of pathology, and little has been added to our actual knowledge in relation to it since the earliest time, although so much accuracy in its clinical aspects has been acquired meantime.

We may pass by the primæval theory of its origin from "the defluxion of an acrid humor, generated in the brain, into the various parts of the body," and at the same time be careful not to drop into this same humoral error presented in a new and more exact chemical form.

We must, however, consider carefully other theories which have been studiously wrought out and have received the sanction of eminent physiologists, pathologists and clinical workers. Of these theories four demand very special notice. First, what may be called the local theory; second, the specific poison; third, the vascular theory; and fourth, the vaso-motor or nervous. The last theory is closely allied to and its views intermingle with the previous one, and also with the electric view of the origin of the disease which has been held by some observers. Recently an infectious nature has been proposed for all the rheumatic phenomena, and since the germ theory of disease has become so popular it has acquired quite a number of adherents and advocates.

These views cannot be discussed in chronological order, and the order in which they are mentioned here is not intended strictly to conform to this method; in fact, these various theories have in point of time com-

mingled and are yet held contemporaneously; at one period or other, each view has had its successful advocate and has for the time become pervasive. At present, at least among English authors, and to a very great extent in other countries, the specific poison theory holds its sway.

The Local or Inflammatory Theory.—Sydenham was the first one to definitely state his belief of the proximate cause of rheumatism and to conform his treatment in accordance with his views of its nature: his pathological notion was that the disease was an inflammation of the joints; that it was a local affection attended with various general symptoms which owed their origin to the joint disease. In this view he was followed by Cullen, and numerous others, and Cullen further expresses the opinion that "the cause of acute rheumatism appears to be exactly analogous to that of the inflammations depending on an increased afflux of blood to a part while it is exposed to the action of cold," and he denies the existence of a "peculiar acrimony" of the blood as a supposition without reason and one founded on the humoral pathology of the ancients.

Scudamore, while holding that rheumatism belonged to the class of phlegmasiæ, speaks more particularly than previous writers of its specific characters and of its constitutional relations. He says that "rheumatism in its true primary character is not so distinctly a constitutional disease as gout;" that "rheumatism is a disease external to the system, which itself becomes affected in a secondary manner by sympathy; but, in turn, it reflects back its influence upon the external parts." "Although it occasionally happens in point of time that pyrexia precedes the development of the local rheumatic inflammation, may we not consider that the constitution has taken such early alarm from a ready sympathy with the textures upon which the offending agent, cold, has certainly made its impression, notwithstanding the characteristic symptoms do not become all at once developed? Yet the interval between the occurrence of the constitutional fever and the local signs of inflammation is never long; and the general fever, inasmuch as it is truly rheumatic, keeps pace entirely with the local disease." "It may be stated that the predisposition to rheumatism consists in a deficiency of healthy tone in the textures, connected with joints and muscles, and in nerves, so as to be affected in this peculiar manner by the influence of variable temperature. If we lose sight of the humoral term 'rheumatism,' we shall come to the simple fact that in a condition of susceptibility cold, or sudden reduction of temperature, makes a particular impression on the vessels and nerves near the surface,

and produces a painful affection of certain textures, which is attended with more or less of inflammation, the phenomena of which are so far of a peculiar nature that we may either consider the disease *specific*, inasmuch as the symptoms differ in their constituent characters from those produced by other inflammations, or we may view the effect in the light of common inflammation, modified on the one hand by the nature of the exciting cause—the external one, cold—and, on the other hand, by the influence of the particular species of textures which become affected."

This view of Scudamore must be rejected, as not grasping in full measure the scope of the disease. At the time it was promulgated his theory was a great advance on previously held ideas of the pathology of the disease, and many of the physiological and pathological notions included in the abstract here given, as well as those found in other parts of his work, were of a nature that have only been formulated quite recently by the light of many newly discovered anatomical and physiological facts. The essential inflammatory nature of the malady is rendered by Scudamore too prominent, and while he admits its general characters, the ascription of these to the effects of sympathy is all too vague to correspond to many of the prominent phenomena of the attacks. The denial of any peculiar alteration of the blood—although such alteration may not be regarded as the proximate cause of the disease—is still less in correspondence with known pathological facts.

We may, therefore, put to one side the view of its local or purely inflammatory origin and proceed to the discussion of the various other theories of its causation. In order to arrive at any common ground for this study it is necessary to start with some initial symptom or occurrence which, by its frequent or almost constant presence, can be looked upon as something either forming a part of the disease or as quite essential to its production. This phenomenon we find in the "chill." By this is not implied that a rigor is universally an initiatory symptom of the malady, but that the person affected suffers, through the application of external agencies, an abstraction of heat, either general or local. This abstraction of heat is so nearly a universally exciting cause, that on it nearly all are able to meet as a common field for discussion; to some of the theories it is an essential feature, but to others it would seem merely incidental, yet the advocates of all the various views acknowledge its occurrence and render its influence a prominent factor in its causation.

The older theories of rheumatism attempt no explanation of the

manner in which the chill develops the phenomena of the disease, except that in the inflammatory theory the bare statement is made that the action of cold, or of cold and moisture, produces the inflammation, and that this inflammation is modified by the nature of its cause or by the texture of the tissues which are involved. Later writers have added nothing to our knowledge of the essential nature of chill which is at all adequate to the explanation of the phenomena which result from it.

The Vascular Theory.—Another theory, which has at times received considerable support, and especially so when the action or participation of the capillary system of vessels in all inflammatory diseases came to be recognized and insisted on, was that the train of symptoms recognized as rheumatic were set in motion by the local or general abstraction of heat, producing a contraction of the superficial vessels. This contraction was succeeded by a marked dilatation of their calibre, acting as a rebound, and a consequent afflux of blood to the parts most strongly impressed by the action of the chill. In cases where rheumatic inflammation succeeded to these phenomena, the original impression of the chill was supposed to have been sufficiently strong to paralyze the elasticity of the capillary wall. The dilatation, by reason of this, was maintained long enough to result in an inflammation. The localization of the disease in certain joints in preference to others was supposed to ensue either from their greater exposure to the agency or from an original inherent weakness, a predisposition peculiar to the part or one induced by functional activity at the time of or immediately preceding the onset of the attack.

This theory is defective in many points. In the first place, it attributed a power to the blood-vessels which is no longer conceded to them. The regulation of the blood supply to a part and the phenomena of chill in connection with the vascularity of an organ are the result of nerve action, and do not result from the state of the capillary walls. Their discussion will follow later. The capillaries are non-muscular tubes and do not possess the power of self-action in respect to contractility and the consequent afflux of blood. A paralysis of the capillaries, therefore, cannot be brought in question; it can only be an overdistention, and if any such effects are to be attributed to cold or chill, they must be carried back to vessels which are possessed of muscular walls. But according to the terms of the theory the effect of the chill was a purely superficial one, involving the superficial network of vessels.

Further, this theory leaves out of account many of the associated

phenomena of the rheumatic attack, while, if its physiological teachings were acceptable, it would harmonize with the inflammatory and local notions of the disease; still, it must be held to be inadequate for the explanation of the visceral complications as well as the marked blood alterations exhibited during and subsequent to its action. This theory in no way differentiates this peculiar inflammation from others, except that its cause is not due to mechanical action, and it does not account for the sudden cessation and oftentimes sudden return of the inflammatory symptoms in an articulation, without there having been in the meantime a reapplication of the same cause.

For this theory, in the terms which it has been stated, very little can be said in support at the present day, and little of it which has not been made more definite in its application under the theory next to be considered. The purely local part of it belongs to the old inflammatory notion, and the participation of the capillaries in producing the effects has, so far as they act, been explained in accord with known physiological laws, by the mechanism of the vaso-motor or trophic nerves.

The Nervous Theory.—As the older notions of rheumatism in the course of time were deemed inadequate to the explanation of its occurrence, when the knowledge of physiological and pathological laws was advanced by numerous anatomical discoveries, and even before we had arrived at a full comprehension of these facts, another theory was promulgated for the development of the attack. This new theory, as at first propounded, hinted at rather than directly expressed the view that the whole train of symptoms was the effect of nervous action.

The Electric Theory.—The earliest of the nervous theories of the causation of the disease had to do with the effects of electricity on the system. At that time it was generally spoken of as the constitution of the atmosphere, and by sudden changes in this rheumatism, as well as other diseases, was produced. Of the many imponderable agents capable of producing diseased action and contained in the air, electricity was the chief, the most subtle, and the one on which the rheumatic predisposition depended. Nervous action was at that time supposed to depend on electric force, if it was not itself actually electricity. It was observed that at times when rheumatism or rheumatic symptoms were most frequent the electric machines furnished but little evidence of electricity, and it was therefore concluded that the rheumatic phenomena were dependent on a deficiency of electricity in the air.

Such a theory found abundant support in days when the knowledge of this agent or force was but very imperfectly known. It was shown that this supposed electric deficiency corresponded to the times of year, and even of the day, when rheumatic diseases were most prevalent and most easily developed from exposure. The fallacy of this view was soon made evident from the fact that although the machine did not produce electric manifestations, yet there was no actual deficiency of this agent in the air; on the contrary, it was not infrequently the case that while but little electricity was manifested from artificial excitation, a superabundance of the agent existed in the atmosphere, and the very conditions which hindered its artificial display, viz., moisture, etc., and conducted the agent away as fast as produced, favored an electric accumulation in the air. Various modifications of the electric theory found believers, and when the dependence of rheumatism on the want of this agent in the atmosphere was shown to be untrue, the occurrence of the disease was referred to its altered intensity under the influence of moist or dry atmospheric conditions.

And again, some thought that a disturbed equilibrium between the atmospheric and the organic electricity manifested itself in rheumatic phenomena; others considered that the disease was produced in those parts of the body by the interchange of the positive or negative electricity of the air with opposite kinds of electricity of the organism; while others again thought that the organism was overloaded with electricity and that this condition called forth the pain and other rheumatic symptoms. "According to my belief the quantitative potent electricity of the air, which passes into the organism, especially through the respiration, and the thermo-electricity generated in the organism itself through changes of temperature, excite the capillaries, and these occasion an abnormal formative action; electricity brings a vital principle, stirs up everywhere activity and life; but when its incitation oversteps the capacity of the incited parts, it occasions an excessive or diseased vitality and decomposition."[1]

The Spinal Theory.—Another phase of the nervous theory of rheumatic affections was introduced by Dr. J. K. Mitchell, of Philadelphia, in 1831.[2] Briefly stated, his view was that "rheumatism might have an origination in the medulla spinalis, and depend on an irritation of that

[1] Eisenmann: Die Krankheits-Familie Rheuma. 1st Band, S. 25, 26. 1841.

[2] American Journal Medical Science, Vol. VIII., 1831: On a New Practice in Acute and Chronic Rheumatism.

important organ." Dr. Mitchell was led to this opinion from the results of treatment of the symptoms of acute rheumatism occurring in patients suffering from spinal curvature. His treatment for these cases was depletion, applied not by venesection or to the affected joints, as in vogue in his time, but to the part of the spine involved in the disease, "with the effect of procuring a prompt solution of the disease." In cases of both acute and chronic rheumatism, coming without any evident spinal involvement, he found that the "local depletion applied to the spinal region benefited the disease, which lay at the peripheral extremities of the nerves," and "that local blood-letting is most potent when applied to that part of the spine which supplies with nerves the parts in a state of active inflammation." Dr. Mitchell did not further express his belief on the universality of this condition as productive of rheumatism; neither did he point out the connection between or *modus operandi* of the so common precedent condition or cause of the disease, viz., cold or dampness, in setting up the "irritation of the nervous masses," which in his opinion led to the articular symptoms. Whether, in other words, he thought that the initial chilling acted directly or even generally on the spinal cord, or whether the altered temperature communicated itself through the usual nerve-channels from the surface to the centres.

His exceedingly clever and original views on the subject of the causation of rheumatism shine out as a brilliant spot to any one reviewing the history of this disease, and distinguish his work as a piece of scientific investigation of this subject which has been rarely, if at all equalled in the centuries which rheumatism has afflicted the human race. No one reading the records of his cases can fail to feel a confidence in his observations or to perceive the true spirit of investigation which he brought to bear on the problem before him. And while this view has found but little concurrence in professional opinion as an explanation of the cause of rheumatic diseases, I think that more attention should be bestowed on it now, a half century after its publication. It will afford a most rational pathology for a number of cases of acute, and perhaps even greater number of the chronic form of this disease. Many cases also of those exhibiting symptoms in connection with the main nerve-trunks, as in sciatica, not improbably will be found to have the condition, supposed by Dr. Mitchell, as their true pathological change.

Rheumatism has been shown to have been, according to the earlier views of its nature, a medley of morbid conditions. A different nature

has been shown between gout and rheumatism, formerly confounded with each other, although both belong to the arthritic family. Gonorrheal rheumatism, pyæmic joint affections, and those occurring in the puerperal state are now well recognized as due to separate causes. Arthritis deformans, or rheumatoid arthritis, as well as certain joint diseases due to chronic spinal cord changes, have also been differentiated from the main bulk of what remains still to be classed as rheumatism. Is it at all improbable that in the course of time we shall come to recognize a rheumatic joint-affection dependent on acute changes in the spinal cord or other central nerve-mass, which at present is classed as an acute or chronic rheumatism? This is what Dr. Mitchell indicated a half century ago, and a perusal of his cases and the effects of his treatment render the supposition quite, nay, more probable than many of the theories which have found acceptance since that time. His observations are the very foundation of all our present knowledge of the concurrence of spinal, or in fact cerebral affections with various kinds of arthritic inflammation, and there is undoubtedly an element of truth in them, at least for a certain number of rheumatic cases, if the test of treatment, which we have applied as did Mitchell, is to be trusted.

Dr. J. K. Mitchell published a second paper in the *American Journal of the Medical Sciences*, 1833, p. 360, on the connection of spinal disease with rheumatic phenomena of the joints, and a quotation from an article in the same journal for April, 1875, by his son, Dr. S. Weir Mitchell, will show the relation which this early work has to the modern researches on the same subject. The latter article is on "Spinal Arthropathies," and calls attention to "the history of spinal and neural arthropathies, a subject which owes its best study to American and French students." "The history of this subject is somewhat interesting, and the more so, because to an American physician belongs the long-forgotten credit of the first discovery that 'an obvious spinal cause may produce a rheumatism characterized by heat, pain, redness, and tumefaction.' The quotation is taken from the second paper on rheumatism by the late Dr. John K. Mitchell. In his first essay he described cases of Pott's disease, in which, below the diseased region, there were acute inflammations of the joints, which proved amenable to treatment directed to the point of spinal lesion. M. Charcot, in acknowledging the first mention of these facts, adds, correctly, that this cause of arthropathies is rare, and, apparently ignorant of Dr. Mitchell's second paper, says that traumatic lesions of the cord are

more often the parent of joint disorders, and refers to Gull for instances of spinal commotion as competent to occasion a like result. Until, in my work on injuries to nerves, I recalled attention to the true author of this clinical discovery, Gull has usually had credit for being the first original observer of these interesting facts, while actually the credit is due to the American author." "Finally, in 1864, I described, in conjunction with Drs. Morehouse and Keen, the joint diseases caused by injuries to nerve-trunks, and again and again since I have illustrated anew this clinical sequence, by numerous reports of cases of manifold forms of nerve injury."

Unfortunately, this theory, first promulgated by Dr. Mitchell the elder, did not attempt to show the relation between the exposure or other exciting conditions and the development of the irritation of the nervous masses; it merely formulated the relationship between the spinal irritation and the arthritic or other rheumatic phenomena. Had this been done the work would have anticipated by ten years or more the theories promulgated by Froriep and Canstatt. Their observations, dating about 1840–5, have only become of value as an explanation of the rheumatic phenomena by the light of our modern, later-discovered knowledge of nervous action.

The Vaso-Motor Theory.—The later developments of the nervous theory, thus commenced fifty years ago by Mitchell, and continued by Froriep and Canstatt ten years later, as well as more recently by others, have been characterized by the attempt to explain the pathology of the disease in more or less strict accordance with the doctrines of physiological pathology. But while the latest developments of this theory insist most strongly on the actions of the nerve-fibres, as influenced by external causes in instituting the initial steps in diseases, it goes farther in tracing their results. According to the older statements, of what has been termed the nervous theory, the articular affection was described as the result of disturbed innervation, and the general symptoms were regarded by some to be dependent on the local affection, and by others as a mixed result, partly due to the local disease and partly expressive of the same nervous disturbance affecting the system generally. It was supposed—and this must be done in order to harmonize the theory with our present ideas—that the cold acted directly on the nervous supply to the joints, or that this impression was conveyed to the nerve-centres so that, as the result either of central or peripheral nervous action, articular inflammatory disturbances were excited.

This theory, which originated at a time when nervous action was beginning to be observed with increasing attention, seemed to supply the wants in the scientific mind by which to account for the erratic character of the rheumatic symptoms. It was also a protest against the universal tendency of the older phlegmasic pathology, which had so long been dominant.

This theory in its bare and simple form has failed to receive any very general assent, principally from a lack of proof of the ways and means of action of the nerve-fibres, viz., the so-called trophic nerves, in producing their effects when subjected to irritations of the character supposed to be brought into play.

The Specific Poison, or Lactic Acid Theory.—The theory of the causation of rheumatism which yet remains to be considered is the lactic acid theory. The suggestion that this acid was the peccant matter giving rise to rheumatism was first made by Prout, and since his time has been taken up by others.

The subject is referred to by Dr. Prout in his considerations on the pathology of saccharine assimilation and secretion in the following manner. In speaking of the development of lactic acid during the secondary assimilating processes, he states that the various acids and other unnatural matters developed in the stomach and digestive tract appear to be absorbed into the system, "where they probably tend to act as exciting causes of derangements in the secondary assimilating processes (nutrition). The symptoms resulting from such combined derangements, like all symptoms connected with derangements of the assimilating organs, have more or less of a periodic character, and show themselves in occasional attacks of bilious congestion, gout, lithic acid, gravel, catarrhal affections, ague, rheumatism, etc., according as exposure to cold, malarious influences, etc., co-operate with the original predispositions and determine their nature."

After speaking of the character of bilious attacks in their relation to this condition, he continues: "In those who suffer from indigestion, whether from excess or from disease, the acid and unassimilated matters appear to accumulate in the system. Such attacks in the strong and healthy are usually displayed in the form of simple feverish excitement, and generally sickness and diarrhœa, but in the delicate, and in those predisposed to other diseases, the weak part, wherever it may be, is usually involved in the affection, and suffers in a greater or less degree. Of all other parts of the system, the mucous membranes seem to be most liable

on such occasions to go wrong; and of all exciting causes, cold is perhaps the most common. Thus every one must have observed that when the system is so charged, he is liable, on the slightest exposure, to get cold; . . . others in such a state of the system from a similar exposure get an attack of rheumatism; others gout or erysipelas; . . . and in all such instances the affection is of a mixed nature, and cannot be advantageously treated without reference to the original derangement of the assimilating processes; and sometimes when this is removed, all the other symptoms disappear. . . . Thus bilious individuals are very liable, on comparatively slight exposure to cold or to malarious influences, to get an attack of acute rheumatism or of ague. . . . The condition of the system above described preceding what is called a bilious attack, being so obviously calculated to predispose the constitution to take on further diseased action, we are neither surprised to find that in such a state of the system the secondary assimilating processes going on in all parts of the economy become more than usually deranged from slight exciting causes, nor that those organs should *superficially* suffer which happen from any particular cause to be predisposed. The inferences, however, we wish to draw from these obvious facts are, that the severe derangements of the secondary assimilating processes going on all over the system are nearly allied to certain forms of *fever;* while the local and specific derangements are identical with certain specific *inflammations.*

"Of what fever and inflammation in general consist we do not venture to offer an opinion; but as no one will deny that certain forms of fever and inflammation are always accompanied by derangements of the assimilating processes, both primary and secondary, and that such forms of fever and inflammation are not only preceded by long-continued derangements of the primary digestive processes, but frequently have their origin in such primary derangements, we may perhaps be allowed to assume without opposition that *some* diseases to which we apply the terms *fever* and *inflammation*, are *practically* speaking at least, what we have above inferred them to be, viz., only severer derangements of the secondary assimilating processes, modified by the peculiar nature of the organs or textures in which such derangements exist—inferences that will enable us to explain the principles on which derangements of the primary assimilating processes predispose to the peculiar derangements of the secondary processes now under consideration; and which we consider to be nearly connected, if not identical, with those forms of fever and inflammation usually denomi-

nated intermittent fevers, rheumatism, and neuralgia; . . . and, moreover, that these derangements of the assimilating organs are usually accompanied by the presence of great acidity of all parts of the system. Thus in . . . rheumatism, during the sweating stages of the paroxysms, immense quantities of acid (chiefly of lactic acid) are thrown off by the skin, and sometimes by the kidneys. In all these cases the saliva is commonly acid."

"In rheumatism, the same derangements, to a less extent, appear to exist in the primary assimilating organs; but, in this case, the secondary assimilating processes, by which the gelatinous portion of the muscular system and its appendages are produced and maintained, may be supposed to be more especially implicated; and the loss of power and the great degree of pain usually present in rheumatism may be referred to the disorder of the numerous nerves of motion and of sense, which, as well as the fibrous portion of the muscles, are likewise necessarily affected by the derangements. Moreover, on these suppositions we may explain the formation of large quantities of lactic acid usually present in rheumatic affections, as well as the swelling, etc.; for as all the organs are more or less involved, and their functions paralyzed, not only imperfect assimilation takes place in the part affected, but the apparatus destined to remove matters which are unfitted, or no longer useful, from the scene of operation, likewise cease to act; and hence such unfitted and useless matters accumulate, and cause swelling in the part affected. In simple neuralgic affections, nearly the same explanation may be given. Derangements of the primary assimilating processes, analogous to, or identical with, those existing in ague and rheumatism, are always present in a greater or less degree in these affections; while the derangements going on in those secondary assimilating processes, by which the nervous substance and its immediate appendages are produced and maintained, may be supposed to be the immediate cause of the pain and other distressing symptoms of the disease." [1]

On this subject Williams speaks in the following manner: "The perspiratory secretion contains lactic acid and lactates of soda and ammonia,

[1] "The reader is distinctly required to bear in mind that rheumatic neuralgia only is here alluded to. Neuralgic affections, arising from mechanical or other injuries of the nerves, are referable to another class of affections; but, even in such instances, the formation and presence of unnatural matters of another sort may contribute to the patient's sufferings."

which probably proceed from the transformation or decay of the textures, particularly the muscular, which the recent researches of Liebig (1847) have shown to contain a preponderance of this acid. Hence these products abound during great muscular exertion ; and when perspiration is checked by external cold they may be retained in the blood, causing rheumatism, urinary disorders, or various cutaneous diseases.

"Rheumatism is especially liable to occur as an effect of cold, when the body is fatigued with much muscular exertion ; and I have frequently observed that the rheumatism chiefly affects the limbs which have been most exercised. Where the skin fails to excrete, an increased task is thrown on the kidneys, . . . and if these organs fail in the task, the lactic acid accumulates in the blood, and, probably acting as a ferment, causes the formation of more, and of the kindred products, lithic acid and its compounds and products; these, in inflammatory subjects, excite rheumatic fever; in cachectic persons, miliary fever, erysipelas, or pemphigus ; and in more torpid frames, various local rheumatic or gouty affections. All these cases are frequently remarkable for the acid character of the cutaneous and renal excretions, and in a few instances the blood has been found to possess acid qualities, or to be deficient in its usual alkaline reaction. . . . The remedy for rheumatism and other diseases arising from defective excretion, therefore, should not be merely antiphlogistic, but also of a kind calculated to eliminate the morbid matter from the blood."

It is probable Dr. Todd has done more to advance this theory and to confirm its belief, at least in England and this country, than any one. It is he who so strongly insists on the diathesis as the basis of the rheumatic state. He says: "The constitution is liable to become modified under the influence of cold, imperfect nutrition or defective assimilation, so as to give rise to what may be called the *rheumatic diathesis.*" After calling attention to its resemblance to other diseases confessedly "due to the introduction of a morbid material into the blood," and insisting that "rheumatic fever"—the term by which he prefers to designate the paroxysms of the disease—is not symptomatic of a local inflammation affecting one or more joints, but is, as Dr. Graves affirms, a disease which may exist without the articular affection, he comes to inquire into the nature of the morbid matter, which he thinks we may infer to be the cause of the rheumatic diathesis, as well as the rheumatic fever.

"This," he says, "is even a more difficult inquiry than that regarding the matter of gout. In the latter disease, we had some guide in the

actual excretion of a peculiar substance, *lithate of soda*, from the diseased parts. The remarkable resemblance, however, between the two diseases, may justify our concluding that a certain similarity may exist between the morbid matter of each." "The two most remarkable excretions in the rheumatic diathesis, or fever, are the urine and the sweat. Both of these are distinguished by the presence of an unusual quantity of free acid. The urine contains a large proportion of lithic acid." "The lithic acid diathesis, however, is by no means so strongly marked in the rheumatic as in the gouty state." "Lithate of soda is never formed in the rheumatic paroxysm, nor in the diathesis." "If with these considerations we take into account the most frequent causes of the rheumatic diathesis and paroxysm, we shall obtain a further clue to the determination of the problem we have proposed. These causes must be admitted to be imperfect assimilation and vicissitudes of temperature. . . . Hard work, exposure to cold and wet, bad food, are strongly contrasted as causes of the rheumatic diathesis, with the ease, comfort, and excess which give rise to the analogous one of gout. If, now, we remember that the skin is the great emunctory of lactic acid, and that bad food, or too little food, may give rise to its undue development as well as too much food, it is no wonder that, as lactic acid is imperfectly excreted through its natural channel, in consequence of cold in checking perspirations, and is too freely developed in the alimentary canal, it should accumulate in the blood and become eliminated at every point. Moreover, the long continuance of the causes which produce the defective cutaneous secretion and the deranged gastric one will give rise to the undue development of the lactic acid in the secondary destructive assimilating processes, thus infecting the blood from every source and tending to perpetuate the diathesis."

It is not, I trust, without great interest to review, in their own words, the teachings of these great men who have mainly promoted this theory of the pathology of rheumatic affections—the view which to-day probably commands more believers than any other. It is especially necessary to remember the state of knowledge on animal chemistry at the time this theory was originated. Prout was among the early students and observers of the animal secretions, and in his first book on urinary diseases, written about 1820, nothing is said in respect to the relations of rheumatism. It is in his second work, written twenty years later, that his views are fully developed and expressed, and it is from this volume that the quotations given above are taken.

It must be remembered, in judging of this theory and the credibility of the opinion of those who first promulgated it, that at the time of its inception the prevalent chemical opinions of the composition of the blood and the urinary products were wholly different from those held since and at present known for facts. Prout and the chemists of his time believed that the blood contained among the other salts a notable amount of lactic acid combined with soda. The urine likewise was held to contain between seventeen and eighteen parts in a thousand of lactic acid and its compounds.

This opinion, of the presence of lactic acid as the peccant matter in rheumatic affection, was perhaps justifiable, or at least not unreasonable or absurd so long as the former chemical nature of the animal economy was held to be true. The opinion, however, continued to be maintained long after Lehmann and others had discarded it and shown it to be erroneous, from the fact that both the blood and urine were partly composed of lactic acid and its salts.

Dr. Prout seems to have honestly held the view that lactic acid was retained in the system by the action of the exciting cause of the disease, viz., cold, or perhaps was generated in increased quantities through the derangements which this agent wrought on the secondary assimilating processes. But when advanced chemical researches show that this acid is not a normal constituent of the economy, this theory must fall at once, unless it can be supplemented with pretty distinct proof that under the abnormal conditions lactic acid is developed. It, therefore, does not seem that Todd and his followers were justified in maintaining this view, since neither they, as clinical observers, nor the physiological chemists of that time offered such evidence as seems to be demanded by the circumstances of the problem.

Physiological and Chemical Origin and Nature of the Lactic Acids.—The origin of the lactic acid, the *materies morbi* supposed to be the cause of rheumatic affections, must be divided for consideration under two heads. The earlier notion in respect to the place of origin of the lactic acid was that it came from imperfect assimilation of certain articles of food, or from altered functions of digestion, and that both of these changes resulted from the effects of cold, damp, etc. This view was held to a certain extent by Prout, almost entirely by Todd, and Fuller even excludes the effects of changes of temperature, etc., in promoting the defective assimilation which he believes to be the cause of the development of this diathesis.

The other branch of the lactic acid theory is of late origin, and its complete statement has only become possible since modern physiology has pointed out the minute details of muscular and articular functions and their organic chemistry. This view of the lactic acid theory has been pretty fully stated, in fact developed, by Senator, and the main features of it are that the acid is developed in the muscles, as the result of their activity, and the effect of the chill is to cause a retention of this as well as other effete products, which by accumulation in the system produce rheumatism.

Let us now consider the mechanism of causation as presupposed under these two suppositions. Fuller, who disregards almost entirely the effect of chill, quotes Headland with approval in respect to the chemical changes which result in the production of lactic acid. His belief was that the starch of the food was converted into lactic acid through the natural operations of digestion and assimilation, and then combined with oxygen to form carbonic acid and water, in which form it is thrown out of the system by the lungs. *Anything* which interferes with this oxidation of lactic acid must lead to its excessive accumulation. As Fuller disregards the influence of chill, he infers that the disease is usually due to causes which prevent this oxidation rather than to the non-excretion of lactic acid by the skin, or to its excessive formation in the primary or secondary processes of assimilation.

The other authors who support this branch of the theory point to the chill as being a necessary factor in the retention, through defective skin-action, of the lactic acid, and perhaps also to the effect of the chill in producing an excessive amount of it, by deranging the primary assimilating or digestive process.

In considering the more recent physiological work and its bearing on this theory of rheumatism, it will be well to speak a little in detail, but as briefly as may be, of the chemistry of lactic acid. Three kinds or forms of the acid have been shown to exist; first, the lactic acid of fermentation, formed when saccharine or starchy matters ferment; and secondly and thirdly two forms of acid which are distinguished by a difference of solubility and by a different optical reaction tested by polarization. Both of these latter are derived from muscular tissue, and may be obtained by the chemical manipulation of beef extract or juice. The fermentation lactic acid is not present in muscular tissue, and cannot be obtained from it, or be derived from the two other isomeric forms. The two others have received the names of sarcolactic and ethylene-lactic acids, of which the

former is looked upon as most prominent and abundant, and is the one about which in this matter we are most interested.

The presence of lactic acid in muscular tissues was discovered by Berzelius, and it was subsequently shown to differ from the ordinary lactic acid of fermentation, as above related. It seems that these tissues in all animals, immediately after death, however recent, exhibit an acid reaction, and it was this fact, as well as the possibility of obtaining the acid in considerable quantity from muscle, which led many authors to believe in its presence during life. It is to Du Bois Reymond we owe the knowledge that the acid is non-existent so long as somatic life continues. "While muscle is alive and in a physiological condition it possesses a neutral reaction; so soon as it dies the reaction becomes acid." Previously the composition of muscular tissue had been studied in its dead condition, and it becomes of immense importance to distinguish between a "tissue which is yet living, though it may be separated from the living body of which it once formed a part, and one which has ceased to manifest the phenomena which it possessed during life."

It would seem, therefore, that there existed in muscular tissue an amylolitic or saccharine ferment, which during physiological action served to convert the nutritive material supplied by the blood into tissue, but which at the suspension of life wasted itself or turned aside to form lactic acid. The very first effect of the appearance of lactic acid in muscle, however, and in fact that which changes its reaction from neutral to acid, is the production of acid potassium phosphate by the decomposition of the alkaline phosphate. This condition is rendered very probable, if we recall the facts pointed out by Lehmann and others, that this salt is ever present in all tissue-forming substances, or as C. Schmidt remarks, is essential to supply the first basis for all new formations of tissue.

It must be remembered that all these studies, which have recently been summarized and still further developed by Gamgee, have been carried out mostly in cold-blooded animals, as their muscles continue their vitality so much longer that fuller opportunities are afforded for careful study. Our conclusion, drawn from such premises, should therefore be duly qualified, and above all, the amount of acid, developed under the most favoring conditions, should be remembered as being actually very small in amount. The older authors, too, speak very confidently of the rapidity of the conversion of lactic acid received into the blood from ingesta, and also of that formed, as they supposed, under physiological conditions in the

muscles themselves. This conclusion is arrived at from the results of therapeutic experiments, where, after the ingestion of certain lactic acid salts, the urine becomes rapidly alkaline, containing undue amounts of the alkaline carbonates, of which the carbonic dioxide is supposed to result from the conversion of the lactic acid.

However, by most authorities the presence of lactic acid even in the chyle of the thoracic duct and also in the blood is generally denied, and has certainly never been shown positively to occur, and this, too, even when its presence is acknowledged in the small intestine, as the consequence of the digestive or other changes in amylaceous or saccharine materials of the food. Still further, the presence of lactic acid is denied, or has never been proved to form a constituent of the lymph or venous blood returning from the muscles, which are supposed to manufacture it in such abundant quantities. It has not been claimed that under physiological conditions the conversion of lactic acid into carbonic dioxide or other products takes place, as do so many of the other nutritive or constructive and retrograde transitions in the muscles themselves, and certainly this conversion cannot be claimed as the normal process for the lactic acid derived from disordered primary assimilative processes, as claimed by Todd and others, as the origin of the supposed materies morbi of rheumatism.

It would seem, therefore, that in deriving the peccant material from ingested lactic acid, the source and the seat of its activity as a morbid or foreign product were separated by a space over which the acid does not show a capacity for overstepping. And this much at all events can be denied to lactic acid from digestion, as the initial factor in the production of rheumatism, however much may be laid at the door of other harmful matters of an unspecified nature, as referred to by Fuller, derived from the disordered primary assimilative process.

Now, with regard to the lactic acid of the muscle—sarcolactic—it is claimed to be present or rather developed more rapidly and in greater abundance during muscular exercise, and it is to the accumulation of these products, as the experiments with frogs' legs show, that the exhaustion of the muscular irritability is due; but how far the presence of this and similar products can explain the occurrence of fatigue, as claimed by the advocates of this more recent phase of the lactic acid theory, is a matter of much doubt.

If these matters are not the cause of muscular fatigue, a very special piece of evidence that lactic acid is the cause of rheumatism is taken away.

The presence of lactic acid, as yet unproved, has been claimed as the materies morbi, and that it was rapidly developed there from the retrograde changes of the exercised muscle—these being almost necessary precedent conditions to the development of a rheumatic attack. If these matters cannot be shown to be the cause of fatigue, the evidence fails almost completely that their presence, and especially that lactic acid is the cause of the rheumatic phenomena.

It is maintained that these products are ordinarily carried off and excreted or decomposed, but that chilling of the surface coincident with or following muscular fatigue prevents the elimination of the muscular products by the skin, which is thought to be one of the usual channels of escape. However, an analysis of sweat, either in health or in disease, has failed to show clear evidence of the presence of lactic acid. The striking difficulties of examination, chiefly consisting in the difficulty of obtaining a sufficient quantity for testing, and in the rapidity with which alteration in this fluid occurs, may well excuse an uncertainty on this point.

The old notions of humoral pathology, especially such as are represented by Todd, held that the peccant matter of the disease was excreted by the skin, and that this acid gave the reaction to sweat which is so often a prominent symptom of rheumatic fever. This idea and a single analysis of Favre furnish the only grounds for the belief in the presence of lactic acid in the cutaneous secretions.

The next conception advanced in promotion of the lactic acid theory of rheumatism was the effort to prove that its symptoms could be developed by the administration of this substance. The experiments of injecting lactic acid into the circulation or one of the cavities of the body have either proved to be failures or the results have not been conclusive. The exhibition of the acid by the stomach in the treatment of diabetes has been quoted by several as effective in producing the symptoms of rheumatism, and that the phenomena disappeared with the cessation of the use of the drug. This result is certainly very striking, and if it can also be shown that the acid enters the system, as such, thus to reach the parts principally affected in rheumatism, the theory must certainly receive its most positive element of support.

It is also known that the rheumatic symptoms produced in diabetic patients by the administration of lactic acid disappear again, although the remedy is continued. This fact at once throws a strong doubt on the

cause of the articular phenomena, and certainly does not point very strongly to their being due to the ingestion of the acid.

Still, I would submit that this item of proof is a very different matter from the statement of the theory as it is now held, viz., that the rheumatic symptoms are produced by changes taking place in muscles as the results of exercise and the retention of these products from chilling of the surface of the body.

The differences between the old lactic acid theory of Prout and Todd and this new belief in the mechanism of the disease have already been spoken of. No allusion has, however, been made to the fact that the other avenues of escape and elimination of the products of metamorphosis are unaffected by the chilling of the skin. The lung and the kidneys under the effects of the chill remain uninfluenced or are at least unrestrained, and are therefore, as we know, capable of assuming a compensatory vicarious action, whereas in the diabetic patient in whom the administration of lactic acid is followed by rheumatic symptoms, these two avenues are especially interfered with as the results of the diabetic disease. In addition—and this is certainly a more important consideration—in the diabetic it is probable that some of the organs—for example, the liver—are so interfered with that the primary as well as some of the secondary processes of assimilation are incapable of assisting in the decomposition of the lactic acid, administered as medicine, or even of producing entirely new products during the course of assimilation. Hence, lactic acid may accumulate, or be capable of producing effects in the diabetic which it does not possess in persons in usual health, and therefore the facts noticed in the abnormal condition cannot, I think, be taken as just conclusions to be drawn in relation to the rheumatic attack, for it is out of perfect health, so far as we can judge, that the supposed accumulation of lactic acid occurs when it produces oftentimes its most striking rheumatic phenomena.

The Defects of this Theory.—In the strictures which have been advanced against the lactic acid theory the argument is not intended to bear so much against what may be denominated the action of effete products, or at least to the mechanism of their accumulation in the system, or even their local action, so much as against the specific substance, namely, lactic acid. I do not think that the acid should be spoken of as the single offending matter to the exclusion of perhaps many others, which we may suppose can be produced by a similar mechanism—a mechanism which, except according to Fuller, is quite an essential part of this theory.

Lactic acid has, I think, received an undue prominence and has been placed in a position which, I have tried to show, it obtained probably through a mistaken chemical analysis. Prout's exaggerated conception of the occurrence of the acid probably led him in the first place to consider it as the materies morbi of rheumatism. He makes no mention of the earlier discoveries of Berzelius, and in no way traces any connection between the lactic acid of digestion and sarcolactic acid of muscular work. There is, in addition, but little unity between the earlier lactic acid theory of Prout and Todd and the later development of the idea of its production from muscular action and fatigue. It would seem, therefore, that having once been accepted as the peccant material, lactic acid has been continued in the rôle, although its source and the mechanism of its production have been entirely changed according to the later notions. It is difficult, therefore, to view the matter as presenting in any way a consistent face; the two ideas are almost as dissimilar as if they related to an entirely new substance. The latter does not receive any support from the former.

It may not be thought consistent to object to lactic acid and at the same time refer to other unknown, unmentioned materials of not dissimilar origin. While at first glance this may seem a just criticism, I do not think on examination it will turn out to be so. The objection against lactic acid is on account of the very slender proof of its presence, so slender as to be almost wholly wanting, and moreover it is a very different matter to object to the specific substance and yet to accept the theory of the mechanism of its production, which, so far as shown, is capable of the accumulation of many other substances in addition to itself. Much better to rid ourselves of the single definite product and accept a host of unknown materials with a probable and consistent cause for their formation, even should that cause be the same as for the origin and accumulation of the single product, namely, the lactic acid. I judge that it is not by any means so desirable to know the exact peccant material as to ascertain the means of its development. It is the desire to correct the abnormal process, rather than to neutralize, chemically or otherwise, the abnormal matters developed by the process, which controls our action and must direct every correct pathological research.

In the old humoral pathology, and in the even older theocratic conceptions of disease, all attention was directed to the discovery of the poisons which were productive of diseased action. Thus there was all the time search being made for the offending material, which must be expelled or

neutralized; according to still older notions, it was a devil to be cast out, and whether called a humor or a devil, it was very much the same thing. The one idea was but a continuation of the other.

The conception that disease is but a deviation from the normal physiological type of action, either by excess or deficiency, is of comparatively recent origin, and its essential truth does not as yet seem sufficiently widely extended. Although many abnormal products of diseased action may be present, or even normal ones may vary by excess or deficiency, it is scarcely sufficiently understood as yet that these products are neither the disease nor the cause of the disease. A very great deal of wasted effort has been expended, both pathologically and therapeutically, in explaining and combating the presence or absence of various materials which had far better be employed in considering the cause and the nature of deviated function.

So it is with rheumatism. Whether we lend support to any one of the various conceptions of its nature—whether we consider it a simple local disturbance (now entirely discarded), or a zymotic disease, or produced by the action of cold on the vascular supply of a part, or (what is much allied to this idea) that it is a vaso-motor neurosis, we cannot too strongly endeavor to disassociate our ideas from the products of diseased action, and concentrate them on the deviated function which leads to their production. Even should lactic acid be shown to cause invariably rheumatic symptoms, when injected into a joint, or that it was always present as the result of deviated action, our study must be to correct the process which produces it. The production of lactic acid has its own cause, and it is to the study of this cause we should devote our attention.

Again, these ideas of physiological pathology, as applied to the study of rheumatism, seem particularly appropriate, and apply themselves in harmony with its peculiar characteristics. No disease bears so little the impress of being a poison-disease. Out of good health suddenly come pain and fever with local phenomena. It comes under conditions of good hygiene with striking frequency. No pathological condition so little leads us to think of any deleterious matter absorbed into the body, and the lapse of time between the exposure to an apparently efficient cause and the onset of its symptoms seems too short for the accumulation of products of an irritant or poisonous nature to take place, at least in a quantity sufficient to produce such striking effects and yet to escape detection and remain through centuries unknown. Besides, the local phenomena are

often very fleeting in character and, when durable, very unlike those we usually connect with the results of an irritant, whether of local or general origin.

Another fact should be alluded to in discussing the specific substance supposed to be productive of rheumatism. According to the later lactic acid theory, this substance takes its origin in waste products of muscular exertion, and is supposed therefore to exist in the muscles themselves. But it is in the articulations that the symptoms first and most conspicuously manifest themselves, and although in muscular exercise the joints necessarily participate in functional activity, yet nothing has been shown in connection with articular processes, normal or pathological, which directly leads us to think that lactic acid is produced in their structures. Although we have no difficulty in tracing the entrance of the lactic acid irritant into the synovial cavities, yet I think we ought to expect to find a preponderance of irritative phenomena in the place where the peccant matter took its origin. Granting, therefore, due weight to the fact that the functional activity of the joints renders them perhaps especially susceptible to diseased action, still I think we should expect to see the brunt borne by the muscles which according to this theory produce the irritant material. And while functional activity of the joints helps to explain their participancy in the rheumatic phenomena, it does not contribute anything to showing that lactic acid is the morbid matter which produces rheumatism.

Infection Theory.—Lastly, let us examine what is the conception of rheumatic disease which has been formulated in accordance with the terms and requirements of the Germ Theory.

This new theory of the pathological nature of rheumatism has been called the *infectious*. Its claims have recently been stated very clearly by Müller, of Zurich. The idea of the infectious nature of the disease was pronounced more than twenty years ago by Hirch, and others have followed him in his views. Müller states that acute articular rheumatism is a general disease, which is due to a specific excitant. The joint affections are only a symptom and depend on infection. The complications are explained by the same cause. Articular rheumatism belongs, therefore, to the febrile infectious diseases.

It will at once be perceived that this theory differs from the usual ones in vogue in attributing the cause, not to something developed within the organism, but to an infectious material imported from without, which either by its presence or by its development within the system directly

produces the phenomena of the disease. Further, this cause is not cold which serves as the foundation of most of the other theories of its pathology, or at least this agent can only be regarded as a determining or exciting cause of the disease. In relation to the influence of cold on rheumatism, Hirch, in his medico-topographical studies, claimed to have shown that this disease was absent in localities especially affected by diseases pre-eminently due to this morbific agent, while in other places where the influence of cold or changes of temperature or climate were but slight that rheumatism was especially prevalent, and that this disease concerned "a specific acute infection, which perhaps has similar relationships to rheumatism as influenza has to catarrh."

The occurrence of so-called epidemics of the disease, to which allusion has already been made, is depended upon to show a probability of its infectious character, or at least its similarity to other diseases of an undoubted infectious nature. The age at which rheumatism most frequently makes its appearance has been pointed to as the same time of life which favors typhus and typhoid fevers.

Not only in the apparent etiological relation of the disease, but likewise in its clinical relations, Müller, in particular, claims that there is a resemblance to infectious diseases. An exact study of a large number of cases, he says, will show that rheumatism does not commence within a few hours after an exposure to which its origin is usually attributed. In other words, it is not a disease without prodromes, but that often for many days the usual febrile phenomena precede the development of the articular symptoms. Ordinarily, however, this condition is so slight that no note is taken of it. This fact, he claims, shows that acute articular rheumatism is not a disease which owes its first existence to local phenomena, or is due to the retention or development of lactic acid. The poison must be an element in the blood, as it is in other infectious diseases, and that the infectious element seeks the joints pre-eminently because here it finds the best condition of its growth and development.

The occurrence of rheumatism subsequently to attacks of scarlet fever, as well as other infectious diseases, is pointed to as favoring the view of the similarity of their nature. Likewise the occurrence of articular disease of a similar character to the rheumatic synovitis, as a concurrent symptom in such purely infectious diseases as pyæmia, septicæmia, and puerperal fever, is strongly insisted upon.

The complications of rheumatic fever, namely involvement of the

serous membranes, the pleura, the pericardium and endocardium, are of the character found in other infectious diseases and quite unlike those occurrences in a simple inflammatory condition.

The occurrence of hyperpyrexia is similar to that coming in other infectious diseases, such as erysipelas, intermittent and typhoidal fevers.

Finally, Müller points to the evidence of pathological anatomy in support of his theory—the occurrence of parasitic organisms, which were found in a patient who died of acute articular rheumatism, in whom small multiple abscesses were present. This is the evidence of v. Recklinghausen in 1871. Fleischhauer, in 1873, found micrococci in miliary abscesses in the lungs, heart, kidneys, and in almost all the muscles, in a patient dying of a disease having the ordinary clinical features of articular rheumatism.

Eberth (1875) has likewise shown a parasitic character for the endocardial changes in a number of cases. Köster (1878) believes that the endocarditis of this disease depends on micrococci-emboli of the valvular capillaries.

CHAPTER V.

DESCRIPTION AND COURSE OF THE DISEASE.

Acute Rheumatism: the Prodromal Interval; Chill; Fever; Pain; Aspect of Face; General Bodily Condition; Skin; Pulse; Temperature; Digestive Functions; Urine; Sleep; Delirium; Menstrual Function; Epistaxis; Course, Duration, and Severity; Result.—Subacute Rheumatism: Its Distinction from other Grades and Forms.

Acute Rheumatism.

The Prodromal Interval.—Almost the first question that occurs in studying this part of our subject is the length of time elapsing between the application of the cause and the onset of the characteristic symptoms. In all infectious diseases this interval is one of great importance, and is called the period of incubation. In rheumatic affections I judge it to be incorrect to apply this term to the period here alluded to, for although the cause of rheumatism is unknown, we are sure that it is not a specific poison introduced into the system from without, as in the case of the infectious or contagious maladies, and there is no materies morbi which requires time for its development by generation, as is supposed to be the case in this class of diseases. This interval in rheumatic affections is therefore of less importance, and is allied in its essential characters to the period which precedes, for example, the development of pneumonia, or let us say, between the receipt of an injury and its consequent inflammatory or febrile phenomena, although this latter instance is not a precisely parallel one to rheumatism. Dr. Haygarth (1805) is the first writer who I find has spoken particularly of this question. He calls it the "latent period" and in twenty-one cases the time between the exposure to cold, which he considered *the* efficient cause, and the commencement of the fever, varied from half an hour to ninety-six hours.

While the determination of this interval preceding the commencement of fever may be a matter of some interest, a knowledge of it is not really

valuable in affording us information either as to the cause or the nature of the disease, in which respects the incubation period of infectious and contagious maladies is so conspicuously of use. And then, too, there is no agreement as to what particular symptom is to be regarded as the real commencement; some would think of the preceding malaise, others would refer it to the febrile phenomena.

The older writers, following Haygarth, nearly all concerned themselves with observing the length of this period, and in accordance with their belief of the true cause of rheumatism it afforded a proper subject of study. It was a simple matter to note the elapse of time between the application of the cause—and it was generally held by them to be cold or some atmospheric influence easy of determination—and the development of the first symptoms. While most of these writers agree with the figures of Haygarth, or quote approvingly his statement, quite a number of observations are recorded of a prolonged period or a delayed appearance of the disease; it is quite natural that such should be the case so long as the opinion held in force is that only one set of influences were productive of the disease. Cases were noted also in which no exposure to the then nearly universally recognized causes was discoverable.

Later writers have been much more guarded in expressing opinions on the duration of this interval, and very justly so. Since the nature and causes of the malady are still in doubt, it is manifestly improper to speak with confidence of the length of the period between the development of the cause and the onset of the first symptoms. Even granting that exposure to certain influences is an efficient cause for development of a rheumatic attack—certainly it does not always thus result, neither is it always a precedent circumstance—it is evident that other changes may be acting than the simple exposure, for example, a wetting, antedating the exposure; the latter, therefore, can only be regarded as a concurrent cause of the attack. The true and just statement, the only one warranted by our knowledge of the disease, is that the onset of the attack, evidenced by the commencement of the fever, is preceded by some trifling symptoms, it may be malaise, disturbance of digestion, or vague general or local pains, lasting for a variable period of time and differing greatly in their intensity. This period rarely exceeds a week, and on the other hand is equally rarely less than a day.

Chill.—The next phenomenon in the development of a rheumatic attack is the appearance of a chill or rigor. From a comparison of a large num-

ber of cases, both of my own and others, it is evident that a well-marked or severe chill is not a conspicuous symptom in the commencement of acute rheumatism. In about one-third of such cases only is it a sufficiently well marked or noticeable feature to be recorded or alluded to, at least by the patient. The more rapidly the onset of the attack succeeds exposure, supposing this circumstance to have preceded the development of the disease, the more frequently the chill is noticed, and likewise is the symptom more marked in intensity. In other cases where the development is slower or without severe or perhaps discoverable exposure, the chill is more generally absent or is more properly described as a chilly or creeping sensation; frequently in these latter cases the rigors, always slight in degree, are repeated.

Fever.—Probably immediately or very shortly after either the chilly or creeping sensation, the bodily temperature is increased and afterward falls to rise again subsequently to the succeeding creep or rigor, if such occurs. The opportunities for observing the temperature oscillations in the stage preceding the onset of acute attacks are not very numerous, for the patients are not usually under observation, and they have not been extensively availed of or recorded. Such, however, have been the results of my own experience, and they correspond with those of a few others. There is nothing characteristic in these thermometric changes; they are only what would be expected in connection with these prodromal symptoms. The longer the precedent stage is drawn out and the more pronounced the symptoms in connection with it are, the greater is the tendency to these slight oscillations of temperature.

Whatever may chance to be the duration or the character of this precedent stage, or if entirely wanting, the important rigor, whether slight or well marked, which announces the immediate onset of the attack ushers in the fever, which thenceforward slowly or rapidly rises and becomes continuous. The initial rigor of the onset is rarely of a marked character, often less pronounced in its subjective phenomena than the sensation of cold from the exposure or wetting, so commonly the cause or precedent of the rheumatic attack; its objective phenomena are also usually of but slight intensity; pallor of the surface, coldness of the extremities and slight shivers, without cyanosis or convulsive movements only are noted.

The fever is most generally of gradual development, and rarely is a high fever heat attained before the second or third day after the real

onset. It is only in very severe cases that a sudden rise takes place, while usually the temperature mounts up slowly but pretty continuously.

Pain.—The pains, usually present in the precedent stage, are vague, flitting, or transient, becoming concentrated, so to speak, in one or more joints; joints which previously were especially painful are apt to be first affected. In some cases, before the onset of the fever, one or more joints are the seat of aching, and these subsequently are the ones to become painful, so that we have the manifestation of a continuous development of the joint affection more or less suddenly accelerated by the accession of the fever. The more rapid the onset of the attack the earlier the joints, alone or principally, become the seat of severe pain. After the accession of fever and the marked concentration of pain in the joints, the fleshy parts of the limbs, as well as other muscular areas still continue painful, and the sensation of these parts not infrequently increases along with the articular pains, although usually this feature is less marked or at least less complained of than the supreme suffering of the articulations.

With the pain, or soon after it, comes heat, swelling, and redness of the affected joints. Of these three symptoms, one or more are generally pronounced, or it may be all three, and are, in accordance with their position, more visible in the joints the least covered with overlying tissues, as in the extremities, which are the articulations more commonly affected, although the same conditions obtain in joints, such as the vertebral, though concealed from observation. In the superficial joints we are able to assure ourselves that the swelling not alone affects the tissues around the joints, but is due, in part and it may be largely, to fluid within the articular capsule. This condition is evidenced by fluctuation, which is always, in this stage at least, of a tense character, and it can be detected only by firm pressure, partly because of the swollen and thickened tissues overlying the joint and partly, perhaps principally, because of the necessary tight spanning of the joint capsule as it yields before the fluid accumulating within its cavity. In fact, so characteristic is the tense fluctuation of an oncoming articular inflammation, or one well developed, that a boggy or loose condition of the capsule becomes an evidence of a lessening or cessation of the articular inflammation and the beginning of an absorption of the fluid. The tense and tearing or rending pains of acute rheumatism, apart from their production as in ordinary inflammation, are thought to be greatly increased by the stretching of the capsule of the joints by the accumulating fluid within them. And it is generally to be observed

that joints which become greatly distended are, during the stage of accession of the disease, more painful than those less swollen, and it also happens that the greatly distended joints, whose ligaments become relaxed, are during the retrogressive stage less uncomfortable than those equally inflamed but not stretched by inflammatory exudation.

The pain of the inflamed joint is probably rarely or never equalled by any other form of articular inflammation except gout. The dread expressed by the patient of the slightest touch of the examining hand, the contact of the clothing or even the imperceptible shaking of the bed by the movement of any one in the room is quite characteristic, and at the same time expressive, of the suffering endured and the severity of the disease. Generally, at this early stage of the attack, the patient is in bed, confined there more by the disinclination or impossibility of rising and dressing, owing to the pain produced by movement, than from the severity of the general symptoms. The disease has been gradually advancing, the malaise has been marked, but not sufficiently to induce the patient to remain in bed, but retiring to sleep for the night he is either awakened by the increasing pain or on attempting to rise in the morning finds it impossible or so painful as to be deterred from the attempt. In other cases the accession of pain, and with it the fever, comes on by day, surprising the patient while dressed. The efforts to remove the clothing are found so painful that undressing is not undertaken until the disease is well advanced. Days and nights are not infrequently spent resting in a chair or lying on a couch or bed, until it becomes necessary to cut the clothing away and put the patient to bed properly, and break up the suffering vigil.

The aspect of the patient is expressive of suffering and a dread of being approached or moved. He acts as though concealing a treasure under cover, which must not be moved or touched, and if compelled to change position the whole body is made to revolve around the painful joint, rather than disturb the affected member. When allowed to remain at rest the face and body assume the air of semi-comfort and intense quiet, but withal there is an air of vigilance. This constantly maintained condition is productive of a strain and is capable of developing a nervous irritability, which necessarily must add to the characteristics of this stage, and also tend to the increase of the general symptoms, apart from the due advance of the disease. The nervous effects vary with the innate constitutional peculiarities of the patient, and become increased or decreased proportionately to these conditions.

Aspect of Face.—Otherwise the appearance of the patient, especially the face, is not indicative of the severity of the disease, and in respect to the fever, does not ordinarily show in this early stage the changed aspect proportionately to the febrile movement present as is exhibited in other diseases. Except after movement, which from the pain endured would induce flushing of the face, no redness or suffusion permanently remains. Paleness or the sallowness of anæmia are very common, but these are to be looked upon as antecedent characteristics rather than peculiarities of this stage of the attack, except, of course, so far as their causes may be considered as equally a cause of the rheumatism; this opinion is held by some writers. In a large number of cases the color and aspect of the face is one of health, altered by acute suffering. An appearance of the face, which I have frequently noted, but which I do not find is alluded to by others, is a turgid, more or less cyanotic one. The condition is one which from its causation is brought about only in the severer cases, where many joints are affected and the pain very severe, or even in the slighter cases during necessary movements or the examination of the patient's physical condition. The turgid face or cyanosis is produced by the voluntary or involuntary efforts in attempting to hold themselves still, to render the painful joints or members immovable, and to lessen the pain due to slight general movements of the trunk or members, produced by the respiratory movements. The cyanosis is entirely apart from, and occurs independent of any cardiac or pulmonary complications and the pains which accompany these lesions. The phenomenon bears the same character as the similar condition seen in any one making a strong or prolonged muscular effort, and is like, though perhaps less marked, to what is witnessed in a patient during the setting or dressing of a fractured bone. In this case the efforts to render the injured member immobile cause an involuntary muscular strain which quickly results in a turgid darkened face.

This phenomenon is, so far as concerns the general condition of the patient or the course or characteristics of the disease, a matter of little moment, although one which is, I believe, rarely absent. Its importance arises from its furnishing an indication of the degree of suffering experienced by the patient. Of course its production is easy, but not likely to be simulated by a malingerer, while the other facial aspects and contortions of pain are familiar, and are but too ready to make their appearance as attestation to the degree of suffering endured. In those patients who, without feigning or meaning to exaggerate their agony, frequently mislead

us, we have a ready test by which to graduate the anodyne or narcotic remedies, if this condition of the face is carefully noted under proper circumstances for proving its reality.

General Bodily Condition.—The general surface of the body is warm but not very hot; it never gives the sensation of dryness and burning heat which are found in other diseases attended with an equal elevation of temperature. In severe, and even in moderately severe cases, accompanied by great pain in the articulation, the surface is bathed in perspiration, often very profuse in amount. The patient in this condition gives off an acid odor and the skin secretion is of acid reaction. It would seem, at least in many, that as the temperature increases, the sweating becomes more profuse, and it is certainly true that the more joints involved and the more severe the pain, the greater is the amount of this secretion.

Skin.—Sudamina make their appearance often in great abundance and in successive crops. Miliaria and other forms of skin eruptions are not infrequently witnessed.

Pulse.—The pulse may be unaffected, but generally increases in frequency from the beginning, and varies from 80 to 100 in moderate cases, and in severe cases runs up to 120 or more in the minute. Its character differs very greatly in different patients, but maintains in general the same characteristics throughout the same attack. It is generally full; it may sometimes be found hard but much more commonly is soft and compressible. In the later stages of prolonged attacks it always becomes soft, small and feeble in proportion to the degree of anæmia. In complicated cases or where rheumatic visceral localizations occur during the course of an attack, the pulse is very liable to become much altered in character, and its frequency is subject to great variations on the accession of new articular involvements.

Temperature.—The temperature in all forms and in every grade, except, perhaps, in chronic rheumatism, is raised above the normal; in general it is found to range in different cases according to their severity within the limits of 99.5° F. (37.5° C.) and 104° F. (40° C.). Cases occur where the heat is so little elevated that careful observations are required, frequently repeated during the day, to detect any thermic excess, and on the other hand cases of hyperpyrexia are not infrequently noted in which the upper limit here given is far exceeded. The rise of temperature is generally a gradual one, and the oscillations during its course, except those due to fresh localizations, are not striking. The termination of the febrile stage

is not marked by any crisis in the temperature curve, but the bodily heat gradually returns to the normal with greater or less rapidity, and more or less irregularity, proportioned to the regular course of the disease.

Digestive Functions.—The digestive organs and functions may in the slighter cases remain intact, and even the desire for food continue unabated, eating being interfered with only by the painful efforts in handling the food. In the severer cases the appetite is lost, the tongue coated with a moist, whitish fur, and often heavily ; vomiting is rarely present and not characteristic ; the bowels are usually confined and their evacuation greatly hindered or prevented by the pain of movement.

Urine.—The urine is acid, high-colored, and scanty ; it lets fall abundance of dark uric acid products, has a high specific gravity, and in uncomplicated cases is free from albumen. Patients often retain the urine voluntarily a long time to escape the pain incident to voiding the bladder.

Sleep.—Sleep is greatly interfered with or entirely prevented by agonizing pains, and is often the greatest source of complaint by patients. This condition, however, varies during the course of the attack ; in the initial stage, before or during the effusion into the joints, the spontaneous agony prevents a moment's rest ; after the full development of the articular inflammation, if a comfortable position can be maintained, sleep is often ample, and is interrupted only by the pains incident to disturbance or movement of the articulation ; in the later stages of prolonged attacks the weary aching of the limbs becomes a source of great wakefulness.

Delirium.—Delirium is rarely present and is by no means a characteristic feature of the disease, even when the higher grade of febrile heat is reached. The mind is clear throughout the attack, except in cases of cerebral complications. Occasionally a slight wandering of the intellect is noticed on the occurrence of exacerbations or complications, or in nervous patients or those rendered irritable by the suffering.

Menstrual Function.—The menstrual function is usually interrupted or the flow is delayed in its appearance by an oncoming attack of rheumatism. If the fever commences but a very short time before the period, a brief or incomplete menstrual flow takes place ; a well-developed or severe attack usually entirely prevents the flow and alters the rhythm of its return. I have seen the menstrual period evidenced during moderately severe attacks by the discharge lasting for a few hours or during one day. The menstrual blood in these cases was very thick, and of a black color ; in these attacks, when promptly recovered from, the next menstrual period

returned in less than the usual time, but with the habitual characteristics. Occasionally it is profuse, if uterine disease exists.

Epistaxis.—Epistaxis is a phenomenon not uncommon in acute rheumatism; it occurs not at the beginning but coincidently with some important new localization of the inflammation and even perhaps more frequently at its remissions. It has been thought that certain drugs, as quinine and digitalis, have an influence on its occurrence. It is, however, without significance in relation to the nature or course of the disease.

Course, Duration, and Severity.—All varieties in the grade of severity of the disease are met with, and are classed accordingly as acute and subacute cases. In some, the general and local symptoms are very slight and interfere but little with the health of the patient affected. In others the local mischief, either in its severity or extent, is greater, and the general disturbance of health is proportionately augmented. In others, again, the disease, commencing even mildly, may attain a severity which, from the degree of fever and the intolerable agony, seem to threaten life.

After continuing for a varying period the disease very nearly always ends in recovery and a greater or less restoration to health. The course which any given case pursues is an indefinite one and without any recognizable type. The mild case may continue its course in the same grade through weeks, or may be prolonged by the involvement of new joints and a renewal of the inflammation in joints previously affected during the same attack. The mild case may become a severe one, by numerous joints becoming attacked simultaneously or by any or all of the many visceral complications. While mild cases, as a rule, end more rapidly than severe ones and a good state of health returns sooner, still the actual rheumatic symptoms may be continued in their subacute form, without exacerbations or complications, as long as in the more severe cases; the return to functional activity of the joints and to general health is always more rapid than in severe cases where the affection has remained for a long period and at the same time has been severe. The visceral complications, while they do occur in mild cases, though perhaps not as frequently as in those of higher grade, do not seem to be as severe in the former as in the latter, although their permanent organic changes in the organs involved are just as lasting in the mild cases as in the severe ones.

A case which at its beginning exhibits high fever and great pain may suddenly mitigate in its severity and even promptly recover. It is these cases which are spoken of as abortive cases and which have been so mis-

leading as to the results of treatment and so confusing to statistics. The initial severity yielding so promptly under the use of a particular drug or plan of treatment has been placed to the credit of the therapeutic agency, and this result has but too frequently not been confirmed by subsequent experience.

The cases in which the fever gradually increases to a high grade and in which joint after joint becomes involved without the complete cessation of the process in the articulations first affected, are more generally apt to be the prolonged and severe ones—severe from long impairment of the general health, and from their visceral, especially cardiac, complications.

However, cases which but too often present little favorable appearance for rapid recovery end quickly and leave no bad results, articular or visceral, behind them, and, on the other hand, cases seemingly promising to be of short duration have their course, interrupted by exacerbations and relapses, greatly prolonged and leave behind a residuum interfering with articular functions or a susceptibility to the renewed occurrence of inflammation, or even worse, a damaged heart or lung.

Result.—Death rarely results from uncomplicated acute rheumatism, and when it comes, the temperature shortly preceding this event rises to an extreme elevation. In fact, a sudden rise beyond 106° F. (41.1° C.) is pretty surely fatal, and is rapidly followed by death unless checked by therapeutic means or quickly lessened spontaneously. The complications, their nature, course, and influence on the general symptoms, will be referred to separately, later.

Subacute Rheumatism.

The classification or distinction of certain cases as subacute, is one that is not used among German writers, and but little among the French. In England the designation is much more common, but less so than with us. Among the older writers the use of the word is scarcely, if at all, to be met with, and it has sprung up comparatively recently to meet a demand for a ready and brief method of distinguishing between the different grades of rheumatic attacks. For this purpose and in this sense the term is a very proper and useful one and has met with general acceptance.

Whatever may be the views held of the pathological relationship between acute rheumatism and the chronic or muscular forms of the disease, it is clear that the affection intended to be designated under the head of

subacute is that which is allied to acute rheumatism or rheumatic fever or that spoken of by the Germans as polyarthritis rheumatica. It is proper, therefore, that a term now so frequently in use should have its range carefully pointed out, and that it should be fully understood whether we mean by it cases which pursue a more or less definite course, within the very wide range which every case of rheumatism has, and are characterized by the presence or absence of certain symptoms or complications, or whether it is intended to indicate merely mild cases of the acute disease as distinguished from the chronic forms, or whether also it is used for mild cases of every form of the collective set of symptoms grouped under the head of rheumatism. In a disease like rheumatism, where the line of demarcation between the various principal groups is as yet vague and uncertain, it becomes a matter of still greater difficulty to distinctly outline the boundaries of a sub-group and to enforce uniformity of designation.

In the present use of the word, the most extended uniformity among both writers and observers obtains in its application to conditions present during the stage of abatement of the symptoms as frequently seen after acute attacks. After the fever has subsided, but not entirely gone, the pains are lessened but yet are aggravated by movements and show a tendency to redevelop, the swelling has not subsided, while the periarticular thickening and the immobility of the joints still exist, the cases which previously exhibited severe symptoms are said to have assumed a subacute form, and it is under these conditions that most generally rheumatic attacks find their greatest prolongation and exhibit their most stubborn resistance to treatment.

Many cases, however, make their appearance and continue throughout their course as subacute, although liable to aggravation, or more or less temporary exacerbation of their symptoms, and it is concerning these cases that the importance of a correct designation of the term subacute is of interest. Many maladies, as well as other rheumatic affections now distinctly separated from acute and subacute rheumatism, have been grouped under the head of subacute cases, and have proportionally warped the essential point of view from which they ought to be regarded.

Of these it is not necessary to speak of many cases of slight, perhaps forgotten, injuries of the joints which run a course without active symptoms and which, from improper treatment, as well as the mechanical alterations of structure, become indefinitely prolonged; these are but errors

of diagnosis. Other cases, as those of rheumatoid arthritis and certain cases of chronic gout, previous to the time of their true nature becoming known, were spoken of as subacute rheumatism. For these and other reasons, perhaps partly from conditions inherent to the grade of the diseased process, it has been customary to speak of subacute attacks as being essentially prolonged, and as showing a less tendency to terminate promptly and thoroughly in a return to health and normal conditions, than a rheumatic attack which has exhibited a greater activity of its phenomena. Hence, not unfrequently subacute cases have been spoken of, and are still regarded as having a nature midway between acute cases and those of a chronic character, and being rather allied to the latter than to the former.

While it may be true that subacute cases have a greater duration than acute cases, this characteristic is not part of their essential nature and is not due to a stronger alliance with chronic rheumatism. This phenomenon of their course is apparently dependent on another cause. Subacute cases do not always have a prolonged course; many of them, mild throughout, end definitely, and the patient returns rapidly to health and normal conditions.

Not a few instances of subacute cases occur in otherwise perfectly healthy persons, never the subjects of a pronounced rheumatic attack. To these cases but little attention has been given, and it is, in fact, not welcomed by the patients themselves, if their medical adviser attempts to apply the name rheumatic. A strong, healthy person spurns the idea of being rheumatic—and spurns with it even more vigorously the physician and his unwelcome diagnosis. Such cases, in the few instances I have been able to study, generally to the patient, under a different name, have been attended with many of the symptoms, very much reduced in grade, found in moderately severe attacks. The history shows an exposure to some one of the causes to which a rheumatic attack is frequently traceable, and after a varying lapse of time, often but very short, preceded or not by antecedent discomforts of a very mild grade, pain occurs, vague and wandering, in one or more groups of muscles, or perhaps affecting one or other of the limbs, and a little later a joint, usually of the same member, becomes the seat of especial pain, of higher grade, or at least more noticeable than the general pain which precedes it; rarely is the involvement, either of the members or the joints, bilateral or symmetrical. With this symptom the feverishness is developed. This phenomenon is of slight grade, proportionate in every respect to the mild local affection, difficult

of detection, and is not accompanied with any acceleration of pulse; in fact, an acquaintance with the personal peculiarity of the patient is essential to the recognition of the very slight disturbance of the normal condition of the circulation.

Such a condition is one of very small importance, and for which the advice of a physician is rarely sought; perhaps even it is not worthy of a name, but if a name is to be applied the term subacute rheumatism cannot be denied to it. It is an acute rheumatism in miniature. Attacks of rheumatism, however, occur in which the disease throughout, at all its stages, assumes a subacute character, and in which the disability and even the sufferings of the patient are very considerable. It is for the designation of this form of the disease that the term finds its most useful and important application.

For entirely unknown reasons, some patients after exposure or the application of causes usually recognized as productive of acute rheumatic attacks, exhibit symptoms of the disease in a milder form. There is pain and tenderness of the joints, very commonly swelling, but rarely redness. The febrile phenomena exists only in their lowest grade, or are entirely wanting. This condition persists for a greater or less period of time, and often quite as long as a prolonged severe attack. The duration would seem to be increased in a manner similar to that seen in not a few other diseased conditions of a low type of activity, and perhaps also because such attacks, demanding neither active remedial measures nor extreme, or even prudent care, become protracted through the want of both.

After an indefinite course, the attack ends in convalescence and a return of normal functions to the articulations involved. That such attacks are of the same nature as acute rheumatism is but too frequently evidencd by their assuming a high grade of severity through a gradual increase of the previously existing symptoms, and again sinking, to be continued in the subacute form. These cases are not instances of either rheumatoid arthritis or of chronic gout, with exacerbations such as both of these diseases are liable to, because after a prolonged duration the joints involved do not become altered, as almost constantly results in each of these forms of joint affection. Besides, neither rheumatoid arthritis nor chronic gout, once fully established, subside so completely that the joints exhibit subsequently no deviation from the normal, as is so frequently seen in these cases of subacute rheumatism.

Again, attacks of subacute rheumatism are not rarely repeated again

and again in the same person without, after the subsidence of the last attack, the joints exhibiting any more permanent alteration of their form and structure than after the first attack. In fact, it would seem that a severe paroxysm predisposes or increases the susceptibility of a patient to renewed attacks, repeated at frequent intervals and from slight causes, of a subacute form of the disease. Frequently, too, the subacute attacks are seen to occur in succession, without being preceded by any severe manifestation of the disease. Oftentimes the subacute form exhibits itself, either preceded or not by acute attacks, as well as interrupted in its successive occurrence by the more severe outbreaks.

Its Distinction from other Grades and Forms.—In subacute attacks usually fewer joints are involved and are less severely affected. It is frequently stated that in this form the articular disease is not symmetrical, but as the joint involvement is so frequently non-symmetrical in the acute attack, this phenomenon is not a very characteristic or distinguishing feature between the two. On the other hand, the subacute form is not rarely seen assuming a symmetrical localization.

The smaller joints do not seem to be more frequently or more exclusively affected than the larger ones ; usually fewer joints become involved, and a joint once affected generally remains so during the attack. The complete subsidence of the diseased condition in a joint and the so-called metastases to other joints are less marked. The pains are less acute, redness is rare or seen only in slight degree, but the effusions within the joint, when these occur, do not usually reach a large amount, are slowly developed, indolent in character, and not subject to rapid change.

The tissues around and in the neighborhood of the articulations involved are very constantly affected, but exhibit the same tendency to indolence and to a low type of activity, and, in the more marked cases, while showing considerable swelling, that condition is not of firm character, and on subsidence leaves the structures less altered, anatomically at least, as judged by their external appearances, than is sometimes witnessed even after prolonged severe acute attacks, although the functions of these parts may remain for a considerable period much hindered. A resolution of this low grade of inflammation cannot be said to take place, at least in the ordinary sense of this word, so slow is the cessation of the active phenomena ; it seems to fade away and there is no point of time at which it can be said to be now here and then gone. It happens, perhaps not rarely, in some purely monarticular cases of this form of the malady, that,

from bad treatment or want of care, the joint disease is unduly and indefinitely prolonged, and the results and the consecutive lesions become strongly pronounced; from a misapprehension of the nature of the disease and from fixation of the involved joint by too strict care and avoidance of movements, false anchylosis from both peri- and intra-articular changes of structure may be produced.

The cardiac and other visceral complications are much more rare, less severe, and when they do occur are of shorter duration and apparently leave behind less permanent alteration. On the other hand, they are sometimes very marked. On this fact a strong point of alliance to the common form of acute rheumatism rests, and by it the differentiation of the subacute disease from chronic rheumatism, and especially from other so-called rheumatic joint affections having a grade or type more closely approaching to it, is frequently to be made. Severe visceral complications in subacute cases are witnessed, resulting disastrously or leaving behind them organic changes, especially of the heart, which permanently disable the patient and finally end fatally.

Patients throughout an attack of subacute rheumatism in its usual form rarely exhibit the profound anæmia of acute attacks. However, not a few of the cases, perhaps rendered susceptible by this state, are already anæmic; their mode of life, occupation, and their general nutrition may be productive of an anæmia, which is so constantly the resultant of the rheumatic disease. Not infrequently, the succession of the subacute attack to the acute disease or the frequent repetition of attacks render the patient more or less permanently anæmic; to this may be added the effects of pre-existing cardiac or other visceral disease, owing to the same cause.

The frequency of this form of the disease as compared to the acute attacks or to chronic rheumatism is a difficult question for determination. From its comparative slight severity it necessarily less frequently comes to observation, especially in hospitals, from whence alone accurate statistics can be obtained. My own impression, taken both from public service and from private sources, is that it is of exceedingly common occurrence.

Subacute rheumatism is, therefore, from its frequency, from its train of symptoms, a well-merited name, and one from its useful application well deserving of retention as a distinctive form of the disease—a form of less severity, one differing from chronic rheumatism and one especially important to separate from non-allied joint affections owning a different pathological state, but which it sometimes closely resembles.

CHAPTER VI.

CONSIDERATIONS OF THE INDIVIDUAL SYMPTOMS OF ACUTE RHEUMATISM AND THEIR PECULIARITIES.

Of the Fever: Chill; Temperature Curve; Modifications from Complications.

It is necessary to speak more in detail, than is possible in giving a general account of acute rheumatism, of some of the characteristic phenomena, as well as of more rare symptoms, which attend its course. We shall speak first of the onset, the course and the decline of the *fever*, as shown by the thermometer, and of some of the alterations of function which attend and are dependent on the febrile state.

Of the Fever.

The discussion of acute rheumatism should properly be opened by the consideration of its febrile phenomena. While we do not look upon the disease as consisting simply of fever changes—the conception which occupies us so largely with the other pyrexiæ—yet most truly acute rheumatism is not merely an articular inflammation. The severity and danger—at least the immediate dangers—arise, not from the local phenomena, but from the general febrile conditions.

Acute rheumatism is never truly apyretic. In mild cases, such as have been spoken of as subacute, the fever may be so slight in degree as to become quite an insignificant phenomenon of the disease, or in fact be undiscoverable, except by careful search and pretty continuous thermometric observations. Although thus in a very numerous class of cases the fever is so slight, and though, too, in another class of cases the suffering from the articular pain is so much greater than the discomfort produced by the fever alone, still we must regard the pyrexia as the leading symptom, and the one which deserves our highest consideration.

It is commonly said that the degree of fever-heat attained in rheumatism is proportionate to the number of joints subjected to inflammation,

yet this observation cannot be regarded as having any great value in either a diagnostic or a prognostic point of view; nor yet can we regard any given case as mild, even if few joints be affected, if the range of temperature is greatly extended.

The observation that the febrile movement runs no given course, and that at any period of the disease the patient is liable, with or without the involvement of new articulations, to sudden and dangerous exacerbations of body heat, is one of much greater value and significance and one that cannot be too constantly held in mind.

Chill.—A chill is a very common though by no means constant symptom at the beginning of an attack of acute rheumatism. My own observation is that the cases are very rare in which, as an initial phenomenon, a sense of coldness is entirely absent. In this matter we are wholly dependent on the account furnished by patients themselves; some would describe a sense of coldness as a chill; others, non-observant of themselves, forget their feelings; while others would not mention a chill as among the symptoms, unless they fairly shook and their teeth chattered. Unquestionably a chill of this latter character is a rare symptom in rheumatism, while I claim that a sense of coldness, whether due to "chill," proper or to a draught of cold air, is almost never absent. Perhaps the only cases in which severe chills occur are those in which the patient has been subjected to a thorough and prolonged wetting; these are the instances, however, which, owing to the precautions usually taken, are not followed by rheumatic attacks.

The chilly sensations are soon succeeded by a feeling of warmth, and fever develops by night or at latest on the following day. Usually it rapidly rises to a considerable height, often attaining the highest point exhibited during the attack. As measured by the thermometer the temperature will be found between 102° and 103° F. In very many cases, however, the thermometer on the second or third night may be found registering even a degree higher, without the general condition or appearance of the patient showing any more marked fever-symptoms than on the earlier day. The degree of fever is greatly modified by the care and surroundings of the patient. Probably nothing tends to keep the fever at a low grade more fully than complete rest in the initial stage of the disease. Every one familiar with hospital patients has noticed the high, often excessive temperature which they exhibit on admission to the ward. A thermometric observation taken of such cases within a short time

after coming to the hospital will show a higher range than at any subsequent period of the attack, although the disease is evidently in its commencement, and later numerous joints become affected. This excessive temperature is evidently due in these cases to the exertion and pain which the patients suffer in removal from their home. It is undoubtedly a misfortune attended with subsequent evil. The high degree of heat is accompanied with quickened respiration and a rapid pulse, and under these circumstances the removal of the patient may lay the foundation for a damaged heart-valve. This suggestion is, I think, in accordance with the fact that rheumatic valvular disease is more common among the lower classes—such as would be likely to seek hospital treatment—than among the richer class of patients. Of course this condition cannot be accounted for solely by this difference in circumstances.

Temperature Curve.—The temperature curve, although not exhibiting any regular evolutions or cycles in its range, may nevertheless be said to have a regular type, notwithstanding this regularity cannot be predicted in any case. It seldom happens that the actual beginning of the disease is under observation, but it may be fairly concluded, from the statements of patients, that the fever does not suddenly rise to an extreme height. This statement is, I think, to be modified, as already said, in those cases which do not receive proper care at the beginning. The height of the temperature is usually attained within the first four days; in many cases, perhaps the majority, a more or less steady decline is seen; in other cases, the most severe ones, the temperature, after attaining a high point, remains steady at or about this point during two, three, or four days before the gradual decline commences.

During the declining stage the temperature exhibits in nearly every case exacerbations of more or less marked character. These elevations are found not only in cases where complications occur—as well as in those cases, which will be spoken of later, of hyperpyrexia—but in regular attacks of acute rheumatism. These exacerbations usually announce the involvement of new joints or the renewal of inflammation in articulations previously affected; sometimes the temperature rises without our being able to point out the direct cause.

In some of these instances I have thought it was due to changes in the weather, and the rise of temperature was certainly simultaneous in a number of cases with the occurrence of a rapid alteration in the barometric pressure, usually its diminution as seen preceding a storm; in other

cases I have thought it due to attempts at exertion, which have caused renewal of pain and discomfort to the patient.

The number of days occupied by the declining temperature to reach a normal level varies very greatly, and the variations in the length of time cannot satisfactorily be accounted for by the apparent severity of the initial articular inflammation. In many cases, showing in the early stage an involvement of several even large joints, and in which the temperature within the first three days shows 104° F.—and this is especially true of hospital cases—the decline may be completed within a week or ten days, and be of steady and rapid character.

In other cases even when the early rise of temperature is less, although quite as many joints are involved, the normal temperature is not attained until after the end of the second or even of the third week. However, it is only in exceptional cases of simple acute rheumatism, without reference to their severity, in which the nursing is good and the surroundings favorable, that defervescence is not completed by the end of the third week. And if cases last longer than this, apart from complications, the protracted period is due rather to constitutional causes, or some pre-existing organic lesion, than to the rheumatism.

In endeavoring to arrive at a conclusion as to the duration of acute rheumatism, many facts must be borne in mind. In the first place, the differences between observations in private practice and hospital statistics must be considered. Again, authors take a different standard in determining the termination of the case; some speak of the termination of the fever, some of the subsidence of the articular pains, and others of the restoration to mobility with comfort of the painful joints. Hospital statistics probably tend at the present time to shorten the apparent duration of the attack; in the first place, by not counting all of the days of illness prior to admission, and secondly, the patients are often discharged before complete restoration. In private, especially among the lower classes, the surroundings and the nursing naturally tend to increase the fever and prolong its continuance. While a mean between these two classes would probably give a correct idea of the duration of the disease, the statistics of private work are so scanty and too often so defective that reliable conclusions are not obtainable.

Here, however, I am speaking of the decline and termination of the fever, and I think the statement is correct as above made, that in uncomplicated cases, no matter how great their severity, defervescence takes

place by the end of the third week. It is probable that this statement will have to be soon modified, even if the time has not already arrived, and the febrile duration of the disease under more recent methods of treatment will be considered as capable of very great abbreviation. But this question will be alluded to further in the chapters on the duration of the disease and also under treatment.

Wunderlich divides the range of temperature in acute rheumatism into three divisions. 1, the ascent or pyrogenetic stage; 2, the height of the fever, and 3, the period of descending temperature. Of the first stage he says that the rise is never sudden, as is common for example in the acute exanthemata, although exceptional cases do occur where 104° F., or even more, is reached as early as the second to the fourth day. Of the height of the fever, many cases, he says, present a solitary peak as a maximum, and this generally occurs between the fifth and ninth days of the disease, though it may occur even earlier, or at a later period in some cases. "Very commonly, however, the summit extends itself into an actual fastigium. But this, in the majority of cases, is very brief in comparison with the whole duration of the disease, and is still shorter when very high temperatures are reached, though the case in itself may not be very severe. . . . . It is only in very exceptional and in other respects severe cases that temperatures of 104° F. (40° C.) or more are reached or exceeded on three successive days."

"The *descending* period shows a varied type according to the form assumed by the decrease and the suddenness with which it occurs." In the favorable cases the descent is quick and generally zig-zag, and perhaps without any evening rise. More commonly the decrease of temperature is protracted with, for several days, a tolerably stationary daily average rise and fall. This may take from ten to twenty days to reach the normal level. Even after this point is reached, or even a subnormal level and convalescence has set in, fluctuations of temperature, usually of slight degree, are not infrequent. Wunderlich further says: "After all, the fever in these cases of acute rheumatism is only of moderate or at the most medium severity. Apart from its brief acme it remains at heights which only exceptionally exceed the bounds of moderate fever."

Modifications from Complications.—The visceral complications in many cases supervene without influencing the degree of bodily heat; when, however, a rise of temperature occurs with or precedes the commencement of a complication, the rise is usually not very great. Occasionally a well-

marked chill ushers in the beginning of a complication, and this comes without additional exposure. The exceptions to this rule are pneumonic and meningeal inflammation, and are pretty much in accordance with what would be expected from the nature of the complication. Meningitis and pneumonia occurring alone give rise to a high temperature, while endocarditis, pericarditis, and pleurisy may and do often occur latently or at least unattended with fever of marked character when these diseases are not complications of an acute rheumatic attack. The temperature subsequently to the occurrence of a complication is modified in accordance with the temperature rule which exists for the intercurrent disease, and in this manner the maintenance of a febrile condition may be seen some time after the polyarthritic inflammation has subsided.

CHAPTER VII.

CONSIDERATIONS OF THE INDIVIDUAL SYMPTOMS OF ACUTE RHEUMATISM AND THEIR PECULIARITIES.—*Continued.*

Of the Joints: Development of the Articular Phenomena; Reasons for the Involvement; Attitude and Position of the Patient and of the Joints; Cause, Seat, and Character of the Pain; Redness; Swelling; Subsidence of Inflammation; Temperature of the Joints; Comparison with Gout; Numerical Frequency in the Different Joints; Relation to Cardiac and other Complications; Duration; Metastasis.

Of the Joints.

After the pain commences in the joints, redness, swelling, and deformity are rarely absent in marked cases. In the very earliest stages we may find a patient complaining of severe pain without these appearances being present, but very shortly some one of them will develop.

Usually one joint is affected first, but this precedence is of brief duration, other joints being rapidly involved in succession. It is commonly said that the larger joints, and especially the knees, are the first to be attacked, but I think this statement requires modification.

Development of the Articular Phenomena.—While it is unquestionably true that in rheumatism, as compared with gout, the larger articulations are more apt to suffer or to be affected first than the smaller ones, I think that the more correct statement is that in rheumatism the joints most exposed, or perhaps most used, are the ones most likely to suffer. Thus in the carrier or postman the knee is more likely to be affected than the elbow; and in the housemaid or washerwoman the wrist, or perhaps even the finger-joints, suffer more than the ankle; or in the maid-of-all-work, who is frequently engaged in washing the front pavement or standing in a damp kitchen, the ankle and foot are most likely to inflame. Still the determination of the question which joints are the most used is very difficult, and the degree of use made will vary with the individual, although engaged in the same occupation.

Reasons for the Involvement.—The number of joints affected varies greatly in different cases and also with the severity and duration of the attack. In severe cases the number of joints involved is usually large, and so too if the febrile movement is of long duration. Although at first but one or two articulations are inflamed, other joints become affected during the prolonged course. However, a familiarity with a large number of cases will show every variety of character as to the number of joints originally inflamed, the number subsequently involved, and also the renewal of inflammation in joints previously affected.

Attitude and Position of the Patient and of the Joints.—The attitude assumed by rheumatic patients and the position in which they seek to place their limbs is a matter of some interest, often in a diagnostic point of view, and varies according to the generalization or localization of the affection, according to the stage of inflammation, and also to the presence or absence of effusions into the articulations. The position both of the patient and of the joints is modified greatly by the freedom or involvement of the muscular tissues.

It may be said as a general rule that in the early stage, as soon as the pain becomes severe, the patient's whole attention is given to maintaining the inflamed joint in a state of immobility, regardless of position, and this is especially true if the muscles or their aponeuroses are affected. In a later stage, after effusion into the synovial sac has taken place, nearly every joint has a rule of its own for attaining the most comfortable position when in this condition ; it is such as will give the greatest relaxation to the ligaments and tendons surrounding the articulation. Thus for the upper extremity pronation, semi-flexion, and the hand flat on the bed will be the most conducive to comfort, the fingers if involved also semi-flexed ; while for the lower extremities semi-flexion at the knee and slight abduction will be the position the patient will seek to assume ; if all these joints are affected and intensely inflamed, immobility is all that the patient will ask for, and nothing will induce him to move until some degree of relief is attained ; in cases where the vertebral joints are attacked the patients lie rigidly on the bed, like a soldier at "present arms."

Cause, Seat, and Character of the Pain.—The cause, the seat, and the character of the pain have always been matters of considerable discussion and about which opinions have differed very much. The older authors, who regarded the disease as a local matter and strictly inflammatory in its character, contented themselves by simply pointing to the inflammation.

In later times, as new opinions arose as to the cause and seat of the rheumatic disease, the question came up whether the pain was intra-articular, peri-articular, or due to changes involving the nerve-trunks and their distribution around the articulation. These questions are obviously very difficult of settlement and are of the nature to test our powers of observation to the utmost. Unquestionably the pain must be due to pressure made upon the nervous filaments, and the only point requiring discussion is the position of the filaments involved. Some would point solely to the nerve-filaments distributed to the synovial membrane, others to the peri-articular nerves, and others again to the nervous filaments distributed to the fibrous apparatus.

It would seem that at times all of these situations were productive of the pain in a manner, so to speak, quite isolated, as well as perhaps others not directly specified; for example, the bursæ and tendinous sheaths. Just as in some cases we find the morbid process localized or concentrated more particularly in one or the other of these parts, so in other cases they may all join when simultaneously affected in furnishing their quota of pain.

It would be quite unprofitable to discuss the character of the pain, as to its burning, tearing, rending, or shooting nature. It may be generally stated that perhaps in the majority of cases the pain commences at night, before the swelling and redness make their appearance. It is apt to cease or lessen with the occurrence of the effusion, and does not reappear after the effusion has reached its maximum and begins to lessen.

Redness.—The redness of the joint makes its appearance shortly after the advent of pain and usually before any marked degree of swelling, although occasionally the red color is not developed until a later period. The color varies considerably in its shade; it is usually pretty evenly spread on the surface affected, over and around the joint. Sometimes it presents a bright red color like an erythematous blush. In other cases it is of a dusky hue; and in still other cases it appears in narrow streaks or lines, often irregularly distributed, and under these circumstances the color is of a bright hue. In general it may be said the brighter the color the more superficial it is, while the dusky hues appear to be connected with changes in the deeper layers of the skin. It is said that the redness is less marked in rheumatism than in gout, although in some cases of rheumatism the color may be very pronounced; and the engorged veins leading from the affected articulation are much less conspicuous in the former than in the latter disease.

The dusky red color occurs especially in those cases where the inflammatory changes affect the peri-articular tissues, while the bright red color comes in connection with the large effusions within the capsular ligament; the streaks and lines of redness call to mind the appearance of a lymphangitis, and in some cases it is very probable that they are due to involvement of the lymph-vessels.

In all the forms of redness, and even when no very distinct coloration is visible, a white line or tâche can be produced by drawing the finger over the skin covering the articulation; it is, however, a torturing kind of experiment which serves no useful purpose.

The color endures in proportion to the intensity and duration of the joint affection. It is usually visible only for a few days and, except in cases where it has been very intense, almost ecchymotic in character. It is not seen to persist after death.

Swelling.—The swelling of the inflamed joints varies very greatly in amount, and unless influenced by pre-existing constitutional cachectic bodily conditions which tend to increase it and prolong its duration, it is more directly proportioned than either the pain or redness to the severity of the general symptoms. The seat of the swelling, as has been alluded to in discussing the cause and seat of the pain, may be due to effusion of serum taking place in either one of three positions or in all of them simultaneously. In the vast majority of cases, if the swelling is at all conspicuous, the cavity of the joint will be found to contain fluid, although sometimes very considerable enlargement may be present when but a small amount of fluid exists within the synovial cavity; in this latter case the peri-articular connective tissue contains the bulk of the distending fluid. In certain unusual cases the bursæ and the tendinous sheaths are found to present a striking amount of effusion, while but little forms in the joint cavity.

Subsidence of Inflammation.—One of the most striking features of a rheumatic attack is the rapidity with which the evidences of an inflammation disappear from within and around the joint. According to my own observation the development of the inflammation, attaining even a high degree, is more rapid than its complete subsidence and restoration to the normal. I think that the general impression conveyed by the statement of authors is the reverse of this; and perhaps the misleading statement and the misapprehension of the facts are due as much to accepting the statement of patients as to errors of observation. Subjectively the joint

is very bad from the moment the pain comes, and very well the moment it leaves, but objectively, the evidence of inflammation is pretty rapid in making its appearance originally or in renewing itself in a previously inflamed joint, but is slow in disappearing.

The sounds and the sensations, both of which may be conveyed to the observer or appreciated by the patient, occurring within the articulation are due to the alteration of its fluid contents. These phenomena have been compared to the sounds produced within the inflamed pericardium, but I think unjustly so, because, except in a few and quite exceptional cases, the products of inflammation within the joints are strictly fluid in character, while those in the pericardium which are productive of the friction sound are solid. The observations on the joint fremitus, as compared with the pericardial sounds, are of little value, are difficult to make, and simply result in torture to the patient.

Temperature of the Joints.—The temperature of the joints, if inflamed in any marked degree, and in fact almost independently of their appearance as to redness, usually indicates from one to two degrees higher than the adjacent portions of the limb, and I believe the difference is less marked in rheumatism than in gout. This is probably in accordance with the general features of the two diseases ; in gout, with perhaps only a single joint affected, the degree of bodily heat is less, and therefore the difference between the adjacent surface and the inflamed joint is much greater than in rheumatism, where the essential fever brings up the surface temperature nearer to the level of that of the affected articulation.

Comparison with Gout.—I know of no observations showing a comparative temperature of the joints in the two diseases, but I am inclined to think that in severely inflamed rheumatic joints the temperature exceeds that of the gouty joint, although perhaps the gouty toe, which is always very painful, exhibits uniformly a higher temperature than the average taken of all cases of rheumatism measured on the surface of the joints. The joints in the two diseases present very striking differences, both during the continuance of the attack and on its decline. The gouty joint is smooth and shining and the skin over it appears stretched and pits on pressure ; subsequently the cuticle desquamates. In rheumatism the swelling is never sufficient or of such a character as to render the skin tense ; pitting on pressure may, however, be observed, though not commonly, and local desquamation does not come after a rheumatic attack.

Numerical Frequency in the Different Joints.—As already stated, certain joints are more likely to be affected than others, and the true reason of this difference has also been given. It is of interest to state the numerical preponderance which one joint has over another, although in naming the order in which they are more liable to be attacked, it is incorrect to convey the notion of the regular evolution of the joint affection; it is simply a statistical statement. That given by Monneret, and quite generally quoted, is that the right side of the body has a preponderance, and of the joints, 1, the knee; 2, wrist; 3, ankle, and then the shoulder, elbow, and hip; the fingers more commonly than the toes and some fingers more frequently than others. It will be found that all joints are liable to involvement, not even the articulations of the ribs and the jaw escape, and that the synchondroses are sometimes inflamed.

The number of joints affected in any case may be few or many, and it is usual for a considerable number to be involved in all protracted attacks. In some cases the disease invades only the large joints, but in much rarer instances only the small joints, with perhaps one, or at most two of the large ones, usually of the upper extremity, suffer. There does not seem to be any rule of relationship between cases with few or with many joints affected and the involvement of the internal viscera in the rheumatic disease.

Relation to Cardiac and other Complications.—In some cases, even protracted ones, and with nearly all the joints suffering, the heart escapes, while in others, mild as to general symptoms, with only few inflamed joints and short in duration, the cardiac complication is serious and may become permanent. In cases of mild character like the latter, cerebral or other nervous complications or sudden hyperpyrexia may develop all out of proportion to the apparent severity of the rheumatic disease and even become fatal in their consequences. It has been thought that when only a few joints are affected the cases showed greater stubbornness, but this rule is certainly not of universal application.

Duration.—The duration of the acute period of the joint affection is not precise—it is difficult to arrive at a limitation of what is meant by acuteness, but in general it may be stated that the maximum is reached and passed for any given joint in from three to seven days, and that after this time subsidence of the phenomena takes place. But for a long time subsequently pain and swelling may remain, though the redness be gone, and stiffness frequently may last much longer.

Metastasis.—The question of metastasis is one which in this disease, as well as in various others, occupied a large space in the discussions of ancient medicine, and does still linger in our phraseology if not in our thought. The transference, or what is apparently such, of the inflammation from one joint to another is not truly a metastasis ; in fact, metastasis, except to a limited extent acting in the case of tumor-growths, is not believed to exist in reality. Theoretically no such thing can exist in such a disease as rheumatism, and clinically the facts are not forthcoming in support of it. The description of the sudden subsidence of the inflammation in an articulation and its immediate appearance at another point has been made very picturesque, but the reality is quite different from the picture. The inflammation does not by any means subside so quickly as described, and if the subsidence is at all rapid, in the majority of cases the patient is well and no more joints become affected. And when a number of articulations are involved in succession, the new inflammation commences before the old ones disappear.

The metastasis from the joints to the viscera, especially the heart, afforded the older authors more plausible grounds of belief in this theory than the external manifestations. The endocardial involvement, and not infrequently the pericardial also, as well as others, come quietly, unattended with symptoms until quite advanced in their morbid changes, so that the period of cardiac symptoms not infrequently becomes coincident with the subsidence of the articular disease. Hence, I say, there was more justification in the apparent fact of metastasis to the internal viscera than from joint to joint. The older physicians depended on the symptoms for diagnosis of cardiac disease, while we recognize its occurrence in advance of symptoms by its physical signs.

But neither in the one case nor the other is there ground for belief in any metastatic relationship between the manifestation of disease in one place and the other, neither internally nor externally. However much we may differ as to the essential cause of the rheumatic phenomena, all are agreed that the cause is a general one and that the change is a blood alteration ; that the manifestations seen first at one point and then at another are exacerbations in a disease which has a remittent cause, and not transference of a morbid material from one place to another.

CHAPTER VIII.

CONDITION OF THE SKIN AND ITS FUNCTIONS.

Excess of Function—Sweating and its Significance: Its Frequency; Its Characteristics; The Difference of the Sweats—The Eruptions: Sudamina; Urticaria; Medicinal Eruptions; Hemorrhagic Eruptions; Rheumatic Purpura; Hemophilia or Scurvy; Embolic Purpura.

The skin in the earliest stages of the disease is hot and dry, and while the fever is developing, presents no peculiarities other than those incident to the conditions found in any continued fever. Very soon after the full establishment of the attack and the development of the articular inflammation, the skin becomes moist, although it still conveys a sense of heat to the hand. But, owing to the presence of the perspiration, the skin never gives a feeling of pungent heat. In fact, the presence of the watery secretion and its evaporation from the exposed parts of the body often gives to these a sense of coldness while the concealed portions of the body, although covered with moisture, are felt to be distinctly hot. The heat, as felt on the general surface of the body, is, of course, of less degree than that of the reddened and tumefied joints.

Excess of Function.

Profuse perspiration is one of the most constant of the phenomena of acute rheumatism, and it would seem, as a general rule, that the more marked the fever and the more severe the case, the greater is the tendency to sweat. Very often the perspiration, at least in copious amounts, is only partial, and while the brow, the chest, and the shoulders are covered with beads of sweat, the lower portion of the trunk and the extremities are found dry, or at least only slightly moist. In other cases the portions of the body exhibiting profuse perspiration may be the reverse of the above, or the whole body may be equally bathed in sweat. It is rare, except toward the termination of the attack, to find all parts of the body dry.

Sweating and Its Significance.

The perspiration, a symptom almost as constant as the articular disease itself, has been regarded (and is yet to a certain extent) as a phenomenon in the disease of very varying significance. Formerly this symptom was looked upon not only as characteristic, but, says Aitken, in 1866, as "nature's cure for the disease. It may be 'wasting and enfeebling,' as excessive perspiration always is, but it is highly sanative; and if it does not occur, the pains are always more excessive and the constitutional symptoms become more severe if the perspiration should unexpectedly cease. The *materies morbi* is obviously removed by the sweating, and the natural cure of the disease is effected by these profuse sour-smelling perspirations. Those perspirations are only useless when they are not of this characteristic sour description. They are then emphatically 'useless, wasting, and enfeebling,' and ought to be arrested." All those who have held views similar to the above insist very strenuously on the markedly acid condition of the skin secretion, and it is probable, although observations are not at hand to prove the question conclusively, that while as a general rule the sweat in rheumatism is in many cases highly acid, yet it occurs not infrequently that the profuse secretion, while acid, is not more highly so than can be found in other morbid conditions and even in those of health. The striking peculiarity of the sweat is its peculiar, disagreeable odor, described as sour, and which may be said to be eminently characteristic of the disease.

Its Frequency.—Todd and Fuller, who held most strongly the acid theory of rheumatism, taught the efficacy of the secretion in removing the poison from the blood, and pointed out that the more regular the perspiration and the more abundant it was in quantity, the more rapid was the recovery. The later opinions held in regard to this symptom are that while the condition may be looked upon as in some respects an essential part of the disease, or at least in most cases, that its occurrence exercises neither a favorable nor an unfavorable influence. It is certain that the sweating of rheumatism cannot be regarded as a critical sweat, such as occurs in relapsing fever, pneumonia, and other acute febrile diseases. Neither is the acid sweating debilitating, as are the sweats of phthisis.

It is really a matter of great difficulty, in the face of such diametrically opposite views to determine which opinion is correct, and it is probable that the truth lies between the two. Perhaps it is true, as the older

authors held, that the excessive sweat-secretion is really sanative, but more than likely not for the reasons which they thought. The old belief that it is an elimination of the materies morbi seems to rest on very slender ground; no one seems to have been able to show conclusively that lactic acid exists in the skin secretions, or in fact in the blood. Any observations to this effect depend mostly on the opinions of men biassed by certain humoral theories of rheumatism.

We can readily believe, however, that although no poisonous material is gotten rid of by this means, nevertheless the sweating is distinctly beneficial. The cooling of the general surface which must be effected through this agency cannot fail to aid in averting one of the greatest dangers of the disease. We now depend, as one of our remedial agencies, on cold baths, packing, or sponging of the body to produce a like result. And this treatment becomes especially necessary in just those cases where the defective secretion of the skin is present, and which Fuller thought were unfavorably influenced by the absence of perspiration and the consequent retention of the morbid product.

The perspiration may be looked upon, in fact, if the expression may be allowed, as critical throughout the attack, for it is in those cases when the skin-secretion ceases, perhaps suddenly, which become characterized by the temperature rising to a dangerous level.

Its Characteristics.—Dr. Maclagan has recently suggested that, as the acid sweats of acute rheumatism are altogether peculiar and constitute one of the ordinary characteristics of the disease, they are one of the common results of the action of the morbid poison. He scarcely believes in the lactic acid theory of the disease, although holding that lactic acid is present as the result of tissue metamorphosis, and that these metamorphoses are brought about by the presence of a morbid poison, in its nature malarial, whatever that may be. This author accounts for the sweating as an effect not due directly to the action of the morbid poison, but indirectly through the presence of the lactic acid produced by it in stimulating the skin to increased action. "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 prejudicial nor beneficial; but as simply one of the necessary symptoms of the disease during whose course they occur."

The Difference of the Sweats.—It may be perhaps well to advert to two different kinds of, or perhaps conditions under which, perspiration takes place in rheumatism. What the older authors regarded as the beneficial excretion, and which is really characteristic of the disease, takes place in the earlier stages of the attack and is attended with heat of skin, a full and bounding pulse, and a loaded state of the urine, and the excretion has a peculiar odor. This is the characteristic, and, as we may say, useful sweat.

Another form of sweating is that which occurs sometimes late in a protracted case, sometimes as the result of injudicious medication or nursing, or in debilitated or cachectic states of the constitution. This form is unquestionably harmful and its effect is enfeebling. The secretion possesses little of the characteristic odor, the skin is sodden, and the pulse soft, weak, and irritable. The skin is apt to be cold and the bodily temperature, as indicated by the thermometer, is little if any above the normal. This condition of the skin may be very properly repressed or corrected by medicinal agents.

The Eruptions.

Sudamina.—The skin, during its excessive activity, is, not infrequently, covered with sudamina. The presence of these miliary vesicles is certainly neither more constant nor characteristic than their appearance in other continued fevers. It would seem as though some peculiar condition of the skin was, in addition to the occurrence of sweating, a necessary precedent of their development. Recently I saw a red-haired blonde woman covered from head to foot with thousands and thousands of sudamina, surrounded at their bases with a red area. The condition lasted four days and then faded away. It was not urticaria. Other skin eruptions are observed to occur at some period during the course of acute articular rheumatism.

Urticaria.—The most common form is urticaria. The duration of this eruption is quite transient and, so far as my own experience goes, the subjective sensations connected with it are much less annoying than in cases of ordinary urticaria with equally severe local lesions.

In one case, occurring in my ward at the Pennsylvania Hospital, I observed a skin lesion which at the first glance closely resembled urticaria, but differed from it in that within a few hours of its appearance a minute

vesicle developed in the centre of the reddened area or wheal ; this vesicle with equal rapidity became a pustule. The skin lesion, too, was attended with no itching and with only a very slight burning sensation. On the following day the color of the reddened area had almost completely faded, and its site was marked by the desiccating pustule, which in the course of a day or two longer disappeared. The patient suffered from a mild attack of acute rheumatism involving several joints ; perspiration was not a marked feature of the case ; the eruption appeared about the fourth day of the disease ; her skin was of a very delicate character.

Medicinal Eruptions.—In describing skin eruptions caused by or accompanying rheumatism, it should be borne in mind that several of the remedies in favorite use are capable of producing eruptions and that the medicinal diseases of the skin must be differentiated from those due to the rheumatic attack.

Hemorrhagic and other Eruptions.—In several older authors, and in two recent French theses, a hemorrhagic form of eruption has been pointed out. These have been variously spoken of as hemorrhagic rheumatism, rheumatic purpura, rheumatic hæmophilia, scorbutic rheumatism, etc. While undoubtedly many of these observations are due to errors in diagnosis, and while the vague pains and the oftentimes considerable febrile movement which may attend both scurvy and purpura as well as hæmophilia may lead to mistakes, I still think that in rare cases genuine acute rheumatism does exhibit patches on the skin closely resembling those of purpura, and which may be denominated very fairly hemorrhagic rheumatism, if any one wishes to use this name in describing an incident in the course of a general disease. It is perhaps as well used a term as epistaxic typhoid fever would be for a case of this disease attended with hemorrhage from the nose.

Embolic Purpura.—I saw this phenomenon of hemorrhagic spots well displayed in a single case. The patient, a middle-aged man, had been exposed during sleep to a strong draught of air from an open car-window for at least two hours. Very quickly he suffered from laryngitis, with vague pains in the extremities, and almost immediately after this a considerable murmur was heard over the apex of the heart, systolic in time. Very shortly, owing to the complaints of the patient, an examination led to the discovery of numerous minute bright-red spots on the inner side of the arms and forearms. These spots varied in size from a pin's head to areas as large as a quarter of an inch in diameter. Their color quickly

became darker, but their further evolution was cut short by the death of the patient within a day or two of the appearance of the lesion. The post-mortem examination showed a fringe of recent vegetation along the borders of the mitral valve. This latter lesion was regarded as explanatory of the appearances of the skin, although it must be looked upon as a very rare result of endocardial inflammation.

Such a lesion is doubtfully to be classed among the skin eruptions of acute rheumatism, while on the other hand it may help to prevent entire discredit being thrown upon this or other forms of a hemorrhagic appearance of the skin in this disease.

CHAPTER IX.

GENITO-URINARY APPARATUS.

Question of Rheumatic Localizations in the Kidneys—The Result of Contact with the Altered Urine—Comparison with Gout—Albumen, its Significance—Rayer's Rheumatic Nephritis—Intercurrence of Bright's Disease and Rheumatism—Lancereaux's Interstitial Nephritis—Embolism, or Secondary Albuminuria—The Bladder, Affection of its Muscular Tissue—Urethra, Ovaries, Uterus, etc.—Rheumatic Orchitis.

Question of Rheumatic Localization in the Kidneys.

It is difficult to arrive at an accurate knowledge of the state of the kidney in the course of an acute rheumatic attack, or the effects which the rheumatic process has on these organs directly. That they are not affected or do not become the seat of localizations of the rheumatic inflammation similar to the heart, pleura, and the lung substance, seems to be pretty generally agreed to, and it is not likely, *à priori*, from the nature and structure of the kidney, that a further knowledge of the post-mortem appearances will show us that changes of a similar character do take place with any greater frequency than is now believed to be the case.

The anatomical affiliations of the kidney are not with the fibrous tissues, which are generally the seat of the rheumatic process, but with the epithelial and mucous tissues. The large amount of fibrous or connective tissue in and around these glands is a congener of the mucous membrane and not of the general fibrous framework of the body; hence the morbid affiliations are with the former rather than with the latter.

On the other hand, however, if we are to judge of the condition of the integral parts of an organ by the state of its functional activity, or the changed character of its secretion, we can hardly escape the conclusion that the kidneys do suffer some alteration, as the result of the rheumatic process. This conclusion, however, does not necessarily carry with it the belief that a rheumatic localization takes place in the organ in the same sense as in the pericardium.

The Result of Contact with the Altered Urine.

Then, too, we doubtless have secondary effects of the rheumatic process exhibited in the kidney more strikingly, and perhaps more commonly than the primary ones. Besides, an alteration of the normal secretion does not necessarily, especially in such an organ as the kidney, imply anatomical lesions of its tissue. A person deprived of water does not perspire, or the sweat is at most of a concentrated character without any diseased condition of the cutaneous glands. So, too, of the kidneys, their secretion may be changed solely from disturbances of the secondary processes of assimilation. But we can scarcely believe that disturbances of assimilation can be long maintained without an effect being produced on the kidney-tissues, and how long this disturbance may be continued and the epoch at which the effect passes over into and establishes a distinctive morbid condition are the points to be considered.

Comparison with Gout.

It is very certain that we do not find the kidneys affected by the rheumatic process, as they are by gout, and this is true both as respects the frequency and the character of the renal changes. A few cases are on record which are quoted as instances of a rheumatic renal localization ; the metastasis of the inflammation to the kidney taking place on the subsidence of the articular symptoms. Formerly this phenomenon was believed to be common, much more so than at present, now that we distinguish the two classical arthrites with greater accuracy.

Albumen, its Significance.

Albumen undoubtedly does make its appearance in the urine in acute rheumatism, and this has been accepted by some writers as evidence of a rheumatic localization—as evidence that Bright's disease is a complication of the rheumatic process. The occurrence of albumen usually takes place, if at all, only at the height of the febrile movement, and lasts only for a day or two, or at most a few days. I do not conceive we are justified in regarding this occurrence as evidence of a Bright's disease nephritis. It is the same phenomenon and due to the same cause as albuminuria in many of the acute pyrexiæ, and it subsides as with them, leaving behind no

after-effects that are appreciable on minute examination. In rheumatic fever, as in other fevers, it comes with the intensity of the febrile movement, and is caused by the tissue-changes in the renal epithelium, suffering from the same sort of parenchymatous inflammation or cloudy swelling which we see in the liver-cells and muscular tissue.

Rayer's Rheumatic Nephritis.

The form of rheumatic nephritis described by Rayer cannot be considered as a localization of the rheumatic process, but is probably due to the embolic occlusion of the renal vessels. The other changes in the kidney productive of albuminuria belong to such changes as above described due to the febrile process, or to the effects of the treatment, especially the cantharidal blistering, which, in the irritated kidney, produce results not likely to occur in a healthy person.

Intercurrence of Bright's Disease and Rheumatism.

Some of the cases of albuminuria, in the course of acute rheumatism, are rather to be looked upon as instances of intercurrent attacks of nephritis, produced by the same accidental exposure that caused the rheumatism, and not as a localization of the rheumatic inflammation. Many more of the albuminuric cases are due to pre-existing Bright's disease, aggravated by the rheumatic fever. The intercurrence of rheumatism in the course of chronic albuminuria has not, however, been frequently met with.

Lancereaux's Interstitial Nephritis.

The form of Bright's disease to be looked for in rheumatism, or as a result of it, is the interstitial inflammation. This form is more closely allied by its genus with the rheumatic inflammation, but Lancereaux, who has studied this question closely, reports that its occurrence is very rare. This author has, however, found thickening of the walls of the renal vessels, especially the arteries, with narrowing of their calibre. This change of the vascular supply is connected so closely with disease of the cardiac valvular apparatus, and also with contracting kidney, that it is difficult to disconnect the change of the arteries, and hence of the kidney, from the results of the rheumatic process. It has been usual to think of the vascular

change as the result of the chronic congestion of the kidney, produced by the defective heart-valve, but the reports of Lancereaux would seem to show that the vascular alteration arrives too early to be accounted for in this manner, and if the reports are confirmed it is probably due to vascular changes, inflammatory in character, occurring in the vessels themselves at the same time as those we are familiar with in the heart-valves or aorta.

Embolism, or Secondary Albuminuria.

The other form of albuminuria is the result of embolism. The epoch of its occurrence is usually late, and it is to be looked upon rather as a secondary or accidental phenomenon of the rheumatic process. Embolism cannot, however, be excluded from the list of complications of acute rheumatism. Endocarditis may occur in any case, and the vegetations from the inflamed surface may find their way immediately into the renal vessels. The first attacks of endocarditis much less commonly furnish vegetations than do the valves already deformed and roughened by previous inflammatory changes, and the kidneys are therefore much less likely to suffer embolism in first attacks of rheumatism than in subsequent ones. Pre-existing or advanced valvular disease, therefore, may furnish the clue to the presence of albumen in the urine during the course of any acute rheumatic attack.

The condition of the urine in these cases is peculiar, and furnishes pretty distinctive evidences of the cause of the albumen. An embolus, occluding one of the larger branches of the renal vessels very soon attests its presence by hæmaturia, smoky urine, albuminous to chemical tests, and showing under the microscope large numbers of blood-corpuscles, not infrequently forming casts of the uriniferous tubules. The larger emboli are usually single, though more than one may successively present itself, and the amount of blood indicates proportionately the area of kidney involved. All the kidney-substance beyond the point of occlusion suffers from a blood-stasis, is congested, and the renal tubules of this area become filled with blood-corpuscles, which find their way into the urine. As soon as the congestion of the infarcted area passes away, that is, reaches the second stage of the progressive changes which tissues in this condition suffer, the blood ceases to appear in the urine. The albumen continues until a quiescent state of the involved kidney is reached.

Many cases doubtless occur, and that too in primary attacks of acute

rheumatism, where very minute emboli are present in the kidney without any evidence in the urine of this accident having occurred. The minute emboli are more likely to be found in cases of fresh endocarditis than large ones, and, if they become sufficiently numerous in the renal vessels, they may give rise to albumen in the urine without, however, the blood-corpuscles, as above described, becoming abundant enough to attract attention and lead us to suspect the cause of the abnormal condition of the fluid.

The Bladder, Affection of its Muscular Tissue.

Cystitis has been described by many authors as the result of acute rheumatism. By some it has been regarded as due to a metastasis or a true localization of the rheumatic process. By others it has been considered the result of irritation communicated by the concentrated urine, perhaps unduly retained in the bladder.

However unlikely we may regard the occurrence of a mucous membrane inflammation, it is certain that we see not very frequently a condition of the bladder in acute rheumatism which can hardly be looked upon as other than a rheumatic condition of the muscular or fibrous walls of the viscus. We always see the patient disinclined to be disturbed to urinate, but we also see in a few cases exquisite pain produced during the act of emptying the bladder, or even when a catheter is used to remove its contents. The pain is located over the vesical region and is not developed until its muscular walls are moving as they close on the decreasing amount of urine in its cavity ; the pain is not associated with rheumatism of the abdominal muscles.

Urethra, Ovaries, Uterus, etc.

Rheumatic localizations in other portions of the urinary and genital apparatus have a much less well-authenticated basis for belief. Various statements are made of the manifestations seen in the urethra, the ovaries, the uterus, and the testicles.

Rheumatic Orchitis.

Perhaps our knowledge of the condition of the testicle in acute rheumatism is more accurate than for the other organs, on account of the freer inspection to which this external organ is open. The testicle and

its appendages, or their enveloping membranes, suffer from inflammatory attacks as the result of rheumatism. The inflammation may precede the outbreak of the articular symptoms or may occur during their course or at their termination. The condition is rapidly developed and attains its height with great promptness, just as we see in the migration of the inflammation from one joint to another. The pain at the commencement is more intense than even in an orchitis, due to the usual pathological cause (though in other respects it resembles it), apparently because the effusion, coming more rapidly, puts the fibrous tunic more quickly on the stretch. The suffering usually is mitigated sooner than in the ordinary inflammation of this part, by the rheumatic process subsiding sooner, and relief comes often without the intervention of treatment. I have witnessed one such case in which the history was as above given, and it corresponds to records made by others. As this description is going to press I have heard a similar personal experience from a friend who has been a frequent sufferer from rheumatism.

CHAPTER X.

THE URINE.

Causes of its Alterations—Its Amount—Its Color—Its Reaction—Its Density—Urea—Uric Acid—Other Solid Ingredients—Albumen—Sugar—"Fibrinous Urine."

The urine in acute rheumatism offers peculiarities which, while not sufficiently pronounced to be diagnostic, differ strikingly from those of other febrile maladies of equal grade; the more severe the symptoms the more distinct are the peculiarities.

Causes of its Alterations.

The characteristic profuse sweating which attends the period of activity of the symptoms would lead us to anticipate changes in the urinary secretion, because, under all circumstances, we know that these two emunctories are complimentary, or supplementary, to each other. And we find, owing to an abundance of the skin secretion, a diminution in the amount of the daily excretion of urine. The change in quantity is apparently to a great degree proportionate to the amount of the sweating; that it is directly so is impossible almost to determine, from the difficulties attending even an approximation of sweat in a person suffering from a grave disease. I know of no observation at all accurate on this question, although attempts have been made in this direction, by weighing the moistened or saturated linen of the patients and comparing the result with the varying daily amount of urine voided.

Its Amount.

However, the fact is well established, as is familiar to every one accustomed to examining the urine in acute rheumatism, that the daily amount is much reduced, and that the reduction is apparently greatest in those patients who sweat the most profusely. It is also known that this

change in amount consists chiefly in a reduction of the amount of water, which, instead of passing by the kidneys, escapes by way of the sweat-glands.

More important questions for solution than the mere quantity of the urine and the deficiency of the watery element are whether the solid ingredients are altered, and whether this alteration is in any manner characteristic of the disease. In all febrile conditions certain changes, greater or less, are to be noted in the quality and character of the urinary salts, but in few if any of the continued fevers can the changes be spoken of as strictly characteristic, and certainly not diagnostic ones. While in many of the local diseases, not of a constitutional or a general sort, though attended with the general symptom of fever, many alterations of the urinary ingredients are well recognized and are of a diagnostic significance, these alterations may be found to consist of a reduction or absence, or of an increase of a normal urine salt, or, on the other hand, of the addition of a new ingredient to the urinary fluid.

In taking account of the changes in the character and amount of the urinary salts, attention must always be paid to the effects produced by drugs, which act either by increasing the amount of fluid, by changing the chemical combination of certain ingredients, by lessening the acidity or changing it to alkalinity, or finally by the addition of new chemical constituents or coloring matters. In either of these various ways the characters of the urine in acute rheumatism may be altered beyond recognition, and this effect has doubtless in many cases caused great variations of opinions in respect to the characters of the urine in this disease. Remembering these facts will help to reconcile the discordant observations which are on record.

The following statement, taken mostly from original observations, supplemented by the work of others, will, I think, give a correct account of the general appearances of the urine, its chemical composition, and some of the peculiarities which it presents, as well as some to which the fluid is subject, under various modifying circumstances.

The quantity of urine is usually very greatly diminished, but the amount of diminution changes from day to day, and is, as has been said, proportionated to the activity of the skin function. In judging of the daily quantity, as well as the diurnal variations, it is well to bear in mind the errors to which such observations are liable, owing to the painful condition of the joints and the disinclination of the patient to be disturbed to attend to this necessity of nature. Patients, during the great

reduction in the urinary secretion, often attempt to retain the urine for a great length of time rather than move; hence the secretion of to-day may not be voided until to-morrow, thus producing a large apparent diurnal variation of quantity.

The acid, and therefore irritating, quality of the urine puts a certain degree of check on this retention, and generally, unless the pain in moving is very extreme, the patient will choose the lesser of the two evils and void the urine to escape the vesical irritation.

Another source of error, I know, sometimes enters into the calculation of the quantity, and is directly due to the voluntary efforts of the patient in delaying to empty the bladder. Urine retained in the bladder unquestionably suffers a diminution in amount through the reabsorption of a part of its water, with the effect of producing a quantitative as well as a qualitative alteration in its composition.

In attempting accurately to ascertain the diurnal variations, strict attention must be given to the frequency and the hour of the day the urine is passed. In ascertaining the amount of reduction during a rheumatic attack, a system of averages, with due attention to the degree of the febrile symptoms, will, on the whole, probably furnish the most accurate notion of this change.

The record of measurement of the daily quantity may vary from nearly or quite normal to zero; the normal quantity of urine may be nearly attained in cases where sweating is but slight, and also where the imbibition of water or medicated diluent drinks has been very great; unless these two factors are conjoined the urinary secretion is much reduced; one of my urine records shows that none was passed during a single period of twenty four hours, due partly to diminished secretion and partly to the voluntary effort of the patient to avoid movement.

Manifestly the above observation is of no value in determining the quality of the *secretion* of urine in acute rheumatism; it merely records the amount passed. There are records published, presumably free from this error, which show that between three ounces and three ounces and a half only have been passed during twenty-four hours, and this condition is almost to be regarded as one of anuria. A reduction below one-fifth or even one-fourth the normal amount is rarely observed. It is probable that the average of a large number of cases will show the daily quantity to be about one-half the normal. This group will include both moderate and very severe cases.

Its Color.

The urine in the active stage of acute rheumatism is always high colored, and it is usually of a deeper tint than in any other general febrile malady. The color of the urine is due to the increase of one or the other or all of the various pigments which are found in this fluid, and therefore the deeper tint observable in rheumatic attacks is of itself indicative of a change of composition of the secretion directly connected with morbid process.

This augmentation of the coloring matter, always present in the disease, influences the character of the urine by increasing its density; for the coloring matter is one of the principal substances comprising the solid ingredients of the secretion during the duration of the attack.

The urine, freshly passed, is, though highly colored, still clear and transparent, but very rapidly on cooling becomes clouded by the precipitation of a sediment, which more or less rapidly sinks to the lower part of the vessel; sometimes, however, the urine, even freshly passed, is clouded. This is usually owing to its extreme concentration, rather than to the absolute increase of any one of its ingredients.

The high color usually presented is yellowish red or reddish yellow, or sometimes red or pinkish; this high color is lessened as soon as precipitation commences. With the cooling of the urine, or in cases in which the urine is turbid when passed, the high color is never so striking in appearance.

It will be found to happen very frequently that a urine, which when passed is high-colored, yellowish red or reddish yellow, and transparent, becomes soon turbid from precipitation, shows after the settling of the sediment a pale yellow or amber color, and is transparent; the sediment in such cases is of a bright red color.

In a few cases, a yellowish red urine after precipitation shows the supernatant clear, transparent fluid of a bright red color; the precipitate usually is of a yellowish red color.

These variations of color show a difference of behavior of the coloring matter in relation to the urinary salts; in one case the pigment adheres or combines with the uric acid or the urates and is carried to the bottom, while in the other instances the pigments show a greater degree of solubility. In some cases the variations must be accounted for by a difference in the amount of coloring matters present in the urine.

In the vast majority of the cases of acute rheumatism, the opacity and the precipitate produced in the urine is due to the presence of uric acid and urates. This acid and its salts are insoluble in cool urine, especially when abundant, and in some cases the concentration of the secretion alone is sufficient to cause turbidity and precipitation, independent of an absolute increase in the amount of these ingredients.

In a few unusual—they might almost be called abnormal—cases the urine is turbid or opaque when passed, but the color is not of a deep tint. It is whitish, and in a few instances I have seen it sparkle. Heat does not dissolve the precipitate, but an acid renders it clear. The urine in these cases is more abundant than is usual in the disease. The chemical and microscopic examination shows the presence of phosphates instead of uric acid and the urate, and that its reaction is neutral or alkaline.

I have not seen the patients who have passed such urine, but have had the samples sent to me for examination, and therefore cannot speak from personal knowledge of any peculiarity of symptoms, if such were exhibited, occurring in connection with this very unusual condition of the secretion. Dr. Fuller speaks of two cases in which the urine showed peculiarities similar to these I have alluded to. In two cases under his care the urine "was abundant, pale, alkaline, and turbid, containing crystals of the ammonio-magnesian phosphate. In both instances the patients were weakly, and their perspiration was of a peculiarly acid nature." He adds that something of the same sort has been remarked by others. It is probable the urine pigments are deficient in such cases.

The question of the nature and condition of the coloring matter is of nearly the same importance as its amount. The condition, or perhaps combination of the pigment in the urine of acute rheumatism, as we have already seen, varies considerably from circumstances with which we are not acquainted. In the majority of cases the coloring matter is precipitated, at least a considerable part of it, along with the sediment which we know to consist of uric acid or urates. In other cases, on the contrary, after the precipitation of these salts the supernatant fluid is of a brighter, usually reddish color, than before the precipitation occurred. Evidently, therefore, there are variations of the conditions under which the pigment exists in the urine. This variation may be dependent on two causes: 1, a variation of its relations to the uric acid salts, or 2, a variation in the nature of the coloring matter itself.

As a rule, we may say that the coloring matter attaches itself to the

uric acid crystals and somewhat also, though to a less degree, to the urates.

In respect to the nature of the coloring matter, it is unquestionably due to the presence of blood-pigments. The blood in rheumatism is, as we will point out later, subject to very rapid changes, especially a diminution of its red corpuscles, which are in part the carriers of the blood-pigment. Therefore, from the destruction of these corpuscles, the urine contains a larger amount of the coloring matter derived from this source. This alteration of the color of the urine may be spoken of as an increase of its normal color.

There is another pigment, which is derived from the changed function of the liver or the chylo-poetic viscera. This pigment also makes its appearance in the urine in other diseases than rheumatism, and also in health. It is therefore not an abnormal urine pigment although the result of abnormal causes, but is one of the special urinary pigments. It is called *urobilin*.

Other pigments have been described in the urine during the acute stage of rheumatism, but their composition and source are but little understood or entirely unknown.

Its Reaction.

The urine in acute rheumatism in the early stage and during the height of the paroxysm, and even later, has an acid reaction, unless affected by remedies, usually of an alkaline character, administered to combat the disease.

The acidity is a very striking feature of the rheumatic patient's urine. The other peculiarities of the urine in this disease partake of the nature common, to a greater or less extent, with those of some other continued fevers, and are therefore to be accounted for partly by the febrile process, apart from any peculiar pathological process due to the rheumatic affection. Quite otherwise is it with the acidity. The acidity of the rheumatic patient's urine is in excess of that found under circumstances of disease or health, beyond that which can be produced by the ingestion of foods or medicine and a condition hard to change or neutralize by the administration, except in large quantities, of drugs calculated to influence or produce an alkaline quality of this secretion.

The more concentrated the urine, the more acid it is usually found.

This rule is, however, subject to variation, as occasionally with a relatively free secretion from the kidney, or at least a larger measured quantity of urine passed, the fluid possesses a more strongly acid quality than when scanty. However, I do not think we are in possession of observations, made under proper conditions, to allow of our making a positive statement of this fact. So much of urine work is done in connection with medication, which necessarily alters the condition of the urine, and so few observations are published of the natural history of the disease unmodified by remedial agents; occasionally, too, under the effects of drugs, a sudden diminution of both the quantity and the acidity results, that we are hardly prepared to say how much the acidity is due to concentration of the urine and how much is directly the result of a febrile process in increasing the absolute amount of acid-reacting material.

Then, besides, our knowledge of which ingredient is the cause of the acidity is defective. Some hold that it is the normal urine products, present in increased amount as the result of febrile process, in which case the increased acidity must be, to a great degree, considered as but a part of the high concentration of the fluid; while others consider the presence of a special acid (lactic acid) as the specific acidifying principle, in which case the heightened reaction is due to rheumatic process as such. But this particular acid has not been identified in the urine, at least in greater amount, if even at all, beyond that found in health, and therefore cannot be charged with producing this condition, or others which have been attributed to it. Perhaps, however, the change which is found occasionally to occur in a urine highly acid when passed to a condition of alkalinity may be accounted for by the disappearance of an evanescent acid like lactic.

Its Density.

The specific gravity of the urine varies very greatly, being influenced by the severity of the attack and showing a different grade at different periods of the disease; it is likewise altered as the result of treatment. Of course, the degree of concentration of the fluid exercises the greatest influence, and in fact determines the specific density of it apart from the special influence of the rheumatic process. But it is nevertheless true that in acute rheumatism the specific gravity of the urine is increased. This is due, as will be shown later, to the increase of its solid contents and not merely the result of concentration. A very simple experiment

will prove this fact; if we collect the whole amount of urine passed during twenty-four hours and add to it sufficient water to raise the whole bulk to the average daily quantity in health, it will be found that the specific gravity exceeds the normal.

I am not in possession of the facts of a sufficient number of such experiments to say how great is the average increase of the specific gravity.

The specific gravity of the urine as passed shows variations within a very considerable range—a range considerably greater than that shown by urine in health and under circumstances in respect to the ingestion of food and drinks not likely to influence its specific density. The range in disease is influenced most markedly by the activity of the cutaneous secretion and to a less degree by other conditions.

Its specific gravity, in any well-marked case of rheumatism, is very rarely found to be less than 1,017. It may, however, attain to 1,036. An average of a considerable number of cases gives the figures 1,026. It is useful, therefore, to speak of the usual range of the specific gravity as being between 1,020 and 1,030.

The foregoing considerations of the urine of acute rheumatism show a considerable similarity, except in two points, to the conditions brought about by any febrile process; the acidity and the greater concentration alone can be looked upon as strictly peculiar to rheumatism.

It remains, therefore, to examine this secretion and to determine the amount of its several solid constituents to ascertain whether the rheumatic process influences their amount in a characteristic manner. As already mentioned, the solid ingredients are increased in amount. Which of them are affected, to what extent, and whether some of them or all, remains to be determined by quantitative analyses.

Urea.

In arriving at a determination of the amount of urea excreted daily in this disease, as compared with health, there are several very important circumstances to be borne in mind. In health its amount is influenced by sex, a considerable preponderance occurring in men, by the nature and quantity of the food, by the amount of physical exercise and by the weight of the patient. Therefore in rheumatism the comparison must be made with a healthy person at rest and taking nourishment of the same character as the sick diet, in order that the estimation may be just and of value.

During the acute stage the daily amount of urea excreted, after making due allowance for influencing circumstances, is increased.

According to various analyses the mean average of the daily excretion in health is from 20 grammes (308 grs.) to 22 grammes (340 grs.). The maximum being about 35 grammes (540 grs.) and the minimum 15 grammes (230 grs.).

During the acute stage of rheumatism, attended with rapid pulse, fever, and sweating, the average daily excretion of urea will be found to correspond very closely to 30 grammes, or about 460 grs., the maximum reaching 50 grammes, or about 790 grs., and the minimum falling to 18 grammes, or about 280 grs.

Dr. Stevenson, in the twelfth volume, third series, "Guy's Hospital Reports," gives figures which differ considerably from the above for the amount of urea, both in health and in disease. He states the maximum of health to be 479.95 grs. (31 grammes), the minimum 377.94 grs. (24 grammes), and the mean 422.85 grs. (27 grammes). Of the seven cases of rheumatism, the urine of which he had analyzed, the maximum is 695.95 grs. (45 grammes), the minimum 211.20 grs. (13½ grammes), the mean is 406.77 grs. (26 grammes). It would seem both from the clinical history and from the chemical analysis that the cases reported by Dr. Stevenson are hardly to be looked upon as fair examples of the condition of the urine in acute rheumatism. The minimum, as furnished by his figures, is lower in the diseased than the normal condition, and his figures are reduced by the occurrence of several cases which excreted less urea during the active stage of the disease than during convalescence. This is a relation which is the exact opposite of the conclusions which he draws in respect to these two stages of the disease from his own figures, and therefore I do not think his results can be accepted as typical of the excretion of urea during the acute stage of the disease, or at all events his figures are misleading in expressing the true proportions.

During convalescence, or at least after the subsidence of the fever, the daily excretion of urea is depressed below the normal. The degree of this depression is about equal to the degree of excess during the active stage. The average of a considerable number of cases taken from various sources shows a mean of 15.499 grammes (236.44 grs.). During this stage of the disease the urine becomes light-colored, increased in quantity above the normal, its specific gravity less than normal, and not infrequently its reaction is alkaline.

Here again Dr. Stevenson's figures, taken from the source above quoted, show a mean average amount of 638.12 grs. (24 grammes) which, while it is below the mean average for health as shown by his figures, is considerably above the mean average as obtained from other sources.

Uric Acid.

All observers agree in pointing out the marked increase in uric acid during the acute stage of rheumatism. In fact, by most it is considered the agent which gives the urine in this disease its strongly acid quality. By some it has been held that the increase of uric acid is in inverse proportion to the amount of urea present during the febrile stage. It is considered by these authors that the febrile process, by preventing the transformation of the urea, increases the amount of uric acid.

We shall find by our figures that this inverse ratio between the two is not shown, but that both of these urinary principles are augmented during the acute stage and both are likewise diminished and even depressed below the normal during convalescence.

In health the daily excretion of uric acid is influenced very greatly by the character of the food, and when this is exclusively or largely of animal nature the amount of uric acid is increased. It is necessary, therefore, in examining the urine of rheumatic patients, to bear in mind the character of the diet with which they are supplied.

Thus, in health, an exclusive diet of beef-tea will greatly augment both the urea and the uric acid excretion.

We may accept as a standard of comparison that the daily amount of uric acid is 0.654 gramme. An average of a considerable number of cases (nearly the same cases as those used in obtaining the average for urea) shows that during the acute stage uric acid is increased to 1.294 gramme.

During convalescence, the uric acid is diminished to 0.384 gramme, nearly one-half less than the average in health.

Other Solid Ingredients.

It would seem, from the consideration of the scanty analyses of the urine of rheumatic patients, that the other urinary constituents, especially the phosphates and the sulphates, show a slight increase in amount,

though to a much less degree than the urea and uric acid. This condition is in marked contrast with the result of analysis of the urine of gouty patients, where the uric acid, especially in the early stage or before the beginning of the gouty paroxysm, is diminished or absent, while the phosphates are markedly increased.

The conclusions which Dr. Stevenson has drawn from his examinations, in respect to the amount of the urinary salts, corresponds to the above statement. His attention, however, was principally devoted to an endeavor to formulate a rule of prognosis from the relative amount of the urinary salts found in the urine. On this point he concludes as follows: "1. In acute rheumatism, when the excretion of solid materials in the urine is large, the patient makes, other things being equal, a rapid recovery; on the other hand, in lingering cases the excretion of solids is usually small. 2. As in this disease the urine is invariably scanty in bulk, but (generally from this cause only) of high density, a useful guide to the progress of the case may probably be found by diluting the urine to the normal bulk, and then ascertaining its specific gravity. According as it is now of high or of low density will the progress of the disease probably be favorable or unfavorable."

The only ingredients of the urine which do not show an increase are the chlorides, which follow the rule in respect to them which obtains in all other diseases of an inflammatory character. Their amount is actually diminished, though to a less degree than in some other inflammatory processes, for example, in pneumonia. Their amount of diminution always seems proportional to the amount of the inflammatory material, especially of a solid character, which is thrown out, although the amount of the chlorides is influenced when the inflammatory material is entirely fluid. Perhaps the more strict statement of the relation between the chlorides in the urine and the character of the inflammatory material is not so much the difference between the solid and fluid character of the morbid material as between the absolute amount of the chlorides contained in it. Thus the croupous material filling the air-vesicles of the lung contains more of the chlorides than the fluid filling the pleural sac, therefore, in pneumonia the diminution of the chlorides in the urine is much greater than in pleurisy and very much greater than in rheumatism.

Albumen.

As a rule, albumen is a rare constituent of the urine in acute rheumatism or in any form of the disease. But nevertheless it does make its appearance occasionally. Its especial significance cannot be said to be very great and its detection does not give greater alarm than that furnished by the other more apparent symptoms of the disease present at the time when the albumen is detected in the urine. According to my own experience, albumen is much less commonly detected in the urine in acute rheumatism than in typhoid fever. The cause of its occurrence and its significance is about the same in the one disease as in the other. This is true at least so far as the anatomical changes in the kidney are concerned. In all febrile diseases we have changes taking place in the cells of the renal tubules—changes which may be briefly described as cloudy swelling—and which occasionally come to our observation during life by the appearance of albumen in the urine.

The amount of albumen is scarcely more than a trace, and its occurrence is of short duration, cotemporaneous with the height of the disease.

Albuminuria in acute rheumatism has been observed to result from what would be described, according to the older authors, as a metastasis of the rheumatic disease to the kidney. Of this we will speak further under the head of complications.

Sugar.

I know nothing of the occurrence of saccharine materials in the urine during the continuance or as a result of an attack of rheumatism; neither have I been able to find any records of such a condition.

Fibrinous Urine.

A rare condition of the urine has been described under this head, but which probably should be spoken of under the head of albuminuria rather than as a separate condition. I have been able to find very few cases of it on record, and have seen but one myself. The urine in this case presented no special differences from those usually described in the acute stage of rheumatism. It contained no albumen, or at least not that kind of albumen which responds to the test of heat and nitric acid. The

whole bulk of the urine at the time of my examination of it presented a feebly clotted or jelly-like consistence.

This condition seems to be similar to that spoken of by Senator and others. My examination of the specimen was but very imperfectly carried out, and I am able to give no further details in regard to it. I know of no better description than to speak of it as clotted urine, and that it reminded me of the white of egg mingled with water.

CHAPTER XI.

COMPLICATIONS, OR EXTRA-ARTICULAR LOCALIZATIONS.

Their Importance in Rheumatism—The Point of View from which they are to be Regarded—The Order and Frequency of their Occurrence: Muscular; Circulatory; Respiratory, Cerebro-Spinal, Digestive, and Urino-genital—Cardiac Localizations: Our Knowledge of them; Their Frequency; Various Forms of Cardiac Lesions Collectively; Their Relative Frequency and Combination. Time of their Occurrence and Concurrence; Symptoms of Endocarditis; Symptoms of Pericarditis—Rheumatic Localizations in the Vascular System.

Their Importance in Rheumatism.

Rheumatism is peculiarly liable to be attended with complications, probably to a greater degree than any other disease, and these complications occupy a very considerable part of our attention, not alone from their frequency, but also from their severity, both immediately in the course of the attack and from the subsequent grave effects which they entail on the organism.

It is a matter of some doubt in what manner these effects should be spoken of. Formerly they were considered as metastases of the rheumatic inflammation from the articulations to the internal viscera, but latterly it has been customary to describe them as complications. It is probable, however, that to look upon them as multiple localization of the same condition will afford us a more philosophical point of view of their nature and true relationships. In other words, the visceral, or extra- or ab-articular localizations are really integral parts of the disease, quite as much as the joint affection, and thus the rheumatic heart holds a different relation to this disease from what perforation of the intestine does to typhoid fever or pleurisy to relapsing fever; the latter are truly complications of those diseases.

The Point of View from which they are to be Regarded.

This view receives confirmation from the fact that extra-articular localizations may occur prior to the development of the joint inflammation, and therefore it would be equally just in such cases to describe the articular disease as a complication of rheumatic inflammation of the heart—manifestly a wrong judgment to make of the question.

The Order and Frequency of Their Occurrence.

Muscular.—The most frequent of the localizations of the rheumatic disease, and standing next in rank to the articular inflammation, are the phenomena developed in the muscular apparatus. So constant is the involvement of the muscles and their connections or appendages in the rheumatic process, during acute attacks, that I am inclined to place these organs in the same rank with the joints. The cases of acute rheumatism are indeed very rare in which the muscular tissues do not exhibit some one or other of the symptoms of the malady to a very considerable degree. The articular inflammation is, however, of so much more severe grade, and, from the nature of the parts involved, so much more painful, that the muscular disease falls into the background in our consideration of the rheumatic symptoms.

The particular manifestations of muscular rheumatism, especially as seen when occurring apart from the articular disease, are very important as well as interesting, and later on special consideration will be devoted to them.

Occurring in the course of rheumatic fever, when they are thus seen, they form so much a part of the general articular disease that it does not seem best to look upon them as a special complication or localization, as we are wont to do in respect to the visceral rheumatic manifestations. And then, too, their treatment as they occur in the course of acute rheumatism, especially in the height of the attack, forms but a part of general treatment, quite unlike the indications when seen in subacute cases or when occurring alone.

Circulatory.—Next in frequency, and of vastly greater significance and importance are the lesions of the circulatory apparatus. And here we enter on grounds which require a high order of skill on the part of the physician, not only to meet the requirements for their immediate recogni-

tion and treatment during the course of the acute attacks, but also during the remainder of the patient's life to regulate the circulatory functions, which have become impaired through the effects of the rheumatic process.

The parts to be considered under this head are three: First, the heart; second, the vessels; and third, the capillary network. The lesions and symptoms of this latter division appertain more properly to the tissues or organs in which the minute vessels find their distribution.

Respiratory, Cerebro-spinal, Digestive, and Urino-genital.—The next in order to the cardiac lesions, and occupying a successively inferior importance, come the localizations in the respiratory apparatus, the cerebro-spinal, the digestive, the urinary, and finally the genital organs.

Cardiac Localizations.

Our Knowledge of them.—No more important question presents itself in medicine than the relationships of rheumatism to heart disease, and yet we find that this knowledge has been in possession but a comparatively short time. To be sure, many early, even very early, authors refer to "rheumatism of the heart," but their references to the condition are so vague and uncertain, that any one may satisfy himself that their knowledge is unimportant to us and was quite useless to them.

It will be found that Bouillaud, by publications between the years 1830 and 1840, was the first one to place the relations of articular rheumatism and cardiac diseases on a firm and useful basis. Since his time great additions have been made to our acquaintance with the conditions and changes in the heart, though but little advance has been effected, except in the manner of viewing the subject, in the essential nature of this rheumatic process.

Since his time the recognition of the coincidence of heart disease with acute rheumatism has been firmly established—nay, it has created an entirely new item in the science of medicine. Lesions of the heart, until then in great degree uncertain, were regarded as accidental effects of quite unknown origin. His researches, aided by the previously developed arts of auscultation and percussion, turned a flood of light on a new and wholly unknown field of medical work, which since then has become one of the most important in medical observation.

Their Frequency.—The frequency of the occurrence of cardiac disease, caused by rheumatism, becomes a most important question for determi-

nation. It was stated by Bouillaud that the cardiac complications are the rule in severe acute rheumatism and their non-occurrence the exception, while in mild cases their occurrence is the exception.

This rule, as absolutely and simply stated by Bouillaud, is entirely misleading, although, like so many generalizations, it is possible to defend it. But as our knowledge of, and at the same time, our greater capacity for the recognition of the various forms of cardiac disease have advanced so have the exceptions to Bouillaud's law increased, until they themselves have almost become the law.

The objection to Bouillaud's rule is that a belief in its entire truth tends to throw an inexperienced observer off guard. Unquestionably the risk that a patient, subjected to a severe and prolonged attack of rheumatism, runs of more or less grave cardiac mischief is exceedingly great, nay, almost amounts to a certainty, and the exceptions to the rule are few, and with our advancing ability of their recognition have become fewer. The more accurately the subsequent medical history of such cases is traced the number of them will be found to diminish still further.

Cases of acute rheumatism, with only a single joint involved, and those attended with very mild general symptoms show so great a liability to cardiac inflammation that it is much safer to have no rule than one which is so faulty, and one which leads to errors so grievous in their consequences.

The numerical comparison has almost always been made in respect to the number of cases of acute rheumatism in which cardiac complications occur, and these statements, furnished by various authors, collated from all grades of cases, show a very great proportion in which a heart affection makes itself known. If, as at present, we are to look upon the visceral involvements as one of the localizations of the disease other than a complication, strictly speaking, the comparison should be made to ascertain the relative frequency of heart localization to the localizations in each of the joints.

Statistics of this sort may have some general interest, but practically they are of almost no value. Face to face with an individual case of rheumatism they may be even misleading and take away attention from a pressing danger. Should the statistical table show that in only seventy-five per cent. of cases the heart became affected, the tendency would be in just that proportion to overlook the information to be derived from physical examination of the organ. Numbers will not help us either in the diagnosis or treatment.

While it is true, as statistics tell us, that cardiac involvements are more

frequent in the severer cases, still the records show that, even in the mildest form of acute rheumatism, valvular lesions occur, and these may precede the articular inflammation, or even be the sole localization of the rheumatic process. The likelihood of cardiac inflammation is vastly increased during the early years of life, and this risk diminishes, but does not disappear, with advancing years; up to the age of twenty-five it remains very great.

Numerical accuracy in determining the number of instances of cardiac complication in acute rheumatism is difficult of attainment. In the first place, nearly all cases in which more than one acute attack occurs must be excluded from the computation—and these form no inconsiderable part of the whole number. They are excluded because when a distinct cardiac lesion has occurred, it becomes a difficult or impossible matter to determine the reappearance of acute inflammation in subsequent attacks. In the second place, the determination of the occurrence of the heart mischief, in its various forms, is not an easy matter, especially in the slighter forms. Observers are not agreed in the matter, and what by one would be regarded as an indubitable sign would be doubted by another. Then, again, during one year the heart-lesions come in nearly every case, while in other years cases of equal severity show little tendency to cardiac complications. The observations must therefore be extended over long periods of time, and allowances must be made for these possible sources of error, before a true conclusion can be arrived at.

Various Forms of Cardiac Lesions Collectively.—The consideration of the cardiac complications of rheumatism present themselves for study naturally under three heads: 1, Endocarditis; 2, Pericarditis; 3, Myocarditis. These three forms frequently occur in combination or conjunctions. Thus, the first two may, and are not infrequently found in conjunction with each other, while a combination of the third with either of the other two, especially pericarditis, is almost a necessity in the more severe cases, and probably in no case is wholly absent.

Although the relative frequency of the cardiac complications, taken collectively, to the whole number of cases of rheumatism is difficult or impossible to compute, and not of very great practical importance, the relative frequency of the individual heart-lesions is pretty well determined and is of great practical importance. Nearly all writers make the statement that endocardial inflammation occurs with very considerably greater frequency than pericarditis, and this is undoubtedly true as shown by records. Some

authors are inclined to doubt it, or at least to think that pericarditis is nearly as frequent.

Their Relative Frequency and Combinations.—It is natural, perhaps, that endocardial inflammation should be diagnosed more frequently than pericarditis, for two reasons—and this refers especially to the slighter cases of the two diseases. First, because valvular murmurs are more easy of recognition, since they are alterations of definite normal sounds, and also that, once developed, they remain more or less constant; once produced, the roughness remains and maintains a murmur-producing surface in contact with the blood-current. And then, too, it must be taken into consideration that the diagnosis of endocarditis may be an error; for inorganic or functional murmurs are not infrequent in acute rheumatism. This mistake would increase in proportion with the frequency with which the diagnosis is made. Whereas, in pericarditis the friction-sound may never be heard, or having been heard, may disappear. The roughened serous membrane may be there, but its two surfaces be separated by a thin layer of serum, not sufficient in amount to increase the area of dulness, but enough to prevent the rubbing of the heart on the sac. This, and other considerations, familiar to all acquainted with post-mortem appearances, indicate how it is possible for endocarditis to show a preponderance in diagnosis, while pericarditis in reality may more nearly approach it in actual frequency. However, conversely to this view, the pericardiac sounds, if loud and extensive, may conceal the lesser blowing murmurs of the valves; doubtless this not infrequently occurs, since the two lesions are frequently coexistent.

Individual Lesions and their Frequency.—So far let these remarks apply to cases of cardiac disease, for the recognition of which we depend on the physical signs rather than the symptoms, and therefore necessarily to the milder type of rheumatic cardiac inflammation. Of the graver cases of heart complication in rheumatism—cases which, by their severity, in the first place give rise to easily perceived or marked symptoms, and secondly alter the course of the rheumatic attack, prolong it, and require active treatment to subdue or even to conduct the patient to immediate recovery—the statement stands quite differently. Then pericarditis comes to the front, with its greater suffering and more immediate danger to life, and requiring oftentimes all the skill at our disposal for successful treatment.

Endocarditis may, on the other hand, when existing alone, cause comparatively little discomfort. The patient may in severe endocardiac inflam-

mation—severe as evidenced by the quantity of the valvular murmur—be comparatively comfortable and the pain due to the heart-lesion be infinitely less than the articular suffering. The patient may not know of the new malady, although the physician recognizes, from the physical signs, the grave peril threatening the valvular structures.

This relation of affairs to the condition of the patient is explainable on an anatomical basis. The space over which the lesion extends in endocarditis is excessively limited, while in pericarditis it is usually coextensive with the large area of the pericardiac sac. It is natural for the more widely extended inflammation to be attended with effects more perceptible to the patient. It is not infrequent to discover an endocarditis unexpectedly, by its physical signs. A pericarditis makes itself known by its symptoms.

In respect to inflammation of the muscular substance of the heart, in connection with acute rheumatism, our information is quite scanty and wholly incomplete for the purpose of its clinical recognition.

In a severe form, or occurring independently of the other two lesions, it is certainly infinitely more rare than either of them. As already said, its appearance coexistently with these lesions, to a greater or less degree, nearly always takes place. Post-mortem evidence shows that the inflammation of the pericardium extends to and affects the muscular substance of the heart; in endocarditis it does likewise, though much less frequently. The cases are very rare—in fact, I have only met with one or two undoubted instances—where the inflammation could be looked upon as a primary disease of the muscular tissue.

The myocarditis, coexistent with the serous inflammations, must unquestionably intensify the symptoms of those lesions, but to precisely what degree is uncertain, since the symptoms of the muscular inflammation are themselves unknown.

Time of their Occurrence and Concurrence.—A question of very great importance is the *time* which these lesions manifest themselves in the course of acute rheumatism. It is now a general belief that the cardiac localization does take place, in certain cases of rheumatism, prior to the advent of the articular inflammation. In fact, some writers hint at cardiac disease of a rheumatic character, unattended throughout with joint manifestations, and even without any general symptoms. If this were true it might serve to explain the origin of many cases of valvular heart disease, or of pericardial alterations from which can be elicited no history of acute cardiac symptoms or of other acute disease.

Symptoms of Endocarditis.—Of the individual lesions, *endocarditis,* in the majority of cases, makes its appearance early in the rheumatic attack. It is not infrequent to find it coming on within a few days, or even a shorter period, after the commencement of the fever. In hospital cases, entering the wards during the height of the illness, the endocardial signs are found already present and often advanced. The patient, once safely landed in the hospital ward, is thought to have avoided one great source or cause of cardiac mischief, viz., the excitement and pain resulting from transportation, which influence unfavorably the heart's action.

But no period of an attack of acute rheumatism, no matter what the severity, is free from the danger of endocarditis, and of course it occurs independently of the patient being moved or not, whether he is in the hospital or is moved to its wards, whether he is surrounded from the beginning of the attack with every necessity and comfort, or is placed in want and misery.

The poorer classes, however, seem to suffer more frequently than the richer. Such is my own experience. It is rare for endocardial inflammation to commence after the general symptoms have subsided, but I have known the murmurs to start at this time and be followed by subsequent chronic valvular insufficiencies. In such cases, it must be supposed that acute changes were present during the height of the disease.

Symptoms of Pericarditis.—*Pericarditis* usually is developed much later than endocarditis. In the majority of cases the second week of attack sees its manifestation if it occurs ; and often, unlike endocarditis, its advent is postponed to the termination of the disease, whenever this may come. The pericarditis thus developed prolongs very greatly, oftentimes, the convalescence of the patient. In the severe cases of rheumatism where there seems to exist a predisposition, as it were, to cardiac complications, the endo- and peri-cardial inflammation may both be developed early and simultaneously, or one of them may precede the other by only a very short interval. In fact, no rule can be laid down in these cases for the order of their occurrence, and it should be remembered that just as a subsequently developed pericarditis may mask the previously existing endocardial signs, so a subsequently developed endocardial inflammation may be concealed under a prior pericarditis.

It must be remembered that rheumatic cardiac diseases may in their turn give rise to complications directly connected with themselves and not strictly dependent on the rheumatic process. We allude to such as em-

bolism of other viscera, connected with endocarditis on the one hand, and to the extension of the inflammation from the pericardium to the pleura and lungs. But of these notice will be taken later.

Briefly the phenomena which notify us of the existence of endocarditis are as follows: The symptoms perceived by the patient are often slight or entirely absent, and their grade varies with the severity and extent of the inflammation. At most, there is felt an uneasiness or pain at the præcordia, some difficulty in respiration is experienced, the heart may beat with greater force or its action may be irregular, and the pulse may not be found to correspond in time or force to the heart's action. The patient is frequently seen with an anxious aspect; with the concurrence of all these conditions the face may be turgid also, and there may be headache, with possibly slight wandering of the mind. The fever is usually augmented, but this increase is not marked and may be overlooked or masked by the simultaneous involvement of a new joint, which would influence the temperature-curve to a greater degree than the endocarditis. Remember that the area of an inflamed valve is much less than even a metacarpal joint.

The general symptoms, both subjective and objective, are so slight, or so lacking in uniformity, that it is from the physical signs the diagnosis of endocardial inflammation is arrived at. A constant blowing murmur, usually soft, heard at the aortic or mitral valves, coming on during the course of the rheumatism, is the only condition which positively allows of this conclusion. If we cannot observe the development of the murmur, a doubt exists of prior origin of the valvular disease; if it is not constant, its cause may be functional and not organic. The soft character of the blowing sound is an essential feature of a simple endocarditis with vegetations, since the character of the lesions in the acute stage cannot produce a harsh sound. The *auditus eruditus* is more serviceable in determining the nature and character of the sound than can be conveyed by description. A very great familiarity, aided by the symptoms alone, will, if at all, lead us to the correct determination of the presence of an acute inflammation in an already spoiled heart-valve—a condition very apt to recur in second attacks of acute rheumatism.

Acute rheumatic endocarditis may occur without the blowing murmur being present. The occurrence of the murmur shows merely that the surface of the valves over which the blood flows is roughened by swelling, the products of inflammation, or the collection of fibrinous vegetations from the blood. The inflammation of the lining membrane of

the ventricular cavity, almost solely the left one, may occur, as well as of the tissue composing the openings and their valves, though its occurrence in this situation is infinitely more rare in the former than the latter. Unless the inflammatory roughening is near the valvular opening, the blood-current does not produce any sound which can be detected. The symptoms which result from the ventricular lesions come much later in the course of the disease usually, and manifest themselves, if at all, more strikingly in other parts or organs than the heart itself, until the fatal issue arrives. The condition of ventricular endocarditis with ulceration (ulcerative endocarditis) is so constantly attended with the formation of fibrinous clots that embolism is more frequent from it than from valvular roughening in its earlier stages. The disease is very rare, as compared with valvular lesion in rheumatism, and is probably of more common occurrence in other diseases than as the result of the rheumatic process. Ulcerative endocarditis is usually fatal at an early stage, and evidences itself by more striking phenomena, both of the heart and also in general, than either inflammation of the lining or the covering of the organ. It is frequently unattended with physical signs which can be detected by the ear.

In pericarditis we have the usual symptoms of an inflammation of a serous membrane, modified by the anatomical peculiarities of this sac and the vital organ which it contains. The general condition does not differ in any marked respect from that of endocarditis. The pain is apt to be greater, and there may be more dyspnœa, especially from the pressure of the distended sac. But, as in endocardial inflammation, the disease may be recognized first through its physical signs. The single or double friction-sound may be found during a casual examination of the chest, and then may disappear. Later, the area of præcordial dulness may be found increased, together with other signs of the presence of fluid, and then the valvular sound may become distant or muffled, though, when heard, remains sharp if concurrent endocardial inflammation does not exist.

The termination of the two forms of inflammation, and the influence which they exert on the course and severity of the rheumatic attack, as well as on the future well-being of the patient, stand out in most striking contrast to each other. Endocarditis, simple and uncomplicated with its own accidents, does very little to prolong the rheumatism or increase its severity, and it tends to terminate simultaneously with the amelioration of the articular inflammation. But its after-effects are most unfortunate and generally result in permanent and ever-increasing disablement of the val-

vular apparatus. Of course there are exceptions to this rule, and cases of well-marked constant murmur become perfectly well and entail no subsequent changes in valves. We may say of it—often mild during the course of the rheumatic attack, permanently dangerous to life ever after.

Of pericarditis, on the other hand, it is often quite the reverse. If severe, it tends suddenly to make the symptoms grave ; coming on later in the illness than the inflammation of the lining membrane, it tends to prolong the attack and to postpone, often very greatly, convalescence. Its termination resulting in complete repair, or by more or less complete adhesion of the two surfaces, it produces a condition which is difficult of recognition and without well-known or only inconstant subsequent effects or symptoms. Recovery from it may be perfect, and if the adhesions are complete there is no possibility of its recurrence in subsequent attacks of rheumatism, whereas in endocarditis renewal of inflammation or further changes in the valves are the rule. The simultaneous or successive occurrence of the inflammation in both of these parts increases in an undue or disproportionate manner the influence which either alone has on the course of the rheumatic disease, but even then the pericardial lesion seems to exercise a preponderating effect and may lead, as it sometimes does singly, to a fatal termination by compressing the heart and interfering with the circulation. And when the two inflammations concur, it seems that the muscular structure of the heart tends to be more deeply affected ; and this occurrence increases, so far as we can judge, the symptoms and exercises a specially untoward effect on the course and termination of the attack.

Rheumatic Localizations in the Vascular System.—The complications of rheumatism on the side of the circulatory apparatus, outside of its central organ, have received very little attention, and but little is known of their existence. In the first place, the study of such condition is attended with difficulty and the symptoms which disease of the vascular system, likely to result from acute rheumatism, would produce in the vessels, or the physical signs which such changes would be productive of, are most difficult to ascertain during life. Let us think for a moment of the difficulty of recognizing a thoracic aneurism, either by the symptoms or the physical signs, or how completely every highly marked atheromatous disease of all arteries, except the superficial ones, almost wholly eludes discovery, and we can at once conceive of the almost impossibility of discovering slight acute alterations of the vascular system in the course of an acute polyarthritis. In the second place, it is probable, nay certain, that rheu-

matic vascular changes, if such exist, are, like endocarditis, at first only slight in degree, and like that disease are productive of changes or leave behind them conditions which, slowly increasing in their effects, are difficult to trace to their source.

There is nothing improbable in supposing that an arteritis—a peri- or end-arteritis—should occur as the result of acute rheumatism, or from the chronic form of the disease. The tissues involved and the conditions to which they are submitted are very much the same. It is certain, also, that we see frequently enough alterations of the coats of the arteries which could readily be accounted for by an acute inflammation similar to that of a rheumatic origin. The symptoms of a local, or even general disease we can readily believe could escape detection, and the presence of the diseased condition may help to explain cases of embolism which sometimes seem to occur without the detection of valvular endocarditis with vegetations.

With respect to the venous trunks and their ramifications our knowledge is equally imperfect. However, on the one hand, while our knowledge of the venous alterations seen post-mortem is less extensive, on the other hand we are more acquainted with symptoms ante-mortem, which come in the course of acute rheumatism and which could be the results of rheumatic localizations in these vessels, than with symptoms connected with the arterial trunks and their branches.

In a very mild form, in truth, it is no unusual thing to see appearances, in the course of acute rheumatism, which can only be accounted for on the ground of a localized phlebitis. This is generally visible around an inflamed articulation, and the condition may extend to the larger venous trunks of the whole of one or more of the extremities. In fact, as I write I have under observation a patient who exhibits on the dorsal surface of the hand changes in the veins, which commenced during an attack of acute rheumatism. The result is that one, or perhaps two, of the larger superficial vessels on the ulnar side have been closed, or at least thickened, by inflammatory alterations, and at one point is felt a hard mass, like a phlebolite. Alterations of the veins are by far more common in gout than in rheumatism. Allusion may also be made in this connection to the changes seen in the smaller vessels of the brain, and even in its venous sinuses, in fatal cases of cerebral rheumatism.

CHAPTER XII.

COMPLICATIONS, OR EXTRA-ARTICULAR LOCALIZATIONS.—*Continued.*

Respiratory Apparatus: The Parts Affected and the Frequency of their Involvement; Pleurisy, its Character and Duration; Pneumonia, its Relation to the Rheumatic Process; its Type and Grade; Mucous Membrane Inflammations, their Relationship to the Tissues Ordinarily Affected in Rheumatism; Rheumatic Angina; Bronchitis and Broncho-Pneumonia—Digestive Apparatus.

Respiratory Apparatus.

The Parts Affected and the Frequency of their Involvement.—Pleurisy and pneumonia, as well as other morbid conditions of other portions of the respiratory apparatus, occur as complications of acute rheumatism. Some of these, especially the first, while not absolutely rare, are very much less frequently seen than cardiac disease. Pleurisy is by far the most common of them, and herein it exhibits, with similar inflammations coming in the adjoining serous sac of the pericardium, the general tendency of the disease to involve the fibrous tissues. These two serous membranes are not infrequently found communicating the inflammatory condition the one to the other. Almost every portion, however, of the respiratory apparatus is liable to be involved.

To determine the frequency of respiratory complications is even much more difficult than it is to answer this question concerning the cardiac localizations, and this is especially so in pleurisy. A thorough examination of the chest, which is necessary sometimes to show the existence of the pleural disease, is almost impossible, often on account of painful articulations. Hence, many cases are unquestionably overlooked. The symptoms of pain and difficulty in respiration are doubtless many times set down to muscular rheumatism and neuralgia.

Pleurisy, its Character and Duration.—Pleurisy is probably more frequent on the left side than on the right, apparently without discoverable cause. Sometimes it results from direct extension of the inflammation

from the pericardium through the walls of the sac, and I have seen the complication result also in the right pleural sac by the same process. Doubtless many slight localized pleurisies coexist with pericarditis and escape detection in the presence of the more grave malady.

A considerable number of well-marked double pleurisies, however, are found in acute rheumatism, which are developed independently and apart from any cardiac complication, or at least are not dependent on this as their direct cause. I am inclined to think that rheumatic pleural inflammations produce inflammatory materials of a less serous and more fibrinous character than is common in perhaps the majority of cases of idiopathic pleurisy. Whether the inflammatory effusion contains much or little fibrin we have but few opportunities of determining post-mortem, but certainly when fluid is present its absorption takes place readily and promptly. Very few instances—relatively to the whole number of cases of pleurisy or absolutely in those occurring in acute rheumatism—pass over into a chronic state, with empyema or other disastrous conditions. Perhaps it may be, as shown from the fibrinous condition of the blood, that fibrin or adhesive lymph is present in increased amount in the effusion of rheumatic pleurisy, and that through its agency adhesions, and the consequent cessation of the pleural disease, take place more promptly. It may be that the inflammatory process ceases here, as in the other localizations, with the abatement of the general symptoms of the rheumatic disease. However, in some cases of pleurisy which I have seen, post-mortem, the thickness of the pleural lymph was very great and also of very firm character, and the inflammation had been continued in a chronic manner with recurrences on the reappearance of the rheumatic symptoms. In another instance, seen post-mortem, there was little or no tendency to the formation of fibrinous material; it was a truly empyemic case and the lymph could only be described as cacoplastic. The patient was, previously to the rheumatic attack, in a debilitated condition. We must conclude, therefore, that this inflammation is not of a uniform type, but is modified by unknown conditions, and especially by the general state of health of the patient.

Pneumonia, its Relation to the Rheumatic Process; its Type and Grade.—Pneumonia is a much less common complication of the rheumatic process than pleurisy. So far as I have seen it, the gravity of the complication was of slight character and was connected with a coexistent pleural inflammation, on which the pneumonia apparently depended for its origin. The records of the cases of pneumonic complications of rheumatism point

rather to the occurrence of the catarrhal form of this disease than to the croupous. The only instance which I have seen post-mortem occurred in connection with chronic rheumatic pleurisy, and was of a mixed croupous and catarrhal character. The partial hepatization of the lung-tissue seems to be due to an extension of inflammatory irritation from the pleura to the alveolar trabeculæ, causing a catarrhal condition of the air-vesicles, and, when of a high grade, a fibrinous exudation also. Hypostatic pneumonic solidifications are not infrequent.

Mucous Membrane Inflammations, their Relationship to the Tissues Ordinarily Affected in Rheumatism.—The other forms of the rheumatic complications of acute rheumatism affecting the respiratory apparatus are mucous membrane inflammations, so called, and the kind of pneumonia above spoken of shows its affiliation to these other catarrhal inflammations seen in the upper portion of the respiratory tract. Rheumatism generally shows a tendency most strikingly to affect the fibrous tissues, such as the pleura, peri- and endo-cardium, as well as the articular structures, and although here the chief phenomenon is a catarrhal process, still the same tendency is, I think, exemplified, for it is apparently the submucous connective tissue or even the deeper tissue which first receives the morbid rheumatic influence, rather than the epithelium; the latter, by its close approximation, becomes involved as the irritative changes develop in the former. Of these bronchitis is the most frequent; a tonsillitis and a pharyngitis also have been described.

A well-marked inflammation of the mucous membrane of the pharynx is probably a not infrequent complication of acute rheumatism, but it is either overlooked or its true nature and significance remain unknown. It is unquestionably many times regarded as the effect of the cold or dampness which produces rheumatism, and such attacks are at first looked upon as a simple case of "catching cold," or "sore throat," but on the later development of the rheumatism are disregarded. In other instances, the throat affection is unheeded in the presence of the graver malady.

Rheumatic Angina.—The condition of the throat usually spoken of as an angina has been carefully studied and described by Lagoanère (1876). The disease is developed in the very beginning of the rheumatic attack and even may be found in some cases as a preliminary or prodromic symptom. Much more rarely it continues after the development of the articular inflammation. It usually appears with the onset of the fever, or attention is called to it just after the occurrence of this symptom, so that

its relationship, in point of time, may appear the same as that of an ordinary febrile sore throat.

The angina comes suddenly with a feeling of stiffness or pain in the region of the palate, especially provoked by movements, such as swallowing, speaking, and even by movements of the neck and head. The pain extends toward the ears, or upward to the nasal region and the forehead, provided the inflammation reaches in either of these directions. The muscles of the neck may become slightly stiff, as in torticollis, and sometimes the glands at the angle of the jaw and below are enlarged.

The throat presents a diffuse erythematous redness of not very intense character, and the mucous membrane is swollen, especially the uvula.

Its course varies ; ordinarily the appearance after full development remains fixed in the place affected, and sometimes it extends to the nasal and aural structures, or to the tracheal and bronchial mucous membranes.

The diagnosis is decidedly retrospective, and the condition finds its explanation through the rheumatic inflammation making its appearance in the joints. To some patients, well experienced in acute or subacute attacks, the angina is diagnostic, or rather prognostic, of such attacks. The appearances presented by the throat are not characteristic, and a dependence on the nature of the occurrence rests on antecedent and concomitant circumstances. The attendant symptoms, too, are usually greater than warranted by the condition of the throat. There are only two conditions, except a simple cold, with which a rheumatic angina could be confounded, viz., erysipelas and scarlatina, but from these diseases it can readily be distinguished by the characteristic symptoms of those maladies.

The nature of angina seems to find its explanation in the similar inflammatory conditions which occur on the skin in acute rheumatism. In the one the rheumatic process affects the external epithelial covering, in the other the mucous membrane exhibits very similar appearances.

Its course is always benign and is usually lost in the severity of the rheumatic symptoms by which its nature is made apparent and into which it generally fades as they develop.

Bronchitis and Broncho-Pneumonia.—The bronchial complication of rheumatism presents nothing to attract special attention, but if it becomes severe the agony of the patient from the racking cough is almost unbearable. The bronchial inflammation occasionally leads on to broncho-pneumonia, and this condition may be followed by a pleurisy.

Digestive Apparatus.

The digestive organs give rise to very few symptoms during life, and after death show few changes which can be attributed to the effects of the rheumatic process directly. Allusion has been made to the rheumatic anginas and coryzas, and these conditions have been observed to affect the œsophagus. Difficulties in swallowing, with or without pain, have been pointed out during the course of acute rheumatism; which have been attributed to a localization of the disease in the muscular apparatus of the œsophagus. The stomach and intestines very rarely give symptoms other than those common enough in any febrile malady. The gastralgia and enteralgia, not unfrequently spoken of, are symptoms easily confounded with localizations in surrounding parts, especially the diaphragm and the abdominal muscles, and must therefore be regarded as of doubtful character. The inflammatory lesions of the mucous membrane of the digestive tube, signalized by diarrhœa, dysentery, or hemorrhagic discharges, much more frequently precede or follow than accompany acute rheumatism, and the rheumatic inflammation is to be looked upon as a sequel of such diseases, or as a distinct attack due to very similar causes.

The liver is sometimes found increased in size after rheumatic fever of a severe and long-continued character. But of the symptoms and changes of function of this organ we know so little positively, except in obstructive disease of its ducts, that we are entirely ignorant of its condition, equally in rheumatism as in other diseases.

The peritoneum, from its anatomical structure, deserves consideration in connection with the fibrous connective tissue rather than with the digestive apparatus proper. This, the largest of the serous membranes, however, requires almost no attention in acute rheumatism; a few cases only are on record in which post-mortem inflammatory changes have been found in it. Such instances acquire importance by showing the wide, almost universal extension of the rheumatic influence rather than from the likelihood of being encountered clinically. It is probable, however, that an effusion of serum not infrequently occurs in the peritoneal cavity, but this is apparently of a non-inflammatory character. The condition seems to occur in cases where a similar effusion is found in the pleural cavities, or in which other localized or general œdemas are seen. Occasionally a peritoneal effusion follows a rheumatic pleurisy or pericarditis.

CHAPTER XIII.

NERVOUS COMPLICATIONS.

Symptoms—Acute Inflammatory, Cerebral, and Meningeal Lesions—Cerebral Rheumatism—Hyperpyrexia—Other Nervous Symptoms—Rheumatic Cephalalgia—Vertigo—Conditions which Tend to Produce Cerebral Symptoms and Lesions.

Symptoms.

In acute rheumatism the symptoms connected with the nervous system occasionally present themselves and form one of the gravest conditions of this malady. The cases with nervous complications divide themselves pretty naturally into groups and sub-groups, which, while acknowledging a common cause, differ so radically in some of their striking features as to appear quite dissimilar and even disconnected.

In acute rheumatism we can distinguish, in the first place, two great classes which are attended with nervous symptoms—one in which inflammatory or other lesions of the brain or spinal cord or their meninges are present; another in which the nervous phenomena are dependent on the general conditions of the malady. As yet no very obvious, or at least marked lesions of these tissues have been pointed out, but nevertheless, in many of these cases marked lesions of the vascular or other organs are present.

The accurate differentiation of these two classes is a matter of great difficulty. Doubtless, in former times cases of the latter class were frequently attributed to meningitis or cerebritis, which now with our greater knowledge and more extended observation would not be thus mistaken. Cerebral rheumatism without brain lesions is now a well-recognized form of the malady, as our knowledge of the symptoms of meningitis has become more accurate.

The two anatomical classes do not, however, afford convenient divisions under which to study clinically these phases of rheumatism, although the distinctions which we have just made are essential to be borne in

mind for a correct appreciation of the various forms of the malady and their treatment.

There are four principal groups of cases which represent the various forms in which the nervous symptoms appear.

First.—The acute inflammatory and similar changes of the brain and spinal cord and their membranes.

Second.—Cerebral rheumatism proper, in which the symptoms occur during the course of the disease but which do not furnish evidences post-mortem of inflammation or other appreciable changes of the brain or its membranes. Sometimes the small cerebral vessels are altered, but much more frequently other organs are the seat of lesions which are supposed to be directly or indirectly the cause of the nervous phenomena.

Third.—Hyperpyrexia. This condition may be thought to be a subdivision of the second group, but I prefer to separate it, because it may have merely the one essential and all-important feature, viz., the excessive temperature, which is almost always lacking in the cerebral rheumatism group.

Fourth.—A set of nervous symptoms which may occur in both acute and chronic rheumatism. They may develop during the acute attack or during convalescence from it. Some of these symptoms are seen in patients who exhibit at the time little or no evidence of the ordinary rheumatic condition. The phenomena are supposed to be due to the same process, although no articular inflammations are present.

1. Acute Inflammatory, Cerebral, and Meningeal Lesions.

The certain recognition of cerebro-spinal lesions in acute rheumatism, is often, as in many other acute diseases, a matter of great difficulty; perhaps even greater than in most others. Because in many of the diseases attended with nervous phenomena we know by extended experience that no lesions which can be recognized as the cause of the delirium, etc., are exhibited. Therefore the conclusion has been reached that such symptoms are only epi-phenomena of the disease.

In any case of rheumatism, attended with nervous symptoms—and it is only in acute cases that these occur—meningitis is the least likely cause. Cases are on record, however, where post-mortem evidences of inflammation have been found—the membranes and the surface of the brain showing injection, with lymph or pus—but they are few in number, very few

even in proportion to the whole number exhibiting head symptoms, which are themselves by no means abundant, and probably even less so now than formerly under a different mode of treatment.

By some the rheumatic nature of even these cases is denied and the condition has been looked upon as an intercurrent attack of meningitis rather than a localization, or a complication dependent on rheumatism.

The spinal cases are of even less authenticity than the cerebral ones. Inflammation of the vertebral joints and pains in the posterior trunk muscles are comparatively common, and the former may even extend to or affect the closely lying tissue within the spinal canal, or by pressure affect the nerve-trunks at their points of exit, giving rise to some phenomena closely simulating spinal inflammatory changes. It is likely also that cases of spinal meningitis, of different origin, perhaps, from cold or exposure, and even epidemic cerebro-spinal meningitis have been mistaken for acute rheumatism of irregular type.

Under this class of complications we must refer to the "chronic rheumatic disease of the brain" which was first described by Griesinger (1860, *Arch. f. Heilk.*). The cases express themselves by symptoms of mental disturbance, in the strict or narrow sense of the term, and not so much by phenomena which are usually connected with meningeal or other gross lesions. In these cases, the few that have been examined post-mortem, no distinctive or plainly causal lesions have been shown to exist, but still I think we may as fairly attribute them to actual, though unknown, alterations as we are in the habit of doing in other cases of insanity dependent on other causes than rheumatism. The common form of mental disturbance resulting from rheumatism is melancholia, and the condition is vastly more frequent, as is this disease from whatever cause, in adults than in children.

The pathological alternations of all the forms of cerebral and spinal disturbances in rheumatism come under one or more of the following classes of changes: 1. The anæmias or congestions; 2, inflammations or effusions of the membranes or of the brain substance; 3, lesions of the vessels proper, either from embolism or thrombosis; and, 4, softening or induration of the nervous tissues.

All these lesions occur comparably more frequently in connection with similar conditions present in other organs, or as the result of prior lesions determined in other parts, than simply from the localization of the rheumatic process in the cerebral centres. Certain portions of the nervous

apparatus seem to be more frequently affected primarily or principally than others, and this preponderance is exhibited in a very striking manner. Thus the dura mater, both cerebral and spinal, are by far less commonly affected in the total than any other portion. The arachnoid, the subarachnoid space, and the pia mater exhibit or give rise to many more symptoms than all the others combined. The cerebral tissue proper and the vascular apparatus, while not escaping from changes during the acute stage, show the effects of rheumatic process secondarily or as the result of lesions in distant organs, especially the heart.

The anæmia and congestion, especially the former, may be divided into two classes, the one apparently functional, so to speak, either by exhibiting the appearances or giving rise to symptoms which we usually connect with an ensanguined state of the vessels ; the other class including the essential anæmia connected with the alterations of the blood, which, during the rheumatic process, becomes deficient in the red-corpuscular element. With these conditions we may also speak of the chemical alterations in the blood, of which we know emphatically little, but which are supposed to be due to its surcharge with the rheumatic poison.

The congestions of the cerebral substance are much more readily appreciated with the naked eye, and their effects are easily shown by the aid of the microscope. They are exhibited under two forms, although the clinical features are indistinguishable ; the effusion in which they result may be either of blood or of serum, and their extent may be quite localized or a general one, and their development slow or rapid. Their causes may be spontaneous or the results of lesions in other organs, or of lesions in the vessels. The result may exceed the congestive stage and proceed to an actual hemorrhage, simple or multiple, large or small. The hemorrhagic effusions are almost invariably minute and are to be seen in the perivascular sheaths of terminal capillaries and also occasionally in patches under the arachnoid. Large cerebral hemorrhages are unknown, and the term apoplexy, not infrequently found in older authors in describing certain rare cases of fatal rheumatism, cannot be attached to this lesion. It relates, probably, to cases whose pathology was then quite unknown, of hyperpyrexia or of uræmia. To these minute hemorrhagic effusions are closely allied cases in which the capillaries are found dilated (capillary aneurism), as the result sometimes of local disease of the vascular wall, sometimes of complete or partial occlusion of their lumen.

The serous effusions are seen more frequently in the subarachnoid

space or tissue than in the ventricles of the brain, but evidence is not wanting that fluid is effused in the substance of the brain—a perivascular distention which leaves its distinctive traces behind it. However, the anatomical arrangement is such that any considerable subarachnoid effusion is accompanied by or even produces, at least post-mortem, an abnormal amount of fluid in the ventricular cavities. The means of communication between the surface and central cavities is afforded through the lymph-tracts. The more frequent occurrence of the subarachnoid fluid, even though a slight amount of fluid is found in the ventricles, shows a peripheral tendency on the part of the morbid process, which is thus in accord with the similar localization of the inflammatory process in the same part of the brain.

The inflammatory conditions may be described under two heads or forms, which are convenient for analysis, but which merge into each other, and are not well differentiated by their clinical features.

The least common form of the inflammation is that which exhibits itself by more or less abundant inflammatory or purulent products on the surface membranes of the brain, and extends into the cerebral tissue itself. It is spoken of as meningo-encephalitis. The other form is seen by the microscope as an effusion along the walls of the capillaries, especially the minute ones in the brain-substance, which appear augmented in numbers and often undergo a considerable dilatation in size.

2. Cerebral Rheumatism.

Acute rheumatism is regarded, and truly so, as a disease which, for its severity in respect to its ordinary febrile phenomena, is singularly free from all those symptoms of a nervous type usually spoken of as cerebral. The patient's mind, even in the height of the fever, is clear and the expression intelligent. It neither inclines to any form of delirium, either active or low, nor is there found dulness or stupor. The vast majority of cases run their course without any nervous symptoms, unless we except a degree of sleeplessness, which cannot be described as insomnia, since it is the result of pain and discomfort rather than the effect of the disease on the nervous centres. In describing the nervous conditions or complications, therefore, we are describing unusual and exceptional cases.

Of those which we propose to describe under this head, cerebral rheumatism is the most important. For what I have here to relate of this com-

paratively rare form of rheumatism I am chiefly indebted to the article of my colleague at the Pennsylvania Hospital, Dr. Da Costa, in the *American Journal of the Medical Sciences* for January, 1875.

This author gives the following as a general description of cases of cerebral rheumatism: "In the course of acute rheumatic fever, usually after it has existed for some time, or even after convalescence and among the first signs of a relapse, appear symptoms of cerebral disorder, manifesting themselves chiefly by restlessness, passing into stupor or coma, or becoming associated with delirium. The former combination is less common than the latter and of much shorter duration. The delirium is preceded by wakeful, dreamy nights, is generally mild, and it is during the restless nights that it shows itself most plainly. Though it may be a continuous, it is scarcely ever a fierce delirium, and is not, as a general rule, linked either to headache, injected eye, or vomiting. It may run a rapid course, delirium or stupor quickly ending in coma, coma in death. But ordinarily it goes on for days, the patient gradually mending or becoming weaker and weaker, and passing perhaps into a condition very similar to that of typhoid fever, excepting that the bowels are constipated. The likeness to enteric fever is heightened by the presence of sordes on the teeth, and the appearence of an eruption. The temperature is apt to be high, the joint-affection persistent, or even showing signs of increase; the breathing is rapid; the pulse is frequent, compressible, and at times irregular. A cardiac difficulty may show itself distinctly as a complication, or again be wholly wanting. In some cases convulsions, in others, local palsies happen; or we may have hemiplegia even suddenly developed. But these features are rare; and it is in the wakefulness and restlessness, in the stupor and delirium, that we mostly find the signs of how decidedly the brain has become disordered." The author in describing the malady limits the term cerebral rheumatism to cases in which the nervous symptoms are prominent and appear to constitute the real features of the affection. He says further that "doubtlessly occasional restlessness and some slight mental wandering at night are not uncommon in decided cases of acute rheumatism at the height of the fever, and are more likely to be found if there be a cardiac complication; but such transitory and subordinate phenomena do not make a disorder which we can call cerebral rheumatism."

The author further says that he does not desire "to speak of the disturbance as one complaint, when we shall find that several morbid states

may contribute to it," and in the general description given above it is used as marking the cerebral type of the malady rather than pointing out a separate and perfectly well-defined morbid condition.

A most interesting point in connection with cerebral rheumatism, as here defined, is the febrile state or temperature of the body. The cases recorded by DaCosta—and the few other cases scattered through medical literature agree with them—show that the temperature at the time of the occurrence of cerebral symptoms is high, and that only a slight rise of temperature precedes the cerebral manifestations. Subsequently variations are but slight, except the morning remission of about one degree, or even less, until recovery, or until the bodily heat increases just before death. The exceptions to this are when, during the course of the cerebral symptoms, new joints become affected, when, as usual, a decided rise of temperature takes place; otherwise the temperature record remains singularly uniform, and cerebral rheumatism, like the visceral complications, for example, endo- and peri-carditis, shows only slight thermometric fluctuations.

It will therefore be seen that cerebral rheumatism, one of the forms of the nervous complication of the disease, differs strikingly from hyperpyrexia, another of the nervous complications, in that the temperature does not reach an excessive height. It is necessary, therefore, to differentiate hyperpyrexia from the brain symptoms strictly—cerebral rheumatism.

Of the strictly cerebral symptoms we may point out three types or classes: 1, stupor; 2, delirium (usually of a mild type); and 3, an active delirium, closely resembling that of acute mania, well characterized by the term rheumatic insanity. It is probable that these three groups are due to distinct morbid causes, and show post-mortem quite distinct morbid changes.

It is probable that a majority of the cases of cerebral rheumatism attended with stupor will give evidence of a renal complication, or perhaps pre-existing kidney disease, and that therefore the condition is not so much one of cerebral rheumatism or of a rheumatic disease of the brain, if we attempt to erect this group of cases into a well-defined morbid condition, as it is the result of the changed function of the urine. It is, therefore, the result of renal disease, either following out its ordinary results, or the involvement of the kidneys in a morbid condition as the result of the rheumatic process.

It is not necessary to conclude absolutely that every case of cerebral rheumatism is the result of renal disease, since other diseased conditions

of the brain or its vessels are productive of a state of stupor. DaCosta points to the probability "that nearly all the so-called apoplectic instances of cerebral rheumatism have as their cause renal disease rather than cerebral alterations;" and also that this cause "suggests the explanation of many of the other nervous phenomena of" acute rheumatism.

The second group, those attended with delirium, find likewise in many instances their explanation in some visceral complication other than the cerebral. This condition is paralleled by the phenomena which are witnessed in other diseases than rheumatism. For example, in pneumonia delirium of a mild type is of not infrequent occurrence, and the same, though much less frequent, occurs in inflammation of the serous membrane. In reviewing cases in this group of cerebral rheumatism, the records frequently show the occurrence of endo- or peri-carditis.

In these cases it will be of service to note the fact that restlessness, which may be considered part of the delirious condition, is often more striking than the mental aberration, and fits in, perhaps, with the early observation of Dr. Bright on "cases of spasmodic disease accompanying affections of the pericardium," and also with the earlier and more recent observations on the association of rheumatic disease with chorea.

The cause or perhaps the mode of action of the rheumatic disease in exciting chorea or other motory phenomena has been very variously viewed. Formerly these conditions were spoken of as true excito-motory phenomena caused by the rheumatic poison acting on the nervous centres. More recently they, and especially chorea, have been thought to be due to the alteration of the circulation of the brain produced by minute emboli of the cerebral centres. And even as yet the whole question remains in a very unsettled state.

3. Hyperpyrexia.

In the third group of cases the rheumatic insanity, or those attended with active delirium, we shall probably find the residuum which we can with accuracy describe as cases of cerebral rheumatism, or a rheumatism of the brain, and which we could, if it were desirable, erect into a group forming a well defined morbid condition. To do this we must separate from the whole number of cases of acute rheumatism attended with cerebral symptoms, on the one hand, those which may be fairly considered as of renal origin and also those in which the symptom is due to visceral in-

volvement, generally thoracic and attended with delirium. On the other hand, as already said, the cases of hyperpyrexia frequently exhibiting head symptoms must be placed to one side. And again, also, all those cases must be excluded in which the cerebral symptoms—and these cases are sometimes attended with motor disturbances of a marked type—can be traced to embolic occlusion of the cerebral vessels, chiefly the smaller ones.

What then have we left to constitute our group of true cerebral rheumatism?

Here again we must separate the rare cases of real rheumatic meningitis. The residuum is, I think, to be regarded as composed of all such instances in which the cerebral symptoms have been considered as due to the action of rheumatic poison on the brain-tissue directly, or indirectly through the changed composition of the blood, or perhaps by both of these ways.

How the rheumatic poison affects the brain-tissue is of course unknown, and if it does so at all, it would seem that the cerebral phenomena should occur with greater frequency in rheumatism than is certainly the case. The instances in which cerebral phenomena appear are comparatively very rare, and these, too, in patients who do not evidence strongly the ordinary rheumatic phenomena, or else in cases during convalescence from an attack. It would seem, therefore, that if these nervous symptoms were the direct effect of the rheumatic process on the brain-tissue, that its effects would be more frequently seen both during life and on post-mortem examination. In respect to the indirect action on this tissue, either from the deterioration of the blood or from its changed composition through the presence of the new material, we have rather more evidence to substantiate a belief. In the first place it falls in with our notions of what takes place in other diseases. Thus, in nearly all the continued fevers, under certain conditions, we are familiar with the occurrence of cerebral symptoms which cannot be traced to evident lesions of tissue.

Then, too, we have very constant and positive evidence of deterioration of the blood. This makes itself apparent in a large majority of cases by the profound anæmia exhibited by patients. The change in the constitution of the blood, especially its corpuscular elements, can readily be discovered by a microscopic examination, which shows a great depreciation in their numbers as compared with health.

A third condition, which may properly be included under the head of real cerebral rheumatism, is an alteration taking place in the lining mem-

brane or the walls of the minute cerebral vessels. The effect of such changes is to produce a partial or complete occlusion of their calibre and a consequent disturbance of the cerebral circulation, affecting larger or smaller areas of the main tissue.

This change is thought to be due to an inflammation or degeneration of the vascular wall, whereby its smooth surface is roughened and presents itself as a favoring spot for the formation of a clot or coagulation of blood.

This is a process of thrombosis of the cerebral vessels as distinguished from embolism, and how far the two conditions can be satisfactorily distinguished requires further study. Minute emboli are at best difficult to identify, and it often depends upon a process of exclusion; if no vegetations are found upon the heart-valves or other roughened surfaces in contact with the blood-current, occlusion of the minute vessels may be said to be due to other conditions than embolism, although it is possible to suppose, and does undoubtedly occur, that under certain conditions minute clots form in the blood, and are carried along in its current, forming plugs in certain terminal vessels. The discovery of degenerative changes of the minute vessels of the brain, accompanied with the roughness of their walls, would, of course, substantiate the thrombotic origin or nature of their occluded condition.

It must be remembered, however, in this connection that minute or partially occluding emboli may, on the one hand, be wholly or partially absorbed, and on the other hand, by adhering to the vessel-wall, produce inflammatory or degenerative changes of its substance. Thus either one or the other of these results of embolism may present appearances or changes closely resembling initial vascular disease or autochthonous embolism with their subsequent changes.

It is certain, however, that a considerable number of the post-mortem records of cases of cerebral rheumatism show evidences of occlusion of the brain-vessels in which no appearances of endocardial or arterial disease likely to give rise to minute emboli were found, and which therefore may be fairly set down to the account of changes in the cerebral vessels due to the rheumatic process.

How far these changes of the cerebral vessels should be viewed strictly as cerebral rheumatism, or how far be classed with such affections, as for example, endocarditis, will be further considered under the head of Complications.

It is especially interesting to note the fact that, in nearly all the cases

of cerebral rheumatism as here described, the brain exhibits so little alteration, and none at all commensurate with the evidences of the grave symptoms during life.

4. Other Nervous Symptoms.

There are a number of symptoms of a nervous character which at various times, and by many authors, have been attributed to rheumatism. These phenomena can, in general, be spoken of as of lesser importance than those previously referred to, although at times they become of severe character or even of fatal severity. Some will be found to manifest themselves during the height of a rheumatic attack, others during convalescence, while others, again, precede, by a greater or less length of interval, a rheumatic paroxysm, or, on the other hand, become one of the sequelæ of such attacks.

Among those coming in the course of a rheumatic attack, DaCosta has observed cases of hurried respiration. He speaks of this symptom as occurring without anything in the condition of the lungs or heart to account for it. He mentions the symptoms as among some of the other morbid manifestations met with in cerebral rheumatism, and it is a condition which is certainly to be observed not alone in cases of cerebral rheumatism, although perhaps in such cases owing to the general nervous condition of the patient otherwise manifested of more frequent occurrence, yet I have seen it in a pronounced degree in severe cases of rheumatism unattended with other nervous phenomena.

Senator speaks of "certain paroxysms of palpitation and oppression which not infrequently occur during rheumarthritis ; they are transient, and do not depend on any cardiac complication." As in these cases no visceral lesions are evidenced by the symptoms of the organs principally concerned, one cannot avoid speaking of the phenomena as of nervous origin.

The occurrence of death during such paroxysms has been reported, but must be exceedingly rare.

I think it will be always doubtful whether such manifestations, when not directly traceable to some visceral involvement—especially such as endo or myo-carditis, as unquestionably is sometimes the case—can be regarded as strictly of nervous origin in the same sense attributed to the other nervous manifestations here discussed. The doubt will remain

whether they are not due to simple exhaustion, the result of pain and loss of rest. If due to any of these causes, they ought not to be spoken of as of rheumatic origin.

Rheumatic Cephalalgia.

Rheumatic cephalalgia has been described by many authors, and especially by Canstatt and Gubler, as the first form of cerebral rheumatism. Boerhaave, van Swieten, and Frank long ago pointed out a rheumatic headache, and spoke of it as one of the early symptoms of rheumatism, but their observations, I think, are not confirmed by the common opinions of later authors.

This rheumatic headache must not be confounded, though it may very easily be and has frequently been so, with neuralgia and with rheumatism of the scalp. Desguin, in a communication to the Belgian Academy of Medicine, gives an observation which I think may truly be regarded as rheumatic headache, and belonging to the group of cerebral rheumatism. He describes the pains as of a mobile character, occurring at various points of the cranium, especially at the level of the occipital and frontal prominences. The pains seem to be within the skull, and pressure on the surface does not modify their character, as is the case in rheumatism of the scalp. The muscles of the neck are more or less rigid, and the movements of the head painful, and at the time often the movements of the eyes cause pain. Conditions of coldness and dampness aggravate the sensations as in rheumatic affections of the joints. Febrile conditions are usually not present. The attacks, as observed by Desguin, is followed by rheumatic phenomena, especially of the articulations of the extremities or by alternations of headache and articular inflammation.

Vertigo.

A condition of vertigo of rheumatic origin, or occurring in connection with rheumatism, has been recognized for a long time, and is spoken of by many of the older authors. Musgrave, Stoll, Bang, Barthez, and J. Frank refer to a rheumatic vertigo, but many of these references are to a degree doubtful, since rheumatism in their nomenclature possessed a diverse signification, and in many of their cases this symptom was unaccompanied by any general affection. Herz, in 1791, was the first to differentiate the dizziness occurring in rheumatism from the quite unlike

condition in cases of gout, which, up to his time, had been combined under the head of arthritic vertigo.

Sovet, in a memoir directed to the Belgian Academy, in 1858, was the first one to give an authentic account of the condition, which met the approval of the learned body to which it was submitted. Desguin also contributes a number of cases.

Rheumatic vertigo cannot be considered as a very important condition, although it may be a very striking phenomenon connected with this morbid process; it is more strictly to be looked upon as an incident of the disease. Its description rounds out the phenomena of the group of cerebral or nervous rheumatism, and it is probably to be looked upon as a sub-group, having to do with the spinal, rather than the cerebral manifestations of rheumatism; neither does the condition seem to be a very rare manifestation.

Partly from my own observations, and partly from the records of others, the condition is characterized as follows: pains localized in the neck, sometimes in the head also, and especially down the spine, and increased by movement of these parts. The dizziness is increased by attempts at walking, and is nearly allayed by complete repose. There is a confused mental condition and a sense of depression; both of these conditions struck me as dependent quite as much on the physical condition, connected with other organs, as on the cerebral state, strictly speaking. These symptoms are continuous, but quickly disappear on the occurrence of articular inflammation, which they usually precede. The vertigo may, however, alternate with the articular phenomena, and may therefore succeed the usual manifestations of the disease, and in turn be followed by their reappearance. In a case under my own observation the vertigo, with staggering gait, was the initial symptom of a relapse, and ushered in an attack of rheumatism affecting the vertebral joints. In this case the muscles of the back were becoming strikingly painful, and I was led to think that the staggering gait was produced by the failure of these muscles to properly balance the trunk. The muscular tissue was probably involved to a certain degree before the extreme pain was developed in them, as occurred later, and this explanation still seems to me not an improbable one.

So excessive becomes the vertigo in some cases, that the patient falls to the ground. Whether this results from a sudden increase of the vertigo —the more strictly nervous symptom—or from the disability of the mus-

cles, owing to pain, which is present in them in many instances, to a high degree, is doubtful. I think from the records of the cases that both of these causes may be held accountable for the phenomenon.

The diagnosis of the condition may be at first very uncertain, and great alarm may be caused by the symptom, but fortunately the nature of the condition rapidly becomes apparent, usually by the development of familiar rheumatic symptoms. The occurrence of the dizzy symptoms, intercurrently in an attack of rheumatism, or as a relapse, would readily find an explanation, but as primary phenomena they would be quite uncertain until the appearance of other symptoms. Occurring in patients having a strong tendency to the disease, the rheumatic vertigo could be distinguished from a gastric, epileptic, or others of a purely nervous origin, or any condition due to grave cerebral lesions.

The appearance of the vertiginous symptoms may take place in cases of very mild, subacute character, or in those of more severe type, and no marked tendency to visceral complications of any sort has been shown in connection with the recorded cases. Especially is this true in respect to endocarditis; in my own case no valvular murmur existed, although the patient had suffered repeated attacks of acute rheumatism, and from a previous attack, a year before, pericarditis with adhesions resulted, but without endocardial inflammation.

Cases occur, but only very rarely, in which symptoms very similar to those above recorded persist in a chronic manner, accompanied usually with vague rheumatic pains in some portion of the body. For example, a patient who has suffered from slight rheumatic pain in one or more joints becomes affected with dizziness or vertigo, which will persist during the whole duration of the slight joint affection; sometimes the occurrence of the vertiginous symptom leads the patient to expect the advent of the rheumatic condition, so constantly is the former the forerunner of the latter.

Some of the other forms of manifestation of rheumatism of a nervous character are chorea, hysteria, contraction of the extremities, tetanus, insanity, and some of the visceral neuralgias, so called.

Conditions which Tend to Produce Cerebral Symptoms and Lesions.

So far as we are able to extend our observations, we are compelled to acknowledge that cerebral symptoms occur in the course of acute rheumatism without lesions existing in the brain, which we are able to define or

which we can place in correspondence with the clinical features. This is true not alone in rheumatism, but also in many acute disorders whose primary and principal manifestations are localized in other organs than the brain. Yet, although we are unable to point out lesions correspondent to the symptoms, we are loath to believe that no lesions exist.

Hence, it has become usual to account for such symptoms as the effect of reflexes. In the case of rheumatism, the efficient reflex has been thought to exist in inflammations of the visceral serous membranes, and the not uncommon coexistence of rheumatic pericarditis with head-symptoms has been quoted in support of this view. By others the irritative effect of a large number of articular phlegmasias is considered as productive of the same result. This view is supported by the tendency of head-symptoms to appear when a large number of joints are involved in the rheumatic process.

The older authors viewed all cerebral cases as the result of the metastasis of the inflammation from the joint to the brain, and they accounted for the absence of the cerebral inflammatory appearances in the same way as the similar disappearance from the joints themselves.

Many cerebral cases unquestionably concur with hyperpyrexia, although it is not clear which is cause and which is effect. Is the hyperpyrexia caused by the localization of the rheumatic process in the brain disturbing the heat-regulation of the organism, or are the cerebral symptoms merely an incident in the course of a case attended with high temperature?

The heart-lesions unquestionably are productive of many cerebral accidents in acute rheumatism at many stages of its course. The changes in this organ, along with alterations in the quality of the blood, cause the large majority of all cerebral symptoms, and to these may be added the effects of certain remedies which in times past have received credit for a greater influence on the cerebral phenomena than at present.

The frequency, relative and absolute, of cerebral symptoms is modified by many circumstances. General statistics show that between three and four cases in every hundred exhibit some form of cerebral disturbance; many give higher figures, and some even lower. Certain seasons and particular years often furnish large numbers of cerebral cases—larger numbers among an actual fewer number of cases of rheumatism. Men are more frequently affected than women, but this results not so much from any sexual differences as from their differences in exposure or from their habits, especially the use of alcohol.

The effects of accidental causes, especially exposure to cold, during the course of acute rheumatism is seen, from a review of cases, to have marked effect in producing cerebral complications. A predisposition to nervous phenomena is to be found in rheumatism equally as in other acute diseases.

The period of the attack at which the phenomena make their appearance varies very greatly. The variety of the cerebral symptom or lesion has its effect in determining this epoch. Allusion has been made to the primary localization of the rheumatic process in the brain or its membrane —those cases in which nervous symptoms are manifested before the articular inflammation—but these instances are exceedingly rare. From the fifth to the twentieth day of the attack the largest number of cases are seen, but it is necessary to remember that no grade of rheumatism, mild or severe as to general symptoms or as to the number of joints involved, shows an immunity from cerebral symptoms in one or other of its many diverse and distinct forms. Generally, too, they make their appearance without definite warning, and are usually rapid in their development, although sometimes slow and not unexpected.

The prognosis in these cases varies manifestly with the efficient cause of the occurrence of the nervous symptoms and with the severity with which this cause is acting. In the majority of cases in all the forms the prognosis is unfavorable.

CHAPTER XIV.

MORBID ANATOMY.

Pathological Changes: Hyperinosis; Condition of Joints; Changes in Cartilages; In Tissues and Skin; In the Blood—Hyperpyrexia—Cardiac Conditions—Endocarditis—Histological Process in Endocarditis—Embolism—Cardiac Murmur—Valvular Vegetations—Inflammatory Products: 1. Inflammatory Resolution; 2. Inflammatory Organization—Chronic Endocarditis—Pericarditis—Acute and Chronic Myocarditis.

Pathological Changes.

A description of the pathological changes in rheumatism is necessarily a brief one. Partly because in a disease in which the number of deaths is small our knowledge is not extended, and partly because the morbid alterations are but few in number. Those that are seen serve to cast but little light on the pathology of the malady.

It is proper in this connection to speak of the mode of death and its immediate cause, or the train of symptoms which precede its occurrence. And these causes can be classed under various heads, in reference to the prominent symptoms which attend them. Thus we see deaths from hyperpyrexia, from cardiac diseases, from pulmonary troubles, from cerebral rheumatism, and to these may be added a few others, which are so rare that it is not necessary to erect for them special classes.

It will be seen that all of these, except the first named, are complications or localizations of the rheumatic process rather than essential features of the typical disease. Exception may be taken to this statement by some who regard the hyperthermic condition as evidence of a nervous complication of the malady. This question has already been partly discussed, and whatever may be its correct solution, the pathological appearances so far furnish no evidence by which it can positively be decided.

Hyperinosis.—Perhaps, in addition to the above given classes, another of often striking importance should be mentioned. It is not one, how-

ever, of which we have symptoms or evidences during life. It is a condition which is discovered post-mortem, although in times past it was familiar enough when venesection was customarily practised for acute rheumatic fever. It is *hyperinosis.*

The fever, the blood-changes, and inflammation of the joints—the three characteristic and almost essential symptoms or conditions of acute rheumatism—either leave behind them little trace of their presence, or else in themselves are not the cause of death. The hyperpyrexia is a frequent cause of death, but leaves behind nothing to show its cause and brings about only such changes in the organs as we are accustomed to see in other diseases attended with high temperature. The blood-changes we have but an imperfect knowledge of, and we are almost without a knowledge of the essential condition or element which is supposed to be present in the blood and to be productive of the rheumatic phenomena. The inflammation of the joints, so striking during life, shows, as a rule post-mortem, only a little increase of their fluid contents ; even this fluid may have almost entirely disappeared, and often none of the other indications of a severe inflammation can be detected.

Condition of Joints.—In describing the morbid alterations found in acute rheumatism, the condition of the joints claims our first attention. On opening a joint shortly after the epoch of an acute rheumatic inflammation there can be seen a greater or less degree of injection of the synovial vessels, which may in places appear dilated, or even ecchymotic spots may be seen. The cavity of the joint contains more than the normal amount of the fluid, although its quantity is much less than the swelling during the acute stage would lead us to expect. The fluid is thinner than the normal synovia and it may be quite transparent, though not infrequently it is clouded or quite opaque. A microscopic examination shows that the opacity is due to fatty or granular cells, floating in a transparent fluid. These cells are derived from the proliferation of the synovial tissues. The synovial fringes themselves show a formative activity and the cells are seen often becoming detached and separating from the basis-tissue. Their substance is highly granular and their nuclei enlarged and they stain with great readiness. These cells are seen to contain spaces, like vacuoles, which are filled with mucin.

A chemical examination of this fluid shows that its normal alkaline reaction is sometimes changed to acid, but a knowledge of the acidifying principle is wanting. No essential difference has been found between the

fluid removed from the joint during life and that found post-mortem, if we except a condition of coagulation which is sometimes seen to take place. And this post-mortem change probably accounts for those cases where a fibrinous inflammatory matter has been described. The presence of mucin forms the most striking feature of the articular effusion, which in other respects closely resembles that found in the pleural cavity as the result of inflammation. These fluids contain fibrin, cellular elements, and a principle like albumen, which coagulates with heat when rendered acid. The fluid in the joints cannot in the strict sense of the word be looked upon as ever being purulent, and the opacity which has given rise to this notion is conferred by the unusual abundance of the cellular elements detaching themselves from the synovial fringes. Suppuration with erosion of the cartilages is absolutely one of the rarest phenomena of acute rheumatism and probably only occurs in conditions of profound dyscrasia.

Changes in Cartilages.—Almost the only changes exhibited by the cartilages, at least to the naked eye, are a slight tumefaction, a loss of polish, especially noticeable in cases where post-mortem coagulation of fluid takes place, and a slight loss of resistance or a softening of their tissue. The velvety condition or villosities, which have been described, are probably nothing more than post-mortem coagulation of fibrin. The other changes in the cartilage which may be present, even when the naked eye appearances of the tissue are unchanged, consist of hyperplastic and proliferative activity, exhibited by the cartilage cells, similar in kind to those seen in the synovial fringes. These cells are swollen, the nuclei enlarged, or even vesicular and the nucleoli very distinct, and cell-multiplication by division has been described as taking place. The changes in the cartilage involve principally their superficial layer, and even here only in isolated areas; the deeper portions of the tissue, however, do not escape, but here, as in the superficial layers, the change is exhibited in a restricted manner. This proliferative activity of the cartilage-cells, if long continued or many times repeated, is capable of distorting the regular arrangement of the cells and of forming striæ which render the superficial layer irregular. The basis substance of the cartilage (intercellular substance) is, however, not seen to become granular as in scrofulous and other inflammations of the joints, although a liquefaction of this part has been described. No deposit of adventitious material is ever seen to take place, as is so noticeably the case in gout.

The periarticular tissues, and the skin lose post-mortem to a great degree their characteristic ante-mortem appearances. The redness and swelling largely disappear. A certain amount of œdema, however, is discerned, and the veins are often turgid with blood. Besides these, in some cases subcutaneous ecchymoses are found, and very much more rarely trifling collections resembling purulent matter are seen.

This condition may extend itself to the tendinous bursæ, and along the intermuscular septa. In these parts, as well as in the periarticular tissues, and even within the joints, thickening, resulting in permanent deformities, may be found in numerously repeated attacks, though in other cases, after quite as frequent repetitions of the disease, almost no alterations are discoverable. The older authors not infrequently described both acute and chronic alterations of the bones and periosteum as the results of rheumatism, but it is probable that the conditions were confounded with other diseases of which, at present, we have a better knowledge.

Changes in the Blood.—The blood, as found post-mortem, is almost always firmly coagulated, and its coagulability is shown likewise ante-mortem. So great is the increase of fibrin, that acute rheumatism has been described as a type of the phlegmasiæ. Its amount has been shown to range from 3 per 1,000, the normal, as high as 6 or even 10 per 1,000. The blood drawn during life shows at once the increase of the fibrin, as well as its tendency in respect to coagulation, by forming a markedly buffy coat. This same condition is exemplified by the highly fibrinous character of the inflammatory products, seen on the serous membranes, and also in the readiness with which vegetations form on the inflamed endocardium, as well as in the laminated coagula found in the heart-cavities. The only exceptions which are met with where the blood is fluid post-mortem are in some of the cases of hyperpyrexia.

The albumen is often deficient in amount, while the whole amount of solids in the serum is increased. The red globules progressively and often rapidly decrease in number, the diminution amounting sometimes to one-half the whole number. Our knowledge of the other constituents, normal or pathological, of the blood is very defective. Urea and uric acid have been found in the blood by some observers, but their presence has been denied by others with equal positiveness. Whether lactic acid is present or not, the blood serum, except in very rare cases, preserves its alkalinity and is certainly uninfluenced by this agent and its presence has never been satisfactorily shown.

Garrod has demonstrated the absence of uric acid or its salts, which are universally present in gout.

In reviewing the records of cases of death from rheumatism it will be found that, with the exception of hyperpyrexia, this event results from some one of the complications in a very large majority of the number. We have just seen that in the one essential symptom of the disease, the inflammation of the joints, there are not present conditions incompatible with life or that are likely to cause death. We will therefore point out the conditions present after death in the various classes of cases.

Hyperpyrexia.

The mode of death in these cases is apnœa, and the death, as shown by the post-mortem condition of the heart and its contents, is not a sudden one ; this statement is apparently in contradiction to the clinical record of these cases, since the observation is not infrequently made that death occurred suddenly. In the majority of them, however, the condition of the blood (hyperinosis), favored by the somewhat prolonged agony, gives rise to clots of ante-mortem character in the right cavities of the heart, which often extend far into the pulmonary arteries. In others of these cases unquestionably death is really sudden, and from this and other causes the blood is found uncoagulated, though it may clot subsequent to its removal from the body on exposure to the air.

In these cases, as well as in others to which the name of hyperpyrexia can scarcely be applied, although likewise attended with high temperatures, the post-mortem examination shows very deep congestion of the lungs, a congestion which is universal throughout their substance though of course more marked at the posterior parts, and therefore due to hypostasis, both before and after death. This congestion is promoted by the conditions above spoken of, both the tendency to pulmonary death and also the fibrinous condition of the blood. But it also has a life history and is recognizable by clinical features prior to the approach of death. The congestion seems to attach itself to the general condition of the patient, and to be a part of the same condition (or to be caused by it) as the high temperature, to be, in other words, of central origin rather than a simple pulmonary disease. The respiration is imperfectly performed, because through the effects of high temperature the nervous centres do not regulate the respiratory act, or perhaps, so to speak, do not perceive the neces-

sity for the aëration of the blood. This description is, I am aware, purely theoretical and has no better basis of foundation than the hyperpyretic theory itself, except that we know there is a respiratory centre, while we do not know of the existence of a heat-regulation centre.

This congestion of the lung is often so intense that a portion of its tissue is deprived of air and it is frequently described as hypostatic pneumonia. Although we find genuine croupous or catarrhal pneumonia occasionally as a complication of acute rheumatism, this condition is something quite apart from inflammation, though doubtless the congestion of intense degree has many times been mistaken for the former. The state of the lung may be defined as one of splenization, for along with the intense congestion which lessens the air-space in the vesicles there is also a bloody or serous effusion, non-coagulable in character, which completes the deaëration of the lung-tissue ; and thus, without collapse and without pneumonic consolidation, the lung becomes nearly or quite solid ; the size of the organ is often considerably reduced, thus furnishing evidence of the failure of respiration.

It is by no means all of these cases which furnish evidence of failure of respiration in connection with hyperpyrexia. In some the lungs are not found thus congested, or at least not to a high degree.

In those cases where the evidence of failure of respiration is absent, it is generally noted that both sides of the heart are relaxed and distended with blood and not merely the right side. In these cases, therefore, it would seem that the nervous failure, if such is really present, affects both the respiratory and the cardiac centres and myocardiac ganglia. The death in these cases is a more rapid one, and whether from this cause or from the changes which take place in the blood coincidently, this fluid is found uncoagulated.

The heart shows evidences of failure—evidences partly derived from its relaxed condition, which may be supposed to be nervous in character, and partly from microscopic examination showing granular and other degeneration of its muscular fibres.

The *spleen* is enlarged, smoother looking, and often very much softened, so as to be nearly diffluent. If pre-existing cardiac valvular disease with vegetations is present, areas of infarction are found in its substance, although the characteristic appearances of this condition tend to become lost in the softening which the organ suffers.

The *kidneys* present the characteristic changes of cloudy swelling of

their tissue. These organs are enlarged, often intensely congested, and the microscope shows the renal epithelium swollen and cloudy, although the granular condition is not as marked as in the inflammatory changes to which these cells are subject. The appearances of the kidneys are of course modified according to the existence or absence of prior chronic changes or present inflammatory disease.

The *liver* shows no characteristic changes, except such as those already given for the other organs and which are common to all diseases attended with prolonged or excessive high temperature. The state of congestion is in this organ, as in all the other abdominal viscera, especially influenced by the mode of death. The gradual failing of the respiration renders these organs very full of blood, and this fulness is solely the result of the mode of death, and is therefore not explanatory of any save the terminal symptoms of these cases.

In addition to these alterations of the internal viscera, the general muscular system in these cases of hyperpyrexia is nearly always found to exhibit changes. The muscular fibres have been described as showing an alteration very similar in appearance to waxy degeneration. This change has been more frequently described in cases of rheumatism with excessive temperature, but it is certainly not absent in certain cases of acute rheumatism in which the fever-heat has not exceeded the usual limit. In typical cases of rheumatism alterations of the muscular fibres have not been described as having any distinctive characteristics.

In cases of hyperpyrexia, post mortem decomposition is often developed with marked rapidity.

Cardiac Conditions.

It is not my purpose to describe in detail all the appearances of the acute changes found in the cardiac apparatus or the conditions which result from them due to rheumatic inflammation of these parts. Both these and the symptoms and physical signs belong to works on heart disease. It will be sufficient to discuss the mode of causation and the results which follow from the various forms of cardiac inflammation, since thereby the effect of rheumatism in the production of the sum total of heart disease is rendered prominent and the seriousness of this complication of the disease is constantly held before us.

The changes which occur in the cardiac apparatus are divided for con-

sideration into three classes, though frequently coming simultaneously, and sometimes one the result of the other. They are 1, endocarditis; 2, pericarditis; 3, myocarditis.

Endocarditis.

The changes in the endocardium vary in their appearance according to the stage of the inflammation, the frequency of its repetition in successive rheumatic attacks, and according to the results from degenerative or other changes which occur in the inflammatory products.

In studying rheumatic endocarditis—and it is by far the most frequent form of this disease—it is necessary in the first place to try to arrive at some notion of the cause of this inflammation or why it is that the endocardium is affected in this manner. To do this it is necessary to remember first that it is the left side of the heart which is affected; its occurrence on the right side of the heart is of the very rarest exception. Secondly that the inflammatory changes and their products are seen to be confined to an exceedingly restricted area of the lining membrane, viz., the line of contact of the valvular leaflets.

The reasons which have been given for the involvement of the endocardium have been very various. The older metastatic notions of the disease can, I think, be at once put to one side. By others, the occurrence of the inflammation has been traced to the direct contact of the blood bathing the surface, bearing in its current the irritant rheumatic material, and the fact that the inflammation was limited to the left side of the heart, has been explained by the physiological difference in the blood before and after its passage through the lungs.

In answer to this it seems quite enough to say that the interiors as well as the surfaces of other organs find quite as intimate contact with the irritating material as does the endocardium, and so far as any physiological—or in this case pathological, since we are dealing with a foreign matter in the blood—difference is in question, it is purely speculative and no facts in support of the theory have been furnished.

To arrive at the cause, and to state it in exact terms, of the endocardial inflammation, we are quite as much at a loss as to account for the articular inflammation, but it seems from every point of view that we are justified in looking at the cause as the same, and that the cardiac inflammation is not in any sense a transference of the disease from the joints. This seems

to be clearly shown by the unquestioned occurrence of endocardial inflammation prior to the articular mischief. It would seem, therefore, that we are justified in regarding the rheumatic process as a general one, which may manifest itself by symptoms at one point of the organism at one time, and at another point of the organism at another. The rheumatic process is going on all over the organism on each occasion of an acute paroxysm, and we are no more bound to show why a cardiac complication occurs in some cases and not in others than we are bound to show why it occurs in the knee-joints in a given case and not in the elbows. I think, however, though not logically called upon to do so, that we can in certain instances show the reason of the cardiac complication, and perhaps also show why one joint is more frequently affected than another.

The point which I wish to insist upon is, that during the course of the rheumatic process the whole body is affected, but through certain accidental circumstances only certain parts are influenced, or that certain results are brought about in them which give rise to the symptoms that point out what we speak of as a rheumatic complication.

I would suggest, in confirmation of this view, what we see in any large series of rheumatic cases. In some cases, which apparently differ in no respect from others, we see the occurrence of skin eruptions, in others nodosities form, in others rheumatic œdema, and in all of these we see but different manifestations of the rheumatic process affecting the dermic or subcutaneous connective tissues.

While the process is thus a general one, and probably affects all parts of the organism, one sort of tissue seems to be, according to the history of the rheumatic symptoms, much more generally, if we are not able to say solely, affected than the others. This tissue is the connective-tissue group. It is usually said that the fibrous tissues are the ones affected chiefly, but I think that the more general statement is better calculated to include all the facts which are witnessed. The connective-tissue group is inclusive of all the fibrous tissues, and includes all those tissues and organs which make up the intermediary nutritive apparatus.

This group, therefore, comprises all parts of the organism which function to move the body as a whole, which move individually, and are especially influenced by pressure in general, as well as in local movements. It includes, therefore, all the general locomotive apparatus, the circulatory organs, and the interstitial connective tissues, especially such as form the boundaries of the great serous cavities. It excludes the epi-

thelial-clad surfaces and the epithelioid organs, commonly spoken of as the solid viscera ; and probably also excludes the greater part of the connective tissues, so called, forming the internal skeleton or framework on which rest the epithelioid elements of these organs.

It will be seen that the rheumatic complications manifest themselves almost entirely in this tissue group. And it would seem that the frequency of the rheumatic manifestations in one part rather than in another was in some degree proportional to the normal functional activity or an activity brought about by pathological conditions.

I therefore would affirm the view that in every rheumatic attack, the more acute and the more profound the affection the more markedly does it occur that the cardiac tissues—and of course with them all the other tissues of the connective-tissue group—are affected by the blood-changes which the rheumatic process is capable of bringing about. This statement does not carry with it the affirmation that in every case of acute rheumatism we must necessarily have cardiac lesions. The lesions in the heart come as do those in any single one of the many joints, probably as the result of functional activity having been greater in one articulation than another during the state of health, or in the case of the heart or other internal organ the functional activity has been increased by pathological conditions.

We can hardly escape the conclusion that in a general disease like rheumatism every part of the body is affected by the blood-changes, and that the special localizations or lesions which result from this general condition are predisposed to such localizations through the greater activity of the circulation, induced by an increased function, normal or pathological. We are not by any means able to point our finger directly to the kind or the occasion of such increased function, but this is probably the nearest we are able to arrive at an explanation of the phenomena witnessed and it appears withal satisfactory. The joints most commonly affected are those which in any given individual seem to be, from the nature of their occupation, the most used. And in the case of the heart-valve, how frequent it is to find, after any hurrying of the circulation, caused by exertion or excitement, a valvular murmur developed at the mitral orifice.

In accordance with these views we are able to indicate the reasonableness of the occurrence of endocardial changes of the left side of the heart, for it is this side which does the most work, and consequently its valves have a greater strain to bear.

That the acute endocardial changes are limited to such an exceedingly restricted area of the valve likewise finds a ready explanation. No one could possibly suppose that in a general disease like rheumatism the inflammation of the valvular tissue would be developed only at the line of contact of the opposed leaflets, an area so restricted that unless the changes are well advanced they may wholly escape detection.

We must suppose that the whole leaflet is affected by the rheumatic process and that for some reason the roughening which we accept as evidence of an inflammation is brought about by some accidental circumstance which does not influence the whole leaflet. This circumstance we find in the fact that the swollen, softened valve beats against its opposing fellow only along the line of contact where we find it roughened. The softened tissues hammer themselves against each other at, it may be, double the normal frequency, and are pressed upon in closing the valvular orifice by blood at more than the normal tension, and thus with great frequency become roughened along the points where they meet. Once roughened they gather up the fibrin from the blood, more highly charged with this ingredient than normally. And when this has occurred the condition is arrived at which notifies the ear placed over the heart that a cardiac lesion has occurred.

I believe that in all cases when the rheumatic process is developed with sufficient profoundness to be distinctly recognized, the cardiac tissues and especially the valvular apparatus is affected to a greater or less degree by swelling and consequent softening, which in these non-vascular tissues we hold to be expressive of inflammation. A valve under these circumstances does not necessarily become roughened, or even if roughened may not collect fibrin on its surface, owing to a less fibrinous condition of the blood, but we can readily see how a valve may thus become damaged if any excitement of the circulation takes place, and can also understand that some valves are more resistant and less likely to be roughened than others.

If, then, we take into consideration all these facts—that some valves are less softened, that less fibrin is present, and that some are more resistant than others, and also that in some cases a given quantity of rheumatic material in the blood produces more softening of the valvular tissues in some cases than in others—we have I think a more reasonable explanation why some cases are affected by valvular lesions when others of equal severity escape, than by supposing, as is generally done, that in one case the heart is affected by the rheumatic process and in the other is wholly

unaffected. Its tissues are affected in all cases, and for the reasons above given lesions manifest themselves in one case and not in another.

This view will help us to explain many cases of valvular disease of the heart, both those which can be recognized clinically, and many more also which we know of for the first time post-mortem. If we can believe that the delicate valvular tissues are affected in all cases of rheumatism, but in only the minority give us physical signs during or shortly after the acute paroxysm, we shall be able to explain those other cases of valvular alterations, many of them of very slight character, above referred to, found in persons who have suffered rheumatism but who during the acute or subacute conditions furnish no evidence of cardiac valvular disease. In such cases it is easy to suppose that the rheumatic process affected the valves, but only after a long period of evolution, that the rheumatic inflammation brought about valvular changes sufficient in degree to give rise to physical signs or symptoms, or the effects remaining quiescent merely show the alterations which we recognize at the post-mortem.

Histological Process in Endocarditis.

Minute examination of a cardiac valve in acute endocarditis gives us the following appearances, provided the valve is in a normal state prior to the inflammation. With the naked eye an injection of the capillaries cannot be made out, since these vessels are by far too scanty and widely separated in the tissue to show this appearance, common to all inflammatory conditions. Microscopically, however, it can be seen that they do participate in the change, since along their course nuclei are seen in increased numbers and those of the vessel-walls swell in dimensions. The presence of increased blood-supply and a condition of injection can be made out when the endocardial lining of the ventricle shares in the inflammatory condition, but this appearance is never conspicuous, because the reddish network has for a background the muscular tissue of nearly the same color.

I have seen a very slight pinkish hue in an inflamed leaflet, but this color can never be accepted as an evidence of inflammation, because of the liability of valves to be stained red by the blood-coloring matters. There seems to be less likelihood of this occurrence in acute rheumatism, however, than in most other acute febrile diseases, since in the former the condition of the blood is not such as to produce staining.

The absence of a capillary supply to the valves has caused some observers to deny the occurrence of inflammation in their tissue, but quite unwarrantably, since if a vascular nutrition is maintained surely a similar sort of inflammation can find place.

The increased blood-supply causes a swelling of the valvular tissue by the presence of the serous fluid in its meshes. This swelling loosens up the meshwork of the fibrous tissue and disassociates its fibres. The interstices of the tissue are filled not only with serum but with cellular elements, both those of a wandering character and those derived from the proliferation of pre-existing cellular elements. The swollen valve is thus rendered not only opaque, but also becomes softened. It is well to note just here, that the histological evidence is in favor—nay, distinctly shows that this inflammation is, so far as the valve is concerned, an internal one, not external. The change commences in its deeper parts, not on the surface, and therefore it is caused by alteration in the circulation of the part, and not by the blood flowing past the inflaming area.

The softening of the tissue and the cellular proliferation quickly affects the superfical or peripheral layers. The valve, thus thoroughly softened, shows unequal thickening, or prominences more marked at some parts than others, corresponding to and caused by aggregations of the fluid or cellular elements. The valve, though showing low elevations at points, is until now smooth, but from the softened condition of its tissues is liable at any moment to become roughened by abrasion of its surface along the line of contact of the opposing segments of the valves, or, if the softening of its tissue is more thorough-going, by a laceration or perforation of the leaflets.

This roughening when only a superficial abrasion, is not sufficient in itself to give rise to a valvular murmur. The perforation or laceration of the valve, a very much more rare event, does of course cause murmurs, which are rapidly produced and are usually harsh in character, or even double in the time of their production. I say that the superficial abrasion of itself does not produce a murmur, and the same is apparently true of the unequal elevations or inequalities of the valve even when they occur at the line of contact. Self-evidently, microscopic abrasion does not present a surface sufficiently uneven to give rise to a sound by the blood passing over it, and the inequalities, even those at the line of contact, since they are in their early stages so soft in character, do not prevent the complete closure of the valvular orifice, and hence, as they allow no regur-

gitation, give rise to no murmur. The soft tissue, although uneven, moulds itself to meet closely the opposing leaflet, and manifestly it is not rough enough to cause a sound as the direct current of blood flows over it. These inequalities, at first soft in character, if they ever produce murmurs must be very large in size, and we rarely ever see such and have little reason to believe they ever exist. Later they may become firmer and, rendering the valve stiffer than normal, prevent a complete closure, and thus allow of regurgitation.

These softened valves do, however, change the cardiac valvular sounds from the normal, and these sounds, so far as they are due to the clashing of the leaflets, are rendered softer, less distinct than normal. This change is often spoken of as muffling, and is due to impact of the softened leaflet as compared to that of the normal firm fibrous tissue. The alteration of sound is frequently noticed during the course of acute rheumatism, even when no distinct murmurs occur, and I have thought that it was present in cases of other acute febrile diseases, in all of which parenchymatous changes pervade the organs. Muffling is, however, produced by other causes than that here alluded to.

The next step in the rheumatic endocardial inflammation must, I think, be regarded as the principal, at all events the most frequently occurring one in the production of acute cardiac murmurs. It is the deposit of fibrin on the surface of the valves. We have seen that the very slight roughening of these structures, taking place almost if not solely along their line of contact, is not sufficient to produce a murmur, but this roughness is especially favorable, nay essential as a starting-point for the adhesion of the fibrin. The fibrinous deposit does not form on the smooth endocardial surface ; roughness is essential to form its clinging spot. The roughening, though not evident to the unaided senses, and though only microscopic in character, almost necessarily, in the hyperinotic condition of the rheumatic blood, begins a fibrinous accretion. The fibrinous deposits are the vegetations of acute endocarditis. These vegetations form the warty or beaded excrescences on the valves along their line of junction, and at all other parts of the surface where roughening takes place, though this latter rarely occurs.

This fibrinous deposit is firm, or becomes firm in character very rapidly, and just so far stiffens the border or line of contact of the valve. It cannot be said in this early stage of the process to render the valve rigid, still, after the fibrin is deposited, the surface of the structure is no longer soft

and easily conformable to other inequalities presented by the opposing leaflet. The closure of the orifice at which they form is incomplete by just the number and size of the vegetations present. After the fibrinous deposits take place, the opposing surfaces of the leaflets no longer meet as do the soft fleshy parts of one's two hands when pressed together, but like the knuckles when opposed to each other. The interstices between the opposed rounded prominences of the vegetations allow of a regurgitation of blood, and a soft, not very loud murmur is heard at the point of greatest intensity, the sound corresponding to the valve at which the change is present. Rarely are the vegetations sufficiently large to produce a sound by the blood flowing over them in its onward course; the murmur is usually a regurgitant one.

These vegetations form a much more conspicuous feature in endocardial inflammation than the changes in the valve which are the direct cause of their existence, but even the deposits themselves require a careful scrutiny to detect their presence, and unless one has a familiar acquaintance with the normal structure, the inflammatory changes in the valve may be readily overlooked. The vegetations on the valve serve to direct attention to the softening of its tissue and to indicate that the opacity of its tissue is due to a recent active condition, and is not the result of past changes.

The vegetations are pretty firmly attached to the valve, and are usually not easily brushed away, though they may be obscured or covered over by clots even of a fibrinous character, though usually soft, which have formed in or extended through the valvular opening during the death agony, or by those of post-mortem formation. These clots when washed away or carefully removed reveal the vegetations beneath them seated on the surface of the valve.

Taking into consideration the cause, the character, and the circumstances of the formation of the fibrinous deposits, and the softened state of the valve and the abrasion which its surface has suffered, I do not think we can regard the vegetations in acute endocarditis as other than protective in their effect, so far as concerns the valve itself. The results which follow from these deposits to the valve, to the heart, and to other organs of the body, as well as the dangers which they bring with them, require separate consideration.

Let us consider their immediate effect, their office, so to speak, in relation to the valve. The softened valves are abraded by pounding them-

selves against each other at a higher rate of speed than normal. Their roughened surfaces collect fibrin which soon becomes hard, firmer at least than the softened valvular tissue. Could any device be found better able to shield the inflamed valve and prevent laceration or perforation? and we have every reason to believe that the fibrinous deposits do thus protect the valves from serious accidents, but on the other hand, their formation may, and generally is attended later on with results which are but too constantly disastrous.

Embolism.

Among the earlier occurring disastrous results, we cannot enumerate embolism as among the more common, although when it does occur the embolic masses give rise to the most striking symptoms. Embolism is much more frequent during the inter-rheumatic periods, or from a second attack of endocardial inflammation taking place in a previously deformed valve, and this condition will be discussed under chronic endocarditis.

Cardiac Murmur.

The immediate clinical effect of the formation of the vegetations is the production of a cardiac valvular murmur, but, as we have seen in describing the symptoms of cardiac complications, the endocarditis and the valvular insufficiency come on often imperceptibly, and are known by the physical signs rather than from any change in the general conditions of the patient. The occurrence of the murmur, and therefore of the endocarditis, is almost without perceptible effect on the heart as a circulatory organ, and almost equally so on the course of the acute affection.

We must look, therefore, to the morbid histological process taking place in the valve to learn the cause of the later-coming disastrous results. We have seen an inflammatory process going on in the fibrous tissue of the valve, attended with the proliferation of cellular and other inflammatory products, and that this process finally extended to the superficial layers of this structure. We cannot but believe that an exudation of these products takes place on the surface of the valve, yet except under certain conditions, to be spoken of later, we never see these materials on the surface.

They are washed away by the blood-current as soon as they are free from the tissue. They are derived from the blood and return to it, becoming mingled with its mass and unrecognizable in it. They seem to give rise to no symptoms or changes in tissue which have as yet been clearly pointed out. Possibly, however, they may in certain cases give rise to those multiple cutaneous emboli which are occasionally seen occurring in rare cases before we recognize the existence of cardiac valvular murmurs. It may be to this condition of affairs that Hueter refers in expressing his views of the pathology of rheumatism.

When, however, the vegetations form on the border of the valve—and it is at this part that the evidences of inflammation are always the most marked, since the mechanical irritation is here superadded to the rheumatic process—the inflammatory exudation is retained under the fibrinous crust, and the products tend to increase the swelling and thickness of the valve at this part. A vertical section made through the leaflet, including its free border and its line of contact covered with the fibrinous deposit, shows the demarcation between the fibrin and the exudative products, and also that at this covered spot of the valvular section the exudative products are more abundant than at other parts. The distinction between the overlying fibrin and the retained inflammatory material has not been always accurately made. Microscopically the difference in their appearance is well marked, the fibrin appearing stratified, from its deposition in layers, while the products derived from the inflamed valve show as highly granular material and often with numerous nuclei or cellular elements imbedded in it. The granular condition often extends into the contiguous layers of fibrin to a slight degree, but not sufficiently to produce any confusion as to the actual boundaries of the two materials. I have described the inflammation as being confined to the part beneath the layer of fibrin, but I do not wish to convey the idea that these products are placed outside the proper tissue of the valve, forming a layer or bed of itself superimposed on the valvular tissue. It is more correct to think of them as seated within the valvular tissue, but coming to the surface and in contact with the fibrin through the abrasion of the superficial lamella of the valve. From the point of abrasion of the valve we see the inflammatory products diffusing themselves in a progressively less and less abundant quantity as we retreat from this point as a centre.

Valvular Vegetations.

However much opinion may differ as to the histological nature or origin of the vegetations found on the surface of the valve, none can exist as to the course followed by both these products and inflammatory materials deposited in the valvular tissue.

Of the vegetations, we can say with certainty that they pretty promptly, in typical cases, organize and fuse with the valve and become a part of it. In favorable cases when the inflammation subsides with the acute paroxysm, or even during its course, the fibrinous deposits smooth off the surface toward the blood-current and also contract in size, and thence the murmurs to which they gave rise progressively diminish and often rapidly so. Judged clinically, it would seem that in some cases—and apparently not a few in number—the formation of this layer of fibrin, deposited from the blood, acts, when it is small in amount and is rapidly organized, like an adhesive inflammation in stopping the friction-sounds of pleurisy. In neither case does the inflammatory process absolutely cease, though some of the untoward tendencies are dissipated by the event.

In unfavorable cases the deposition of fibrin becomes exaggerated, the inflammation does not subside, the softening and swelling of the valve continues, and above all the fibrin fails to consolidate and to become organized. Under these circumstances, so far as concerns the fibrinous deposit, portions of it may be separated and washed off in the blood-current, giving rise to infarctions. The portions which adhere to the valve may by their great size cause marked insufficiency of the orifice or form a considerable obstruction, in either case producing disturbance in the onward movement of the blood. Again, the adhering fibrin may, flapping backward and forward with movement of the valve, abrade the surrounding parts of valvular endocardium as well as such portions of that lining the cavities as are within its sweep. Thus new centres of endocardial inflammation and roughening are established, which in turn give rise to new fibrinous deposits with a collection of inflammatory material in the subjacent tissue. These latter class of changes are by far more frequent in chronic endocarditis or in acute changes occurring in a damaged valve than in primary acute inflammation even of the greatest severity, although we cannot exclude them from among the possibilities of primary acute rheumatism with endocarditis. Finally, adhesions may also take place between the fibrinous masses, and fusion of the portions of the leaflets result.

Inflammatory Products.

The course pursued by the inflammatory products in the valvular tissue varies with the severity and the duration of the inflammation, and can be classed under the usual changes which all inflammatory products undergo in the process of recovery.

1. *Inflammatory Resolution.*—We can describe a distinctive *resolution* of the inflammation. Such cases as those spoken of above as resulting favorably are here to be included, and also those in which we may believe an inflammation occurs in the valve without a valvular murmur being heard to attest its existence. The resolution—the abortion of the inflammation—may be more or less complete, and in proportion to its completeness does the inflamed part return to normal conditions. With the abortion of the inflammatory process the newly formed or emigrating cellular elements are removed. No new connective or fibrous tissue is laid down and no cicatricial thickening or contraction is brought about. The pre-existing permanent connective-cells of the valve are necessarily influenced and will invariably show alterations, but these changes may be completely effaced by time in the most favorable instances. These cases furnish, therefore, a group, in which, after a substantial inflammation has existed, a return to the normal is effected ; this normality may be complete anatomically, and in very many more cases is complete functionally, though not anatomically. How numerous such cases are we have no means of determining absolutely, partly because the criteria of the existence of the inflammation are imperfect and partly from the rarity of post-mortem inspection. But judged of clinically, it is within the experience of all, that cases of rheumatic endocarditis recover, become free from murmur, and never subsequently have cardiac symptoms. Post-mortem evidence is not wanting of cases of valvular inflammation which, dying of other disease in subsequent years, show altered valves which are functionally normal.

2. *Inflammatory Organization.*—In all cases of endocarditis in which the inflammatory materials become abundant and are present for any length of time, *organization* of the inflammatory process takes place. The new cellular elements lay the foundation of new fibres, and the original tissue suffers a hyperplastic increase. The permanency of this increased tissue, thickening the valve, is assured through the development of new capillaries or the extension and enlargement of the old vascular apparatus.

The embryonal or rounded cellular elements become by complete organization a cicatricial tissue, and added to this we have the thickening of the valve by the hyperplasia of the original tissue of the part. The contraction or deformity of the leaflets of the valve depends primarily on the development of the new elements, while its bulk may be augmented through simple increase of the old tissues, without cicatricial retraction manifesting itself; of course the cicatricial tissues themselves still further augment the mere bulk of any affected valve.

The changes in form which the valvular orifice and the leaflets of valve undergo depend in great degree on the position in the valvular tissue in which the inflammatory cicatricial material is placed, and on the subsequent changes, constructive or degenerative, which the tissue suffers. The forms which the orifice and leaflets assume under these influences are legion, and the actual changes, beyond the mere facts ascertained by the physical signs or symptoms, of valvular insufficiency cannot be predicted clinically. Their description belongs to the study of cardiac disease and the work of the pathological anatomist.

Chronic Endocarditis.

Besides the acute endocardial inflammation and the changes which progressively result from it, there occurs an inflammation which can only be described as *chronic* throughout its course, which is likewise very potent in altering the forms of the valves and entailing the mischievous consequences of cardiac valvular disease. It, like the acute form, is by far more frequent in the valves, though its frequency at other parts of the endocardium very far exceeds the acute manifestation at these parts.

The chronic endocarditis is described as a process essentially inflammatory, though wanting in certain steps and chronic throughout its whole course. It has its analogue in the chronic endo-arteritis or atheroma with which we are so familiar. When describing a process as chronic, a certain amount of doubt must rest on its history, since by no possibility can we see its early stages, and this is especially true of chronic endocarditis. We know of the possibility of its commencement by an acute process so slight in its inception that from the vague criteria by which we judge of its occurrence it passed unrecognized. Then, too, the distinction between chronic and acute is one of quantity and rapidity rather than of duration. In speaking of chronic endocarditis we must remember that the acute

process may, as the result of a "slowing up" in its rate of progress and certain transformation of the products, present us with the essential conditions to which we give the title of chronic endocarditis.

The induration and the deposit of lime salts are to be looked upon as characteristic features of the chronic process, but these are not wanting in the transformations or retrograde degenerations which are exhibited in terminal stages of inflammations which we know to have been acute during a part of their course. Our knowledge of chronic endocarditis succeeding the acute form as the result of acute rheumatism is, however, pretty complete. The thickened, the contracted, and the deformed valves nearly always show evidences of chronic inflammation, if, in fact, their condition is not entirely produced through this slow process succeeding an acute disease, and the valves in this state are peculiarly liable to exhibit acute inflammation on the recurrence of acute or subacute paroxysms of rheumatism.

The insufficiency of a valvular orifice—the obstruction or regurgitation of the blood—is not alone determined by the altered form of the leaflet, but is conditioned also by the fibrinous deposits which are more or less continuously forming on the roughened surfaces; a valve which, although thickened or stiffened, is still capable of completely guarding the orifice is rendered insufficient through a collection of vegetations on its surface.

Pericarditis.

The appearances presented by this serous membrane when inflamed in the course of an acute rheumatism show no substantial difference from those produced by inflammations owning other causes. It has been thought by many observers that the fibrinous character of the lymph is more marked in the rheumatic cases than in others, and this observation is to a certain extent true, whatever may be its explanation. But certainly a considerable number of cases are seen of what may be considered as idiopathic pericarditis, in which the lymph is quite as abundant as in the most marked rheumatic cases. In the Bright's disease, or uræmic form of the inflammation, the lymph is much less abundant than in the rheumatic form, but the explanation of this difference rests on the shorter duration of the process in the former than the latter. The pericarditis of contracted kidney is usually seen as one of the terminal phenomena of that disease, and lasting but a short time just previous to death, and hence less likely,

other things being equal, to develop large quantities of the inflammatory products.

The rheumatic complication of this malady frequently terminates in general or partial adhesions, and it has been thought that many of the white patches seen on the surface of the heart or the sac walls are the result of localized inflammatory changes of short duration in the course of rheumatic attacks. These latter are certainly seen with great frequency in patients who have suffered from rheumatism.

Acute and Chronic Myocarditis.

Inflammatory conditions of the muscular substance of the heart are nearly always seen accompanying inflammation of both the lining membrane and the pericardium. It is invariably present to a greater or less extent in the latter, but is not common in a noticeable degree in the former, except when the lining of the cavities is involved in the disease; when endocarditis is centred on the valvular tissue naturally we would not expect to find a coincident inflammation of the muscular tissue.

Myocarditis then, as we see it in rheumatism, is generally the result of an extension of the inflammation from the lining or the covering of the heart. Of its appearances or symptoms as a primary disease of the cardiac muscle we know almost nothing. In fact, if we transfer the phenomena seen in muscular rheumatism to a similar affection in the heart, we can readily believe that even the initial period of the disease is incompatible with life.

A state of hyperæmia of the tissue, and even some slight degree of cloudy swelling of the muscular fibres, has been described in those rapidly fatal cases of hyperpyrexia, and these conditions have been pointed to as instances of acute myocarditis. The irritability of the heart and the pain and some other symptoms seen in certain cases and not in others of endocarditis, and seen in pericarditis, have been explained in a similar manner. The palpitations which occur unconnected with the usual inflammations of the heart, likewise have been thought to be due to similar conditions seen in the peripheral muscles, though not necessarily in either case regarded as inflammatory in character. But of any of these states, we have little or no knowledge from post-mortem evidence to guide us to a correct conclusion.

Acute and chronic changes of the muscular tissue are seen in both

acute and chronic endo- and peri-carditis. The inflammation of the surfaces extends to the intermuscular fibrillar connective tissue in which its effects are principally exhibited. In the acute condition of the endocardial form of the disease the earlier stages correspond with acute endocarditis, but with its immediate effects on the muscular tissue in this situation we know little more than the results. In the favorable cases beneath the thickened endocardium of the ventricular surface we find the subendocardial tissues thickened, and if this condition extends far below the surface the muscular fibres are missing. In other rare cases a process which can only be described as *ulceration* occurs. A true melting down of the muscular fibres takes place, and the surrounding tissues are found infiltrated with inflammatory products and purulent matter. In other somewhat similar cases, instead of the ulcerative excavation, a large fungous outgrowth results from the deposits of fibrin, but the subendocardial tissue and the muscular fibres show the effects of the inflammatory process.

Another effect of myocarditis of rheumatic origin is the indurative change which is not infrequently exhibited. This change is certainly a chronic one in its course, and is found as a diffuse and also as a local process. It occurs as the result of the extension to the muscular tissue of both endo- and peri-cardial inflammation, though it is also found with any exhibition of the effects of either disease.

CHAPTER XV.

DIAGNOSIS AND PROGNOSIS.

Diagnosis: Pyæmia; Gonorrhœal Rheumatism; Acute Rheumatoid Arthritis; Acute Gout; Pain; Age: Duration of Attack—Prognosis—Death from Violence of the Fever; Death from Cardiac or other Visceral Complications; Disability from Joint Changes; Liability to Second or Recurring Attacks, or to Chronic Rheumatism—Predisposition to the Disease.

Diagnosis.

The diagnosis of the nature of a well-developed attack of rheumatism or rheumatic fever presents but little or no difficulty, and there are few maladies with which it can be confounded. The existence of fever of greater or less degree, with pain localized in or about one or more of the larger joints, with swelling, redness, and tenderness of these articulations, taken in connection with the history of the patient, can hardly fail to indicate to every one the occurrence of a rheumatic attack.

In the subacute and mild cases of rheumatism, and those in which the manifestations of the disease are limited to particular organs or localities, the recognition of the disease often presents great difficulties, which, at the beginning of such attacks, baffle diagnosis. In fact, it is just such cases which, from the absence of a certain means of recognition, are the cause of much of the confusion in relation to the cause, pathology, and treatment of the rheumatic affections in general, and compels, perhaps much less at present than formerly, the classification under one head of many dissimilar diseases, or at least diseases which, having in common the phenomena of pain or the symptoms, owe their occurrence to quite unlike causes or morbific agents. In respect to chronic rheumatism—of whose differentiation from other chronic muscular and articular affections more in detail will be given subsequently—this confusion is perhaps even greater than in cases of the milder forms of the acute type. As has already been remarked, the dissimilarity of the chronic from the acute type

is so great that many have been led to disassociate the two forms of disease, as owning different pathological causes.

Speaking first of the diseases which may possibly be confounded with acute rheumatism or rheumatic fever, there are to be mentioned, pyæmia, gonorrhœal rheumatism, acute rheumatoid arthritis, acute gout, acute synovitis, puerperal synovitis, glanders, milk leg, trichinosis.

Of these, however, there are only a few in which even the most superficial observer would overlook the manifest distinguishing points of difference. The three diseases, first mentioned above, which belong to the arthritic group, will be spoken of later, and their diagnostic features given. An acute synovitis, due to prolonged exposure to cold or to traumatism, may resemble acute articular rheumatism in the phenomena connected with the joint, the tumefaction and tenderness being the same, but in all the other symptoms, except perhaps fever, the two diseases widely differ. In trichinosis, the confusion between severe acute muscular rheumatism and the muscular pains and irritation due to the invasion of the parasite, might possibly exist at the beginning, but the subsequent symptoms belonging respectively to the two diseases would soon settle the question.

Milk leg is easily to be distinguished from the history of the case, as it occurs usually in pregnant women, or shortly after delivery, or after some continued fever, and a puerperal synovitis may be distinguished by the same means.

Pyæmia.—Although pyæmia has an irregular febrile type, like acute rheumatism, the presence of an external injury, or more rarely of internal lesions, at once points out the difference. In addition, the chills in pyæmia are usually much more conspicuous, severe, and frequently repeated, at least this is so by the time the disease has advanced to a stage when many joints are involved. In other cases, where few or none of the articulations are involved, no difficulty arises in distinguishing the malady from one of rheumatism. The non-acid character of pyæmic perspiration will almost invariably differentiate the two conditions. Death, preceded by typhoidal conditions, rapidly solves all doubts, which can exist only at the very onset of a pyæmic disease.

Gonorrhœal Rheumatism.—In gonorrhœal rheumatism, the presence of the specific discharge will almost certainly lead to a correct differentiation of the conditions. Although usually the course of this form of rheumatism alone will enable us to reach a correct conclusion, yet without a positive history of gonorrhœa, we may for a time remain baffled. And it is sur-

prising how often it is nearly or quite impossible to obtain a correct history. I have known men who, apparently with all sincerity, were unaware of their condition until the actual voiding of drops of purulent matter from their urethra demonstrated to them the existence of a specific disease—men who, without a particle of desire to conceal their condition, recognized the meaning of the urethritis from previous personal acquaintance with its symptoms, but could with difficulty recall the occasion of their exposure, which, not unfrequently, had taken place during alcoholic intoxication. In women, the presence of a vaginal discharge which to them was a condition not unfrequent or was acquired unknowingly, and had no significance, can oftentimes only be made certain by ocular inspection.

Gonorrhœal arthritis, as will be shown later, is usually attended with less fever; fewer joints, perhaps only one, are involved, and there is less tendency for the inflammation to shift from joint to joint.

Acute Rheumatoid Arthritis.—Acute rheumatoid arthritis is, in my experience, the most difficult condition to distinguish from acute rheumatism. In fact, except certain general features which may, in mild cases of rheumatism, be so modified from the typical standard as no longer to form distinguishing characteristics, there are only two things which positively differentiate the two diseases. First, its longer continuance fixed in the joints originally involved; secondly, the great tendency which the rheumatoid disease has to permanently damage the articulation. Nearly all the diagnostic marks of these two diseases which are usually given may occur, as exceptions, in either malady, saving the two above mentioned. The usual features of rheumatoid arthritis—its more frequent occurrence in women in debilitated conditions, uterine or menstrual, its affecting more frequently the small joints—will be spoken of more particularly under that disease.

Acute Gout.—Acute gout is much rarer in this country than rheumatism; it occurs in middle age; it affects the small joints, more frequently the great toe; its local pain is more intense, the redness greater, and the skin is dry and subsequently apt to desquamate. These and other features, to be given under gout, generally will conduct us to a correct conclusion.

The patient attacked with acute rheumatism generally gives the history of exposure to wet or dampness, or of muscular exertion with perspiration and sudden cooling of the skin; others speak of nervous shock or

exhaustion; and still others—perhaps a much larger number than is commonly thought—in whom the disease develops without apparently any antecedent cause of this nature, and who have perhaps been frequently exposed to cold or even constantly to dampness without untoward results.

How much value is to be placed on such antecedent circumstances has already been indicated; we must, however, always be prepared to meet cases of rheumatism of all forms and grades in which no such account of its development is given us, partly no doubt from forgetfulness of the patient, and also partly because of the absence of such events.

Pain.—Unquestionably, in the vast majority of cases, the first symptom noted by the patient is pain; in a few cases fever precedes the pain. The pain is rapidly followed by swelling, redness, and heat. The large joints are principally affected and begin to suffer first. The temperature rises very soon to 102° or 103° Fahr., and remains at about this point; variations occur according to the severity of the attack at evening, and with the onset of complications and the involvement of new articulations. The pain and stiffness usually remain after the temperature has subsided to the normal.

The peculiar character of the swelling and inflammation of the joints, taken in connection with the tendency to shift about from one joint to another, form eminently distinguishing features in rheumatism which usually will prevent confounding it with other diseases.

In rheumatism very rarely are the cerebral functions interfered with, and never is an attack accompanied with the hebetude or the delirious excitement of other febrile diseases of equal severity or continuance, except in cases where hyperpyrexia supervenes. The only exceptions to this rule are those cases of cerebral rheumatism, or in others in consequence of a complication involving some one or more of the internal viscera. Perhaps, also, there may be mentioned in this connection rare cases of rheumatic involvement of the scalp, in which from the intense pain of its swollen fibrous tissues maniacal excitement occurs; it would seem that the symptom is due to the intense suffering rather than, as in other cases, to changes of the cerebral functions directly.

Age.—The age of the patient is of use in determining the nature of the disease. Manifestly, however, this consideration is of comparatively little value at the outset; too many cases of acute rheumatism occur at both extremes of life for the nature of an individual case to be determined by the age of the patient. Acute attacks of rheumatism are, however, more

rare in childhood than in those past adult life, so that in one of tender years exhibiting some of the usual rheumatic symptoms, doubts would more strongly hold, simply from age, than in an old person; then, too, there are many more diseases occurring in the young than the old, in which a simulation of rheumatism is more close. In respect to the chronic forms of rheumatism, old age is the period of greatest frequency, while childhood is practically exempt.

Duration of Attack.—The duration of an attack is frequently an important factor in determining its rheumatic nature. The time is, however, very variable; in the acute attack the symptoms may end in two weeks, or be prolonged to five weeks, or even longer with the complications; the milder attacks and those of lesser grade end usually much sooner, but on the other hand, seem sometimes to become indefinitely extended, and result in a chronic or almost permanent rheumatic condition. These cases of lesser grade must be carefully distinguished from rheumatoid arthritis, which will be spoken of later.

These points, taken in connection with the general symptoms of the disease, will serve to diagnose all cases of acute or subacute rheumatism.

Prognosis.

Acute rheumatism, the most painful and the most discomforting of all diseases probably, has a strikingly low rate of mortality. Of chronic rheumatism one may say, suffer and be patient, but you will never die. Would that one could.

In considering the prognosis of any particular attack, there are three points, or possibly four, to be examined: 1, death from violence of the fever; 2, death from cardiac or other visceral complications; 3, disability from joint changes; 4, the liability to second or recurring attacks or to chronic rheumatism.

Death from Violence of the Fever.—The death from acute rheumatism is, as we have said, rare, and results either from hyperpyrexia or from cardiac or other visceral complications. It is comparatively recently that the former cause has been distinctly recognized; the use of the thermometer, now nearly universal, has brought this source of danger very prominently to attention, and has likewise brought with it the use of means to combat this direct source of danger. This condition of hyperpyrexia has recently, by some writers, been claimed as the only *direct* cause of death in acute rheu-

matism. They have attempted to exclude the visceral complications, when they produce death, classing such results as indirect or due to complications rather than to usual and necessary, though exaggerated, symptoms of the disease.

This distinction is, we consider, an improper one, because the visceral phenomena which precede death—and the cardiac and cerebral are the most common, though still rare ones—do not belong to the same order as the frequently occurring valvular changes and pericardial and pleural inflammations. These latter are truly complications, and the conditions and their symptoms persist often a long time subsequent to the rheumatic attack.

Death from Cardiac or other Visceral Complications.—The cardiac and cerebral conditions which tend directly to death are of a different order, coming during the height of the disease, rapid in their development, and subsiding with the rheumatic symptoms when they do not speedily produce death. When they make their appearance, they are as much a part of disease as the joint mischief, the fever and the sweating. This cardiac phenomenon, as distinguished from the usual endocardial complication, is not well understood; its duration is short, death usually resulting so rapidly after the appearance of its symptoms that its clinical study is imperfect. Some regard it as a pure rheumatic myocarditis; others as an implication of the cardiac ganglia or as a mixture of both these conditions; others, again, as the result of an implication of the nervous centres. Perhaps, however, it is not altogether clear wherein these conditions, whether due to muscle or nervous involvements, differ from other visceral complications; perhaps they are merely complications occurring earlier in the rheumatic attack and involving parts which are more necessary to the continuance of life than cardiac valves and serous membranes. We know less, certainly, about such conditions than the other and more frequent organic changes, and in respect to prognosis of the immediate continuance of life they are still more widely distinguished from each other.

The cases of cerebral rheumatism, which are frequently fatal, are also to be viewed as the direct effects of the disease rather than instances of visceral complications; the cerebral symptoms are the effects of vitiated blood-supply or of exhaustion of the nervous centres rather than those of meningeal inflammation. Perhaps, too, certain cases of kidney failure belong to the direct rather than the indirect effects of the disease; these cases are, however, more rare than either of the others.

Of the visceral complications, the cardiac is by far the most frequent source of a fatal result, and, with the exceptions of which mention has just been made, the mischief which leads to death is usually late in producing this result. The mischief is an inflammation of the endocardium, chiefly affecting the valves of the left side of the heart, and comes as a true complication of the rheumatic attack. How frequently death results from rheumatic heart disease, implicating its valves, is a matter of very great uncertainty. Statistics of sufficient extent to be valuable are wanting to show how frequent is the occurrence of endocarditis in rheumatism; after eliminating the cases in which, after the occurrence of valvular disease, complete recovery takes place, it will be found difficult to obtain a distinct and clear history of fatal cardiac cases to show which are of rheumatic origin and which depend on other causes. Then, too, it is but imperfectly known in how many cases of those who, having suffered previously of mild attacks of rheumatism, but without cardiac symptoms, and coming under observation later with acute attacks are then found to have valvular disease, whether the cardiac lesions are due to the former rheumatic malady, or whether they acknowledge an independent cause. It is not definitely known whether valvular disease of the heart occurs in those said to have a rheumatic diathesis, independently of an acute manifestation of the joint disease.

Death from rheumatic valvular heart disease usually results at a considerable period subsequently to its primary occurrence. But a valve once altered by rheumatic endocarditis, unless the affection is but very slight, usually advances with steady, however gradual and slow, steps to a fatal termination. Many circumstances tend on the one hand to hasten, and on the other to delay, this result. One patient with severe valvular disease may postpone the event by a careful life and the avoidance of exposure to a recurrence of rheumatic attack; while another, unaware of the presence of cardiac disease, so slight are its symptoms, may by over-exertion damage the diseased valves, or by exposure and want of care bring a new attack of rheumatism with renewed endocardial inflammation.

The other internal visceral complications of rheumatism rarely if ever lead to death in this delayed manner; on the one hand because they do not possess the same tendency of gradually increasing in damaging and deranging consequences, and partly also that none of them involve organs or parts of organs so necessary to life. The pericardial inflammation is much more commonly fatal very shortly after its onset, if fatal at all; but

still it occasionally leads to death from the gradual changes effected in the relations of the heart by hindering its freedom of motion or by the damage inflicted on the heart muscle itself. Both of these changes are gradually effected, and make themselves felt after the lapse of considerable time, and they are much rarer results to meet with than the deaths from the endocardial changes. The pulmonary and pleural complications, too, are usually speedily fatal, if ever; but they possess little if any power of entailing subsequent gradually developed damaging consequences.

Disability from Joint Changes.—The third element of prognosis, viz., disability from joint changes, rarely come to consideration as a result of acute rheumatism. Any change of a permanent character said to be the result of a single uncomplicated attack of acute rheumatism, is to be viewed with the greatest suspicion. Nothing is rarer than a permanent damage as the result of rheumatic fever. The older authors recorded many such cases, which we know now to be errors of the diagnosis. The diseases with which they are connected have, with a fuller knowledge of pathology, been separated under different heads. Under the class of mild or subacute attacks many instances are still pointed out of permanent and often progressive articular changes; these likewise are probably all errors of diagnosis, or will in time come to be viewed as such; some are rheumatoid arthritis, others articular changes connected with nervous diseases affecting nutrition—the cerebral or spinal arthropathies.

Liability to Second or Recurring Attacks, or to Chronic Rheumatism.—In respect to the liability to second or recurring attacks of the disease, it is very certain that one acute attack neither shields the patient from subsequent attacks nor from the chronic manifestations of the disease. Nay, many hold that one attack directly predisposes to a second. Predisposition is, like the question of inheritance, so much a matter of opinion and can be so unsatisfactorily determined as a fact by statistics, that it behoves all who wish to arrive at the truth, rather than state opinions confidently, to be guarded in their expressions. Except on purely theoretical grounds, nothing has been advanced to show why one attack of rheumatism should predispose to a second or subsequent attacks, and if a predisposition is brought about by the first attack, it would seem that two attacks should increase such a tendency. Certainly no increasing predisposition from these circumstances has been shown, either to subsequent acute attacks or to the chronic form of the disease.

It is probable that in patients who exhibit a recurrence of attacks there

is nothing more at most than a strong original tendency to the disease, or more probably a mode of life followed and continued by them which tends to excite rheumatic disease. While it is true that a very considerable number of patients, who have suffered multiple attacks, are met with, it may be said that the sufferings endured *dispose* such who are wise to try and avoid the repetition of them. It is certain that we see very many more persons who, damaged by rheumatic valvular disease, escape second attacks of acute rheumatism, than there are of those who have recurrent acute symptoms. If one attack predisposed to subsequent ones, we should expect such attack to occur with especial frequency in persons enfeebled by a damaged heart, although allowance, of course, must be made for the greater care which the rheumatic cardiac patient is likely to exercise for the maintenance of health.

Predisposition to the Disease.—The belief in a predisposition to the disease, and to recurrent attacks, arose from the theory of a diathesis, and has since then been held, but, as we have said, without proof of the grounds on which its truth is considered tenable.

From a purely anatomical point of view this belief gains little or no credence. In diseases of a type closely allied to inflammation, and in which, in addition to the general symptoms of fever, blood-changes, etc., there are local lesions of a marked character, a predisposition may be brought about by the changes which result from the inflammatory lesions. A residuum not infrequently exists, which serves as a favorable basis for renewed or recurrent attacks, and thereby a predisposition to the disease may truly be said to be created or increased. A bronchial inflammation, especially in attacks long continued, furnishes a striking instance of such predisposition. With acute rheumatism it is quite otherwise; the rheumatic inflammation of the joints is of the most evanescent character and leaves behind no traces of its existence. Of the changes in the blood we have no comparison to make, as we are equally ignorant of them in this disease as in all others of an acute character.

CHAPTER XVI.

TREATMENT.

Treatment of Rheumatism in the Past—General Remedies: Venesection; Cupping and Leeching; Purgatives; Diuretics; Sudorifics; Woollen Applications; Moist Applications; Revulsives and Counterirritants; Blister Treatment—Drugs: Colchicum; Guaiacum; Cinchona Bark; Quinine; Opium; Antimony and Mercury; Veratria and Aconite—Alkaline Treatment: Nitrate of Potash; Bicarbonate of Potash; Citrate of Potash; Lemon Juice; Bromide of Potash—Salicyl Treatment: Salicin; Salicylic Acid; Salicylate of Soda—Mode of Action of the Salicyl Treatment: Action of Salicin; Action of Salicylic Acid; Action of Salicylate of Soda—Anæsthetic and other Topical Applications—Diet and Regimen.

Treatment of Rheumatism in the Past.

The treatment of rheumatism has in the past, as in the present, been as equally unsatisfactory as its pathology. In different ages the remedies and means have been varied according to the prevailing notions of the causation and nature of the malady, and at all times a great deal has been done in a purely empirical or haphazard manner. At one time as a new notion of its pathology arose, the treatment was ordered in accordance with that belief; at another time—and an attempt has been made at this lately—a successful mode of treatment has introduced new notions of its pathology. This most frequent disease, most unsuccessfully treated, especially in its chronic forms, has given birth to hordes of cure-all specifics, of which many are in themselves good, but like most remedies given in ignorance of the pathology of the disease intended to be benefited, are useless if not harmful.

Our efforts for relief divide themselves for consideration into two classes, the general and the local. The general remedies and measures have varied in times past from the most active and even violent procedures or drugs to the most inactive or inert, from bleedings sufficiently large or frequently repeated as to drain the body of this fluid, from almost poison-

ous doses of antimony to the "do-nothingness" of mint-water and milk-whey. Both of these extremes, as well as the intermediate grades, have found their advocates and supporters, and during long years one after the other has found acceptance to the exclusion of the rest.

Of the local remedies, we find those whose object is to modify the morbid process in its local manifestations, and those which tend simply to the comfort and repose of the patient.

The greatest possible uncertainty exists in the judgment of the value of a remedy or course of treatment in rheumatism on account of the uncertainty of the duration of an attack. Our studies of the natural history of the disease, uninfluenced by remedies, are completely baffled, and the only conclusion we arrive at is that variability is its most striking feature. On this account, even a very large series of cases give us no positive assurance that the favorable or unfavorable results are due to the means employed to combat the disease. The statements which have been made of the value of a certain drug in preventing, for example, endocardial complications, are, for similar reasons, not to be relied upon, since we are not positive what proportion of cases that escape are naturally free from this liability.

In addition to this, our statistics are constantly vitiated, because it is well known that at certain times, or during some seasons, greater severity, or, on the other hand, greater mildness of the symptoms, or greater liability or exemption from visceral localizations and complications exists than at others. This variation comes from unknown causes, and in respect to visceral complications is not proportionate to the severity or mildness of the general symptoms. This variation finds its parallel in other acute diseases.

General Remedies.

Venesection.—The oldest recognized method of treatment of rheumatism, especially in its febrile manifestations, was venesection. This practice prevailed as early as rheumatism was recognized as a distinct disease, but it may be questioned whether it could be looked upon then as in any sense a distinctive treatment for rheumatism, since the habit was in all acute, and nearly all chronic, diseases to use the lancet. The variation was not in the remedy, but in the quantity of blood drawn.

It is probably just as erroneous to condemn or universally prohibit general or local bleeding in rheumatism, as it was, according to former

methods, to universally make use of it. It is certain that very few cases occur now which indicate its requirement, although now and again a patient is seen about whom the thought occurs, would not some of the congestions, the intense pain, or even the restless discomfort, be relieved by the abstraction of blood locally or by venesection?

The old method of bleeding, as practised by Sydenham, though afterward abandoned, and as systematized by Bouillaud, could not fail to be harmful. And it became acknowledged, finally, that the use of the lancet fails to effect even an abridgment of the acute symptoms; and as we now view the results, it is evident that the convalescence was greatly prolonged

The notion of the disease on which the treatment was based is now contested or denied, though, perhaps, too much stress has been laid on this fact in condemning this mode of treatment. Undoubtedly rheumatism is not a local inflammation, as then thought, but still some of its various phenomena are those of inflammation. It would seem more philosophical to condemn the venesection on the ground that the chief inflammatory symptoms, which it was intended principally to combat, are not of a sufficient grade of violence to require such violent or severe measures.

Venesection was practised also on account of a condition of the blood, which it was supposed that copious bloodletting would remedy. The *buffy* coat, which was noticed to be more marked in rheumatism than in most other diseases, formed a prominent reason for this practice, and it was kept up until a supposed reduction in the amount of this blood component was thought to be effected.

It is hardly necessary to discuss the therapeutic influence of venesection since this method of treatment does not correspond with our present, or in fact any rational belief of the nature of the malady. It must be insisted on in respect to this particular measure, as well as for all others tending toward depletion, that the constant result of rheumatism is to produce anæmia, and that, therefore, whatever may be the present gain in abating the acute symptoms, depletion in whatsoever manner effected may do in the end more harm than good.

Cupping and Leeching.—The local abstraction of blood, as distinguished from the general, depends on many special considerations, and its use is to be decided on other grounds than those directly connected with the rheumatic process. The question of cupping or leeching over or around a joint is one that now very rarely would be raised, at least in the

acute stage of the disease. The generally fleeting character of the articular disease would contra-indicate to every one's mind the use of so strenuous measures, and yet local depletions of this character were formerly practised. In cardiac and even pulmonary complications the use of leeching and wet cupping is frequent. The propriety of these measures must be decided by the individual case, and the settlement of the question is out of place for discussion here. Each physician will have his own views on the subject; and few will be found who will deny the service sometimes most strikingly obtained from local depletion in severe cases of pericarditis in its early stages.

The use of dry cupping to the lower vertebral region has been thought to modify the severity of the articular phenomena in the lower extremities —just the parts in which it is most severe. I can testify to its utility in some cases. Although I speak of this measure under local depletion, the effect is undoubtedly produced in a general manner through the nervous centres located in the spinal cord and connected with the affected articulations. This procedure has often a very striking effect on the muscular and nerve-pains so common in acute articular rheumatism, and also in acute sciatica.

Purgatives, diuretics, and sudorifics, and even emetics, were formerly much used in all classes of rheumatic ailments. Bleedings were never solely depended upon to subdue the inflammation or to remove the inflammatory condition of the blood, and even by the older physicians a long list of drugs were employed to expedite the cure.

Purgatives.—Of purgatives alone, or in conjunction with other remedies, magnesium and sodium sulphates have been the most employed. This plan of treatment has little to recommend it. It is probably based on wrong notions of the disease, and is withal so discomforting and painful to the patient that both the moving and exposure incident to it become sources of positive harm. Purgation is to-day the fundamental element in the treatment, especially of chronic rheumatism, by quacks and nostrum-venders. The idea of it is connected with the old humoral notions, and by it the peccant matters are supposed to be removed from the blood by exciting an excessive action of the intestinal mucous membrane. Its injurious influences are very well stated by Dr. Aitken. First, it is not necessary to the cure of the patient, and, like bleeding, tends greatly to reduce his strength and protract recovery. Second, from the nature of the complaint the patient is quite incapable of moving, and his sufferings

are aggravated, his irritability is increased, and his heart's action accelerated by the repeated shifting of his position, which is rendered necessary by the calls of nature. And, third, it necessarily gives rise to more or less exposure, which must be prejudicial to a person bathed in perspiration.

In respect to the movement of the bowels the usual direction is to secure a daily evacuation of the bowels ; but while theoretically this injunction is a correct one, practically it cannot be carried out during the acute stage of suffering. The febrile condition is in itself a considerable obstacle, and besides, the patient, by voluntary efforts, is able to resist the effects of any moderate exhibition of purgatives. It would seem best, therefore, in view of the small amount of food taken and insignificant fæcal residuum from it, to rest content and abstain from purgation until a period of quiet and comparative comfort is reached. A movement of the bowels every third or fourth day will probably suffice for the requirements of nature, and then if necessary—if the stool does not take place spontaneously, as it does in the vast majority of cases—a mild purgative is to be given, or an enema, if skilfully administered, will suffice for the purpose. During convalescence greater care must be exercised to secure a thorough daily evacuation, as this does much to restore the tone to the digestive functions, which have been in abeyance or disturbed as the result of the acute paroxysm. The compound liquorice powder of the German Pharmacopœia serves an excellent purpose, although in the selection of the drug the personal peculiarities of the patient should be respected.

Diuretics.—The use of diuretic remedies has been advocated in all grades and varieties of rheumatism, and by some observers as a specific form of treatment in rheumatic fever. It is certain that in acute rheumatism, and often in other forms of the disease, the secretion of urine is defective both in quantity and in the amount of the urinary salts, and the object of the diurectic medicines is by increasing the flow of urine to promote the general elimination, and also specially of morbid products suspected as the cause of the disease.

This plan of treatment is less to be criticised than the purgative method, and is certainly without any special deleterious effects. It is perhaps, in all cases, well to bend our efforts toward its promotion. But nothing has been shown as the result of it which can be claimed as a specific influence on the course of the disease. In fact it can be shown very conclusively that so long as the febrile condition is maintained, the secretion

of the urine remains uninfluenced by the most strenuous diuretic measures. The most efficient diuretics, under these circumstances, are those which tend most rapidly to influence, by depressing the fever, remedies which ordinarily exercise no power over the urinary secretion.

As already said, many simple diuretics, or even other remedies, which without influencing the quantity of urine change its qualities, are very useful in many cases of rheumatism. These remedies are most useful, however, in certain of the rheumatic complications, for example, the dropsies which result from the inflammations of the serous membranes, especially in their chronic stages.

Sudorifics.—The characteristic and profuse perspiration has been considered as one of the principal efforts of nature in getting rid of the morbific materials productive of the acute paroxysm of rheumatism, and this circumstance has been seized upon as an indication for treatment.

The idea, as a specific means of treatment, is based upon mistaken notions, a legacy of the humoral pathology, and the results obtained by this method show no alterations in the course, the severity, or the duration of the disease. The amount secreted by the skin, in ordinary cases, is so great it would seem that the perspiration could not be advantageously increased. The circumstances in which sudorific measures offer advantages are those in which the skin is deficient in its action, but the cases of acute rheumatism with a dry skin are exceedingly rare.

The sudorific remedies which have been employed are those which act generally, and those applied for local effects. The most common agent, the one which even now finds great acceptance, and in many cases proves itself useful in meeting a variety of indications, is Dover's powder. This remedy is especially applicable in the commencement of an attack, though by many practitioners its use is kept up throughout the acute paroxysm, and its narcotic effects are useful even when its sudorific action is not called for. Dover's powder finds frequent use in connection with other remedies, especially when the alkaline treatment is pursued.

Woollen Applications.—Another sudorific measure which has commanded, and still commands approval in all forms and grades of rheumatic disease, is the use of means for enveloping the whole body or only joints in warm clothing or wraps. This treatment has been recommended as the only measure to be adopted for acute rheumatism, or it has been tried in common with some simple diluent drinks, and the results have been warmly praised.

Its object is held to be twofold, first, in promoting cutaneous action, and secondly, in shielding the body from the effects of cold and draughts during the perspiring condition. The material recommended is woollen, especially flannel garments, and many hold to a special virtue in red flannel. The shops contain varieties of medicated red flannel for sale, many of which are positively dangerous from the presence of irritating or poisonous dyes used in their manufacture.

In acute rheumatism certainly nothing can be more discomforting to the patient than the contact of woollen raiment, and add to this the disgusting condition in which such clothing soon becomes from the abundant sweat and the necessity for changing it frequently, with a greater liability to cold, there is certainly a question of the propriety of its use. In chronic rheumatism and during convalescence this habit of dressing is generally a necessity, and the value of flannel worn next the skin in preventing rheumatism, especially for those predisposed to attacks, is well known to all.

During the presence of the articular inflammation it is a common practice to envelop the joints in raw cotton or in wool; on the other hand, the procedure is condemned by many, under the notion that by increasing the warmth and the consequent flow of blood the conditions for inflammation are augmented. The majority of patients, however, find comfort from the application, and those accustomed to rheumatic disease crave its use.

Moist Applications.—Frequently in addition to the simple envelopment of the painful parts, lotions of various characters are used. Both sedative and alkaline applications, or their combination, find many advocates, and their effect is often very salutary in allaying the pain. In making moist applications to the inflamed joints it is necessary, so great usually is the heat of the skin, to surround them with an impermeable covering, such as waxed paper, gutta-percha cloth, etc., to prevent the rapid evaporation and the necessary renewal of the application. The impermeable covering retains the moisture, increases the heat, and consequently the flow of blood to the parts thus enveloped to a much greater degree than the dry application, and owing to this fact a greater objection is made to them than to the former. It is certain that on this account the moist applications are not serviceable in all cases; many cases are not benefited by them, and they even increase the pain and discomfort of the patient. It has seemed to me—and recently I have made a pretty careful trial in

order to find the class of cases in which moist applications are useful—that only those are benefited when the intra-articular inflammation was marked, when the capsular ligament resisted the expansion of the accumulating fluid, and when the peri-articular tissues failed to swell. These are the cases in which the pains are generally the most severe. The moist and warm application served its useful purpose by softening or causing the peri-articular tissue to swell, and thus relieved the pressure of the effusion within the joint. In addition, we can believe that the narcotic—usually opiate—lotions have their ameliorating influence here as under other conditions in assuaging the pain. In respect to the alkaline fluid, though originally used for its antidotal or neutralizing action on the acid effusion supposed to be present, we know of nothing confirmative of such action, though I believe that it is often useful; for example, a strong solution of sodium carbonate, in softening the otherwise hard and tense skin, especially around the knee-joints. For this purpose and under these circumstances we find local applications to a limited degree serviceable.

Revulsives and Counter-irritants.—The application of revulsives or counter-irritants to the inflamed joints is a method which has been always more or less in vogue, from very early times even to the present. With our present knowledge of the therapeutic action of irritants applied to the skin, the use of a severe application over an inflamed rheumatic joint does not appear to be a rational procedure. The effects of such measures would seem rather to increase the tendency or the conditions favoring inflammation in the adjacent synovial membranes. The more severe the irritation of the skin, the more marked would be the effects within the joints, whereas, according to our present belief in the mechanism of trophic nerve action, the counter-irritation should be of the mild character, continuously or frequently applied in order to produce the best results. The milder applications can be applied directly over the inflamed joints, while the more severe should be made to the adjacent parts.

As a specific treatment of acute rheumatism none of these applications have met with more than a very slight approval, and by nearly all writers are spoken of in combination with other remedies of an internal character. In chronic rheumatism, however, they find a very constant use, and also in the subacute forms as well as in the late stages of an acute attack where the articular disease lingers.

Blister Treatment.—In June, 1864, Herbert Davies delivered a clinical

lecture at the London Hospital "On the Treatment of Rheumatic Fever in its Acute Stage, exclusively by free Blistering." Dr. Davies' method was not applied by him in view of the principles above spoken of, but, as he says, "believing it probable that the virus localized itself for a time in the inflamed joints, and that the intensity of the local inflammations was a measure to some extent of the amount of poison collected by a species of affinity in the parts attacked, I determined to attempt the local elimination of the materies morbi, wherever any external manifestations of its presence existed. I ordered blisters, varying in width, but of considerable size, according to the locality, to be applied around each limb, and in close proximity to the parts inflamed; and I hoped to relieve the affected joints, partly on the principle of derivation, but *mainly* and really by affording through the serous discharge from the blistered surface a ready means of exit for the animal poison. Armlets, wristlets, thighlets, and leglets, and even fingerlets, were applied near to, but not upon *every joint inflamed*, at the very *height* of the inflammatory stage when the local pains were the most severe, and the constitutional disturbance the *greatest*."

It was claimed for the blister treatment that it effected a shortening of the duration of the acute paroxysm, and especially lessened the liability to heart complication. This suggestion of Dr. Davies', and especially his manner of applying the blisters, attracted great attention, gave a new impulse to this plan of treatment, and gained many followers. The mode, however, never received general acceptance, and has since fallen into disuse. Although according to Dr. Davies' report, and those of many others, the blisters relieve the pain, quiet the pulse, and after their use the urine becomes alkaline, still nothing specific has been shown from their effects, and it is self-evident that under many other plans of treatment, and even no treatment, the same result follows in equally severe cases with equal rapidity. The severe blistering is regarded by many as barbarous, and while this ought not to preclude the use of them, if distinct advantages result, still the idea forms with many patients a bar to the application.

The production of strangury has been made an objection to the use of cantharidal blisters; but the reports of the cases show that this condition is a rare one resulting from their use, and in a disease attended with so great deficiency of urine, and also difficulty in passing it, there is great liability of misjudging the effects of such a remedy.

It must be mentioned in this connection that in the serum of the

blisters no lactic acid has been shown to be present. This acid is, however, one most difficult to obtain from any organic fluid, and the failure to obtain it in the few experiments which have been made with this object in view, cannot be quoted as positively proving its absence.

The application of blisters has always been in use in the treatment of various visceral localizations; for example, pericarditis, pleurisy, and the cerebral inflammations, etc.

Drugs.

Of the very long list of drugs which have been employed and have been discarded, or still find occasional use, a few require special mention and discussion. Many of this class were used purely empirically, others in accordance with some pathological theory.

Colchicum.—Colchicum and its preparations were held at one time to exercise a specific influence on the rheumatic process, or to promote the elimination of the rheumatic poison. This belief probably arose out of the similarity of this affection with gout, and at the time when the two diseases were imperfectly distinguished. Later the use of the remedy was mainly in connection with other drugs as an adjuvant to them, or in the promotion of some particular organic function. In small doses it has been thought to promote elimination by the kidneys; but in this way acts independently of the rheumatic condition. In large doses the remedy is regarded as useless, if not positively dangerous, in acute rheumatism. It is without influence in altering the course of the paroxysm of fever, very unlike its influence on the gouty inflammation.

The favorable reports given of its influence in acute rheumatism have arisen from the mistaken diagnosis of the two diseases, or very probably from its unquestioned effects in depressing the heart's action, and the relief of pain, etc., due to the lowering of the vascular tone. It is for this reason that colchicum is often a dangerous agent in rheumatic affections in which the heart is especially liable to be diseased.

Guaiacum.—Guaiacum is another remedy which has been recommended in acute rheumatism, but apparently with even less reason than colchicum. It has never been shown to alter the course of the disease, and during the acute symptoms usually shows less influence on the secretion than during health. This remedy finds more use in chronic conditions.

Cinchona Bark.—Cinchona bark was formerly considerably used in the

treatment of acute rheumatism. Its first employment was limited to cases of the disease exhibiting an intermittent type. But later, especially after the use of the alkaloid became common, and rendered its employment more convenient, its systematic and routine use was advised, and the remedy was given in heroic doses.

Quinine.—Quinine has never found general acceptance as a remedial agent in acute rheumatism ; but about forty years ago, especially among French writers, it was highly recommended as having a most marked effect on the course of the fever, and in allaying the articular inflammation. To produce these effects the dose must be from twenty to ninety grains, given during twenty-four hours, and in these quantities it is usual to find that both the stomach and the nervous centres are unfavorably affected. By allaying the fever it was thought to lessen the liability to cardiac complications ; but if these were present it seemed not to affect the general condition of the patient or the complication unfavorably.

The use of quinine, in both large and small doses, very soon fell under the suspicion of inducing cerebral symptoms, and was mostly abandoned even by its earnest advocates. This suspicion was probably in great degree an unjust one, and although unquestionably in this, as well as in other febrile maladies, the nervous susceptibilities, and even certain nervous phenomena are excited, it cannot be held that the graver forms of cerebral rheumatism are due to its use. The giving of quinine must always be subordinated to the effects which it causes on the side of the nervous system.

Primarily its use is directed, from the point of view of a rational pathology, by the control which it exercises through the nervous system on the febrile process, as well as on the articular disease. It cannot be shown that the drug has any specific influence in controlling the rheumatic process, and hence we are driven to accept this explanation of its action. It is therefore to be considered as an *antipyretic*, and from the valuable qualities of the drug in this respect, in so many diseases and under so many circumstances, it is deserving of investigation.

We cannot deny to it very serviceable effects in certain conditions, in what may be described as a regulating action of the nervous system, although, as alluded to above, its inopportune or excessive administration serves to exaggerate or even to develop untoward nervous phenomena. Again, it may serve well, mitigating the febrile and the articular processes in acute rheumatism, as well as in many cases of the subacute or chronic

forms of the disease; but except in its effects in this direct manner, it is inoperative against the development of the visceral complications.

In very many cases of rheumatism the tendency to remittance in its symptoms is of very common occurrence. These recurring phenomena are frequently, though probably nearly always, incorrectly set down to the effects of so-called malaria; the phenomena are commonly of a nervous character. The use of quinine is most auspicious in breaking up this so-called periodicity, and acts apparently by its regulative effects in modifying the nervous functions. In the same manner it is serviceable in warding off relapses of the diseases. There are also a number of minor symptoms of the disease for which quinine may be given, according to certain general principles furnished for its administration, apart from the presence of the rheumatic disease. One of these is the excessive sweating, in the modification of which quinine is useful.

Viewed as an antipyretic agent, quinine may be considered a very serviceable agent in the treatment of acute rheumatism, but to effectively modify the temperature it is necessary to employ often very large doses. In this respect it is less certain in its action than other remedies, or than it is itself in other diseases, and while less certain in its effects the large doses of the drug often produce perturbation of the nervous functions or of the digestive functions.

More recently quinine has been used in combination with other remedies. Garrod, in "Reynolds's System of Medicine," has proposed this modification, and no one is more entitled to be heard on this subject than this careful observer and writer. Let us quote his opinion. "During the last ten years (writing in 1867), since the publication of his paper on the treatment of rheumatism with large doses of bicarbonate of potash, the writer has made a very extensive use of the following plan, from which he thinks he has obtained more valuable results than from any other; it may be termed the quino-alkaline treatment, and is thus practised: Sulphate of quinine is ordered to be rubbed up with a solution of bicarbonate of potash, to which a little mucilage and some aromatic, as tincture of cardamoms or spirit of chloroform, is subsequently added; each ounce and half dose contains five grains of quinine and thirty grains of the potash salt, the quinine being reduced to the state of carbonate. To the adult the above dose is given every four hours, and persevered with until the joint affection and febrile disturbance have completely abated. When the quino-alkaline treatment was first made use of, a few days were allowed

for the exhibition of the alkali alone, and then the quinine was added; but of late the quinine has been given from the first; it neither increases the thirst nor the furred state of the tongue, and its influence upon the heart is to lower the pulsations, but not to weaken them, and hence, when peri- and endo-carditis are present, its employment is not contra-indicated. If cardiac complications exist, local depletion and counter-irritation may be made use of." "If desired, the citrate of potash, or some other alkaline salt with a vegetable acid may be substituted for the bicarbonate." "Although the results obtained from a large number of trials have not yet been tabulated, the writer feels assured that the above treatment is much more efficacious than the simple alkaline plan; that there is far less tendency to the occurrence of relapses, and that the patient is left in a more satisfactory condition after the cessation of the febrile disturbance."

Opium.—Opium has been used for two purposes in acute rheumatism; first, for its narcotic effects, and secondly, in combination usually with other drugs, for its diaphoretic action. Of the latter an account has already been given in speaking of Dover's powder. For its narcotic effects it is legitimate to use opium or some of its preparations under all circumstances and in every condition, when the pain, usually of the inflamed joint, but occasionally of other parts, is so severe, and so long continued, that the suffering of the patient prevents sleep or renders his condition unbearable.

In early times opium was used for its specific effects on the rheumatic inflammation. It was thought, by its calmative action on the circulation, or by other means, to tend directly to lessen the inflammatory process; but later authors, especially Cullen, condemned its use during the febrile stage of the disease.

However, its use was continued for this and other purposes, and in combination with other drugs its applications were innumerable and became almost universal. These combinations were made with the purpose of checking the over-action of many remedies, which in themselves were thought to serve a specific purpose in the treatment of the rheumatic condition. Its combination with calomel was made in order to check the action of the bowels, and, as it was said, to prevent this drug from being drained off and thus failing of its proper absorption, and its effects on the inflammatory process. Opium has also been used in connection with colchicum, quinine, digitalis, veratria, and belladonna or its alkaloid atropia.

Later, the use of opium was highly recommended by Corrigan and

others, but we fail to discover from their accounts any further effects from it than those already detailed. It served to assuage the pains of articular inflammation, leaving the patient comfortable until the subsidence of the disease in its natural course.

Antimony and Mercury.—The antimonial and mercurial remedies have been advocated for their direct effects in modifying the course of the acute paroxysm; but their use has now been almost entirely abandoned, except to meet some special indications. The former were received with favor, doubtless owing to their general depressant action, which was often carried to such extremes that no active morbid process could for the time be carried on by the overpowered vital forces. The latter were employed for their general antiphlogistic effects, and especially for the prevention as well as the treatment of cardiac complications. Their influence on the rheumatic process has not been shown to exist, and their effects in cardiac complications is based both on mistaken notions of the morbid process and on the general effects of mercurials; this is especially true in regard to endocarditis. The effect of mercury is not perceptible in preventing or arresting the inflammation, and may do harm in breaking down the collecting fibrin which shields the roughened and inflamed endocardium. A comparison of the results of mercurial treatment with those of other remedies shows no preponderance in their favor; in fact the condition of the patient during convalescence, if ptyalism has been induced, is much less favorable.

Veratria and Aconite.—Both veratria and aconite have been recommended for their favorable effects in combating the fever and for allaying the pain of the acute paroxysm, but have only met with temporary acceptance. Although they have unquestionable action in both these respects, given alone or in connection with opium, the indications which they fulfil are generally much better met by other remedies.

Alkaline Treatment.

The plan of treatment, which consists in the administration of the alkaline bases, is divided for consideration into two heads: the one, in which merely various saline materials were given, the other, in which these salts were especially intended to produce their effect by their alkaline characters. The action of these two classes of remedies however, does not differ probably very materially the one from the other. Given in the usual

manner and in ordinarily used doses, they are intended to produce alteration in blood, and hence are both directed to the same end, and their effects may be discussed together.

Nitrate of Potash.—The earliest used saline was probably nitrate of potash, and its employment arose from the common practice of giving saline mixtures in all febrile maladies, especially those of an inflammatory type. At first this salt was brought into prominence by Brocklesby, who used it in connection with venesection. The blood-letting was intended to subdue the inflammation and the fever, and the alkaline qualities were to serve the purpose of breaking down the inflammatory products and promoting their absorption. The practice was introduced more than a hundred years ago, and has been continued more or less intermitting up to the present time.

The results of the treatment have been reported by many to be very favorable, shortening the duration of the malady and favoring a rapid convalescence. Fuller and others who have employed this remedy do not agree in this view, and in quoting the results obtained by Basham and others who used the nitre treatment, criticise their reported favorable results as not confirmed by the details of their cases.

The dose of the nitre, in order to exercise any influence, must be very large. One ounce or two, or even three ounces, are given during twenty-four hours, very largely diluted either in water or in water gruel. Taken in divided doses at short intervals, it was said to produce no disturbance of the digestive function or of the abdominal organs; others, however, do not confirm the mild effects on the stomach here spoken of, and alluded to nausea, vomiting, and diarrhœa, which compel the discontinuance of the drug.

My own experience in the use of nitre is very small, and is confined to the use of much smaller doses, which seemed to be without influence on the symptoms, and which, in fact, given even in the reduced doses, quickly disagreed with the stomach, leading to the abandonment of its use.

The exact therapeutic effect or the chemical reactions of nitrate of potash are not known. It is one of the salts which under all circumstances is found in urine shortly after its ingestion.

The *alkaline treatment* proper has found its most earnest advocates in Dr. Fuller and Dr. Garrod. It took its rise from the gradual development and acknowledgment of the new view of the pathology of rheumatism, as well as a greater accuracy of the knowledge of its symptoms. It was the

answer to the demand that the acid condition of the body should be neutralized by opposing it with an alkaline base with which it could combine and thus be rendered innocuous. It was the attempt to cure disease by chemical reactions rather than by controlling vital action. It was the acceptance of the fact that lactic acid was the cause of the disease rather than that both the disease and the supposed lactic acid were the outcome of a perversion of normal action. It was an effort to treat one of the products of disease; and not the disease itself. All of these ideas led to the systematic use of alkalies, which in the hand of Fuller, Garrod, and a host of others, has constituted the chief means of combating rheumatism during a quarter of a century.

Garrod says that they were not "systematically" employed until the year 1847, when Dr. Wright published a communication on the subject. Since that time both Fuller and himself made full trials of their use, and after the publishment of their results, the use of these remedies became common. "The form in which," says Dr. Fuller, "I usually prescribe the remedies, is that of an effervescing citrate of potash or ammonia draught, with an excess of from forty to sixty grains of the bicarbonate of potash or bicarbonate of soda, to which, if the patient be strong and exhibits the usual symptoms, I usually add a drachm or more of the acetate of potash, and ten or fifteen minims of the vinum colchica." "This draught is repeated, for the first twelve or twenty-four hours, at intervals of two, three, or four hours, according to the strength of the patient and the severity of the attack." "In proportion as these symptoms of amendment manifest themselves, so is the dose of the alkalies decreased, until, after the lapse of three or four days I feel justified in commencing the administration of quinia or bark, in combination with small doses of bicarbonate of potash." The author did not confine his recommendation exclusively to the alkaline treatment: "Alkalies in many cases prove unequal to restore a healthy state of assimilation and to prevent the further formation of acid in the system," "but no certainty can be felt as to arresting the disease, without the aid of other medicines to assist in modifying the function of assimilating and to act on the various excretory organs."

Bicarbonate of Potash.—Dr. Garrod's plan of treatment consisted of "administering a dilute solution of bicarbonate of potash in about thirty-grain doses, every four hours, until the joint symptoms and febrile disturbance have completely disappeared. These doses produce no inconvenience either to the stomach or bowels; the urinary secretion is not nota-

bly increased, but its character is completely altered; and the reaction becomes either neutral or alkaline; it usually remains clear, but occasionally gives rise to a deposition of the triple phosphates. Upon the heart the alkaline bicarbonate acts as a sedative, reducing the frequency of the pulse sometimes forty-eight beats in the minute, but not causing any faintness. When the patient is fully under the alkaline treatment, the blood is distinctly altered, and the coagulation of the fibrin takes place more slowly." The author has since his first trials with the simple alkaline treatment found cause to change his method, preferring the results obtained from his modification of it, viz., the quinia-alkaline treatment, the details of which I have already given.

Other forms of the alkaline salts besides those mentioned have been employed, and still find use in the treatment of the disease. Both of the salts mentioned are combined either with a fixed mineral acid or carbonic, and these find direct entrance into the blood and are eliminated from the kidney in the same state. The alkaline carbonates are supposed to have the advantage, in addition to their effects in and on the blood, of neutralizing any acid with which they come in contact in the digestive tract. Thus they operate on the acid state of the system both in the primary process of assimilation and on the acid products which are derived from the secondary assimilative process.

Citrate of Potash.—The other forms of alkaline salts which are used are those in combination with vegetable acids. The type of these is the citrate of potash. Their character is that of neutral salts, and they are without effect, at least so far as we are acquainted, on the acidity of the mucous membranes, but their action on the blood is similar to that of the alkaline carbonates, into which they are decomposed. They appear in the urine as basic carbonates of the salt used.

The results of their action in rheumatism appears to be equally good with that of other alkalies, and indeed they appear in many cases to serve a better purpose.

Lemon Juice.—Before closing the subject of alkalies let us consider the treatment by the use of lemon juice. The connection between the two modes of treatment is a pretty close one. The difference between the administration of an alkali and an acid, at first glance, seem as great as possible; but the similarity of the two kinds of remedies is very great, especially the latter class of alkalies just discussed. Dr. S. Owen Rees, who first discussed its action (1849) and carried out the use of the rem-

edy, thought that the acid juice containing a considerable amount of oxygen by decomposition, and the blood, favored the conversion of the uric acid into urea, and perhaps carbonic acid, and thus promoted its elimination. The urine, under its action, becoming neutral or alkaline, and the excess of urates disappearing. The presence in the lemon juice of the potash salts, to a certain degree conforms its action to the alkaline mode of treatment.

The remedy is given in doses of three to eight ounces of the fresh juice at intervals during each day. It was claimed that its use moderated the heart's action and afforded relief to the articular pains. Given in these large doses, although the contrary was claimed, it certainly tends to disturb the stomach, exciting colicky pains, and often producing diarrhœa. If it removes the peculiar smell of the acid perspiration, it replaces it with the acid smell of the lemon, which hangs around the patient even a long time after the remedy is discontinued.

This treatment, from its novelty and simplicity, attracted general attention, and was for a time extensively used, but has now fallen entirely out of favor. It is oftentimes hard to carry out the treatment, both in private and in hospital work. I have found lemons particularly expensive, difficult to obtain, or lacking in juice at seasons when acute rheumatism is especially prevalent. A number of substitutes have been proposed and used for the juice of fresh lemons ; for example, lime juice and citric acid, but these preparations have not been found more serviceable than lemon juice, and have been used only when that article was not to be obtained.

The use of lemon juice, according to the common judgment and the results of subsequent experience, has not been shown to modify the course, duration, or the occurrence of complications of acute rheumatism, but it is not to be summarily dismissed from the list of agents useful in this disease. On many occasions and for different purposes it will be found applicable, and its administration may be carried out as an adjuvant to other treatment, or when a failure results with other methods, in mitigating the patient's sufferings.

Bromide of Potash.—Bromide of potash, subsequent to the introduction of the bromides into the Pharmacopœia for the treatment of other diseases, found a frequent application in cases of rheumatism. It was frequently given in place of opium for its general soothing effects, but especially in cases attended with various nervous phenomena, and also when actual rheumatic localizations developed in the nervous tissues, in which

instances it was thought that opiates tended to increase these symptoms.

The remedy is of great service in patients addicted to the use of alcohol, in whom congestion, and, perhaps on account of the rheumatic attack, the condition of alcoholic delirium is threatening to develop. In cases of simple delirium and extreme nervous agitation, occasionally seen in acute rheumatism, the effects of even a few doses of the bromide given as required suffice to calm the patient completely, allowing the disease to continue its usual course, and the patient's general condition and the excretory function is undisturbed by the exhibition of the drug.

However, no attempts were made at the time of the introduction of the bromides into medical use to confine the medication of acute rheumatism to this drug until 1865, when Da Costa, having "while treating some patients troubled with great restlessness and pain during rheumatic fever, prescribed the bromides to allay these symptoms, and finding them to act favorably, was led to study their influence on the course of the disease." The particular drug employed by him was the bromide of ammonium, although the salt of potash was also tried. His early observations extended over more than thirty cases of acute rheumatism; "all the cases were marked ones; most of them would be called severe ones." The records of these cases were published in the "Pennsylvania Hospital Reports" for 1869.

The results of this plan of treatment are certainly as favorable as shown by any other that has been proposed. The mean duration of the attacks, from the initial symptoms to full convalescence, was 22.5 days, and the group of cases include two long cases, ill and under alkaline treatment for a considerable period before admission to the hospital. The mean time under treatment in the hospital was 14.16 days. The period bears a very good comparison to that reported by others.

The influence of the remedy on the pulse showed the reduction in the number of beats was promptly affected, "rendered decidedly slower, and often also loses in force." On the skin "its influence was not very decided;" "but, in so far as it seemed to have any, it appeared to promote perspiration. It certainly seemed to do so in the cases associated with a dryish skin." The temperature does not appear to be markedly influenced by the remedy; the thermal curve usually gradually returning to the normal level with the subsidence of the joint disease. The influence on the respirations was not particularly studied, since this function, owing to pain

or to the constrained position in which the patients are apt to lie, prevents any accurate appreciation of whether its frequency is altered by the malady or by the drug. "On the pain the influence is very striking. Not only is the local distress often stated to be ameliorated, but the general restlessness and uneasiness are decidedly benefited." The remedy gave rise to no gastric symptoms, nor did it constipate. In a few, indeed, it produced slight diarrhœa. The urine remained acid during the administration of the remedy, but its quantity was decidedly increased. The bromide made its appearance in the urine, in very small proportion or none at all, and "certainly out of all proportion to the amount swallowed."

In respect to cardiac complications the report shows "that not a single one had endocarditis originating under treatment; in not a single one was it met with . . . . in which it had not existed at the admission of the patient into the hospital." Of pericarditis "four cases are the only ones among the thirty under analysis." "Nor was there a special tendency to relapse; for a relapse only happened in three." "As regards the immunity from heart affection, the bromide treatment ranges itself alongside the alkaline treatment, which it seems to me to surpass in the greater influence on the pain, in the healthier, less anæmic look of the patient on recovery from the acute symptoms, and the speedier convalescence." The bromides "were, for purposes of observation, given alone." "They could be conjoined in special cases to alkalies, or to any remedy that the particular symptoms of the case might seem to render most advisable. Moreover, as we find with every treatment, there will be cases in which it may not suit. It is but a plan of treatment, attended, I believe, with good results. But it remains to be worked out to what cases it is the most applicable, and in which its success is most striking."

Having personally seen a good deal of the results of the bromide treatment in the hands of others, and especially Dr. Da Costa's, and having used it considerably in my own work, I am prepared to confirm its good effects, and to testify that its merits are such in certain cases that no other remedy quite fills its place. After becoming familiar with its effects in rheumatism generally, it becomes comparatively easy to recognize, without perhaps being able to define or portray the class of cases in which the remedy is likely to be useful and successful.

Salicyl Treatment.

The treatment of acute rheumatism with this class of remedies, of which there are a large number, though only a few of them have been actually employed, dates back about six or seven years. Since then they have come very largely—nay, almost universally—in vogue, and have received the very highest praise.

Salicin.—The first one of these compounds to be used in acute rheumatism was salicin. Maclagan, of London, commenced his observation on the subject in November, 1874. The other compound, the salicylic acid, was first used in Traube's clinic in Berlin, and the account of its action was published two years later in the *Berliner klinische Wochenschrift*, January, 1876.

Since these compounds are comparatively new remedies, at least in their present applications, let us glance at their nature and the history of their use in this disease. Salicin was discovered in the latter part of the first third of this century, the willow bark from which it is derived having been used for similar purposes nearly two centuries. Salicin being convenient for administration, was very considerably employed as a substitute for the cinchona alkaloids, and favorable reports were made of its antiperiodic effects in inter- and re-mittent fever, and also of its febrifuge qualities.

Maclagan was led to the employment of the willow-bark alkaloid, as he says, from consideration of the nature of the disease, and the qualities and nature of this drug, and not purely empirically. The method of reasoning which controlled him was as follows: "In connection with the action of quinine on the various forms of intermittent and remittent fever, and indeed in connection with the action of the cinchonaceæ generally on the diseases of tropical climates (ipecacuanha in dysentery, for instance), one fact had always strongly impressed me—that the maladies on whose course they exercise the most beneficial action are most prevalent in those countries in which the cinchonaceæ grow most readily—nature seeming to produce the remedy under climatic conditions similar to those which give rise to the disease."

"Impressed with this fact, and believing in the miasmatic origin of rheumatism, it seemed to me that a remedy for that disease was most hopefully to be looked for among those plants and trees whose favorite habitat presented conditions analogous to those under which the rheumatic miasm seemed most to prevail.

A low-lying damp locality, with a cold rather than warm climate, are the conditions under which rheumatism is most likely to arise.

On reflection, it seemed to me that the plants whose haunts best corresponded "to this description were those belonging to the natural order Salicaceæ—the various forms of willow."

This form of argument is not new, it has been applied many times to other diseases and natural products, and often with not greater accuracy, or perhaps with as much of pure imagination as in the present instance. The details of this special argument are defective, and to criticise it on no other ground than the error of topography, on which the author quoted seems to rely, is quite sufficient. The quinine-bearing trees grow on mountain elevations, far from swampy regions and in a climate approaching that of the temperate zone, and the willow's favorite haunt is the swamp, which if it produces rheumatism, is also the birthplace of the intermittent fevers.

Imaginative reasons like this should cast no imputation on the value of a drug, whatever else they may do. The therapeutic means must be tested by its results, and not by the way they were suggested for use. However, when one starts with *à priori* reasons, perhaps dictated by a prejudice, in favor of a method of treatment, it is well to be cautious in judging of the results and of the effects on the disease for which it is administered.

The manner of administration Maclagan describes as follows: "The salicyl compounds are so rapidly eliminated that their full beneficial effects can be got only by giving them frequently, as well as in full dose." "It takes about an ounce of salicin to remove the acute symptoms. That quantity should be taken within the first sixteen or twenty-four hours, in doses of twenty to forty grains, at first every hour, and then every two hours, as the acute symptoms begin to decline. A second ounce should be consumed in the next forty-eight hours. After that, twenty to thirty grains may be taken every four hours, for two or three days; and for a week or ten days more that quantity should be taken three times a day. By that time the patient will most likely be safe. Any threatening of a return of the rheumatic symptoms must be at once met by a return to large and frequently repeated doses of the remedy."

Two years later this author gives an account of his experience with the drug. To abbreviate this account the statement is as follows: "From so small an experience of salicin as I have had, I would not assert in anything like a dogmatic manner the full extent of its usefulness.

"1. We have in salicin a valuable remedy in the treatment of acute rheumatism.

"2. The more acute the case, the more marked the benefit produced.

"3. In acute cases its beneficial action is generally apparent within twenty-four, always within forty-eight, hours of its administration in sufficient dose.

"4. Given thus at the commencement of the attack, it seems sometimes to arrest the course of the malady as effectively as quinine cures an ague, or ipecacuanha a dysentery.

"5. Relief of pain is always one of the earliest effects produced.

"6. In acute cases relief of pain and a fall of temperature generally occur simultaneously.

"7. In subacute cases the pain is sometimes decidedly relieved before the temperature begins to fall; this is especially the case when, as is frequently observed in those of nervous temperament, the pain is proportionately greater than the abnormal rise of temperature.

"8. In chronic rheumatism salicin sometimes does good where other remedies fail; but it also sometimes fails where others do good."

Writing in 1881, he further says: "Subsequent experience and more extended observation have necessarily led to a fuller knowledge of the therapeutic uses of the drug; but they have not led me to recall, or even to modify, any of these conclusions." "The patient, of course, is confined to bed; the bowels relieved by medicine, if necessary, and the diet is light and simple, consisting mainly of milk and farinaceæ." "The salicyl treatment should be begun as soon as possible; for, in the interest of the heart, every hour is of importance. Twenty to forty grains should be given every hour, till there is decided evidence of its action. It will generally be found that before an ounce has been taken there is a marked improvement. With the continuance of the remedy improvement progresses; and often within twenty-four, generally within forty-eight, hours from the commencement of treatment the pain is gone, and the temperature is at or near the normal standard." "As the symptoms decline the dose may be diminished; but it is well not to do this too quickly or too early; for if the remedy be omitted too soon, or given in inadequate dose, the symptoms are apt to recur. The object in view is to keep the system persistently under the salicyl influence." Treated thus the course of uncomplicated acute rheumatism is arrested, the pain is abolished, and permanent convalescence begins frequently within twenty-four,

and generally within forty eight, hours of the time that treatment commences. In first attacks, and in young subjects, such is almost invariably the course of events when there is no cardiac complication.

"How different from the duration of the disease under other methods of treatment! Under all of them it was common for the acute symptoms to last for three or four weeks, and twice that time frequently elapsed before the patient was free from pain, or able to leave his bed. Now it is often difficult to keep him in bed for more than two or three days."

Salicylic Acid.—Before considering the mode of action of salicin, and the results and the judgment which others have obtained and maintained, let us glance likewise at *salicylic acid*, which belongs to the same group, and which in many respects is similar to the former drug.

This acid has been known since 1844, when it was prepared from the oil of winter-green. Its scarcity and great cost prevented its use, and its quality remained to a great degree unknown. Through subsequent discoveries, especially in 1852 and 1869, it became better known, and finally in 1773–74, by still further improvements in the process of manufacture, it became possible to produce it cheaply. Kolbe in the latter year showed its antiseptic properties, and Professor Thiersch, of Leipzig, employed it in the treatment of wounds, substituting it for carbolic acid in the Lister method.

Subsequently, for surgical uses, salicylic acid was very extensively employed, and is yet to some extent, though now nearly relinquished.

Then came the application of this remedy and some of its salts in various febrile diseases as an antipyretic agent. Its antipyretic value is acknowledged in cases of pneumonia, typhoid fever, scarlatina, pleurisy, and phthisis; but by most it is regarded as inferior to quinine, and in the acute diseases the remedy shows no power of essentially altering the course of these diseases or of modifying the local process.

Stricker was the first who, in Traube's clinic in Berlin, 1876, used salicylic acid in acute rheumatism. His work and Maclagan's with salicin were entirely independent observations; and both seem to have arisen in answer to the newly worked idea in medicine of the germ theory and its teachings. Buss and Riess had both published, a year previously to either Maclagan's or Stricker's publications, the effects of salicylic acid in acute rheumatism as an antipyretic medicine; whereas both Stricker and Maclagan were controlled by theoretic considerations, and their dominating ideas were on the germicide theory.

Stricker's conclusions are given as follows:

First.—Salicylic acid appears to be a rapid and radical remedy in recent cases of genuine acute rheumatism of the joints.

Second.—It is not injurious to the human organism when administered every hour in doses varying from seven and one-half to fifteen grains.

Third.—It can be given in these doses for a longer time to young and strong individuals than to the old and feeble.

Fourth.—In the latter it produces toxic symptoms more readily than in the former.

Fifth.—The toxic symptoms vary in degree.

Sixth.—Those most commonly met with are noises in the ears, difficulty in hearing, and diaphoresis; when these occur, the administration of the medicine should be discontinued.

Seventh.—If salicylic acid be found to fully answer the expectations entertained regarding it, the internal administration of a certain quantity may be expected to prevent the occurrence of fresh attacks in hitherto unaffected joints, and also secondary inflammation of serous membranes, especially the endocardium.

Eighth.—To prevent relapse the medicine must be continued in smaller doses for some days after the termination of the main treatment.

Ninth.—Salicylic acid is of doubtful utility in chronic articular rheumatism.

Tenth.—It is not likely to be of use in gonorrhœal or diarrhœal rheumatism, or in the polyarthritis attending septicæmia.

Salicylate of Soda.—In respect to salicylate of soda, which is now used as much, if not more than the acid, there seems to be no essential variation in the effects of the two preparations.

The similarity of the reports of these two independent observers of the effects in this disease of these two closely allied remedies is very striking. And if but the half of these observations were true, the millennium of rheumatic sufferers would certainly have arrived.

Numerous observers in this country and in England reported nearly equal success, and coincided with the conclusions here given. And now the salicic treatment of acute rheumatism, including the use of salicin, salicylic acid, and salicylate of soda, was fully launched in the medical world and has now been tried by every one, and very largely praised.

Forty years ago, when Furnival first proposed the alkaline specific treatment of rheumatism, there were but few medical publications, and

the common mind of the profession was slow to receive new indications in respect to either pathology or treatment, and hence but little *furor* could be created about his results. Nowadays the mail steamer carrying the clinical reports of Stricker's work in Berlin, brought also the supply of medicine to every country to which the news was carried, and every clinician is at work immediately testing the results. An announcement in a medical congress is now telegraphed around the world, and some conclusion is arrived at in countries thousands of miles away before the delegates have dispersed and arrived at home.

The reception given to the alkaline treatment on its announcement thirty or forty years ago, allowing for the greater slowness of communication, resembles so much that now accorded to the salicyl compounds, that one cannot but pause and reflect, make a comparison, and wonder what will be the fate of the new remedies forty years hence.

Their application arose, so to speak, at dictation of the terms of the germ theory, just as the alkaline treatment sprung from the now fallen lactic acid theory. And I think that the salicyls will stand or fall as stands or falls the test that will be soon applied to the germ theory. We would not be without alkalies in meeting indications in rheumatism, and so it will probably be in respect to the salicyl compounds.

Mode of Action of the Salicyl Treatment.

Now that we have seen the favorable reports of the effects of these remedies in this disease let us look to their mode of action.

Action of Salicin.—First in regard to salicin. It is a crystalline, bitter substance, having various therapeutic as well as other analogies to quinine. It is only slightly soluble in water. It may be transformed by the use of reagents into many of the other members of the salicyl group. Administered internally, it has long been known to have slight tonic properties, to act as febrifuge or antipyretic; hence one of its analogies to quinine, for which it has been used as a substitute. Of its general effects or of its mode of action we know nothing. If we believe that intermittent fever is produced by germs, then quinine acts by destroying the germs, and as Maclagan would have us believe, salicin acts in rheumatism as a germicide also.

Taken into the system, salicin is converted into some one of the other members of the salicyl group—it is generally believed into salicylic acid.

Thus, according to Senator and other observers, salicin when acted on by various organic ferments is changed immediately while in the stomach into one of its congeners, and that this substance on its absorption into the blood is oxidized into salicylic acid. This latter substance makes its appearance in the urine within a half hour of the taking of the salicin.

This conversion of salicin in the blood into salicylic acid is denied by Maclagan, claiming that the difficulties of this transformation are too great to be effected in this manner. He also denies the accuracy of the test by which Senator, Ranke, and others propose to show the presence of salicylic acid in the urine. This question has not as yet been decided, and the urine reaction shows so far only that some change is effected in the salicin on its passage from the mouth to the bladder. What this change is precisely, what effect it has on the system, and what effect it has on the diseased condition, has not been pointed out.

Salicylic acid is a very disagreeable, irritating substance when inhaled or applied to the mucous membrane. It occurs in needle-like crystals, which dissolve freely in hot water, but only slightly in cold. The acid rapidly appears in the urine, and passes with that excretion entirely unchanged.

Senator concludes that salicin acts in fundamentally the same manner as salicylic acid, since the former is converted into the latter during its passage through the organism. Maclagan argues strenuously against the fact that salicin owes its therapeutic virtues to its conversion into the acid, and in support of his view quotes the results of clinical experience. That salicin has properties that salicylic acid does not possess may be taken for granted ; but that it acts in the same way—whatever that action may be—on the rheumatic process seems to be pretty certain.

In judging of the relative value of these two compounds there are several things to be borne in mind. First of all, the use made of salicin is much less extensive than that of the acid, and with our deficient experience there is consequently less knowledge of possible unfavorable, dangerous or defective results from its administration. Secondly, a natural product, like salicin, is much more likely to be pure, or at least to contain matters which are in themselves harmful, than a manufactured article like salicylic acid, derived from the source which it now is. Thirdly, the former is nearly certain to be a stable compound, while the latter, perhaps not even containing a residuum of matters used in its manufacture in themselves harmful, may, by defective reactions, not be the precise molecular body which it purports to be.

The first difficulty in this category can only be overcome by further tests of the relative merits and defects of the two remedies. The use of salicin in the large doses recommended has not yet been extensive, though many have reported as favorable results as those obtained by Dr. Maclagan. This author says, "The only unpleasant effects which I have found follow large doses of salicin, are tinnitus, partial deafness, and headache, such as frequently result from the administration of quinine." The advantages of its use, as stated by him, are its equally prompt action, its tonic properties; "that cases treated by salicin are less debilitated at the end of the attack, and convalesce more rapidly," that it produces no nervous symptoms, except those noted above, and that it has no disturbing effects on the heart or circulation. Salicin also has the advantage that it "may be freely administered while the system is still depressed from the toxic action of the salicylic acid, and that under its use the rheumatic and the salicylic symptoms both disappear."

Senator, in a review of the whole subject comparing the relative merits of the two medicines, concludes that the antipyretic qualities of salicin are less marked, and that it is slower to depress the temperature, but that its effects in this respect are more lasting. This drug is less likely to affect unfavorably the circulation, and hence in the presence of any cardiac disease, especially fatty changes in the heart-muscles, it is free from danger.

Many writers have confirmed these advantages of salicin over salicylic acid, and the former is generally regarded as free from any unpleasant or alarming effects; but it is thought to be less powerful and more uncertain in producing the results desired. It is more soluble than the acid, but is less readily dissolved than salicylate of soda, and is therefore less easy of introduction in the system than the latter. It causes almost no local irritation, and is almost always retained by the stomach without producing nausea and vomiting.

Salicylic acid and salicylate of soda are regarded as identical in their action. The difference in the two preparations in respect to the amount of the free acid is about 1 of the former to 0.68 of the latter, and usually a variation in the quantity given per dose is regulated accordingly.

The preference is now pretty generally given to the sodium salt, on account of greater solubility, its less irritating qualities, and its less disagreeable taste.

Its effects, as given already in Stricker's summary, are now generally

acknowledged. The differences of opinion which exist are in respect to its toxic effects (which are now pretty well recognized), and also concerning its curative action on the rheumatic process, that is, if we may so describe it, its destructive action on the rheumatic poison.

In respect to its toxic effects—the alarming and dangerous symptoms which sometimes supervene in the course of its administration in the doses in which it is recommended have been frequently observed and well recorded by numerous writers. Some of these effects, especially those recorded at the commencement of its use, were undoubtedly due to the impurities or the imperfect manufacture of the remedy, and even yet it is necessary to guard against dangers from the same cause. Nevertheless, alarming conditions are frequently seen from the use of the pure drug, and which, if not averted by treatment and the cessation of its administration, threaten disastrous consequences and death.

Nervous symptoms, similar to those recorded from the use of salicin, are pretty frequently met with. In addition to these, delirium is spoken of as occurring, but it does not seem to be of a very marked character. If proper watchfulness is exercised on the effects of the remedy, the danger of the occurrence of this condition can be averted by stopping the use of the drug. The cause of delirium has been supposed to be the failure of its excretion by the kidneys, and the consequent supersaturation of the system by accumulation of it in the blood. Attention to the renal secretion and the chemical examination of the urine at the outset of the administration, would at once settle the question of the propriety of its use, and its continued use can then be determined by its effects. The occurrence of delirium I have never seen which could be fairly attributed to the drug, while the minor nervous phenomena I have witnessed daily.

The chief source of danger is the effect of the salicylic compounds on the circulation. They are a real source of danger. They are not evidenced plainly by striking phenomena, as are the nervous symptoms, and are not complained of by the patient, often until an advanced stage of development. They require for their detection a careful examination, a knowledge of their character, or a familiarity with the physical status of the patient. This depression of the circulation I have seen develop so gradually, the patient meanwhile becoming more comfortable, that the condition was easily mistaken for improvement. And it is only by a careful examination of the heart that the weakening of the cardiac action is discovered. In such cases I have not found much alteration of the radial

pulse, at least not such changes in its character as would warn one of the coming or existing danger; the softness of the radial pulsations, under these circumstances, could be interpreted as an improvement, and are quite compatible with the ease and comfort felt by the patient. I have found that the pulse, by no means in all cases, and not even in many cases, becomes increased in frequency, and neither does it have the feebleness of the failing, frequent pulse of the exhausted patient.

In such cases, at the moment we are congratulating ourselves on the relief of the patient, another dose of the remedy may place us on the brink of a serious or fatal depression of the cardiac action, especially if this organ has deformity of its valvular apparatus or fatty changes in its muscular fibre.

The changes in the action of the heart which I have noticed in these instances of depression are a feebleness in the apex-beat as felt by the hand, an absence of the tone or volume of the sounds as heard by the ear, and a general lagging of its contractions.

Oftentimes the patient commences to complain of a depression or sense of sinking before any depression of the heart's action can be detected by the observer. This is a full warrant for stopping the medicine and pursuing another line of treatment. However, with some patients the first dose of the salicylate compounds produces nausea, which in many is accompanied by a feeling of extreme sinking, and this false depression passes away, as by continuing the medicine the stomach tolerates its presence, and the nausea ceases. If we can distinguish the second form of nausea, we are warranted in continuing the treatment, in hopes of having the stomach retain this desirable drug. I have noticed that the cardiac depression produces a sinking feeling, which pervades the body generally; the depression of nausea and the sinking sensation is confined to the epigastrium but I do not think this rule is universal. The danger of the remedy consists in the possibility of grave depression coming unconsciously to the patient, or even with a sense of comfort.

There is another form of depression seen occasionally in acute rheumatism treated with the salicylate remedies which I do not think ought in fairness to be attributed to the toxic agency of the drug. It is a condition of affairs which is seen in some cases of both intermittent and remittent fevers treated with quinine. It is also seen in many cases of other continued fevers at the period of their crises; in typhoid fever lasting through the third week, three or four days before the temperature

curve finally descends to the normal it is very common to find the temperature fall often in the course of a few hours to a subnormal level, and with this comes a condition of collapse. Other fevers exhibit this, but to a much less marked degree than typhoid, and in none of them do we fear evil from it. In rheumatism untreated, and even with most remedies, we see nothing of the kind; but in giving salicylic acid, so strong is its antipyretic action, whatever may be the cause of this, that occasionally we see the same result produced, and manifestly it must be regarded in the same light. If the condition become too pronounced we treat it, as in typhoid or other fevers, by very mild stimulation, or perhaps better by concentrated nourishment.

The grave depressions of cardiac action due to the toxic effects of the salicyl remedies require prompt and energetic means to combat them. I have found brandy the best remedy, but black coffee serves well; and as with this depression, and really a part of it, free action of the skin takes place and the surface becomes cold, a warm blanket and external heat are good adjuvants to the internal remedies.

Action of Salicylic Acid.—Let us now speak of what seems to be the mode of action of salicylic acid. It has not been possible to give any account of the physiological action of salicin, since no observations have been made with accuracy, and no record of its effects has been made beyond its somewhat slightly tonic ones, and the effects which it produces in rheumatism and intermittent fever.

In regard to salicylic acid we know a little more, and having reviewed its effects in rheumatism and seen its danger, we are better prepared to understand what is ordinarily spoken of as its physiological effects. In speaking of the effects of salicylic acid, we will include those of salicylate of soda, which appear to be exactly the same. This salt was proposed for medical use by Mœli, of Rostock.

The first experiments were performed by Koehler, who published his account in the *Centralblatt für Wissenschrift Medicinische*, 1876. He injected the drug into the veins and found that the blood-pressure was lessened; this effect was in proportion to the dose. The pulse at the same time decreased in frequency. The same phenomena he found occurred after section of the pneumogastric nerves of the spinal cord, which seemed to indicate a direct and special action of the drug on the ganglion apparatus of the heart itself. There seemed to be also a decrease in the respiratory action, and the subjects of the experiments died of asphyxia. This

seemed to result from a diminution of the excitability of pneumogastric nerves perhaps, resulting in insufficient oxygenation and the accumulation of an excess of carbonic acid.

Action of Salicylate of Soda.—In a memoir to the Academie de Médecine in 1877, Dr. Laborde concludes that "the physiological action of salicylate of soda is predominantly shown in an elective manner, as it were, on the phenomena of the sensibility to pain, or consciousness." "The mechanism of this action resides, he thinks, in the influence exercised by the chemical substance not only on the conductive power of the sensitive nervous cords, but on the receptive centre and elaborator of the peripheric impressions." "This action of salicylate of soda on the functions of sensibility, and consequently on the cerebral organic seat of this phenomena, is the reason of the effects induced on the painful symptoms in disease, and it is principally as an *analgesic* that the drug acts in the cure of articular rheumatism." (Deseille: Thesis, Paris, 1879.)

In the same year we find numerous communications to the French Academy from Professor Sée. His results are obtained from clinical sources alone, and are as follows: 1st. The rhythm and the number of the cardiac pulsations remain normal. 2d. In healthy people no modification of temperature is found, and in some exceptional cases the use of salicylic acid has even raised the temperature to a febrile level. In febrile conditions its febrifuge power is the most limited and the most doubtful. 3d. The respiration is not compromised by moderate doses. 4th. Finally, in doses of 6 to 10 grms. daily, the patient suffers buzzing in the ears, a sort of deafness, sometimes a staggering, or rather an uncertainty in walking, but never is the sensibility modified in a constant manner, nor are the reflexes.

Bochefontaine and Chabert conclude from their experiments that at first the salicylate acts on the brain and the medulla, the organs of sensibility and motility; then on the cord and the reflex phenomena; next, on the peripheric nerves and those of excito-motor action; finally, on the movements of the heart and on muscular contractility. In other words, its first effects are seen in lessening the activity of the encephalic gray matter, and probably also the gray matter of the cord, and that thus it exercises a special paralyzing action on the sensitive nervous fibres of the peripheric nerves. They also note its effects in producing nausea, vomiting, and diarrhœa, sometimes these discharges containing blood. There is also a state of general feebleness, a more or less complete paralysis of

movement, sometimes convulsive movements, and death comes from the arrest of respiration and the cessation of the heart's action.

Other French experimenters have noted the vomiting and diarrhœa often streaked with blood, probably due to the irritant action of the drug on the mucous membrane. They have noted the action of the drug on the respiratory centres, and its effect in reducing the temperature at least 1° C. in the non-febrile condition, and a lessening in the frequency of the pulse. The urine, too, becomes lessened in amount, and an albuminous condition of its secretion is developed.

These observations are somewhat contradictory, and are not sufficiently definite for us to conclude with certainty on the action of salicylic acid in the normal condition, or to determine the nature of its effects in acute rheumatism. All the reports, however, in a general way, point to the facts that, by a paralyzing or depressing action on the nervous centres or on the peripheral fibres, or both, it lessens the activity with which tissue-changes take place.

Having given the account of the effects of these two medicines on the normal organism, and also their effects in controlling the disease, it is proper to consider what is their mode of action upon the morbid state. But in order to reach a clear conception we must definitely state in what consists the rheumatic process. Heretofore our conceptions of the pathology of this disease have rested under the blackest cloud of traditional notions handed down from the earliest times. Finally, the conception of a distinctive "rheumatism" freed itself from a medley of other conditions having somewhat similar symptoms, but there remained attached to it the unfortunate name showing its origin, and constantly misleading our pathological notions of its real status. The name will probably always remain.

Then came a chemical age in which the product was mistaken for the cause, and the lactic acid theory held us in its fetters for forty years or more. The insufficiency of this theory was not so much disproved as that without any one stretching forth a hand to tumble it over it sank out of sight.

To-day we stand in the presence of a germ theory, and every disease is completely furnished out with its own particular zoology or botany—insanity almost alone stands as an exception—and this malady, according to past notions, should have been supplied first of all. The rheumatic humor of early times, and the lactic acid of more recent periods, the rheumatic poison in general has been displaced by "a minute organism."

If it is not too late or too old-fashioned already to protest against this so-called new idea now is the time, before the peculiar "minute organism" of rheumatism has been identified. These views on the nature and action of minute organism remind one strongly of the old theological notions of pathology looked at, as is the custom nowadays, with a microscope. The theologians, who controlled medicine in former times, taught that most diseases—and insanity stood at the head of the list—were due to the possession of the devil or of some evil spirit, which entered the system and took up its residence there. The symptoms of disease were owing to these perturbing influences.

The size of these successors of the evil spirits has, in accordance with our methods of study, been reduced to microscopic size. We are taught to believe that every microscoporon found in our blood or tissue is a peculiar germ productive of the disease in connection with which it is found.

The evidence as it stands is quite as strong in favor of the minute organisms serving the function of scavengers as that they produce the disease. The whole scene of life, as we see it around us, conveys but one idea—that life and growth and activity tend to sweetness and purity, and not to foulness. In higher life the lower orders take the offal from those above them; the eucalyptus tree renders the stagnant swamp fresh; the maggot in the putrid mass leaves it sweet and clean.

Without pursuing this idea further, it suffices to state that the proposer of the salicyl treatment endeavors to saturate the system as thoroughly and rapidly as possible in order that the minute organisms may be completely destroyed.

It seems possible to explain the curative effects of the salicyl compounds without calling up the presence of unknown organisms with the prospect of rapid death before them. It is possible to think of disease, not as a foreign entity which takes up its residence in our tissues, producing disturbance and destruction by new and foreign method. It seems much more likely that disease is a misdirection given to our normal functions by agents other than minute organisms. We have been accustomed to think of disease as changed, depressed, or exaggerated function, but still always to be recognized as function, and not to think of medicine as a ferret sent in to hunt out and destroy an interloper in our bodies. We have always sought to think of medicine as guiding the formative and retrograde processes of nutrition according to the normal physiological

methods, and by changing the nervous action or influence to alter the blood-supply, which is the fountain of nutritive activity, normal or pathological.

So it would seem to be in rheumatism. If we may even in slight degree trust in the evidences of the experimental effects of salicylic acid, or of the results obtained clinically, this remedy causes a cessation of the rheumatic process by controlling the misdirected process of nutrition. Whether it does this by its action on the central portions of the nervous gray matter, or on the peripheral fibres, or on the intermuscular ganglia of the heart, thus directly, as it were, altering the blood-supply, or by a combination of these effects, we are not able to determine. It would seem that, in some cases at least, that the latter of these effects is the most pronounced, since it is not infrequent to observe a cessation of the general febrile excitement of the circulation without a corresponding proportionate diminution of the articular inflammation. And then, too, after the discontinuance of the drug, the articular disease is apt to return—a so-called relapse—without a corresponding return of the circulatory excitement. It looks as though the normal function had returned completely to one part and continued normal, while at another part the balance was not completely restored.

Anæsthetic and other Topical Applications.

The value of local applications to the inflamed joints has been very variously estimated; by some as useless, by others as serviceable, by others as harmful. None of them are probably efficient in removing or preventing a return of the disease, but nevertheless probably many of those proposed are of use in modifying the local inflammation. The question is to be judged of on general principles.

The list of remedies which have been used for this purpose is almost endless, and their effects are generally indicated by the nature of the article employed. And the nature of these articles have been of the most opposite and contradictory characters. The softest of down and the hardest of immovable bandages have been applied, the coldest of refrigerants and hottest of stimulants or actual heat have been used. The most soothing lotion and hypodermic injections of morphia have found their opposite in similar injections of carbolic acid and pure water. Leeching, scarification, and blisters have all found advocates. The swollen joints have been

rubbed and moved, and they have been compressed by contractile bandages or fixed immovably by splints. The joints have been treated, according to the varying pathological views of the disease, by salines to overcome the acidity, and by carbolic acid and salicylic acid to destroy the minute organism.

The governing principle in the local treatment seems to be this—and we know more of the natural history of a rheumatic joint than we do of the general disease as a whole—a joint becoming inflamed and swollen is painful, and when the swelling has attained its maximum the pain is less. The inflammation never or rarely becomes so excessive as to lead to suppuration; on the other hand, unless the articular disease is of very short duration or of very slight degree, it will leave at least some temporary stiffness, but this generally passes away without treatment pretty promptly. If a joint is many times involved, and severely so, if affected for a long time with severe inflammation, or if after a severe inflammation a milder type lingers in the articulation, the parts within or without the joint cavity may become so much affected or suffer such changes of form as to be permanently sore, stiff, or even altered in outline.

There seem therefore to be three indications: *First*, to assist the loosening or distention of periarticular tissues when the pain is severe or unbearable by means of heat and moisture, both of which should be proportioned as in other inflammations to its severity. The heat and moisture may be assisted by anodynes and by other remedies like alkaline lotions, which soften the tissues in these cases as well as in others.

Second.—It is not necessary to treat the inflamed articulations with a view of preventing a possible suppuration.

Third.—If the inflammation is slow in subsiding completely, or if thickening and stiffness continues, iodine or other similar remedies hasten the return to normal conditions.

For the comfort of the patient the limb may be elevated or fixed immovably, and the joints should be disturbed as little as possible, since movements will increase both the local disturbance and also the general symptoms.

Diet and Regimen.

During the acute attack no solid food can be digested thoroughly, and hence the diet should be entirely fluid. Milk and light meat broths are all that is necessary. Usually abundance of water is desired, and of this

the effervescing waters are found often most agreeable, and they may be taken separately or mingled with the milk, by which they serve the purpose of promoting its digestion. Others crave acid drinks, as lemonade and lime juice. Citrate of potash in solution, toast water, and many similar drinks are in demand. Alcohol in any form is, as a rule, disadvantageous, though craved by and perhaps necessary to some patients who are in the habit of using it to excess.

During convalescence, or even just after the subsidence of the fever, light farinaceous articles may be given with advantage, and still later some of the delicate meats, until the return of complete digestion and a normal appetite makes its appearance.

The patient with acute rheumatism should be well protected from cold air ; but it is quite as important that the bed-covering is not made heavy. The sweating, febrile patient is easily overheated and exhausted. In the convalescent period warm underclothing is essential, and a neglect of this precaution often causes a relapse with very serious developments. Under no circumstances should the patient be allowed to get out of bed, and all the wants of nature should be attended to under cover.

CHAPTER XVII.

CHRONIC ARTICULAR RHEUMATISM.

Symptoms and Course.—Morbid Anatomy.—Diagnosis.—Treatment.—Local Applications.

Chronic rheumatism is a term which has been most variously employed. By some writers it is used to describe all forms of rheumatic phenomena not attended by fever; by others, for those changes confined to the joints and their immediate surroundings which show not an active, or but a very slightly inflammatory process, excluding the rheumatic symptoms of the muscular tissues. It seems best, at least from a clinical point of view, to add the word "articular" to the title, because the symptoms exhibit themselves almost wholly in and around the joints, although the muscles and their connections are very frequently simultaneously involved. There is no inappropriateness in the use of this word, since, unlike acute rheumatism, the internal viscera, and especially the heart, show no tendency to participate in changes which we describe as rheumatic, or at least such changes never exhibit active symptoms.

Doubtless very many pains and aches and alterations of the joint pass under the designation of chronic articular rheumatism which have little or no relation to this disease. Still, I think the formation of a group of articular rheumatism of a chronic variety enables us to differentiate this complaint from others of a different nature more sharply than when the muscular forms are arranged with the articular in the same class. But because of a certain indefiniteness of which the group consists, and which also inheres to our notions of it, we are not prepared to abandon it. Rather is it the more necessary to prune and garner the various members to render the whole more homogeneous. The less pronounced the symptoms of a malady, the less able we are to be positive in respect to its nature.

The components of the group of chronic articular rheumatism are formed of several classes of cases. After separating all the joint diseases of neural origin, the gouty cases, and the instances of rheumatoid arthri-

tis, as well as the surgical cases connected usually with injuries, etc., we have left two classes of chronic joint disease which form the bulk of the group. First, those which are dependent on results from previous attacks of acute rheumatism; and secondly, those which develop slowly, gradually receiving accretions from time to time, until the joint or joints become permanently altered in their function and structure. To these two classes some are inclined to add the chronic joint changes which are superinduced by such maladies as gonorrhœal rheumatism; and others, those cases also which in their earlier stages were looked upon as rheumatoid arthritis, or neural arthropathies, perhaps, which ceased to develop, and even other maladies which attack the joints.

Many writers, as already mentioned in speaking of acute rheumatism, are disposed to separate entirely all the classes of this group from the rheumatic disease, and to consider them as of a purely local nature. The grounds for doing so must rest on the opinion that all the changes which are to be described are to be looked upon as the results of disease, and not as an acting process of disease; in other words, that the conditions which we consider as chronic articular rheumatism are only alterations in function and structure produced by past diseases, but which are in themselves inactive, or have arrived at a stage which the changes have ceased.

The justice of this view depends on facts, both clinical and anatomical, which are open to observation and study. If we define chronic articular rheumatism as a lower process of inflammation—as a subacute inflammation below the grade of the sub-form of acute rheumatism—it is not proper to deny to it a rheumatic origin. If we acknowledge that the changes result from the repetition of acute attacks, unquestionably rheumatic, the disease itself is certainly rheumatic. If changes of the same character, attended with the same symptoms, develop slowly, though its activities never rise to an acute grade, to deny its relationship is unjustifiable.

To place this form of the disease in the class of local ailments merely because it so very exceptionally develops fever or visceral complications, is saying nothing more than that the condition is not accompanied by such phenomena, but it is not showing that it is a distinct disease. It is rheumatism, but it is a local form of it; local because in activity it is of low grade, and simply because it manifests no general or visceral symptoms does not prove that no changes in the blood or other organs than the joints do not slowly and unobtrusively take place.

This form of the disease, occurring as it has been shown, exclusively, or

almost so, in the latter periods of life, may be dependent on the state in which the tissues generally involved in rheumatism have been placed by advancing age. We can readily understand how the joint tissues are less prone to exhibit acute inflammatory phenomena, and also how, when once inflamed, even though but slightly, they less easily return to their normal conditions. That a heart valve in age is less liable to acute endocardial inflammation is even more ready of explanation.

The disease is very frequently found, however, in those who, earlier in life, have suffered from cardiac or other visceral complications, and these past inflammatory changes often form a serious barrier to the restoration of the health of the chronic articular rheumatic. But the disease exists in patients who have not been crippled by any of these accidents; in fact, I am inclined to think that the most typical instances often are seen in those who have escaped such complications, and perhaps also that, numerically considered, the larger number of the cases living are without a damaged heart or other viscera. Possibly this fact is due to the cardiac cases dying before the joint changes become advanced, and thus also the number of patients with valvular disease are diminished.

That the disease follows from the acute form, and is produced by the same general causes, show that the malady, though local in its manifestations, is of the same general nature. That when the disease develops slowly it does not manifest the usual striking symptoms is due to differences in the application of the exciting cause, and the receptivity of the patient, and the age and stage of nutrition of the tissues. When the development comes after acute attacks it results from improper care and after-treatment, to exposure too soon after recovery, to improper regimen, and it may be from injuries or over-exertion, and to the original severity of inflammation which have perpetuated such tissue changes as form a suitable anatomical basis for continued inflammatory changes. This relationship of primary inflammation to the subsequent tissue changes in and about the articulation finds its parallel in very many other diseases.

The local nature of the chronic articular rheumatism is defended only by those who support the humoral pathology views, and who point to such specific agents as lactic acid or "minute organism" as the cause of the acute form. It is naturally difficult to suppose that a small supply of lactic acid or of "germs" remains over, and more or less permanently affects only one or two articulations or a single nerve or muscle. Germs live and multiply, and are rarely local in their disturbing effects. Look-

ing, however, on rheumatism as a perverted functional activity, the localization of the malady and its indefinite prolongation are easily explainable.

Symptoms and Course.

The grade of the symptoms presents an infinite variety; this is modified by the duration, the severity, the stage of development, and very especially by the mode of life of the patient. Typical instances are seen where, by an easy life, the patient is merely conscious, but nearly continuously so, of having one or more affected joints. The consciousness consists in tenderness, or pain and aching, either spontaneously or on movement only, or a stiffness, or all of these combined, and usually with these articular sensations there is a radiation from the joint into the tendons or muscles of the neighborhood. Under these circumstances the affected joint may exhibit to palpation and to the eye little or no change of its external characters, and its motion may be but slightly if at all hindered.

From this slight affection, rapidly or slowly, the symptoms may increase, and the form and functions become altered in the highest degree short of dislocation, although anchylosis may occur. In many cases there are periods of more or less complete alleviation of some or all of the symptoms, but this is but imperfectly seen in cases where the anatomical alterations are well marked. The periods of immunity from symptoms vary very greatly, and their time is influenced by the usual rheumatic influences, so-called, climate and season, moisture and cold. Exercise, injuries, and the state of general health, as well as unknown causes, often lead to exacerbation of the malady.

The joints most frequently affected are generally the large joints, although the smaller ones are not exempt. The rule in respect to acute rheumatism here also prevails, that the most used or exercised ones are preferably diseased; but this rule is not more universal than it is in the acute form.

In some cases the shifting character of the disease is often exhibited, but even here the shifting does not occur after a joint becomes much altered, although its vagrancy is still seen in the other articulations. A more or less symmetrical character is also found in many cases, and perhaps even more strikingly so than in the acute disease.

The aggravations of the disease often come suddenly, like a flash, and are naturally attributed to a slight strain of the muscles, tendons, or joints.

The increase of the symptoms may consist simply of greater pain and stiffness, though frequently it exceeds this limit, and the case becomes one of subacute rheumatism, attended with slight swelling, redness, tenderness, with effusions within and without the articular cavity, and a slight febrile movement is evidenced by the thermometer, pulse, and even other conditions, which are wholly absent from the chronic articular phenomena.

These often slight exacerbations, which frequently pass without medical care and observation, undoubtedly have the capacity of developing visceral complications, and this fact may account for the belief expressed by many, that chronic articular rheumatism often produces valvular heart disease. If such clinical facts are substantiated, they go to establish the essential unity of the two forms of the disease, and to disprove the purely local character of the chronic form.

In speaking of the complications of acute rheumatism the belief has been expressed that the localizations were oftentimes accidently developed, and that, in all cases of the rheumatic paroxysm, tissue changes were present, which are the essential foundation of the precedent condition which by accidental aggravations gave rise to the symptoms or physical signs which enable us to recognize the facts. In every well-marked rheumatic paroxysm the tissue of the heart valves were changed, but not always do they become roughened, and thus collect fibrin which produces the initial murmurs. If in acute rheumatism the tissue swells and inflames, but does not collect fibrin, the cardiac valve may subsequently undergo deformity, although it has not during any prior stage given the physical signs of endocarditis and roughening.

This development is certainly of rare occurrence in comparison with the initial production of a murmur with roughening and subsequent deformity. But if it can be shown to occur it will help in the explanation of those cases of cardiac valvular disease, which must be known to all, found in patients suffering from chronic rheumatism. I have known cases in which no history of a paroxysm could be ascertained, but in which advanced valvular disease existed.

The duration of the disease is exceedingly indefinite. Many of the cases, those especially coming after acute rheumatism, gradually pass from this grade of the joint affection through a more or less prolonged subacute stage into an indefinite chronic form. These cases progressively may improve until all symptoms cease, and their past existence is evidenced by only a little thickening around the joints. This condition is commonly more

perceptible to the patient's than to any one else's senses. Many other cases, however, continue indefinitely without signs of improvement; in fact, from time to time, as the intervals of pain recur, become worse.

A considerable number of instances are known of patients who, having suffered many years from chronic articular pains, gradually—and I have known them even suddenly—lose all sense of pain, stiffness, or ailment. All of the cases above referred to are those instances in which but comparatively little alteration has taken place in the joint or its surroundings.

In the larger number of cases—although this number, I am inclined to think, is not as large as usually represented—a condition of thickening of the capsular and other ligaments, as well as the cartilage perhaps, is permanently altered, and the sufferings of the patient are terminated only by life. This termination may come as the result of other rheumatic visceral disease, or from other maladies disconnected with the rheumatic process. These permanent alterations unquestionably may keep up the impeded function of the affected articulation, and it is difficult to determine where to draw the line between a rheumatic inflammation and its deformity. When, however, the pain and the evidence of the very lowest grade of inflammation ceases, we may conclude that the rheumatic process has ceased. We know that a very much altered joint will in time accommodate itself to great changes, and will again function comfortably and conveniently, although its external appearances are deformed and clumsy.

Morbid Anatomy.

The cavities of the affected joints are never found to contain more than the usual amount of fluid, unless a subacute condition has supervened just previous to death, on the chronic changes, and even then the increase is not a notable one. More frequently, in fact, I have seen such joints drier than normal. The character of the synovial secretion is sometimes altered, becoming thicker or more glue-like in character, and to this inspissation of the secretion a part of the description of dryness belongs. In addition to the greater consistence, the synovia is sometimes no longer clear and nearly transparent, but has a slightly yellowish hue, reminding one of the color of slightly smoky urine. This color is, I believe, due to blood elements, which make their appearance in the fluid owing to the interference with the circulation in the synovial membrane and the peri-articular structures. The secretion of the joint shows no tendency to become purulent.

The tissues around the joint—and these are the parts which in my experience show the greatest changes in chronic articular rheumatism—are thickened and denser in character than normal. The skin shows a varying character of changes, none of which are constant or characteristic. The subcutaneous connective tissue is increased, its fibres are coarse, and it presents the appearance often of œdematous subcutaneous tissue, although it rarely allows of any fluid being pressed out from it. The vessels traversing the neighborhood of the affected articulations are enlarged, and this is especially shown by the conspicuousness of the veins, which during life are often to be traced by their dark line and the channel they form in the sometimes denser skin.

The capsular ligament loses the shining iridescent appearance of its interlacing fibres, and all the ligaments become thickened, dense, and cut more nearly like cartilaginous tissue. They have evidently had new material added to them, the result of inflammatory proliferation and by hyperplasia, and the new tissue becomes cicatricial.

The changes within the joint vary in character according to the severity and the stage of the disease. There are two classes of change with which I am familiar. The one shows itself as a roughening of the cartilage. This roughening consists in ridges and furrows on its surface, so that to the sense of touch the once normal smoothness becomes ribbed or fluted; the extent of this change is very varying; in some cases it is wide-spread and nearly universal, in others displaying only a few irregular elevated lines.

In the other class, the articular surface is covered with a layer of fibrous material superimposed on the cartilage. In some cases this material forms adhesions between the opposing surfaces; in others it is free from attachments, except perhaps at the margins; in some cases the adhesions are short and hamper, or almost entirely prevent, the play of movement, and form a more or less partial fibrous anchylosis.

The cartilage in nearly all cases becomes more opaque and denser white, and generally it is thickened. In this form of the disease I have never seen erosion of the articular cartilage, but sometimes it is thinned where the fibrous adhesions are thick, dense, and well organized, probably due to the encroachment of the inflammatory tissue, at one period of its existence highly vascular, on the non-vascularized tissue of the cartilage. The cartilage, so far from breaking down, as in suppurative changes, tends to become calcareous; of the constancy and characteristic features of the calcification not much can be affirmed, since in so many of the cases

we are dealing with a tissue which from the age of the patient has a natural tendency in this direction.

The inflammatory thickening tends to extend, in well-marked cases, on the one hand, to the periosteal surfaces of the adjacent bones, and on the other to the sheaths and bursæ of the tendons, and even to the tendinous structures themselves, and calcification may make its appearance in both of these positions.

Of the nature, affiliations, and tendencies of chronic articular rheumatism, examined in the light of its morbid appearances solely, there are several conclusions to be drawn. Nothing in the appearances throws much light on the nature of rheumatism—and the same has already been said of acute rheumatism—but there is no discredit thrown on their rheumatic character, if the pathology of this disease has been read aright. The affiliations are all toward the same character of inflammation as we have to deal with in unquestioned rheumatism—an inflammation attended with a severe, though not high grade or active class of inflammatory phenomena—though lasting for a long period the alterations are quite disproportioned to the symptoms.

The tendencies are those of a formative character, at least so far as this process proceeds, and are away from the destructive characters seen in other forms of arthritis to which the name rheumatism and rheumatoid have become attached. And this is quite in accord with the general physical status of the patients in whom the diseases respectively make their appearance. The chronic articular rheumatisms affect people whose general health otherwise is good, and whose nutrition is well carried on, barring the effects of crippling. In the other form of arthritis, the destructive process of rheumatoid arthritis, and the general nutritive changes start below par, and continue to degenerate.

Diagnosis.

The recognition of chronic articular rheumatism is effected without much difficulty, from the history of the patient and a close scrutiny of the symptoms and course of the disease. In the cases of slow development it is probably nearly impossible to say that the condition is not one of some of the other articular maladies; but after a short time its nature becomes apparent and the question is easily solved. In cases following paroxysms of acute rheumatism the continuation of the disease is self-evident.

The nature of traumatic and tubercular arthritis should be at once recognized by the history, or by the general condition of the patient, as well as of the other viscera. The scrofulous disease, at some period at least of its development, expresses itself both locally and generally as a malady of entirely different type; it is destructive in its tendencies.

The forms of arthritis which cause confusion or difficulty in the diagnosis of this disease are certain cases of gout, rheumatoid arthritis, and the spinal arthropathies. Gout, mostly a disease of paroxysmal character in its articular manifestations, does under certain circumstances lead to deformity and thickenings, attended with pain, discomfort, and inflammation of a very low grade affecting one or many joints; and it is matter of the very greatest difficulty to distinguish between these two forms of articular disease. Rheumatoid arthritis, besides presenting a different general condition of the health, shows more joints affected, and pretty rapidly leads to deformities. As already alluded to, it is a malady with a different type of nutrition. The distinction often spoken of, that rheumatoid arthritis does not proceed from an acute attack, is not altogether a very serviceable criterion. The differential diagnosis will be recognized after the description of this disease is familiar.

The spinal arthropathies nearly always, and usually early in their course, present symptoms which no one, unless very careless in ascertaining their history, could fail to see were indicative of spinal cord disease rather than an articular malady. The cerebral arthropathies are at once self-evident, or rapidly make themselves known.

The prognosis is never favorable to complete recovery. Cases do, however, become entirely well; but this is effected more through changes in the character of nutrition than from the influence of medication. The degree of comfort to be obtained is very largely influenced by the circumstances of the patients obtaining for them immunity from unfavorable conditions, and very great temporary, or even permanent relief, if combined with these circumstances, can be effected by proper hygiene and drugs.

Treatment.

Probably the large majority of patients suffering from this disease are on foot and about, and a very large element of their treatment consists in shielding them from further harm. This is effected by removing them from, and warning them against, the causes which develop rheumatism—

cold and damp ; for those who have suffered from the malady in any form the necessity of over-exertion and insufficient protection by clothing are paramount considerations. The methods of enforcing these measures must be adapted to each case.

To all those who discard the rheumatic nature of the disease, and believe in its purely local character, the administration of internal remedies is regarded as useless, at least so far as such measures are of an anti-rheumatic character. If no rheumatic diathesis exists, there is no purpose to be gained in giving remedies whose object is to change or do away with the diathesis. But all the same, neither believing in a diathesis, a specific poison, nor a minute organism which the remedies are supposed to change, neutralize, or destroy, there is great gain to be obtained by internal medication.

No one nowadays fails to recognize the fact that processes of nutrition, excessive or diminished, or even normal, can be modified by the use of drugs. We do not know much of these effects except in a few instances but we seem to be on the brink of finding out a great deal more. Hitherto, in studying the action of medicine, we have been contented with certain catchwords which we have accepted as knowledge. We have been contented to say a drug was alterative, without being interested in how it "altered." When we know whether a morbid process is affected by sending more blood, or by diminishing the supply to the part, we shall have an item of knowledge of the right sort, and be able to influence inflammatory actions similar in character to that of chronic rheumatism. In this way internal treatment is, and is likely to become, a matter of still greater importance. By such agencies we may hope to quell local manifestations of a morbid process without detriment to normal nutrition, since function tends of itself to return to its usual channels and morbid processes are more easily affected than normal ones.

Hitherto various arthritic, rheumatic, or gouty specifics have always been considered essential in the treatment of chronic articular rheumatism, and nearly all the lines of treatment recommended for acute rheumatism have been employed with a degree of vigor proportioned to the decreased violence of the symptoms. These procedures have returned but little results, or have been considered valueless. Colchicum or its derivatives, alone of all of them, to-day maintains itself in higher estimation than the rest, but it is by no means certain in its effects. The more active the symptoms the better the results obtained from the use of remedies pro-

posed for the acute form of the disease, and the lower the grade of symptoms the less do our efforts seem to be of avail.

In addition to these remedies we find recommended, and very constantly employed, measures calculated, under ordinary conditions, to improve nutrition and increase the appetite. It is true that many patients, suffering from this complaint, have impairment of appetite, take less food than usual, and show often considerable emaciation; but this condition is to be looked upon as a secondary effect, the result of confinement brought about by crippling. Very considerable numbers of patients are, however, well nourished. These remedies are appropriate when the general condition of the patient seems to indicate them, and they are often essential to improvement of the local condition. They cannot, however, be looked upon as specific in their action on the local inflammation or the conditions resulting from it. In fact, they are often wrongly administered, and fail to do good because they cannot be absorbed in the disordered state of digestion so often concurrent with this disease. Nevertheless, cod-liver oil, quinine, and arsenic often are most useful The latter is held by some to exercise also a direct effect on the articular inflammation.

Of the remedies recommended in rheumatic inflammation, propylamin, which has been used in both the acute and chronic forms, seems often to exercise a most striking influence. It will sometimes cause the pains, even when severe, to disappear very rapidly, and the swelling, and often slight heat of the articulation, are lessened. This medicine, which is more reliable in the form of hydrochlorate of propylamin, has a better effect in the chronic cases attended with exacerbations than in the slowly developed continuous cases attended with deformity and thickenings, though even in the latter it is not without beneficial results.

The iodides, in the form of iodine of potash, are a very universally applied remedy. It gives the best results in cases attended with chronic thickening and swelling—cases without a tendency to marked exacerbations—and is to be avoided entirely when any febrile phenomena are devoloped. This medicine is very frequently combined with the bromides, and the latter are of great service often when given alone.

Recently the salicyl compounds recommended in acute rheumatism have been used also in the chronic form of the disease. Their effects have been reported to be favorable, but their action is less striking and conspicuous than in the acute malady. In a number of cases I have found them of service, but this efficiency is not necessarily to be attributed to

any specific or germicide action. Salicylic acid can be supposed to be a drug which, by its action on the nervous system, is capable, under certain conditions, of changing the blood-supply not only in the joints but in other organs. By this action the perverted function of the rheumatic process may be modified. This drug, like most others, except iodine, shows little or no influence in removing the thickenings around the joints, and consequently the best results are obtained with it in cases showing active phenomena.

Local Applications.—Remedies applied over and around the joints have received more general approbation than internal medication. Their effects are most serviceable, and they are essential and necessary in the treatment. Their application is to be directed according to general principles: 1st, for the relief of pain and stiffness; 2d, to promote the absorption and removal of inflammatory thickening; and 3d, to prevent the recurrence or continuance of the inflammatory process.

The haphazard manner in which local applications have been made of remedies, recommended as "good for rheumatism," renders the work of reducing them to order, and explaining their appropriateness one of the greatest difficulty. The method of their use, and the mode of applying many very serviceable remedies are so entirely wrong that the object expected to be obtained is defeated. Thus an anodyne liniment is often rubbed on so roughly as to increase the pain and soreness; a sedative, cooling lotion is applied in such manner as to increase the heat and inflammation; and a counter-irritant irritates so deeply the tissues that the swelling is permanently increased.

Every one has a favorite remedy to meet certain indications, and the good results obtained are proportional to the skill with which it is applied to its appropriate condition. This skill is obtained by experience and by a proper recognition of the pathological condition, and a knowledge of these cannot be conveyed by words.

Anodyne liniment and morphia, or morphia with atropia, usually suffice for the relief of pain, and are indicated when this symptom becomes severe. Very often they fail, and a resort to heat and moisture is necessary. This may be carried out by wet cloths covered by impermeable sheathings or by local or general baths of simple or saline waters. Some one of these measures usually suffice to render the patient comfortable.

The deformity and thickenings represent two distinct conditions: first, the enlargement or swelling due to a very slow and low grade of in-

flammation; second, the thickened tissues left behind, which result from the inflammatory process. The first is histologically composed of comparatively soft tissue materials and considerable fluid; the second is pretty hard, firm, well-developed fibrous tissue, and these two conditions are found mingled in every degree of variety, and our success in treatment depends on the recognition of the preponderance of one condition over the other.

The typical conditions of each of these are to be met by unlike, often opposed, modes of procedure. A sub-inflammation rubbed and handled becomes greater; a fibrous tissue has its circulation uninfluenced by heat and moisture, and though it may be thus softened it soon becomes as hard and fibrous as before.

The first of these conditions is oftentimes relieved and dissipated by iodine and mercurial applications, provided the inflammatory process is not of a marked character, as this is then apt to be increased by their irritation. Their effect is produced apparently in preventing the further development of the new cellular elements into fibrous tissue. The cells which pack the tissue are destroyed, or failing to develop are carried away, and thus the disturbed function of the circulation is restored to its normal status. As long as the circulation is sluggish new formation is continued, and hence tends even to increase itself. The influence of iodine as a mild counter-irritant is not to be despised even when tissues have become firm and fibrous.

In the softer condition of the tissue the prevention of cell increase and development may be arrived at by a different method, namely, by lessening the flow of blood to the affected parts. This method may be carried out by general remedies which lower the tone of the circulation, such as aconite, etc.; possibly the salicyl and other compounds effect their good results in this manner. Or the means may be of a local character; of these firm compression of the limbs, or of a particular joint (where it can be done skilfully) has served me the best. An elastic band of rubber to the knee joint or to the leg is very useful, and simple bandage to the wrist and arm are very comfortable, and in time change the whole character of the disease. Contractile collodion may be used for the fingers.

In opposition to this latter method is another which at times in this softened condition of the tissues serves an equally useful purpose, though it probably is more frequently called for in the second class of conditions than in the first. Instead of removing the swelling and thickening, the

results of the sub-inflammation, by compression lessening the amount of blood, or by the application of iodine, breaking down the forming tissue, we seek to effect the object by increasing the flow of blood, endeavoring to restore the normal condition by rapid changes of nutrition.

This object may sometimes be brought about by heat, more frequently by dry heat than the moist. Frictions with the dry hand may be tried; but just here we are on the brink of increasing the inflammation by rubbing, and all such measures must be advised and carried out with the utmost skill. The counter-irritant method by blisters of small size—in truth flying blisters—carried successfully over the whole periphery of the articulation are frequently seen to be followed by the very best results. They have the great advantage of producing their effects in a circumscribed manner, and if the result does not seem favorable they can be discontinued. When favorable, the surface of the joint over the area vesicated, even very superficially—the more superficial the better—will appear like a cicatricial spot, depressed beneath the surrounding by the removal of elements produced by the rheumatic sub-inflammatory process; nothing can be more striking than their effect as occasionally seen. The same effect may be occasioned by acupuncture. This method is even more dainty, and its influence more circumscribed than that of blistering. It often gives very beautiful results. It is not a cruel procedure if done skilfully, and the easement of pain, even from a single puncture, far more than compensates the momentary discomfort from the needle. The needle possibly produces its effect by nerve-impression in the timid, and the threatened puncturing maintains the vaso-motor influence; or possibly results may follow later from cicatricial changes in the deeper tissues.

In the hardened condition of the tissues, the second class above referred to, the surroundings of the joints are much more difficult to influence for good or bad, and our remedies may be exhibited with greater vigor and with less danger of doing evil. It is just as necessary, as in the other class of conditions, to have always clearly in mind the result we expect to effect if we are to do any good.

Our object here is to soften the firm, cicatricial-like tissue of the ligaments and the surrounding parts, to prevent their pressing on the nerve-filaments which course through or in their neighborhood, and which this tissue squeezes very painfully when it swells even very slightly under influence of over-exercise or the changes of damp weather. It is also desirable by softening it to promote the freedom of movement of the muscles and

bones which are concerned in the movements of the affected articulation. For the changes in the cartilages, when roughened, we can do little except wait for time and exercise to promote the accommodation of altered surfaces to each other so that they will again move with freedom and smoothness.

This object can sometimes be effected by one of two methods, or by a combined use of both. First, by promoting tissue-change and absorption by increased vascularity; second, failing or abandoning the accomplishment of the first, to manipulate the tissues into a supple condition to allow of a partial restoration to normal activity and movement.

Dry heat, moist heat, general and local baths, or heat alternated rapidly with cold can sometimes be depended on to cause and maintain an increased vascularity, or at least a more rapid flow of blood. The constant use of counter-irritants, or more properly irritants producing a deep and lasting impression, are essential, and frequently the only method open to us to reach the desired end. Their use must be intermittent, else their effect is simply to drive the blood away. A temporary swelling of the parts must be induced and allowed to subside before a renewed application is made. During the interval of subsidence the treatment may be directed as indicated for the softer state of the tissues. The remedies proposed for producing irritation range from a slight vesication through all grades to actual cautery, and great circumspection is required in making a suitable selection.

The second method consists in massage, manipulation, and of passive and active movements of the joint and contiguous limbs. These means may be employed, so far as possible, simultaneously with procedures already recommended, but they should always be used in the intervals between such treatment, so that the two constantly alternate with each other. In addition to the massage as ordinarily practised, the use of oleaginous compounds is especially advantageous. An easily flowing oil is better adapted than a thick ointment, and the former may be medicated if desired. The use of this means is unquestioned, and its advantages have been exemplified in all periods of history, from the Grecian athlete to the modern dancing-master.

This method is too frequently disregarded in the treatment of this disease; its use cannot be too strongly advocated for the purposes for which it is indicated, but its practice must be restricted to these, for its value is limited to these alone. Hitherto the direction "to rub," so easily given

and so little understood apparently, has constantly been doing more harm than good; it has been resorted to to employ the hands and thoughts of the sufferer in the intervals between the medical visits, and the patient resorts to it unadvisedly unless strictly forbidden.

The opposite of this method, enforced rest by fixed apparatus or other means, is distinctly contra-indicated in chronic articular rheumatism, unless the condition approaches an acute stage of inflammation. Active movements often are impossible on account of pain, but passive movements carefully made are better in this condition than long periods of immobility, and are even serviceable in the relief of the pain.

The subject of the natural spring waters and baths, which so often are made useful in this connection, is a long one, and a mere list of springs without special directions for their use would be of slight value.

In speaking of patients suffering from chronic rheumatism, the directions for treatment have been given irrespective of the presence of any other morbid condition or visceral complication of rheumatic or other origin. Great care is necessary to be exercised in applying many of the remedies which have been recommended in cases in which cardiac disease exists; this is especially true of the baths. This complication, through its effects on the general circulation, renders many of the local measures abortive, since it becomes impossible to correct the defective heart's action. Pulmonary disease, such as emphysema and chronic bronchitis, which are no infrequent accompaniments of chronic rheumatism, are nearly equally harmful with valvular disease. Other diseases require to be met according to their special indications, but, in addition to these mentioned, no others are of constant or even frequent occurrence.

Another condition which I have frequently met with, I deem it important to mention. I know of no casual connection of it with chronic rheumatism, yet the relief of its symptoms or appearances is so frequently followed with removal of the articular conditions that it may prove interesting, and it is unquestionably beneficial to the patient.

The condition forms one of the lesser group of the disease, and by many it would unquestionably be banished altogether from among its number, thence to find no resting place whatsoever in any nosological catalogue and to remain unbenefited. The number of instances of the condition are large, but do not come to the attention of the physician, partly because the patients think it a small ailment, not worthy of notice (perhaps they have been put off on former occasions with directions to use some liniment),

partly because its nature has never been distinctly apprehended. I have no very satisfactory statement to furnish of its pathology, but of its reality I am well convinced.

It occurs not unfrequently in those who have suffered from rheumatic paroxysms, but also in those who have never suffered in this manner. The uncertain pains and stiffness are the same as usual, but deformity or thickening I have not seen developed. In all respects, therefore, but the latter, it shows its relationship or affinity to chronic rheumatism, and hence I have included this statement here, this condition which I have found that always goes along with the pains is the very positive evidence of an abnormal state of the mucous membrane. Ordinarily the patients do not complain of disturbed digestion, and very often no symptoms of such disorder can be elicited by close questioning; they usually describe their health as perfect, but perhaps not robust, though this may be accounted for by their being past middle life; it occurs, however, early in life also. An examination will show, even if no complaint of digestive disturbance is made, that the tongue is coated or dry and leathery, that it is swollen and marked by the teeth along its borders; in addition, there is evidence of some sort or other that this condition is present throughout the digestive tract. We have then an instance of the very lowest form of a catarrhal irritation, or even an inflammation, so low in degree, however, that it does not excite the usual dyspeptic symptoms which the higher grades always produce.

This inflammatory condition, vast in extent, though low in grade, we may readily suppose is capable of disturbing the functional activity of other parts of the organism. That it does on occasions influence the sensory organs, for example the eye, we well know, and that it may disturb the cerebral circulation, producing aural phenomena and the variety of vertigo described as gastric, we have ample evidence. Is it less probable that it may also produce nutritive changes in and around the joints in patients whose point of least resistance is situated in these parts, those with the so-called arthritic diathesis? I think not, and I have found that whatever would remove the catarrhal condition banishes the articular symptoms, as it does the ocular and cerebral phenomena.

This suggestion of the pathology of the condition does not, I consider, lend a probability to any humoral views of the nature of this complaint or of acute rheumatism. In the first place, we have little or no evidence of such disorders of digestion as would be productive of noxious matters in

the digestive tract furnishing the materies morbi; secondly, the relief is often so sudden under the treatment to be suggested that no opportunity is afforded of purging the blood of poisonous or irritating matters. On the other hand, everything about the condition favors the idea of the production of it by tissue changes or alteration of function through nervous influences reflected from one position to another. Furthermore, I have nothing to offer in opposition to the view which traces both of these irritative or inflammatory conditions to one common source or seat.

The means of relief for this condition consists in the endeavor to find a remedy or a plan of procedure which removes the catarrhal state of the mucous membrane. Many such remedies are found among the numerous groups of chloride compounds. Muriate of ammonia, chloride of arsenic, and several other of the metallic chlorides are often useful. The best results are to be obtained from the mild chloride of mercury given in very minute doses and at frequent intervals. The diet requires but little change, unless some dyspeptic symptoms are present, or unless the dietary is manifestly improper. The use of the corrosive chloride of mercury in still smaller doses (one-fortieth of a grain) and at less frequent intervals than the former, has also yielded favorable issues. The effect of neither of these drugs can be attributed to their mercurial action, since given for a period of a month in the small doses and at very frequent intervals, even two or three hours apart, no mercurial action is seen to result from their use. Mercurials have been recommended for chronic, as well as acute rheumatism, but the dose has always been a large one, mercurialism has been induced, and the results have not been favorable. In the small doses their effects are very different, and nearly always favorable, and prompt when the morbid condition does not exist for a very long period. No striking symptoms or phenomena are brought about beyond the removal of the catarrhal condition, and in this instance the relief of the pain.

CHAPTER XVIII.

GONORRHŒAL RHEUMATISM.

Liability to, and Frequency of its Occurrence—Symptoms, Course, and Duration—Its Results—Treatment.

This disease did not pass without recognition by many of even the older writers, but their observations on the subject afford very little information which is useful. Among English writers Cooper was the first to so graphically describe it that general attention was directed to it, although he was not the first to observe and mention it. It has attracted a large share of attention among French writers. But from all the very thorough discussion which it has received at various times very little has been added to our knowledge of the malady given in its early description.

The condition has received its name from the two facts that an articular inflammation, not dissimilar to a rheumatic one, makes its appearance, and that this inflammation comes on sometimes during an attack of gonorrhœa. The frequently observed concurrence of these two inflammations led to an establishment of a separate form and to the distinction of this articular inflammation from the ordinary rheumatism. Previously the rheumatic phenomena were classed with acute or chronic rheumatism according to the grade of its severity, and the urethral discharge, when not regarded as an accidental or concurrent attack, was thought to be a rheumatic localization. In the latter case the rheumatism was looked upon as the cause of the urethral discharge.

At present the gonorrhœal urethral inflammation is regarded as the cause of the articular disease, although there is some difference of opinion in respect to its mode of action in producing the joint inflammation. The majority of observers favor the pyæmic mode of causation, and I think rightly so. Others refer the effects to the results of reflex irritation, and many instances have been cited in support of this view.

The occurrence of the articular conditions does not seem to require for its production any of the usual exciting causes which lead to the ordi-

nary rheumatic attacks. The mode of life, the circumstances of the patient, and the general surroundings, as well as climate, seasons, etc., do not seem to have any influence in its causation. A patient exposed to cold, irregularity of habits, and intemperance, may thereby develop an attack which otherwise would be escaped, but these or similar conditions are not necessary concomitants of its development.

Men suffer gonorrhœal rheumatism very much more frequently than women, and this preponderance is relatively greater than the proportion of attacks of gonorrhœa in the two sexes. Women having gonorrhœa show less liability to the articular disease, and this exemption has been accounted for by the less sensitiveness and greater thickness of the mucous membrane of the vagina and urethra in women than the urethral tissues in man.

The period of life during which the disease occurs is limited by obvious circumstances; in men it has been noted to occur shortly after the age of puberty, and from this time on until past middle life; in women the cases which I have seen have been in the quite young or those who have been abused sexually at the time they have acquired the gonorrhœa. In women the vaginitis may be a subsidiary feature of the disease, and they may make no complaint of its symptoms.

The frequency of its occurrence in relation to the whole number of cases of gonorrhœa we have no means of determining, though we have felt sure the proportion is very small; some patients develop the rheumatic symptoms on the occasion of each successive attack of gonorrhœa, and having once had gonorrhœal rheumatism the articular inflammation returns with every urethral irritation or excitement.

The condition of the general health has probably more influence on its development than this circumstance seems to have in acute rheumatism. The weakly, debilitated, and exhausted have, in my experience, furnished the larger number of cases. The disease is, however, met with in those apparently in robust health, excepting, of course, the gonorrhœal complaint.

We have no facts by which to show conclusively the mode of causation of the articulation and its direct connection with the specific urethritis. The original theoretical explanation of the disease was that the inflammation was transferred from one place to another by metastasis; but this theory has fallen to pieces in reference to every morbid action, and is now abandoned. Other theories have been proposed, but have passed away with the hypothetical notions on which they were based; for example, organic sympathy

The two theories which to-day divide attention are that of reflected irritation and the pyæmic theory. The former of these was proposed by Fournier, and was based on his observations that other irritations besides the gonorrhœal inflammation developed articular symptoms ; for example, catheterism and other inflammations of the urethra of a non-venereal origin. Such cases, however, have been very infrequently reported, and when they do occur the phenomena, especially the general symptoms, but likewise the articular ones, are not typical of the usual cases of gonorrhœal rheumatism. Reflex irritations, when such truly occur, are, as a rule, promptly developed and usually as promptly subside on the removal or lessening of the cause. This result is not the characteristic of the course of this disease, otherwise the articulation should come and go when the urethral disease had reached its acme. And then, too, while irritations at distant parts are seen commonly enough through nerve-channels, inflammation requires for its production something more forcible—it requires irritation at the root or source of the peripheral nerve which is distributed to the seat of the inflammation.

The spinal arthropathies are seen when the irritation is in the cord itself, and not under the influence of reflected action.

The symptoms and course of the disease favor the pyæmic theory much more strongly than any other. While the phenomena are in accord with this view in by far the larger number of cases, the evidence in its favor is not without its defects. In the first place, it is very infrequent to find pyæmic or septicæmic symptoms of any sort result from a mucous membrane inflammation, unless such inflammation is very long continued, very extensive, or else some purulent depot is formed in the neighboring tissue, organs, or lymphatic glands. Secondly, no evidences of a gonorrhœal pyæmic disease in other organs have been pointed out or are known to exist. Neither are the articular inflammations widespread or numerous, and the changes in joint-structures are by no means typical of pyæmic inflammation. Still, withal, the symptoms and their course in the absence of any extensive data of the morbid anatomy of the disease seem much better explained on this ground than any other as yet proposed.

In the absence of post-mortem details, it would be quite out of place to discuss further the possibility and the method of infection of this pyæmic process. Our knowledge of the state of the joints in the early stages of the disease is likewise very limited, although the facts, so far as known, point to it as an inflammation chiefly affecting the synovial membrane and

also the articular cartilages. The peri-articular structures seem to be but little affected by marked inflammation, although they exhibit often great swelling. The inflammatory effusion very frequently shows a tendency to fibrinous deposits. In the later stages, the joints sometimes present a condition of false anchylosis. The disease therefore belongs to the class of arthro-meningo-chondritis.

Symptoms, Course, and Duration—its Results.

At some time during the continuance of the gonorrhœal urethritis, the patient feels a sense of heaviness, aching, stiffness, or sharp pain (the sensation is variously described) in one or other of the joints. The knee-joints are far more commonly the first to be affected, and the records show that the left knee is the favorite one, and it is not uncommon for the disease to be limited to these articulations, and even to one of them. Any other single joint may form the sole localization of the inflammation. In some cases, however, a number of joints may be successively involved, or all at once at the outset of disease.

The urethral inflammation and discharge have usually existed some days, at least they are well developed, and it may be subsiding before the articular disease commences. I have seen a patient among my hospital cases, who was entirely unaware of having, or of having had, gonorrhœa, and who on admission to the ward was suffering from a monarthral inflammation of some days' standing. The urethral discharge was pointed out to him, and he gave a consistent account of its acquirement twelve days before the articular symptoms were noted. A young woman, with an articular inflammation, who was unaware of having gonorrhœa, and who did not suffer inconvenience from it, gave the history of the probable contraction of the vaginal disease, and of great sexual excess at the time of its acquirement. She had felt local discomfort from the excessive frequency of coition, but none of the usual symptoms of gonorrhœa in young women.

The articular symptoms come without usually any of the common exposure which precedes acute or subacute rheumatism. And the patient frequently tells of waking in the morning with the sensation in the knee, or perhaps some other joints, which prevents rising or using of the affected limb. The sensations increase only slowly in severity, and sometimes do not increase, and the parts do not at first exhibit any alterations in their external appearance. Movement is more painful than is handling

of the joint, and the aching not unfrequently extends into the limb, at first above the implicated joint, and later below this part also. The redness, so constant in acute and subacute rheumatism, is often, in fact most generally, absent through the whole course of the inflammation.

The inflammatory process is, throughout its course, of a subacute type, and is generally slow in development. It is often many days in reaching its height, but it is very much increased and rapidly hurried forward in its course by movement, especially walking, when the knee is involved, and pressure in addition to movement is inflicted on lining structures of the joint cavity. The swelling is consequently several days in arriving at any very noticeable degree of distention of the capsule of the joint. While the fluid is increasing in amount, the aching is often intolerable, preventing sleep without anodynes, and is of deep-seated character. This symptom does not seem to cause the same degree of restlessness or other nervous symptoms as is the case often in acute rheumatism. The patient is wont to be apathetic.

The general symptoms in the early stage are usually slight, the temperature is normal, or but little elevated, and always subfebrile (99° F.). I have known the thermometer to indicate 102° F., taken in the axilla, on the fourth day of the attack, when only one knee-joint was affected, and I have seen it a degree higher when the knee and elbow were simultaneously involved.

Sweating is generally not a marked feature, but in severe cases, and in the debilitated, I have seen it presenting strikingly pyæmic characteristics. In these cases, too, marked rises and falls of temperature without the involvement of new joints, or even an exacerbation of inflammation in ones already affected are seen. But this feature is not striking or common in the usual cases. In fact, in gonorrhœal rheumatism, the flitting from one joint to another is of rare occurrence; it never occurs in the manner so noticeable in acute rheumatism. The joints, if successively involved, pursue the inflammatory course irrespective of each other, and little, if at all, modified by the general symptoms.

The inflammation, when at its height, distends the joint implicated often to a marked degree, so that distinct fluctuation is perceptible, and if it is the knee-joint, the patella is floated upward. The character of the fluctuation apparently varies with the sort of fluid which is present in the joint-cavity; at all events, sometimes the fluctuation is tense, elastic, and easily produced, as though the effusions were thin, and at other times

doughy, flabby, and only slightly perceptible, apparently from a thick, sero-fibrinous fluid. Of course these distinctions may be modified by the conditions of the coverings of the joint. The skin over and adjacent to the articulation is sometimes œdematous, at others, nearly unchanged in appearance, and almost never tense, smooth, or shining.

The articular inflammation runs an indefinite course and may be very prolonged, or if the inflammatory symptoms cease the effusion may remain for a long period. It is always of some weeks' duration and it may be months before the joint returns to normal conditions. There are two unfavorable terminations to the attack : the one where a chronic effusion remains long after health is apparently restored, which resists our efforts for its removal, the other in which apparently the fibrinous element predominated in the effusion, and where as a result great stiffness and even a more or less complete fibrous anchylosis results ; the latter condition produces great crepitation or crackling on any movement of the limb, and it may be perceived on moving the patella with the fingers. The joints show no tendency to suppurate, although pus may form in the connective tissue in the neighborhood ; possibly the latter is due to injudicious treatment or severe and ill-applied remedies, since the inflammation in this tissue is rarely severe. The urethral discharge has commonly ceased before the articular symptoms have disappeared, but not infrequently a gleet may remain.

Recurrence of gonorrhœal rheumatism does not come without the renewal of its cause, and the course toward recovery is continuous though slow, and pauses often come with complete inaction. Except through imprudence, from inattention or undue disturbance of the joint, the inflammation rarely tends to return after partial or complete subsidence. Another attack of gonorrhœa or other marked irritation or inflammation of the urethra is perhaps to be regarded as a more efficient cause of a return of the articular symptoms than the primary urethritis, or at least that the joint once subjected to the inflammatory changes is more likely to have its nutritive activity disturbed than prior to such occurrence. Of this, however, it is difficult to be positive.

Second attacks of gonorrhœal rheumatism are probably less common than a recurrence of ordinary rheumatism ; possibly because its exciting cause can be more surely avoided. When second attacks come they are said to be attended with less marked inflammatory phenomena, but the joints are more likely to continue longer affected, to be more stiffened and even permanently affected than in primary attacks. It is said also that

more joints are liable to become involved, even to the disease spreading all over the skeleton. Such statements must be scrutinized very carefully, and it is certain that in many of the older cases the disease has been confounded with both acute articular rheumatism and with arthritis deformans. The possibility must be borne in mind that some patients, with a stronger rheumatic susceptibility, may be suffering the results of illicit intercourse and who pretty constantly during the activity of the virile function have a gonorrhœal inflammation.

The secondary localizations of the disease are, unlike acute rheumatism, rare. The eye and its appendages may become affected, and this fact occasionally serves to call our attention to the character of what might otherwise be an obscure case of articular inflammation. The eye affection, however, seems to be a direct transference or inoculation of the conjunctiva by the irritant pus from the urethra being carried by the fingers or clothing to the palpebral fissure. It does not appear to be an inflammation of the sclerotic, such as is seen resulting from ordinary rheumatic causes.

The other serous membranes do not show a tendency to become the seat of inflammatory changes as in acute rheumatism, and the valves of the heart do not suffer from endocarditis. Very recently, however, one or two apparently authentic cases have been reported of gonorrhœal endocarditis. If this fact should be confirmed it would strengthen the pyæmic view of the nature of the disease, and if we believe in the pyæmic theory we should not be surprised at the occurrence of both the endocardial inflammation and of that of the serous membranes. It is a fact, however, that these complications rarely have to be combated.

The diagnosis and prognosis of the disease are sufficiently evident from the preceding remarks. The certainty of the former depends on obtaining a history, or on the presence of the urethral discharge. The prognosis is never unfavorable to life; even the prolonged duration does not lead to fatal or extreme exhaustion. A crippled joint may in time be induced to resume its function.

Treatment.

We are here presented with two considerations, first what shall be done for the gonorrhœa, and second according to what principles shall our remedies be directed for the relief of the articular inflammation. We may in some cases be called upon to treat the general condition of health.

In respect to measures directed against the urethral discharge, very

frequently the advice has been given to do nothing for it lest the remedies or the means of their application serve to increase the irritation of the mucous membrane, and thus an aggravation of the articular disease be brought about. Surely if this advice is sound we must either doubt the value of the remedies in gonorrhœa, or give up in great measure our pyæmic theory of the nature of the disease. If this cautious advice means merely that our local applications to the urethra or those internally administered shall be of such character as not to increase the irritation, the directions unquestionably apply equally in gonorrhœal urethritis as in gonorrhœal rheumatism.

If we believe that the disease is of pyæmic origin, that it is caused in some manner by the pent-up discharges of the urethral mucous membrane, surely it cannot but be proper to remove the inflammatory condition or wash away the discharge. Under ordinary circumstances, patients and their physician are desirous of "stopping" the discharge immediately, and remedies are often exhibited which do more harm than good, but in gonorrhœal rheumatism the urgency is much less, and a mild astringent injection or even a simple saline one used with a view to cleanliness generally will meet all the indications. The enforced rest in bed is exceedingly beneficial.

The development of the rheumatic symptoms has not been thought to be in any way due to the methods instituted for the cure of the gonorrhœa; cases occur after all modes of treatment as well as without treatment; neither can the occurrence be charged to the neglect of treatment. As a practical fact, so many cases of the articular complaint come under treatment, both among hospital and private cases, so late in the gonorrhœal disease that the discharge is lessening and with or without previous treatment is disappearing, so that no remedies are required. Frequently it must occur in private cases, that, owing to the inability of the patient to attend to himself, to the absence of a proper attendant, or for the purpose of concealment, nothing can be done for this inflammation.

The joint disease does not seem to differ from any other inflammation or inflammatory effusion in the cavity of these structures; it is not a specific disease, although it is due to a *special* cause. The treatment must be guided accordingly, and directed to allaying the inflammation, to the promotion of the absorption of the effusion subsequently, and to the restoration of the movements of the joint if they become impeded by adhesions.

Many things have been recommended, as though they were specific remedies, and others have been applied in so haphazard a manner, or irrespective of the requirements or stage of the inflammatory process, that,

judging from the records of some published cases, the disease has been prolonged beyond the time in which it would probably have recovered unassisted. The complaint is a stubborn one most generally, and requires assistance very judiciously applied.

Remembering the disease is an inflammation which, untreated, usually runs a moderate course, it does not appear judicious to make use of depletion, either local or general. The local depletion by leeches or scarified cups is apt to be harmful in the thinly covered joints such as the knee; perhaps in the deeply seated ones, as the hip or shoulder, which, owing to their thicker covering can be less easily affected by other remedies, bloodletting may find its uses, and for the same reason is less likely to do harm. From the use of leeches I have seen both peri- and intra-articular suppuration, and from the use of the scarified cups, both an increase of pain, inflammation, and superficial suppuration. Besides its liability to harm, the mode of procedure is certainly not frequently required.

Cold, applied in the form of ice, is likewise not usually indicated. Cooling by evaporating lotions is much more frequently serviceable, and this may easily be carried out by a lead-water lotion.

Rest is essential, and the limb should be elevated when this can be done. I have found a bed of pillows raised above the bed quite as useful and much more comfortable than any fixed apparatus or splint which requires bandaging. When the shoulder is affected, but little can be done by these means, but a pillow, or pillow and splint, is easily arranged for the elbow. The knee-joint is much more frequently affected than any other, and I have met this indication for it by a light extension apparatus attached to the foot and ankle, with the limb properly elevated on the pillow and allowed to bend at the knee, since this is the most comfortable position for the joint when an effusion is present in it. The slight extension serves not merely to keep the limb quiet during sleep, but also to separate very slightly the inflamed surfaces. It is to be recommended as a source of ease to the patient; a similar appliance might be found of use when the hip is affected. Splints must be used with caution, since their fixation of joint favors a false anchylosis, unless their angle is frequently changed.

An application which has served well in the earlier stages of the inflammation—in fact as long as the inflammatory process continues—is a mixture of lead-water and laudanum. The full strength of these drugs in equal proportion or slightly diluted with water is to be used. Let a cloth or piece of lint be saturated with the mixture, and placed over the in-

flamed joint. This may be left uncovered, and thus serve as an evaporating lotion, but more generally it is well to cover the whole with an impermeable sheathing of gutta-percha. The closeness with which this is sealed up can depend on the degree of warmth developed; if it becomes too warm the pain may be increased; but later, as the inflammation abates, a bandage exercising slight pressure can be applied with advantage. Under this treatment the inflammation will subside, and sometimes all the effusion become absorbed.

After the inflammation has ceased—it may be a long time—a considerable effusion very frequently continues present, and is often stubborn to treatment. For this condition various measures may be put in force, according to causes which seem to maintain or to continue its presence. Rest is just as essential now as at any period, although passive movements are often useful or necessary. Iodine in the form of one of its solutions, or perhaps as an ointment, mercurial ointment or plaster, the various means of counter-irritation, or alteratives and absorbents will find their use. Local and general hot water, or vapor or air baths are highly recommended in some cases. The measures would seem to be more useful when the condition was prolonged by a low grade of inflammation. The more stubborn cases and the simple dropsies of the joint will rarely resist the application of pressure carried out by straps of adhesive or lead plaster, or by bandages; these can be combined with some one of the above remedies.

I have seen several cases where the local condition seemed to be maintained or prolonged by the feeble or exhausted condition of the patient, due either directly to the disease or to previous debaucheries; sometimes the patient is very feeble, even when the joints recover pretty promptly. In the former cases it is necessary to attend to the general condition in order to insure a favorable result. And in every patient who does not exhibit robust health, it is safer, especially remembering the pyæmic character of the disease, to administer tonic remedies. Quinine and tincture of chloride of iron meet these indications, and they should be given in full doses. Other symptoms and conditions are to be attended to as they arise, according to general principles.

No case need be abandoned as hopeless, even should anchylosis occur, and in nearly all cases it should be prevented from occurring by early and constant daily movements of the affected joint. The adhesions, if they become firm, can be readily broken up under an anæsthetic, and this should be done early and thoroughly, and the treatment continued as indicated.

CHAPTER XIX.

GOUT.

Causes and Causal Factors.—Age, Sex, Heredity, Customs, and Nationality.—Varieties.—Symptoms, Course, and Duration.—Acute Gout, Chronic Gout.—Visceral Manifestations.—Pathology and Morbid Anatomy.—Diagnosis.—Prognosis.—Treatment.—Treatment of the Paroxysm.—Treatment of Chronic Gout.

Gout has, in past ages, and even up to recent times, received the greater share of attention, and been supposed to constitute the greater bulk of the cases forming the extensive arthritic group. From this group have from time to time been separated, one member after another, as their characteristic differences became recognized. We have seen that rheumatism was thus first recognized in the seventeenth century, but more than four centuries before this disease received the name of gout, and it was under this title that all the members of the arthritic group were discussed.

The derivation of the word is obscure, and I believe the work of the author, Rudolphus, from which we might have derived information on this point, who first used the term, has been lost. Its meaning is to be referred to old humoral pathology which taught that the disease was a material effused or dropped (gutta) into the joints.

The nature of gout has been always a question very much discussed, and no hotter battles have been fought than about the pathological condition which produces it. Passing by the notion of the ancients concerning the humors of an uncertain and vague character, which were looked upon by them as the cause of all forms of disease, we find that the humoral theory of the disease has always been the prevailing one, and its factors have been presented in many different aspects. The variations in the humoral theory have consisted principally in the question whether there was merely a retention of the offending material, or whether there was, in addition perhaps, an increase in the amount of it produced.

The opponents of this school of believers have found but few followers, and they have presented their views with great cogency. Cullen was the

one who traced the cause of the disease to changes in organs other than those chiefly or most strikingly affected during the paroxysm of the attack.

Recently, and chiefly through the work of Garrod, the almost universally prevalent humoral belief has become confirmed. And it is not strange that this theory should so universally receive approval, since in no other disease are the phenomena so much on the surface, and after its attack are the results so completely placed to our hand as a means of verification of the morbid process. So simple does the problem seem that, knowing the ways of nature in health and disease, this apparent simplicity is almost enough to raise the doubt whether we have actually arrived at a thorough solution of the question.

Causes and Causal Factors.

The chief etiological factor of gout is the presence in the blood and the tissues of uric acid and its compounds. How it is produced, and under what circumstances the material is retained in the organism, constitutes one of the important parts of our studies of the disease. And in the first place we must look at what are the favoring circumstances and conditions of life, such as age, sex, habit, etc., under which the morbid action makes its appearance.

Age.—Primary attacks of the disease are very much more frequent in middle life than before or after this period. There are very few attacks before the age of twenty, but such have been reported in young children, and after the age of puberty they become more frequent. After the age of forty-five and fifty primary attacks decrease in frequency, but they have been observed at a very advanced age. The declining period of life furnishes the greater number of cases of the disease, and this is the age in which its development and its changes are the most marked.

Sex.—Women have gout very much more rarely than men, and this is especially true of the articular form with its distortions. And in women it has been thought to come on later in life than in men. Some writers have explained this fact by the different peculiarities of the sexual organs, the maintenance of the menstruation through a long period of their lives, which discharge is supposed to shield them from its attacks. Some claim that in the majority of cases the disease does not make its appearance until after the climacteric period has arrived.

That men should be affected with gout more frequently than women is

evident from the fact that the habits and mode of life which are especially productive of the disease concur more frequently in the former than the latter.

Heredity.—By the old authors gout was always spoken of as an hereditary disease, and by them this was looked upon as the chief cause of its production. In fact, gout has been pointed to with more or less pride as an heirloom in the family, like a piece of plate, and as a disease which conferred more or less respectability on its sufferers. With our more democratic notions of the present time, and also the fact that a well-marked form of poor man's gout has become known, this pride has become somewhat lessened. More than once I have had the diagnosis of gout given me by a patient in a case of chronic rheumatism, and once, where to confirm the gouty nature of the complaint a bastard paternity a generation before was confessed, and a relationship with an English nobleman was claimed with some pride.

Undoubtedly gout furnishes a larger number of instances where the complaint is found in both parents and children than any other disease of the same type and character, but by no means as frequently as a number of other diseases of whose heredity no one can doubt. We have already spoken of the true basis of heredity, and in making an estimate of gout's hereditary transmissibility we must remember these characters unless we wish to be misled. It is necessary also to think of the tendency of patients to represent rheumatic or other articular affections as gout. This disease, too, is one that develops not in early life, the time we should expect its manifestation if inheritance of function and tissue, and not merely habits of life, was the determining factor in making it manifest. On the other hand, gout is unquestionably a disease which is more influenced by habit and mode of life than almost any other, and which takes considerable time for the development of its effects. Again, hereditary gout, it is claimed, is frequently seen to skip a generation, and afflict the grandchild of a gouty grandparent. This fact, if substantiated, shows a very mild or doubtful kind of heredity, and the statement is quite as truly interpreted in another way, namely, that the gout of the grandchild was in reality acquired, his parent escaping by avoiding the habits of the grandparent.

The statistics on this question, as given by Scudamore, show that in 58 cases out of 113, the disease was not known in any ancestor. "The cases of acquired gout, in which no family reference could be traced, were

to the rest as 58 to 55; and in the examples contrasted with those immediately hereditary, as 58 to 44." Eleven cases of so-called heredity were in grandparents or more distant relatives.

In the report of the commission of the French Academy of Medicine (1840), 34 out of 80 cases showed the hereditary predisposition. Garrod says, "That more than half the gouty subjects can distinctly trace their ailment to hereditary taint; and if patients in the upper class of society are exclusively selected, the percentage is found to be considerably greater."

Our opinions on the strength of the hereditary nature of the malady will vary with what we consider the essentials for the proof of its transmissibility; whether it depends on the transmission of function, or on the inheritance of habits and customs; and also whether we demand more proof of this quality than is furnished by numbers.

Customs and Nationality.—The occurrence of gout in certain nations, climates, and classes of people is a very striking feature of the ailment. The disease is rare among the natives of warm and moderate climates. This fact is strikingly seen in Europe, where the dwellers in the northern countries are almost the only sufferers. In England, the disease is more common, and has certainly attracted more attention than anywhere else. In Holland, and among the Scandinavian races it is also frequent. In this country the disease is not common, though by no means rare in its various manifestations, so that it is scarcely possible to contrast the differences in the various sections.

The differences produced by habits of ease, rich foods and drinking, as well as by regular bodily exercise, are probably more striking than any other factors in causing the disease, but no one of these acting singly is sufficient for its development.

The use of various kinds of liquors, and of a diet composed principally of meat, has been spoken of by many writers as though a proportional amount of gout existed potentially in each beverage or article of food. Unquestionably some of these are more potent than others in disordering the functions, and must be entirely avoided, or used in moderation by all who have suffered from the disease or by those who expect to escape its occurrence or repetition. Those distilled or fermented drinks, rich in alcohol and other ingredients, and food highly nitrogenized, are regarded as most likely to be baneful in their effects.

Indigestion, whether directly traceable to improper articles of diet or

arising from other causes, is frequent in gouty persons. It is not all forms of dyspepsia that are harmful, and certainly many patients suffer from this ailment all their lives without exhibiting any gouty tendencies. By many others the effects of hepatic congestion seen in many forms of digestive disturbance, especially when connected with congestion of the digestive tract, are strikingly potent in the production of gouty conditions. The state of the abdominal organs not unfrequently exists unaccompanied with symptoms which patients recognized as dyspeptic, and consequently the statement is often made that they have never felt the sensation of indigestion.

Exhausted conditions, especially those connected with the nervous system, frequently determine an onset of the gouty paroxysm, but of late it has been doubted if any nervous condition alone was capable of developing the disease. Cullen and his followers, however, looked to the nervous system as one of the prime causes of gout.

Gout in many persons shows as great a predilection for certain seasons as does rheumatism, and in probably the majority of cases the initial outburst comes at about the same time of year as do the attacks of acute rheumatism, and often for succeeding years the gouty inflammation returns at the same time that it made its appearance on the first occasion. As the morbid condition becomes established the attacks may return at quite irregular times and with greater frequency.

Lead impregnation has for a long time been observed as a predisposing cause of gout. It of course especially occurs among those who work in some one of the many occupations in which this metal is used. We know the influence which lead poisoning has in affecting the mucous membrane of the digestive tract, and the conditions of these organs when thus affected are very similar to those of gouty patients, when the disease is due to other causes.

Varieties.

In dividing the groups of symptoms, as manifested in different patients for the purpose of study into varieties, it must be remembered that gout is essentially a time-lasting or chronic malady. It is the rarest of occurrences to see a single paroxysm or a visceral phenomenon cease never to recur. It may not be so uncommon for a patient to be cured or for the results of the disease to become quiescent, but it is not until after the

paroxysms or other manifestations have been continued during a considerable period.

The numerous divisions proposed by the older writers, though very expressive, tend to confusion, and their names form merely a list of the numerous principal symptoms and accidents to which the disease is liable. Without following up these minute ramifications, to which allusion is here made, gout is most usefully to be studied under the following heads: First, acute; second, chronic gout; and third, visceral manifestations. To these must be added the condition of lithiasis or uricæmia.

The divisions often overlap each other; thus both acute and chronic gout may be attended with visceral lesions other than those of the joints, and the patient with chronic gout almost never escapes. The disease is hardly ever one of a single paroxysm, and therefore in the acute form we are in the midst of a chronic disease. The chronic form frequently has acute paroxysms. Visceral disease is rare without both chronic and acute articular phenomena. And none of the first three are supposed to exist without the latter being more or less constantly present, but the latter occurs, whether continuously or not we are not well informed, without any articular symptoms resulting, at least any of a marked character; it is probable, however, that not unfrequently visceral lesions result from it, without at any time articular changes being presented.

Another useful method of distinction is under the two heads of regular and irregular gout; the former designates the articular malady, whether acute or chronic; the latter is reserved for non-articular affections, both the lesions of internal organs, and those apparent acute inflammations which come in the chronic gout. The irregular class is somewhat vague in signification and undefined in its boundaries, since it is in some respects uncertain whether any of the visceral manifestations occur without chronic, and often not specialized, lesions having preceded; in other words whether an acute inflammation of the internal organ ever comes in this disease without a pre-existent chronic lesion being present. There is some objection to the words *regular* and *irregular*, on the ground that visceral lesions are quite as frequently found post-mortem in gouty patients as have been the articular inflammations during life. These words, as commonly used, signify little more than internal and external gout. Nevertheless these terms serve often a useful purpose.

The distinctions between these varieties can be given in the clearest

manner by detailing their characteristics. Therefore we will proceed to consider their

Symptoms, Course, and Duration.

Until recently it has been the rule to speak of disease as developing suddenly in the form of an inflammation in or around one of the joints ; as though this inflammation were the initial stage of the disease. It is difficult to believe that the morbid condition is thus sudden in its formation. Evidently the premonitory symptoms or conditions of gout are not to be come at from the feelings of the patients, for so many of them speak of perfect health and of unusual sensation of strength and good feeling, and for these reasons, as well as others, there are difficulties in obtaining opportunities for the study of the condition. Other patients, however, complain of various dyspeptic conditions or unusual cardiac, respiratory, nervous or urinary sensations which, if they have suffered a previous gouty attack, they recognize as premonitory phenomena.

In others, again, there is the complete absence of all prodromal symptoms and the articular inflammation ushers in the attack. It is possible in these latter cases, if opportunities could be obtained for observation, that some clues, if not the complete solution might be derived, for in the other cases the dyspeptic and other phenomena do undoubtedly mask the underlying conditions not unfrequently.

Acute Gout.—The attack is developed sharply and violently, usually in the latter part of the night, waking the patient from sleep with the severe pain in the affected joint. The pain is variously described by patients, but is always severe, more so, as a rule, than in acute rheumatism, although I have seen the rule reversed. "Screw up the vise as tightly as possible—you have rheumatism ; give it another turn, and that is gout," has been said of the relative amount of pain. In judging of the amount of pain it is well to bear in mind the condition and characteristic of the patient who is the sufferer. In gout there is often a very large element of irritability produced by the morbid condition itself directly, and to this must be added that the class of patients in whom gout occurs most frequently are not wont to bear discomforts patiently. In rheumatism neither of these circumstances concur. Pain is better borne by the poor man, as I have had opportunities of seeing, than by the man who acquired or promoted his gout with two or three bottles of port wine daily, with the surroundings which such luxury implies. The attention of the patient

can be more easily diverted from an inflamed gouty joint, than a rheumatic inflammation.

The metatarso-phalangeal joints are the favorite seat of the commencement of the attack, and its phenomena are most often confined to the single joint, especially in first attacks. The disease, however, does make its first appearance in other articulations, and may subsequently involve still others; this is more frequently true of the chronic disease than of primary attacks.

The affected joint, within a short time, becomes red and swollen; the skin over it becomes more and more smoothed out and shining. The swollen parts either pit on pressure with an elastic feeling, or there is fluctuation from the fluid accumulating within the articular cavity. The veins over the joint or on the adjacent parts of the limb become conspicuous from their enlarged size and dark purplish color; the swelling often extends to the limb where the veins are affected, and here also there is pitting on pressure, but of a less elastic character. Spasmodic contractions of the muscles frequently add greatly to the pain.

According to the severity of the local inflammation, there is a greater or less development of the general symptoms. Rarely is there no fever, although in some cases it may be quite inconsiderable. The initial rigor is slight. The tongue becomes coated or is already furred or whitish. The bowels are confined. The urine is scanty and usually high-colored, depositing a sediment of reddish or pinkish urates or uric acid.

During the course of the day following the nocturnal attack, the pain abates, especially if quiet and proper precaution have been observed. The inflamed joint, however, remains sensitive to touch or movements, and the general symptoms, as the local appearances, continue unchanged.

As the second night approaches, or sometimes during it, an exacerbation takes place of all the symptoms, depriving the patient of sleep from then until about the same hour as the previous day, when a remission again occurs. The same succession occurs with as great or less violence during five or six successive nights, when the paroxysms commonly end.

During the latter portion of this time, when the swelling has fully stretched the tissues, the nocturnal exacerbation are attended with less pain, the articular cavity is less distended, the fluctuation is more perceptible, and the surrounding parts pit more distinctly under the finger; the skin becomes wrinkled, and when the inflammation has completely subsided, desquamation takes place.

Such is a typical attack of articular gout, which has been described so often, so graphically, and eloquently, made eloquent by personal suffering.

The grade of the symptoms and the variations in the joints affected are very considerable, although less in the latter respect than the former. In a vastly greater number of cases it is the great toe joint, more frequently the left, which is alone affected than another; sometimes other joints are invaded secondarily during a paroxysm; again, this joint may never suffer either in the first or in subsequent attacks. Unlike rheumatism, the joints attacked do not appear to be those which are chiefly brought into severe functional activity, although traumatism does sometimes appear to control the selection. I have, however, seen gout affecting only the finger in such persons, as penmen or tailors. Trousseau has described a form of gout—"gout of successive paroxysms"—which Charcot has spoken of as acute primitive general gout. This form of the disease invades many joints simultaneously, and its paroxysms are apt to be of longer duration. Charcot speaks of the great liability of mistaking the ailment for rheumatic polyarthritis.

The general and local symptoms in some cases present but slightly pronounced characters; this has been spoken of as asthenic gout. It occurs in the weak and debilitated, and is more common proportionally in women than men. There is very little fever, the swelling and redness are slight, but the ultimate changes or deformities are often just as marked as in severer cases.

When the paroxysm ends, the patients describe themselves as feeling unusually well, better than before the attack; this is especially true of those who have had marked symptoms during the pre-paroxysmal stage. After the primary attack, the function of the joint, in the vast majority of cases, is completely restored, but sometimes a considerable degree of swelling or stiffness, or both of these, remain for a period which may become indefinitely prolonged. In other rare cases, partial or complete anchylosis results, or concretions may form in the periarticular structures.

As has been remarked, gout, even in its acute manifestations, is essentially a chronic disease, and after one paroxysm we are ever looking for a recurrence of an attack, or for the development of the chronic form of the disease, or else some visceral lesions with their attendant symptoms. Usually another onset of articular inflammation occurs, and the period of immunity varies very greatly. The second attack rarely occurs sooner than the same season of the following year, in other cases it may be postponed

for two or three years, or even longer. In two or three of the next succeeding paroxysms the interval may be equally long, but afterward the attacks come twice a year, and finally at shorter and more irregular intervals, following no rule of time or season. The patient, unless measures have been rigidly enforced to remove the liability to the ailment, finds himself, with every indiscretion and even without apparent cause, on the brink of or in the midst of another attack.

In the earlier paroxysms, the same succession of symptoms is shown during the attacks, with some remission of the sensation, and during the intervals the same complete restoration of health, both local and general.

Later, the tendency is for more joints to become affected either primarily or subsequently during the paroxysm. Joints previously unaffected suffer, and the disease appears in the hands or knees or other joints. The general symptoms become of a subacute type. The intervals are shorter in duration, and during them there are nearly all the time either some articular symptoms or some of the many functional disturbances, directly connected with the morbid condition. The disease thus loses its acute form and becomes chronic.

Chronic Gout.—Practically the chronic stage of the disease is characterized by the deposits or by the alterations which occur in or around the joints, as well as by lesions of the internal viscera which come in connection with the disease.

As has just been stated, the chronic form may arise through frequent repetition of acute paroxysms, and sometimes this condition may be brought about after a single particular attack by the maintenance of the functional disturbances which are so destructive to health.

The chronic state is also characterized by general feebleness—and it is here difficult to draw a distinct line of demarkation—more or less marked, but which may amount to a positive cachectic condition. This feebleness tends rapidly to various organic degenerations, not unlike those usually seen in old age, but they often present characters distinctly gouty, and under these circumstances the disease most frequently makes rapid progress. The organic changes are doubtless due to the constant condition in which the blood is found.

Sometimes also gout exhibits the feebleness from the very start, and in these cases the visceral changes commence very early and rapidly advance, and the deformities of the joints and the deposits in other parts of the integumentary surface are prompt in making their appearance.

The most important circumstance in chronic gout, in connection with the articulations, is the deposit of concretion and the changes which they undergo, and the alterations which they induce in the form of the joints. After the deposits have formed they lead the existence of foreign bodies in the organism, but, like all foreign bodies, they, during all the stages of their formation and existence, give rise to active phenomena. During their formative stage the accumulating fluid produces soft fluctuating elevations of the skin. At this time there are rarely any changes to be witnessed except those directly connected with the gouty process, namely the further increase and the consolidation of the materials. I have seen a case, however, in which the accumulation of the semi-fluid matter took place so rapidly that the skin burst, allowing of the escape of the milk-white fluid ; the large subcutaneous vesicle collapsed and cicatrized without any purulent formation. Later, the hardened masses constantly give rise to inflammatory changes, and as constantly are added to by the further deposits of the same material. They may encroach on the articular structures, but more commonly the skin over them is destroyed, often without marked inflammatory changes, but also frequently by an ulcerative process. The destruction of the skin exposes the whitish deposit, and if any fluid material is present it drains away, or, if completely solid, it remains for an indefinite time and may be removed *en bloc* or undergo a gradual liquefaction through the process of ulceration. Renewed attacks of gout, during this period, very constantly add to the amount of the deposited material. The ulcers or abscesses thus formed are very difficult to heal, and may remain open for long periods, without, however, causing marked general symptoms, or even any, and without exercising unfavorable influence on the structures of the joint. Occasionally the subcutaneous deposits extend deeply into the bone or cartilage, or are continuous with similar deposits in the latter structures, in which case the ulceration leads to very great destruction and deformity when cicatrization is effected.

Visceral Manifestations.

In speaking of the symptoms, coming in connection with gout, which develop in other organs than the joints, it is necessary to be especially guarded. Formerly it was the universal custom to speak of many kinds of visceral changes as gout, even when no external symptoms had occurred. The expression "gouty" has been used to cover a multitude of errors.

In respect to the term "retrocedent" gout, it is clearly a misnomer and a misapprehension. First, gout is a constitutional malady, and therefore must pervade the whole organism, and, besides, we cannot put our faith in any so-called metastatic phenomena. Secondly, a vast number of the symptoms ascribed to a gouty cause have, in reality, nothing directly to do with this malady.

The real question is whether the symptoms and the organic lesions—and there are very many occurring in gouty subjects—are due to an actual invasion of the gouty inflammation, to functional changes coincident with the disease, or, in respect to the visceral lesions found post-mortem, to deposit of gouty matter in degenerating tissue altered by other morbid processes.

It is unquestionably true that a genuine gouty process invades the viscera, or certain of their composite tissues, and this may occur in acute or chronic gout, or even may be seen in cases which have exhibited neither of these forms in connection with the articulations. The gouty process in these cases leaves its attestation behind it, which can be found post-mortem, either in the form of a deposit or of other changes, but these conditions are not numerous and are confined to very few of the organs.

The quite numerous functional disturbances coming prior to, or in conjunction with, an attack of gout, are, in all probability—nay almost certainly—not gout. Some of them, for example the dyspeptic symptoms, may be looked upon, perhaps not strictly speaking, as the cause of, but rather as one of the foundations of the malady; and the symptoms of a similar character which follow acute or chronic articular manifestations, while some of them may be due to gouty localizations, are in the great majority of instances the result of tissue alterations of a degenerative character, very similar to those seen in old age, but they certainly fail to attest their gouty nature by the appearances which they present.

Every degeneration may, in a gouty subject, receive a special deposit of the gouty matters, and yet in these we fail to recognize their gouty origin from this fact alone. In the gouty subject whose blood is surcharged with uric acid, we could but expect that at least one of retrograde metamorphoses would lead to such deposits. Very many, if not all of the organic degeneration observed in gouty patients belong to some one of the great general classes of tissue-changes, some of which are due to special causes, but the most of them own many causes; one of these many causes can be, under certain circumstances, gout, but there is really nothing special in the

appearances by which we can unequivocally pronounce them of gouty origin.

The so-called functional derangements, on which so much reliance has been placed to prove the existence of visceral gout, are of course not purely functional; they unquestionably rest on an anatomical basis, and yet the organ thus functionally disturbed, while not failing to show morbid changes, fails to show the connection of such changes.

While such changes have been pointed to as gouty, no attempt has been made to prove them to be due to an invasion of the gouty inflammation, or at least the attempt has not been successful, unless in the case of one or perhaps two organs. Dependence has been placed chiefly on the succession of symptoms; for example, in the case of the stomach, disturbances which precede articular gout have been regarded as gout—instead of merely the cause of the malady—because the symptoms disappear from the stomach to manifest themselves in the joint. Such symptoms are very common in the stomach, and constantly lead to the same anatomical changes which are found in the gouty subject; in one set of patients gouty inflammation of the joints never occurs, and neither are there any other symptoms which can be connected with this disease. Can we construe the anatomical alterations of the stomach as gout in such cases? If we cannot consider them as gout in all cases we have naught to depend on to prove them in the gouty subjects.

The so-called retrocedent gout of the stomach—derangements of its function coming after a gouty paroxysm, which are sometimes so severe as to cause death—shows nothing more definitely gouty than does the precedent form, except that the anatomical alterations are more advanced, so advanced that, after a violent or long-continued course of gout, their function cannot be resumed to a degree compatible with continued life.

In respect to other organs and the symptoms connected with them in gouty patients, the case is very much the same as with the stomach. In the respiratory tract acute inflammations are of rare occurrence, and do not seem to be connected with the gouty state. On the other hand, such chronic maladies as bronchitis and asthma are of very frequent occurrence, but not more so than in many other conditions; moreover, their type and their symptoms are the same without the presence of gout.

In former times the cerebral symptoms of this disease formed a very long list and occupied great attention. Gouty apoplexy is no longer considered an attack of inflammation retrocedent to the brain; it is a

cerebral hemorrhage from degenerated vessels which are present in patients whose tissues are poorly nourished as is likely in gouty subjects.

The habits of life productive of this disease favor the development of chronic headaches, but this form of suffering is not more common in the gouty than in others. Many other acute functional disturbances, such as delirium, are quite as well accounted for by chronic alcoholism as by gout. Acute alcoholism and excessive venery both have among their rarer symptoms certain forms of spinal paralysis, which have been charged to the account of acute and chronic gout.

The liver must necessarily be changed in all subjects whose digestion is disturbed in the manner common to all those who acquire gout by the most usual cause ; in those who suffer from it by heredity or who do not indulge in excessive drinking the organ is much less affected. The lesion is not as frequently as we might suppose a cirrhotic contraction, but more especially atrophic changes of the liver-cells. But of the liver—the chief organ concerned in the production of uric acid—we know less than of almost any other organ, and are less well able to explain physiological and morbid states of activity.

The urinary organs, chiefly the kidney, suffer not only chronically but also directly and acutely, from the effects of gout. The changes are apart from those due to calculous formations which are exceedingly common. It seems not surprising that the kidneys, and even other parts of the urinary tract, should suffer in this disease, since these organs form the principal outlet of the excessive morbid matter present in the blood in this malady. The inflammation of the joints are not to be regarded as the *summa* of the disease, but rather as a local trouble incited by the presence of the uric acid. The other organs of the body may occasionally, as we have seen, be disturbed by its influence, but in general to a very slight degree. The kidneys, however, do, as we should expect, present evidences of both acute and chronic manifestation of the gouty process.

It is probably an exaggeration to speak of any functional disturbances of the organ as metastatic alternations between the kidneys and the joints. In acute gout their function may become disturbed, as it does sometimes in other acute diseases, for example, the continued fevers, but we do not in these cases speak of typhoid or other fevers of the kidney, neither should we in the case of gout. When the kidney is invaded by the inflammation of the gouty process, the organ responds with symptoms somewhat similar to those seen in nephritis of various forms due to other causes.

The character and severity of the symptoms vary, as in other cases, with these causes and the mode of their application. The lesions in the kidney form one of the members of a well-marked group in Bright's disease; it is one of the forms of contracting kidney. In addition to this the effects of the gouty process leave the attestation behind them of deposits of urate of soda.

In the heart we never see such changes and symptoms as result from attacks of acute rheumatism. The functional derangements commonly alluded to in gouty subjects are palpitation and certain irregularities of the pulse; but these are frequent in many forms of disturbed digestion and in other conditions, and therefore are in all probability dependent on the same cause in all these maladies. The fatal cases formerly attributed to retrocession of the disease to the heart are probably all to be explained by cardiac failure due to fatty degeneration of the muscular fibres. In chronic gout this condition is not unfrequent and is owing to the general poor nutrition, the tendency to fatty changes and accumulations in all parts so frequently seen, and perhaps also to the overaction of the heart from constant stimulation; the condition is more common at all events among those whose potations have been free and abundant.

I have taken the ground denying the existence of most of the so-called localizations of the disease in the internal organs which have been so frequently and freely assumed by most observers, because of the almost complete failure to signalize the present or the results of the gouty process by anatomical evidences. Not a few careful observers, with wide experience, have assumed the same position; the majority have, however, seen fit to coincide with the older opinions on the subject, and to day a large number of conditions, whose pathology is unexplained, are classed under the manifestations of latent gout. One of the principal reasons for this classification is the therapeutic test, or rather the therapeutic failure which has attended the measures applied to combat the symptoms.

Pathology and Morbid Anatomy.

The views of the nature of gout and the theory of the disease are to-day dominated by the presence of the uric acid and its salts, which play so prominent a part in the phenomena of the malady. It is almost the only disease in which the existence of the apparent materies morbi has been shown with clearness, and its presence is so very unequivocal

that it is quite impossible to escape from it in the discussion of the theory of gout. The evidence is everywhere and always complete in favor of its development in the blood and tissues in every instance of the malady. The question arises, however, whether in certain morbid conditions this material, which is a normal excrementitious matter in limited quantities, may not be present in an abnormal quantity, and even form deposits, and yet no such trains of symptoms to which the name gout has been applied result.

While the presence of the uric compounds is unquestioned, the theory of gout must account for their presence from retention or from their development in increased quantity, or by both of these factors. In other words, the consideration of the nature of gout concerns itself almost entirely with the process set in motion, by which an abnormal quantity of morbid matter finds itself within the organism, and only secondarily with the effects of this matter on the various tissues and functions.

The ancient writers declared that there was a morbid matter in the blood—and this to them was true of all diseases—and pointed to the articular deposits in proof of the truth of their assertion. Hence, with the products of the disease in hand, their views of the nature of gout, as gathered from their writings, seem much more definite and positive, whatever may be said of their accuracy, than for other diseases. They were, however, unacquainted with the nature of this material, and it was not until the end of last century that its chemical composition was recognized.

After this time it was by nearly all spoken of as the morbid matter, but its presence in the blood was not shown until Garrod, in 1848, carried out his investigations. As the result of these investigations it became evident that the amount of uric compounds removed through the kidneys lessened during the attacks of the disease; that uric acid was present in the blood in all cases of gout, as was shown by the causing it to crystallize from the serum, and that there was a deposit in the articular tissue of this matter, even in first attacks of gout.

"It must not, however, be supposed," says Garrod, "that an excess of urate of soda in the blood constitutes gout;" but still no physiological theory has been until quite recently attempted to account for the disease. And every question in relation to the malady has revolved about the increase of the quantity of this material, and the disease remains *par excellence* the poison-disease of our present nosological catalogues. This poison was by them supposed to be effected almost entirely by the ingestion of

various articles of diet which were capable of being converted into uric acid.

The questions then to be considered, if we attempt to formulate a physiological theory, are, first, Is there an increased production of uric acid ; is it due to ingested matters which interfere with digestion, and which either before absorption or afterward by failure of assimilation increase the amount of this product? Second, Is there simply a retention of the normal product of the system, which, as in the former case, acts to disturb its functional activity, and set in motion the morbid train of symptoms. Or third, Is there a process set up wholly within the organism, whose action is favored or promoted by the character and quality of the food and by the general mode of life.

There is little or no evidence to support the speculation that the increase of the uric compounds is due to the failure of renal excretion. We know of no condition of the kidney in the antecedent period of gout which lends the support of facts, and we do know, moreover, of diseases of these organs causing retention of this material in which no gouty phenomena are recorded.

In respect to the origin of uric acid and its excess in the blood in gout, the theories of it have been very similar to those of lactic acid in rheumatism, and the older theories of both have passed through the same transformations and alterations of their terminology in recent times. Formerly it was supposed, and this was especially insisted on by Todd, that uric acid was derived from an abnormal condition of the stomach and duodenum. Todd, however, did not consider that uric acid was essential to gout, but included along with the uric compounds many other materials as the offending matter.

As in rheumatism, so now in gout, according to more recent physiological teaching, the uric acid is derived from errors in the secondary, rather than primary, processes of assimilation. Some look to the failure of the oxidation of the albuminoid materials of the food, which ordinarily are converted in part into urea, as the cause of accumulation of the uric acid. Others direct attention to certain organs, as, for example, the spleen, which when enlarged, as in leukæmia, give rise to increased amount of this substance. It has also been sought to trace it to the joint structures, the cartilages, and fibrous tissues, in which after gouty attacks the deposits are first seen.

But at present our knowledge of the physiological and pathological

chemistry of the formation of uric acid and urea, their relations to each other, their relation also to the ingesta, on the one hand, and to the metamorphosis of tissue on the other hand, is so imperfect that no satisfactory theory can be formulated.

Of the third speculative question suggested above, there is nothing as yet been suggested which supports it. And in absence of a knowledge of the life history of uric acid, I do not see on what grounds we can proceed in its discussion. Following the analogy of all other morbid processes it seems but rational to think of a misdirected function as the basis of the gout, and that also the production of the morbid material is the result of an excessive activity of the process by which uric acid is normally manufactured in the system. I speak thus of the presence of uric acid because it is so constantly present, but it does not seem unlikely that gouty symptoms may occur without uric acid, and also that uric acid may be present in excessive quantities without gout. It would, therefore, seem that there must be some underlying process which is the essential of the disease, which is apart from, or at least only a part of, the process which produces uric acid. On the whole, therefore, it would seem that we know more of the results or products of gout, and less of its essential nature, than of almost any other disease. In some respects we seem to know more of its essential cause, and can produce or reproduce its train of symptoms—in other words, produce the misdirection of function which leads to it with greater certainty than in most other diseases of the same class.

The post-mortem history of gout concerns itself almost entirely with a description of the uric acid deposits, the changes which they induce, and the situations in which they may be found. We have therefore mostly to do with the joint structures, for it is almost exclusively in and around them that the deposits occur. Besides this, there are various changes found in the internal organs, but these are so inconstant that no particular description is needed further than the allusions already made.

It has been shown by a number of observers that the crystals of urate of soda form in the articular cartilages of the affected joints, and, as Garrod says, they are present from the very first attack, gradually increasing as the attacks are repeated and the ailment becomes chronic. The deposits persist indefinitely even should the attack never be repeated. The seat of the earliest deposit is shown to be the superficial layers and around the centre of their free surface. The surface of the cartilage shows a white patch. It has been suggested that this position is one

most remote from the vascular area of the articular tissues. Finally, however, the whole cartilages are invaded, and, in the chronic stages, the synovial membrane and the ligaments become incrusted with chalky material.

The concretions, or tophi, which are found around the joints, and which sometimes, though rarely, occur early, or even at once, after first attacks, or in purely chronic cases, but nearly always later on, after frequently repeated attacks, form in the bursæ, along the tendons, or simply in the connective-tissue interspaces. In their very earliest state they consist of fluid more or less richly impregnated with uric acid compounds, which, from their deficient solubility, crystallize in the form, when it is urate of soda, of acicular crystals. I have tapped such swelling with a fine needle, or examined exudation from such swellings, which have opened spontaneously, and found the milky fluid composed of a clear serum in which floated innumerable needle-like crystalline masses; besides these, there were a few large and small granular corpuscles, similar to those seen in any serous fluid.

Later, these fluid collections inspissate, and finally become often exceedingly hard and dry. In this stage the tophi, as well as the deposits in the cartilage or other places, sometimes fail to show their crystalline character, but present a confused opaque agglomeration of the same material, which, by proper chemical agents, can be made to assume its typical appearance, or can be converted into uric acid.

These concretions have been found in a number of other places besides the usual one around the joints. They have been described in the bony tissues, and not unfrequently in the periosteum. The skin in various parts of the body, generally the legs or thighs, probably arising from deposits in veins passing near veins from and around the affected joints. The cartilages, or skin covering the ears, is a very common seat of their occurrence, and here they may serve a useful purpose in clinical investigations of doubtful cases. The eyelids, the nose, the cheeks, and along the tendinous structures of the palms of the hands, where they produce sometimes the characteristic contraction of one or more fingers (Dupuytren's contraction), also exhibit their presence. Virchow speaks of a concretion in the larynx, one record has been made of them in the lungs, and Charcot mentions the penis. Even the spinal dura mater and spinal nerve-roots receive incrustations of uric acid compounds.

The concretions are also found in the kidney, as sometimes rounded

masses in the cortical substance, but more commonly as streaks or points extending along the straight tubes as they ascend from the medulla into the cortex, and also at the summit of the pyramids. In the specimens which I have seen, the deposit was almost intertubular, and it was seen in the region of the convoluted tubes as well as the pyramid portion of the organ. The kidneys usually exhibit a more or less advanced stage of interstitial nephritis (contracting kidney) in cases of chronic gout, while in the early stages the records indicate that little or no changes are visible.

Diagnosis.

The recognition of a joint inflamed with gout is nearly always a simple matter, and it is only in the cases where unusual joints or a large number of them become involved simultaneously, as in acute general gout, that the diagnosis is difficult to make.

In gout the small joints are usually alone affected; the febrile disturbance is moderate; the sweating not profuse. The joints present a different appearance, which has already been described. The habits and mode of life generally furnish a useful clue to the nature of the malady, but it must be remembered that there is a poor man's gout as well as a rich man's—one might say almost a starvation as well as a repletion gout.

The serum of the blood, according to Garrod's investigation, furnishes us an infallible test when gout is well developed. The fluid from a blister, and probably, also, from the subcutaneous connective tissue drawn by a needle, serve equally well for the purposes of Garrod's thread test.

The chronic varieties of the disease are much more difficult to distinguish from other maladies. The presence of tophaceous deposits around the joints, and in the other situations already mentioned, will often serve a useful purpose.

The various functional disturbances, which have been discussed among the symptoms of the disease, furnish in themselves very little evidence of their nature, and without the presence of external deposits must be regarded as doubtful. On the other hand, the functional disturbances connected with obscure articular symptoms are often sufficient to make clear the character of the latter.

The lithæmic condition, with its vertigo and mental symptoms, its heartburn and eructations, is revealed when sufficiently careful, and perhaps continued, examinations of the urine are carried out.

Prognosis.

The outlook for a gouty patient is hardly ever favorable. The malady probably never causes death in its early or acute stages. One attack of gout is so constantly followed by others, and by many repetitions, that a chronic condition with its attendant discomforts and dangers is induced. However, many patients suffer from frequently repeated attacks, and even severe ones, and yet in the intervals maintain a very good state of general health and live long lives; some even become free from the gouty paroxysms and remain in good health. More commonly, however, the course of the disease is ever a downward one, and may be cut short at any period by the rapidly coming organic degenerations, or by many of the accidental intercurrent maladies to which these patients seem especially liable.

The prognosis of the hereditary form of the disease would appear to be less favorable than of the acquired malady, and is also perhaps—and this is the reason of its unfavorable tendency—more liable to visceral alterations.

Prevention by a rigid course of life and of diet seems to have more influence, or perhaps this is more easy to carry out with those who have acquired gout than with those whose ancestors have suffered from its disturbances.

The more limited the acute manifestations, and the more strictly they are confined to single joints, the better is the prospect of preventing renewed attacks, or without this, of living to an old age. The earlier in life gout makes its appearance the more unfavorable is it for the subject.

Garrod says that "if the urine was formerly turbid with urates, or if it gave rise to a deposit of crystallized uric acid, and has become of late clear and of a paler color, the change probably indicates that the kidneys have to a considerable extent lost their power of eliminating uric acid, and that which seems to the patient a favorable change is in reality a sign of serious structural alteration in an important secreting organ." "It is a grave sign in gout to find the urine pale, the specific gravity exceedingly low, and the fluid devoid of uric acid; and if, in addition to this condition, albumen is likewise present, the indication becomes still more unfavorable."

Treatment.

The treatment of gout presents itself to our consideration under several heads. First, the institution of measures to prevent the development or continuation of the malady; second, the remedial measure in an acute paroxysm; and third, in the stage of chronic gout.

Too much stress cannot be placed on the enforcement of a proper mode of life, avoiding the conditions which have been described as so constantly prevailing in the habits of gouty persons. Whatever may be the views on the production of uric acid, whether due to the improper conversion of the albuminous matters, or whether this sort of diet prejudices certain organs or processes engaged in producing this material, it is certain that no medicinal agents will be effective in removing, or even checking, the perverted turn which the organic processes have received or acquired.

What the measures are which must be enforced has to be decided for each patient, and reference must also be had to the time of life and the stages of the malady. It must be remembered that starvation measures are nearly, not quite, as disadvantageous as the opposite of this extreme. A mixture of food is unquestionably what is required, and the vegetables, toward which the diet list must be mostly directed, should not be of the bulky or watery sort, and the meats must be of lighter, easily digested kinds. Fats and oils, and articles or meats containing them, eggs, potatoes, and most of the farinaceous articles, and tea and coffee, strong or in large quantity, must be avoided.

In regard to the number and times of taking food, it requires most careful management; some do better with more than the usual three meals, others can, to their advantage and greater comfort, be restricted to less than the ordinary ones. The heavier meal should come early in the day, and this is often best effected by a full breakfast early in the morning.

The gouty patient must be warmly clothed. How many of the chronic cases suffer from more or less constant pains, which ought to be avoided by attention to this simple matter! How many, too, of old gouty patients exhaust their strength in warm weather, or in warm rooms, sweltering from the heat of extra wraps! Both sets of patients have their theories and prejudicies in regard to clothing and temperature, which are exceedingly hard to overcome.

Clothing suited to the circumstances is the most useful adjuvant we have to exercise in promoting the activity of function not only of the skin

but of other organs, and should always be superintended with great care.

Exercise, which by its activity is suited to prevent a gouty development and to promote healthy digestion, as well as to consume certain elements, especially the fatty ones, which are necessarily present in more or less amount in the diet, is quite unsuited to chronic gout with its visceral and articular complications. And where active muscular exertion is impossible it should be replaced by passive movements and other measures helping to promote a proper circulation of the blood.

The various forms of special diet treatments is useful often not only in themselves, but frequently as a means of regulating the dietary, which patients so frequently transgress when merely general directions are given. Often, too, these special diets and cures can only be carried out in certain countries, which by their salubrious climate and attractive scenery are of benefit to the patient. Of these the grape and whey cures serve as good illustrations.

Probably the successful treatment of gout in any of its numerous forms and stages can never be profitably carried out without the aid of some one, or even many, of the natural or artificial mineral waters. Ordinary drinking water is often found very useful, and is too often omitted from the dietary list of these patients. The natural mineral waters are of course much more valuable than their artificial imitations, and when it can be done, they are much better taken at the springs.

The waters serve many useful purposes. As purgatives of a thorough yet unirritating, undisturbing character, they may always be used, and constipation is at nearly all stages of the disease a difficulty which requires careful management. In addition to this, many waters serve to relieve the catarrhal conditions of the stomach and bowels, which must be allayed before any improvement in the gouty tendency is effected—if these conditions even do not stand as a cause of the disease. This improvement effected, the tendency to an increased production of uric acid is lessened or removed. Further, the use of water is essential in promoting or maintaining the solution of the uric acid. Pure drinking waters contribute to this, but there is a greatly increased efficacy afforded by many of the salts contained in the natural mineral waters. This effect we may consider serviceable during all stages of gout, both before the threatening paroxysms and after them if required, as well as after deposits have formed, although it may be regarded as doubtful whether anything will affect these concre-

tions ; sometimes they may gradually lessen if the process of accretion is stopped.

Probably no disease has had more infallible remedies proposed to its cure than gout. The quack remedies in themselves form an innumerable host, the old ones passing away and new ones taking their places.

The alkalies and alkaline salts have always enjoyed a high reputation in the disease, and were used as a remedy for its acidity long before the presence and the nature of uric acid were pointed out. Theoretically much has been said in favor of one or other of two common alkalies over the other; but both of them, under certain circumstances, exercise a very beneficial influence. I have always thought that their disadvantageous effects were greater on stomach digestion than their ultimate effect on the blood, and it is on account of the former rather than the latter that their exhibition has to be discontinued. Of their neutralizing action, and the consequent benefits, there is, however, no doubt.

Better in its effects, and apparently without the disadvantageous effects of the former alkalies, is lithium and its salts. The existence of this alkali has been known for many years, but only recently has it been introduced into the list of drugs for this disease. It is very common to find it in the natural mineral waters along with the other alkalies. Taken in this form, it too, like the other natural salts, produces its best effects ; but it is very common to administer it—and it is very useful in this way—in the form of a carbonate. The combination of lithium with uric acid forms the most soluble of all the urates.

Treatment of the Paroxysm.

We know pretty well that a paroxysm of typical character will end, uninfluenced by remedies, if unprejudiced by wrong regimen, within a few days. This at least is the rule in typical cases, and knowing this fact a gouty inflammation presents itself to us as a morbid process which recovers more rapidly than any other of equal severity. We know of no other inflammation which passes away so soon, whatever may be its treatment. It is a question, therefore, whether it is not better to stand by with watchful care than to interfere actively. The results of active treatment, especially local measures, are known in many cases to be harmful.

Nevertheless it is not proper, whatever may be our theoretical or practical conclusions, to do nothing during the continuance of an acute attack.

This is especially true in the earlier paroxysms, occurring in healthy persons, although it is sometimes best to follow the do-nothing policy in the milder paroxysms of chronic gout, and especially in those patients in whom we know that there exists visceral degenerations or lesions. In the latter class all our procedures should be of the most guarded character.

There is one drug, and it is perhaps the only one, which has at any time maintained the character of a specific for gout. It is colchicum, and it should be given in an acute paroxysm, whenever there are not pretty certain contra-indications to its use, such as known irritability of the digestive organs, fatty or other lesions of the heart, or kidney disease, and perhaps other less common concomitants of gout. In almost every primary attack, and many of the earlier ones, its exhibition may be made with freedom in proper doses. Its effects are usually good and pretty prompt, and when otherwise it not only does no good in the paroxysm, but its general and ultimate influence on the patient is harmful.

The more chronic the disease and the further advanced the visceral changes, the less useful is colchicum, and the more carefully watched must be its administration. There are three results to be especially guarded against, its purgative and otherwise disturbing effects on the digestive organs, the disturbances on the nervous system, and its depressing effects on the circulation.

Combined with this drug it is often well to administer moderate doses of the alkalies, either as carbonates or in combination with some of the vegetable acids ; these may be given as effervescing draughts, and this form is applicable to the nausea and vomiting often present. These salts can be made to serve at the same time as purgatives, diuretics, or diaphoretics.

The local measures, as has been said, are not essential to recovery, but it is well to try and mitigate the suffering when intense. It is necessary to remember the character of the inflammation with which we have to deal, an inflammation in which the tendency to suppuration never has to be combated, and also one which, if we may speak so, runs a regular rapid course to recovery if not disturbed. It is the comfort of the patient and the after-results of the local disturbance which require attention, and hardly at all the violence of the inflammation.

If the inflammation does not tend to run its regular course we cannot force it to do so by violence, but only direct or persuade it by the assistance of gentle means. Such is its natural history. In order to abate the

inflammation when violent, we do not require bleeding or cold, but the soothing influence of heat and moisture, and these two must be proportioned to the severity of inflammatory process. Combined with these the anodyne lotions are of great service.

When the inflammation lingers, slight stimulation is of use, carried out by means of blisters and similar agents, proportioned in strength to the result desired. It is probable that the effect of stimulation, especially vesication, is due to the influence exerted on the circulation of the part rather than to the removal of irritating fluid by the serum. Blisters and even moxæ or cautery have been employed to check or abort the gouty inflammation, and in some cases it is reported very successfully. The ultimate effects on the articulation cannot but be bad. Although these agents may check the inflammation, the cicatrization resulting from them places the joint in a very unfavorable condition on the renewal of the gouty paroxysms, even if its immediate effects do not become unfortunate by inducing sloughing, etc.

Treatment of Chronic Gout.

"Colchicum is found equally efficacious in subduing the exacerbations in chronic gout as in combating the early fits in the acute disease, due regard being paid to the strength of the patient, and dose regulated accordingly," says Garrod.

There seems to be a pretty general consent given to this statement; but the limitations imposed on its use by the dangers to the chronic, enfeebled, gouty patient, are such that many have completely abandoned its use under all circumstances. The drug does much to relieve the pain and inflammation in the chronic stage, though probably less effective than in the earlier attacks; but in its use we are brought face to face with dangers which it is almost impossible to recognize or foresee. If these dangers are recognized, colchicum must not be prescribed, or else immediately abandoned. There are other methods of controlling the inflammation and preventing to a great degree further danger to the articulations by the long continuance of a paroxysm without resorting to this oftentimes dangerous remedy.

The use of *alkalies*, as already described, will here serve a good purpose, and be in reality of more service than in the earlier attacks, and at the same time free from danger. Another remedy is *guaiacum*, but its

immediate effects are but slight during an acute attack. It is serviceable for the purely chronic cases, or when the articular inflammation tends to remain long or become continuous, assuming a subacute type.

The various iodides, especially iodide of potash, are used to produce the well-known effects of these compounds, and they are also recommended for their influence in controlling the inflammation. This latter effect is somewhat doubtful, except in its very low grades, and their influence on the special uric acid deposit is unproved.

The vegetable tonics and bitters are not merely useful, but frequently quite essential to the gouty subject. None of them appear to exercise any direct or specific action on the gouty process ; but in connection with other remedies, and in assisting and maintaining the digestive functions is where they show their best effects. Cinchona in its various forms and preparations has long been used, and it is thought by some to diminish the quantity of uric acid excreted by the kidney. This drug is recommended in combination with colchicum, and it is also prescribed along with gentian. The latter has also been extensively used both alone and with the bicarbonates of the alkalies. A tea or decoction of common ash has long been known as a household remedy for gouty subjects, and the list of preparations taken from the trees and plants of different localities and countries forms a very extensive one.

In addition to the various medicinal agents here spoken of, and very many more, the treatment of chronic gout must be supplemented by the adoption of a most careful mode of life, habits of eating and of exercise, and by the use of many of the agents spoken of under the prevention of the disease, such as mineral water and baths.

INDEX.


BOUND BY
W & E PICKERING
BATH.