Acute Rheumartthritis
Its Pathology and Treatment

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Acute Rheumatoid Arthritis
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There is perhaps no disease to which the human frame is liable that has since the days of
Sydenham attracted so much attention in this
country as rheumatism, especially that form
of it known as acute rheumatoid arthritis, articular
rheumatism, or rheumatic fever.

Its frequency among all classes of the community,
the painful and often terrible maladies that
frequently follow in its train, combine to give it
a notoriety that few other diseases possess.
Added to this the mystery that has for so long a
period hung over its origin, and which still
endeavors to baffle us, makes it an object
worthy of a little study and reflection.

Having had the misfortune last year to be
prostrated with an attack of acute rheumatoid
I can speak with some degree of experience
concerning this most painful disease.
The word Rheumatism was first employed by Monsieur Bailleau, a French physician, and is derived from the Greek word σάρκας signifying a stream or flood. It is not the purpose of this thesis to deal with the different forms in which the disease manifests itself, but merely to discuss that form of it known as acute articular rheumatism, with its subacute variety, the two forms being manifestations of one and the same disease with different degrees of severity. These two forms I shall speak of as rheumathritis, this term being, I think, more expressive of the disease than any other. Muscular rheumatism is usually of a chronic form, continuing sometimes for a lengthened period, and differing in many respects from acute rheumatitis. I have however in the course of the latter seen the muscular form develop, not in the muscles themselves, however, but in the aponeurotic sheaths, fasciae, and tendinous structures, having thus probably spread from the joints itself by continuity and contiguity.
This I shall also include under acute rheumathritis, being quite different from the muscular rheumatism of middle and old age.

**Definition of Acute Rheumathritis.**

It is defined by Prof. Granger Stewart as being a specific constitutional disease, consisting in a peculiar kind of inflammation of joints, fibrous tissue and serous membranes; characterised by inflammatory oedema and acute pain in the affected joints, great constitutional disturbance and perspirations; frequently attended by inflammation of the pericardium, endocardium, and muscular substance of the heart; more rarely by pleurisy and by violent head symptoms.

It is caused, according to some, by the production of lactic acid in the system; it is readily induced in some constitutions by exposure to cold and over-fatigue. It results frequently in recovery, frequently also in permanent injury of the heart, and sometimes directly fatal.

This then gives a concise but good idea of the
of the chief features of the malady.

Rheumatism a "Constitutional Disease."

There can be little doubt that rheumatism must be ranked under the so-called "constitutional" diseases. By this I mean that there must be lying latent in the system of certain individuals, a something, which when stirred into life by some internal or external circumstance, will give rise to manifestations of the disease peculiar to the individual constitution. Thus, a chill, which to a healthy man would mean nothing, would in all probability in one of the rheumatic constitution be followed by pains in the joints, &c., and all the symptoms of rheumatism.

In speaking of constitutional diseases Dr. Walsh says it seems strongly probable that each member of the group has its specific morbid principle in the blood, uninterchangeable with the rest, just as any one virus is uninterchangeable with others; and that further there may exist for each constitutional disease its specific curative agent - an antidote for each poison.
In the greater number of these diseases, a more or less obvious disposition may be traced to symmetrical arrangement of the anatomical character of the local lesions, whether these be external or internal.

Hereditary transmission of the rheumatic taint is admitted by all. We know a gouty parentage gives a liability to gout, and children born of tubercular parents are in turn liable to suffer from the same disease. Rheumatism is inherited in the same way. By inheritance I do not mean that the man is born with 'the something' already in him. The gouty subject does not develop his tendency till middle or old age; the rheumatic one not till advanced youth or manhood. It is not the actual disease that is transmitted, only the tendency to it, a weakness that renders the inheritor more liable to contract it. What is this weakness? Wherein does this tendency consist?

Rheumatism we know to be peculiarly a disease
of youth and manhood, when the motor powers of the body are most frequently called into play, and when risks from exposure to cold are greatest. In rigorous manhood the motor apparatus of the body is most frequently and often most violently called into action. This action encourages and brings out the rheumatic weakness. The fibrous tissues of the joints of A. and B. are anatomically and chemically alike, but if A. is heated and then chilled, he develops an inflammation in his joints, whereas B., under precisely the same circumstances escapes this consequence. The fibrous tissues then, must play an important part in the production of rheumatism. But the joints with their fibrous connections must be regulated by some power in their nutritive functions. This power then will also be called into play in the production of the disease. The name we give to this power is the "trophic centre" of the articulation. It must be in the "trophic centres" of the articulation
and in the fibrinous tissues, therefore, that we must look for the weakness; and remedies for the weakness must act on these parts.

What causes this we shall discuss fully afterward.

The weakness or tenancy may miss the children and appear in the grandchildren.

This is known as the law of "atavism." I have noticed this interesting fact myself in dogs. A fox terrier I possessed named "Lill" had rheumatism in one of her hind legs. In wet weather it appeared to be very bad, but in warm dry weather, it was fairly well, the being able then to put it to the ground. This "Lill" had a litter of puppies which were without exception healthy and handsome specimens of the breed, so much so that I kept one for breeding purposes. In due time this one had a litter of puppies and, strange to relate, the law of "atavism" asserted itself, for every one had "kennel lameness," as it is called, or in other words, exhibited the rheumatic constitution.
and this so markedly that they had to be destroyed.
I have not had time to try any further experiments
in this way but for the above I can vouchsafe.
Rheumatism then we may admit is most distinctly
a constitutional disease.

Symptoms of Acute Rheumatism:

An attack of rheumatism generally
commences with the sensation of having taken
a cold; often a distinct rigor is felt, the
patient declaring “he cannot get warm all he
can do.” He may sometimes have several of
these rigors. This chilliness is followed by
a feeling of heat; the joints now beginning
to turn painful, swollen, and inflamed.
The complexion is sallow and bilious looking,
the eyes sunken or yellowish. The countenance
altogether presents rather an appearance of anxiety.
There is loss of appetite; the tongue is white and furry.
The bowels are confined. No headache is, however,
complained of; a fact worthy of note as dis-
tinguishing it from the eruptive fevers,
The urina is beautey, of high specific gravity, and deposits on cooling a copious cloud of red lithiate. The only complaint on the part of the patient is the arthritis which now becomes the most marked symptom. The pain generally commences in the knees and insteps. From these parts it generally tends to take an upward course, the hips being usually next affected. Simultaneously with the knee or following shorty after, the wrists and fingers may become attacked by the inflammation, which now extends to the shoulders, till nearly every joint in the body is more or less involved. Sometimes it begins on one side of the body in this fashion, then in similar order attacks the opposite side.

The pulse varies from 90 to 130 per minute, is full and bounding, but soft in character. The perspiration pours from the body of the sufferer, requiring to be mopped off his face very now and again as it streams from his forehead. He is unable now to move hand or foot.
the slightest attempt to do so causing him to scream with agony. The weight of the bedclothes even oppresses him. On his countenance is depicted the expression of intense suffering as he lies there, the victim of an attack of acute rheumathritis.

The foregoing is a brief sketch of an ordinary ease of the disease. When the pains begin to abate somewhat in severity and the disease is on its decline, the swelling of the joints gradually disappears, and sleep may be obtained without the use of opiates. The sweating too, lessens in quantity, the sour disagreeable smell as noticeable at the commencement of the attack also disappearing. The urine becomes increased in quantity and better coloured. The patient is able to move his joints with some freedom; take more interest in things in general, and if he be of a strong constitution gets better rapidly. If however, he is of a weakly nature or has been weakened from any cause
prior to the attack, the pains are much slower in their exit. They may even assume a sub-acute or chronic character and remain for many months in defiance of all treatment. In every case the joints are for some time more or less painful, stiff, and weak, the result of the inflammation that had previously affected them. This wears away, however, as the patient gains in strength. In my own case it was fully a month before I regained the proper motor power of my limbs, and even after that lapse of time, a little more exertion than usual, produced some amount of pain and swelling in those joints which had been chiefly affected.

For the sake of simplicity I shall divide the model case above narrated into three stages, and analyse the individual symptoms occurring during each stage.

**Analysis of Individual Symptoms.**

**First Stage.**

In eleven cases of acute rheumatoid arthritis...
out of twelve we find the patient positively affirming that "he thought he had merely caught a cold." He does not pay much heed to this, thinking that it will pass away as colds generally do, but he is reckoning without his host, and that host shows no mercy to his guest. Once he gets him in his clutches he does not rid himself of him till he has paid the utmost gashing of his reckoning!

There is no doubt that the initial symptoms are those of cataract, and in a non-rheumatic constitution would pass away as such, but in one constitutionally predisposed they do not. This feeling of "malaise" is most trying and is most debilitating. In my own case it was most dreadful. For some time previous to the attack I had been very hard worked, a number of serious cases on hands, with their attendant anxiety and worry, I had overworked and overthought; if I may use the expression—For many nights prior to the illness I had
never been out of the saddle. For many days I had jolted over the country in a gig, till I felt myself to be in that stiff immovable condition that Dr. Oliver Wendell Holmes describes country practitioners as sometimes being in. But to make a long story short, I got a cold. I was weakened at the time from this hard bodily work and mental strain and the cold instead of getting better got worse, till about a week or so after the initial stage I had one evening a distinct rigor, which I shall call the commencement of the second stage.

Second Stage.

The rigor determined my going to bed and remaining there; in fact I was able for nothing else. The feeling of rigor or chill is very marked. There is not heat to be obtained one thinks even under a load of blankets and numerous hot bottles.

State of Digestive System.

The tongue is very dirty. It is generally coated with a thick, whitish-brown fur. There is a
feeling of thick mucus all over the gums and teeth, and a decidedly bad taste in the mouth. I have frequently seen a brown fur all over the tongue, a symptom which along with yellowish brown conjunctivae and constipated bowels would seem to point to a sluggish liver as being occasionally at least, an adjunct to the second stage of rheumatism.

The pulse is very quick, 100 or 20, full and bounding, but not absolutely plethoric. It may sometimes even be soft in character.

State of Urinary System:

The urine is scanty, the secretion of water by the kidneys being markedly diminished, the solids being increased. According to Parke the uric acid is considerably augmented. There is no proof whatever that lactic acid exists in greater quantities in the urine of rheumatism than in the urine of health. The quantity of urine voided varies of course greatly as to the amount of fluid drunk and the action of the skin.
It is always abnormally acid, the litmus paper being immediately reddened when introduced into it. The specific gravity is from 1020 to 1030. From its concentrated condition it deposits a copious colloid humour, which contains large crystals of free urea acid in addition to the urate of soda. The high colour which is one of the most striking manifestations of the rheumatic urine is due, according to Sage, to an increase of one of the normal urinary pigments viz.: urobilin. It has been observed that sometimes albumen is present, but this is due probably to slight hyperpyrexia and not to any inflammatory condition of the kidney.

These are the most notable symptoms in the second stage, but the majority occur in the third stage.

This is the stage of pain, and embraces the chief symptoms of rheumatism proper. I have called it the stage of pain, and I think it is worthy of the name. No one who has gone
through it, will, I think, ever forget the agony he endured at this time. If it can be imagined—a location of every joint in the body, and a blacksmith pulling at them with a pair of hot pincers,—this would give some idea of the pain of acute rheumatism. The pain is generally worst in those parts of the motor apparatus that had previously most work to do. Thus the blacksmith will feel it most in his arms & shoulders, the pedestrian in his legs; the washerwoman in her back and so on. In my own case I felt it first in the spinal column, and in the nerves of my cases of rheumatism, I find that I can generally trace the pain as beginning first in the back and spreading in a vague way down the limbs. I merely mention this matter now, as the long we shall have to discuss it more fully when looking into the cause of the disease. In my own case I blamed the jolting about in the saddle. We may put it down as an axiom, that the
pain generally referred first to the back, takes up its abode next in the joints most exercised previous to the attack, and is in direct ratio to such exercise. In my case it extended from the spine to the ankles and knees of one side; the hip was next attacked; then the shoulders and wrists.

This shifting or fleeting about of the joint pains is very characteristic of rheumatic arthritis. Today the knee which is red, painful, and swollen, may by tomorrow present hardly a trace of these symptoms, whilst the wrist or elbow which had hitherto escaped becomes affected.

In regard to the nature of this phenomenon of the disease Senator says:—"the signs of inflammation often disappear quite suddenly from one or more joints, other joints or else internal organs being attacked with equal suddenness. This may be explained in one of two ways: He may suppose that some phlogogenic matters endowed with a special
affinity for the joints and certain serous membranes, and with a special tendency to cause the detion
of serous fluid are introduced into, or generated in the system intermittently. Or we may
imagine that various trophic centres, especially related to particular joints are successively
attacked." I believe this latter to be the true reason. No local cause could explain this
flitting about; we must fall back on the nervous system for an explanation.
The pain sometimes returns a second time in this way to the same joint, I noticed that
whenever it did so it remained longer, as a rule, and sometimes even became chronically fixed.
The first symptom of the joint affection is passing twinges of pain, which at length
fixes itself in one of the larger joints, primarily at least. This joint becomes next hot and
inflamed; after some hours effusion takes place and the joint apparently enlarges. The
skin which till now was hot and dry, becomes
moist. If we examine one of the joints we find it very much swollen and inflamed. The swelling depends partly on effusion into the joint, and partly on oedema of the skin and subcutaneous tissue.

It is generally observed that there is an increase in the intensity of the pain towards evening, and when morning comes there is a slight remission. In my own case about 10 or 11 P.M. the agony was almost unendurable, but towards morning it gradually lessened, as the morning advanced it was much more bearable. This increase of pain at night I specially noted.

The reason of this may be, that... the gradual abstraction of the sun's rays from the earth as night comes on renders the air more chilly, and this cooling down affects the trophic centres of the articulations. The feebler state of the body at night would also aid this. Another point observed was, that when a fresh
Joint was attacked the pain was more severe before the redness and swelling commenced. Then the latter symptoms became pronounced the pain abated slightly, and when free effusion took place the decrease of pain was still more marked.

The order of events in a joint attacked with rheumatism would thus appear to be as follows:—first, intense pain, then redness and swelling, then effusion into joint, followed by oedema of surrounding tissue. This oedema is followed by abatement of pain.

It would appear as if the inflammation suddenly appeared in the joint, inflamed its synovial membrane, and had gradually expanded its force as effusion took place.

The same thing we notice in pleurisy. When the two surfaces of the pleura—a and the serous membranes are closely allied to the synovial—rub against one another on inspiration, the pain is most severe, but as effusion takes place
it becomes much more bearable. The effusion into the joints I believe acts as a soft cushion, preventing adjoining inflamed surfaces from rubbing against one another, and thus allowing the ligaments and fibrous structures freedom of movement. Such at least was my impression at the time. It seemed as if the effusion lubricated the sheaths of the tendons and prevented the pain a little. It also allowed a little movement.

Another point I noticed was the red lines radiating from the affected joint, showing where the rheumatic inflammation had followed the sheaths of the tendons, thus evidencing its fondness for the fibrous tissues.

The pain I have mentioned is present before any swelling is apparent, showing that the inflammation of the fibrous tissue is the primary change, the effusion, edema, and perspiration being secondary. The pain also is not in proportion to the cutaneous hyperemia, as it is often most severe before the redness of the skin manifests itself, in fact by
the time the redness is seen the pain is often much abated.

State of cutaneous system.

Profuse perspiration of a sour smelling nature, and highly acid in reaction is one of the most characteristic symptoms of acute rheumatism. As a rule the more severe the case, the greater the perspiration. These sweats are of no critical importance. By some they are looked upon as weakening by some as salutary. With such opposite opinions, expressed by those who believe in the lactic acid theory, we may doubt if either expresses the truth. They do not appear to relieve the pain. In fact, the pain is often most severe when the sweating is most copious. If the acid were the cause of the rheumatism its secretion would relieve the joint pain, but this is not the case in practice. A small quantity of this acid given by the mouth to a diabetic patient will cause his dry, branny skin to perspire, so in rheumatism where the skin acts as well
it is no matter of surprise that profuse perspiration is induced. From the large quantity eliminated by the skin it is evident that a very large quantity is formed during the course of the disease. In health it is being constantly formed in the tissues, but it is then oxidized, converted into carbonic acid and water and thus thrown off by the lungs and skin. In the disease it is seen to be eliminated unaltered, which shows that there is produced a larger quantity than can be so converted. These perspirations therefore are merely due to the stimulant action of the acid on the sudoriferous glands and are a necessary symptom of the disease.

The skin is sometimes covered with a morbid eruption resulting from an irritation of the orifices of the sweat glands. Sometimes a red papular eruption is visible where the sweating is not excessive. The free use of Salicylates equally I have seen produce a characteristic rash during the course of the disease.
Temperature and State of the Nervous System.

The temperature varies from 100° to 105° Fahr. In the usual run of cases it is between 101° and 103° Fahr. When it goes beyond 105° Fahr, we have the dangerous condition known as hyperpyrexia produced. It may then even reach 110° Fahr, but these cases are usually fatal. The temperature unfortunately has not the distinctive character seen in other fevers. Wunderlich points out that 102° Fahr. is usually seen when at the worst in an ordinary case, the evening exacerbation, which I mentioned as always occurring, sending the mercury up to 104° Fahr. He also states that in cases admitted to hospital, it is usually highest on the day of admission or the subsequent day. The fall of temperature is usually very gradual. Complications of any kind are generally preceded by a rise of temperature.

The state of matters that gives rise to hyperpyrexia has never been thoroughly explained.

In the Med. Times and Gazette of Oct. 5th 1867, Dr.
Sydney Ringer records notes of three cases in which this was very marked. It is needless for our purpose to relate these cases, suffice it to say, that after the usual symptoms of rheumatism had run their course, there was suddenly developed a remarkable and sudden rise of temperature. After this rise delirium set in, which rapidly lead to profound coma, from suspension of brain action, and death in a very short time. There is, strange to say, never any appreciable lesion found after death to account for this state of matters. Let us look into the nature of this disturbance. Senator thinks the phlogogenic matters in the blood may stimulate the centres presiding over heat production.

Although I am averse to giving a special centre to every thing that demands an explanation, still modern research has all but demonstrated the existence of such. I have already hinted at centres presiding over the articulations, the existence
of which has been ascertained by Chareot and Rosenthal, and I may state that Brown-Séquard has made it pretty certain that a thermic centre, regulating heat production, also exists. This centre, he says, has a distinct set of thermal nerves, apart from the ordinary sensory nerves which convey to the brain impressions of heat and cold. The experiments of Ludwig and Schiff show that the vaso-motor centre, which controls the blood distribution, has its centre in the spinal medulla, so from the connection of this centre and that of heat distribution it is likely that they are in close connection with one another. It follows then that irritation of this part of the nervous system will cause changes both in circulation and in temperature. Now if the centres of the joints are in a state of irritation, which is able to produce an inflammation in their fibrous texture, might not the irritation affect the thermic centres higher up in the cord, the consequence being also a loss of controlling or restraining
power, or in other words hyperpyrexia? I think it may. The various duties of the cord in this respect are so blended one with another, that any disturbance in the one must ... almost as a necessity affect the other. Hyperpyrexia being of course a comparatively rare occurrence, this state of matters does not often occur; but in the case of a patient already suffering, or recovering from an attack of rheumatism, injudiciously exposing himself, the second chill, in the then existing state of matters, when the centres have already been exhausted, instead of a relapse as is often the case, a fatal result might ensue. Perhaps some peculiarity of constitution may determine it in one person more than another.

The hyperpyrexia in itself might not seem so fatal as it does, if other centres in close communication with the thermometer were not likewise involved; but as I have already stated, they are so blended together that this cannot...
possibly happen, more especially in the spinal medulla. It is a fortunate thing that the centres of the joints are not situated in the medulla, which controls life itself, but as we have seen they may possibly irritate sometimes.

Although the disturbances in hyperpyrexia must be most profound, still there remains no trace of it in the cadaver. That no lesion should be discovered on section may be explained by the disturbance being of a functional character. This holds good in all disturbances of the cord in rheumatism, but even in distinct spinal hyperaemia the same thing happens.

In mentioning hyperaemia, it may not be out of place here to mention that in the notes I possess of cases of rheumatism, I almost invariably find the first symptom of pain complained of to be referred to the lumbar region, with vague pains in lower limbs, from thence proceeding to the joints that have been in the habit of being
most need. Now we know that a hyperemic condition of the cord gives rise to these symptoms. Inflammation of the fibrous textures of the intervertebral joints will likewise do this, and especial meaning this has been thought by many to be a complication of rheumatism, because the former disease has begun with these lumbar pains. It may be reasonable, therefore, to suppose that a disturbing condition of the cord, has something to do with the ushering in of rheumatism, and that the lumbar pain may be a direct consequence of this disturbance, or, by inflaming the tissues of the intervertebral joints first, before the irritation is sent to more distant parts, make itself evident in this way.

If the chill be directly applied to the spine, this would easily explain this state of matters; and I believe the chill does so act, the complaint of cold "shivers" down the back being a common one. If we have the starting point here, it is easy to explain how all the articulations
may in turn be affected, and why the inflammation may suddenly leave one joint and fix itself in another; fibrous tissue in any part of the body being all that essential for its development. This could also explain why the inflammation should leave fibrous tissue of the articulations and attack fibrous tissue in the valves of the heart; as it is in the deeper structures of the latter that it originates, in the rings and valves before extending to the endocardial covering; it being the friction, due as the result of the fibrous inflammation and infiltration, that really causes the endocarditis. If the sub-structure was not thus irritated, it is probable endocarditis would not be the terrible sequence of rheumatism that it does is. However, in rheumatism—which means inflammation of fibrous tissue—it is very difficult to avoid.

Microscopic examination proves this view of the formation of endocarditis to be the correct one.
On making a section of a granulation, through fibrin, endocardial membrane, and adjacent fibrous tissue, we find the small nodule to consist of a cloudy swelling of the endocardial membrane pushed up from a multiplication of the cellular elements of the adjacent fibrous tissue. The granulation that caps the projection is fibri whipped up from the blood, collecting on the foreign body.

We shall presently look into the nature of fibrous tissue, but before doing so, it only remains for me to state in connection with symptoms referable to the nervous system, that delirium tremens has sometimes followed rheumatism, the latter probably acting as the exciting cause.

Sometimes, too, we have fatal cases of rheumatism in drunkards, in whom we have reason to suppose the existence of fatty degeneration of the heart and other internal organs. In these cases we have insomnia, very quick pulse, very hurried breathing, face cyanotic, and death occurs in a short time.
Another symptom I have noticed which appears to be purely nervous, is a palpitation, with a feeling of oppression, sometimes even a sense of impending death. This symptom I have seen quite apart from any endocarditis or pericardial effusion, and is probably caused by the pain the patient suffers reacting on the nervous system.

Seat of Rheumatism.

Acute Rheumatism chooses for its manifestation the fibro-serous tissues of the body. Elastic or yellow fibrous tissue.

When examined under the microscope this takes the form of spirals, the ends of these spirals having a tendency to curl up. They are composed of elastic and are unacted upon by acetic acid. The nutritive process is not very active in fibrous tissue, the blood vessels being situated in the periarticular structure.

Acicular or white fibrous tissue.

This consists of bundles of wavy fibres crossing one another, and composed of minute filaments.
or fibrillae. Acetic acid renders them very transparent, they are composed of gelatin. There are two kinds of fibrinous tissue, are widely distributed over the body, especially the white fibrinous, which forms nearly all the lining membranes, such as fasciae of muscles; pericardium; the neurilemma or sheath of the nerves; the aortic and choroid coats of the eye. The yellow elastic, however, enters largely into these structures as well. The tendons and ligaments are formed of white fibrinous interspersed with yellow elastic tissue. The latter also forms almost exclusively the ligaments of the vertebrae and the fibrinous tissues of the base of the brain.

Serous membranes.

These membranes are formed of fibro-cellular tissue. This forms a membrane which contains the blood vessels, the free surface of which is covered with a layer of epithelium. Between these two structures is situated the basement membrane. There are two kinds of serous membranes, those which
line the various cavities, as the pleurae, pericardium, peritoneum, &c., and those lining the joints, sheath of tendons, ligaments, and synovial bureaus.

This latter, viz. the synovial membranes, are to all practical purposes identical with the serous membranes.

The office of serous and synovial membrane is to furnish a smooth, soft, membrane lining for the various cavities they take part in, so preventing their being injured by friction. This end is further promoted by the fluid which is secreted from the free surface of these membranes. This in health is merely sufficient for this purpose, but in diseased conditions is sometimes greatly increased.

As before stated rheumatism claims for its manifestation the yellow elastic and white fibrous tissues of the joints, the serous and synovial membranes.

The parts most commonly attacked are the capsular and synovial membranes of the joints, the ape-
onuritic sheath of the muscles, the tendons, the valves of the heart, which are composed of
white fibrous tissue; and the serous structure of the pericardium. Although the white fibrous and serous elements that in all parts of the body, it is in the above mentioned spots in which rheumatism chiefly manifests itself. The reason of that is that these parts are subject to most movement, and we know that the inflammation shows a preference for such parts, where a free and definite movement takes place. The dura mater we know to be a serous membrane, but how seldom is it ever affected in rheumatism, compared with the pericardium! The reason of this is that the former is never subject to any degree of movement, whereas the heart with its investing membrane never ceases its movements as long as life lasts.

Predisposing Causes of Rheumatism.

Rheumatism is notoriously a disease of temperate climates, although it is by no means unknown in the tropics. Even South Australia which is marked in the map as being free from rheumatism, is really
not so. I had lately under my care a gentleman who had contracted two attacks of rheumatism in Adelaide.

Climate and season have an influence in the production of rheumatism, as predisposing causes, it being always most prevalent in those climates where damp and changeable weather predominate.

It is said to be unknown in some parts of the world, Cornwall, Guernsey, and the Isle of Joge being among the number of favoured spots. It is more common in the East than the West coasts of the kingdom. Exposure to heat does not appear to have much influence in lessening the amount of rheumatism, as Sir A. Tulloch found that it was greater in the navy than in the army. It is seen more frequently in the month of May than in the month of December. It is more prevalent in the Cape of Good Hope than in Nova Scotia and Canada.

The reason of these seeming improbabilities would appear to be that in December the body is generally
better protected against cold and exposure than in the deceptive month of May. In Nova Scotia and Canada, the same thing holds good; the system too is likely to be in a healthier and harder condition in a place like Canada than the Cape. In this latter place, the system would be more relaxed and unwrapt, consequently more liable to be influenced by a chill.

Occupation and mode of life are mentioned as factors in the production of rheumatism, also neglect of the ordinary precaution of changing wet clothes, sleeping in damp rooms, between damp sheets, etc. Rheumatism is found therefore more among those exposed to such causes. It is seen more in the lower than the higher ranks of life, and more frequently in men than women.

Age is another factor in the production of the disease that must not be overlooked. Dr. Thorne found very few cases in those under 15, and still fewer after 50, the acute form never after that age.

From this we see that rheumatism is essentially
a disease of manhood. The reason for this we have already slightly looked into. The exposure to cold and wet is greater then that at any other period of life. A larger amount of bodily exercise is indulged in then; the consequent fatigue and wear and tear of the motor apparatus of the body is greater then than in youth and old age. The boy has not the same amount of work to do, the gaining of a livelihood has not yet begun to influence his mode of life; in short the wear of body and mind that aids in the production of the disease by lowering the whole system, does not yet influence boyhood. Advancing years always tend to diminish the chances of an acute attack of disease. Chronic forms are now more common. That very activity that causes the disease in vigorous manhood is wanting as age advances.

We are therefore that in early youth the disease is seldom seen; it is almost unknown in old age; but in manhood it is one of the commonest of maladies. I have said it is almost unknown at
the age of 50, but this spring I had a case in a woman of 65. I must add, however, that she was a rheumatic subject, and at the time of the attack had been greatly weakened by the change of life. This leads me to notice what was long ago pointed out by Dr. Todd, that disordered uterine function, and the premenstrual state aid in the production of the disease. Todd says, "that the secretions of the uterine, if of an unhealthy character, and not duly thrown off, may be absorbed into the circulation and contaminate the blood, producing symptoms of greater or less urgency, according to the nature and quantity of the morbid secretion which may have been absorbed."

Todd it would thus appear is of opinion that the unhealthy uterine secretion be absorbed it is sufficient to produce an attack of rheumarthritis. From what I have seen of cases of this sort I am of opinion however, that mere must always be present the constitutional tendency to the disease, and that the deranged uterine merely acts as
an aid in the production of the disease, by weakening a system already prone to contract the disease. The woman I have mentioned above is a good case in point. She was of a marked rheumatic constitution, having had a previous attack some 12 years before, after her last confinement. Now here was the rheumatic tendency asserting itself after the parturition, when the system would be weakened and lowered. There was not any absorption of morbid matter then, as she had merely in her weak state injudiciously exposed herself to cold. The same causes had no doubt produced the second attack.

Rheumatism, like its twin-brother gout, is very apt to attack the system when it is lowered for any cause. It appears as if, in those predisposed, it was the body only requires to be below par, and exposed to cold to induce an acute attack. Gout takes the same mean advantage of its victims. Should there be a joint that years before had been sprained, or a bone that had been broken, mat
to the part subject hold of which most anxiety.
Rheumarthritis has been known to follow dysentery
and scarlatina, which further shows its liking for
a debilitated constitution. Cases have also occurred
where it has followed prolonged blood letting. Had
it been due to a poison in the blood surely it
would not have happened then!
Anything that tends to weaken the constitution,
we therefore see, tends to invite an attack.
Overfatigue, grief, mental emotions, sudden shocks
and all tend, by lowering mind and body,
to favour Rheumarthritis.

Pathology of Rheumarthritis.
From time immemorial the pathology of this disease
has been shrouded in mystery. Many attempts have
been made to assign a reason for its production,
but all have more or less signally failed. The theories
advanced are so different that we shall require
to look at each in detail, and then endeavour
to gather from some of their grains of truth, on
which we shall hope to build a good and rational
theory, the outcome of which ought to be a rational and successful treatment.

**Theory of cold.**

Since the time of Hippocrates, cold has been alleged to be the cause of rheumatism. It is a convenient word to employ when assigning a reason for an attack. It is very much easier, however, to say that cold causes rheumatism, than to explain how it does so. We still hear old physicians talk in this way. It is sufficient for them that cold brought it on. They give a prescription for a potash mixture, and very probably never think any more of the matter.

It behoves us, however, in this thesis, not to rest content with any such superficial explanation, but to endeavour to trace the disease to its fountain head, to get a glimpse of its true pathology, and on that pathology to build up a successful treatment.

That cold, or rather the irritation it produces in the system, plays its part in the morbid process I do not deny; on the contrary, believe
that it is a most important factor in the causation, but only a factor; so we must be careful not to raise it from its secondary position as such to the rank and dignity of a cause. Simple exposure to cold could not explain all the phenomena of the disease; there must be some other state of matters present to produce such a peculiar train of symptoms known as acute rheumatism.

The symptoms following exposure to cold are indeed quite different.

Cold when applied to any part of the body produces a chilling of that part. There is a paleness of the surface visible from the blood being sent to the more internal parts. If the cold be now removed, an increased circulation takes place with a feeling of warmth and comfort. This may be exemplified by a cold plunge or a shower bath.

If a greater degree of cold be applied the part becomes white; the skin wrinkles and sensation is diminished. After a time these symptoms pass away; the skin becomes
red and the part so treated may swell. There
is a peculiar itching felt.
If a still greater cold be applied complete gangrene
and frost bite supervene.
These symptoms are vastly different from what
we know as rheumatism. There are no splitting
pains, no pycrosis, no cardiac complications,
in fact none of the characteristics of rheumatism.
Many however tell us that if dampness be combined
with the cold the disease will follow, and there
is some truth in this as I shall afterwards show.
If cold per se were the cause of the disease, and
did cause it when the body was exposed, how
would the fact be explained, that thousands of
working men are daily exposed to cold and damp,
yet do not contract acute rheumatism.
Again if cold was sufficient to account for it
we should expect to find it in the young and
aged who cannot so readily withstand it;
but this is not the case as we have seen
when looking into the question of age.
Still further, how seldom does rheumatic inflammation end in suppuration, whilst this is the ordinary termination of an inflammatory mischief produced by cold. When the theory of cold was discarded as being unable to explain the phenomena of rheumatism, the one introduced was the Lactic acid theory.

One of the most characteristic symptoms of acute rheumatism is the occurrence of profuse acid perspiration, the urine is hyperacid, the very saliva has an acid reaction.

That lactic acid was the matter morbi of rheumatism was first suggested by Dr. Prout, and later by Todd, Fuller and Wells. These physicians believed that lactic acid is generated in the system as a product of malassimilation or faulty metamorphic action, and accumulates in the blood from defective cutaneous action. This view has been received by the profession generally as giving a satisfactory explanation of the evolution of rheum arthritis.
It has been demonstrated that lactic acid is formed in muscle during exercise, and after such exercise there is excess of acid in the system. If a checks or chill be given to the cutaneous apparatus, it is not excreted as it ought to be; it accumulates in the system, and symptoms of rheumatism result. Such is the perfection of the lactic acid theory, but we cannot accept it without first giving it a careful consideration.

Lactic acid is very difficult to discover in the perspiration on account of the readiness with which it decomposes. It has, however, been demonstrated by Favre. The acid appears to act as a stimulant to the subcutaneous glands; the larger the production the more active the elimination.

In the year 1857 Dr. Richardson injected lactic acid into the peritoneal cavity of dogs and cats, and about two hours after the experiment the heart's action became irregular, and next morning the animal was found dead. No peritoneal mischief was found, but marked endocardial inflammation
There was no swelling of the joints or pericarditis. Dr. Richardson states that in his experiments the right side of the heart always suffered, and not the left as is the case in endocarditis following rheumatism. He explains this anomaly by saying that in his experiments the poison was introduced into the body by an absorbing surface, and found its way into the circulation by the venous blood. It would consequently come in contact with the inner surface of the right side of the heart first. In rheumatic endocarditis he explains the fact of the left side of the heart being most affected by saying that the poison is a product of respiration; consequently in traversing the circulatory canals comes in contact first with the inner surface of the left side of the heart; while in the systemic circuit it undergoes less. Further on he says that the lactic acid being present in the blood causes the mischief by direct contact with the endocardial surfaces. It acts like a local irritant.
The results of his experiments have been supposed to be most favourable to the lactic acid theory. Indeed they are quoted in most of the text-books as being conclusive proof of the truth of the theory, and as explaining the phenomena of the disease. But it only requires a little examination into the matter to show that Dr. Richardson's statement is erroneous.

She says that in rheumatic nodular disease lactic acid is produced in the pulmonary and destroyed in the systemic circuit.

I think we shall be able to prove the very reverse, viz.: that it is formed in muscle, blood or in the systemic circuit and destroyed in the pulmonary one.

Let us look into the process of lactic acid formation in health; because the acid is the product of the disintegration of tissue and as such is a normal ingredient of the system. The blood containing the products of digestion is collected from the capillaries of the intestines.
by the portal vein which carries it to the liver. In the liver it undergoes some changes. One of these is that the sugar it contains is converted into glycogen or liver sugar and as such is stored up in the liver. This liver sugar however can be produced from the secretion or elaboration of the hepatic cells of the gland itself as was shown by Bernard. This liver sugar passes on by the vena cava into the lungs, but is not, as many suppose, burnt off as such there. The experiments of Lehman & Ludwig tend to show that grape sugar injected into the veins of an animal, causes little or no increase in the Oxygen consumed, or the carbomé acid given off from the lungs. The sugar now goes on into the muscles and is there destroyed. In its destruction lactic acid is produced. The following equations will show the process:

\[
C_6 H_{10}O_5 + H_2O = C_6 H_{12}O_6 = \frac{2}{3} C_4H_8O_2 + C_2 H_4O_2
\]

Liver Sugar  Water  Glucose  Lactic acid  Lactic acid

The lactic acid thus produced is got rid of
as carbonic acid and water by the addition of oxygen.

\[
2C_3H_6O_3 + 6O_2 = 6CO_2 + 6H_2O
\]

Lactic acid + Oxygen → Carbonic acid + Water.

To effect the formation of this acid a good deal of oxygen is consumed and a good deal of carbonic acid given off. From the muscles, the blood, by this time deprived of its sugar and substances derived from it, is gathered up and taken by veins to lungs there to meet with other blood from liver, and again goes the round of the system playing the same part.

These changes in the tissues—the formation of lactic acid and its oxidation—go on so smoothly that no acid reaction can be detected in life. When they die however, or from any irritation of their centre, the disintegrating power is diminished, the lactic acid is not oxidised, they become at once acid and this acid is given off unaltered in large quantities in the sweat.

We see therefore that his conclusions drawn by Richardson are faulty. He fails to show any relationship between induced and spontaneous endocardium.
The disease he says he produced too, was endocarditis, not rheumatoid arthritis which is the subject under discussion. There is no mention made of arthritis or pericarditis, which are as important symptoms in the disease as the endocardial mischief.

Again, we know that experiments on dogs do not always hold good in man. Dogs do not perspire like the human subject, and drugs administered to them may not affect them in the same way. Reyer and Vereshow, also, found that endocardial mischief was common in dogs without any previous injection of lactic acid.

Fallacies such as these therefore may creep in and tend to invalidate experiments of this sort. Richardson would imply that lactic could not be present in the blood without causing endocarditis, but our clinical experience teaches us differently. We know it exists in the blood in rheumatoid arthritis always, but endocarditis only is noticed in a few cases.

That it is a local irritant is also absurd, because
if it were so we should have endocarditis in every case. I do not deny that lactic acid is present in large quantities in the blood in rheumatic arthritis—every one must admit that, but I hold it to be only a symptom of the disease, and not by any means the cause of it. I trust to be able to show in due time that its production must be regulated by a higher power to which we must look for an explanation of the cause of rheumatism. We must, however, examine first, some other evidence recorded in favour of the lactic acid theory. Since lactic acid has been given as a cure for diabetes, some cases of rheumatism have followed its administration.

In his Clinical Medicine (574) Dr. B. Foster records a case in which the administration of lactic acid was followed by arthritic attacks.

"The phenomena," says Dr. Foster, "corresponded in all respects to those which are characteristic of acute articular rheumatism. They came on when the acid was taken, and ceased when it was dis-
-continued. When moderate quantities of the acid were tolerated, an increase in the dose was succeeded by the painful inflammation of the joints, coinciding with the development of the articular affection; the appearance of perspiration, at first only slight, but afterwards, in the more severe attacks, copious and acid."

When this case of Dr. Foster's was published, it was immediately laid hold of by the supporters of the lactic acid theory, especially by those who had some doubts of Dr. Richardson's experiments, as being conclusive proof of the accuracy of the theory, and at first sight it undoubtedly appeared striking testimony.

But here again the same mistake was committed; a symptom of a disorder was raised to the dignity of a cause.

Dr. Foster's evidence is to the effect that lactic acid given by him to a diabetic patient, caused articular pains, i.e., resembling rheumatism. Let us examine again into this matter.
Loeic acid, we have already seen, is a product of the retrograde metamorphosis of tissue. It is therefore a normal constituent of the tissues, the muscular more especially, as we have seen. It is decomposed into carbonic acid and water and easily got rid of. This process, the formation & decomposition, goes on so smoothly in health, that the acid gives rise to no disturbance whatever in the economy.

There are two conditions however, in which this equalable process is interfered with, and in which an increase of acid takes place. These are active muscular exercise, and acute rheumatism.

After active muscular exercise, there is an increase of acid in the muscles. Whenever this acid is produced it acts as a stimulant to the organ which eliminates it. It has no wish to remain in the muscles and joints, increased formation being always attended with increased elimination. It is thus easily got rid of.
This is how the acid is disposed of after exercise. The lactic acid theorists hold that the acid is the cause of the inflammation of the joints. This is in fact the foundation of their theory of rheumatism. The association of the occurrences is the sole cause for the belief. I hope I shall be able in due time to show that the inflammation precedes the formation of the acid.

I have said that lactic acid is formed in the tissues in health. These tissues are the muscle and fibrous tissues. The two are in reality one as far as their function, conservation, etc., is concerned. The sheaths, tendons, ligaments, etc., are so intimately connected with one another, that changes that take place in one are very apt to take place in the other, and after an attack of rheumatism the loss of weight in the muscles shows how deeply they have sympathized with the joints. It follows therefore that inflammation or disturbance of the fibrous textures of the joints, will be accompanied
with inflammation or disturbance in the muscles. As a direct consequence of this there will be increased production of acid, which, not being oxidised and got rid of in the usual way is eliminated unchanged. These acid sweats we have seen are one of the chief symptoms of the disease. This is how retained excretory products act.
by blood.

Inflammation or disturbance in the muscles.

Now after inflammation or disturbance in these tissues, lactic acid is not oxidised but passes off unchanged in the blood. Its presence here does not produce any toxic effect, only, by the ordinary laws of diffusion, interference with the retrograde tissue change, causes an impediment to the passage backwards into the circulation of the products of tissue waste. It so interposes itself in all ordinary tissue change.

The same as retention of carbonic acid is a bar to the passage backwards of that gas from the system. Its retention therefore in Dr. Foster's case would cause disturbance in the joints and muscles, in the former especially as they would give more evidence of functional disturbance, and any weakening of the latter would be lost in the already weakened and wasted muscle of diabetes.

We should expect to find pain too in these cases of retention in the febrile tissues; the disturbing agency would be sure to cause it, the same as retained excreta elsewhere cause different
symptoms in the respective organs in which they are retained.

Dr Foster's case was one of diabetes, a disease where the skin— the great eliminating medium — does not act at all freely. After the death, the body was toxic in its lungs. Lactic acid therefore given to a patient of this sort, would not likely be very rarely absorbed and eliminated. It was certain to become a retained excrement and behave as such, viz:—by giving rise to disturbance in the part in which it was retained.

It is a mistaken idea to regard the excess of lactic acid that is undoubtedly present in rheumatism as an accumulation simply. What we mean by accumulation is that it is being formed but not properly eliminated. This elimination goes on most profusely in rheumatism, the more severe the case the more profuse the elimination, so that no accumulation really takes place.

It may be said that if you admit the presence of lactic acid in the blood you admit the lactic
acid theory to be true. But this is not the case. An excess of acids in the blood every one must allow, but it does not follow that this is the cause of the disease! The lactic acid theorists say that is the materia morbi, the special something which sets against the malady. But it cannot be the cause of its own presence! It is merely a symptom of the disease, which is almost always present, and a consequence of the process which initiated it. Lactic acid is in excess in the blood, but what causes the acids? Dr. Foster's experiments throw no light on this, the most important point in the pathology of rheumatism.

Weak points in the lactic acid theory:

1. It cannot account for the excess of the acid.
2. If excess of acid is the cause, we should expect neutralizing it with alkalis would relieve the pain and cure the disease. It does not do so.
3. Sulfur, which undoubtedly relieves the painful joints, has no action whatever on the acids, the secretion often remaining acid for many
days after the pain has gone.
We have thus seen that the lactic acid theory cannot explain the causation of rheumatic fever or its peculiar symptoms, so let us look at some other. The next best known one is the Malarial, bacterial or Fungoid theory.

The "Lancet" of Nov. 1867 in a leading article says:-

"In the last number of the American Journal of Medical Science Dr. Salisbury has professed a startling hypothesis relative to rheumatism. He states that the blood contains masses of minute algoid filaments (zymotoic translucent) and he believes that it very probably gives plasticity to the colourless corpuscles, and favours their mutual adhesion into masses which lead to embolism in the vessels, and the symptoms of rheumatism."

This startling doctrine was soon refuted by Dr. Wood another American physician, who declared that these fungi could not exist in the blood under the circumstances inherent to acute rheumatism, and showed that the peculiar history of rheumatism..."
as compared with that of a disease undoubtedly produced by vegetable organisms showed how essentially different they were.

We cannot therefore say much of this fungoid theory of the disease. Later experiments throw no light on it, in fact, they tend to contradict it.

The theory however is interesting as showing that so far back as 1867 the idea of spores in the blood was mooted as a probable cause of the disease.

Malarial theory.

Malarious remittances had long been mentioned by physiciens as a probable cause of the disease, but the matter was never fully discussed till Dr. Macleagan in the 'Lancet' of Nov. 1846 reintroduced it to the profession as the explanation of the success which attended the treatment of the disease with Salicin.

In his paper at that time he says:— In the course of an investigation into the causation and pathology of acute fibrile ailments which he for some time back engaged my attention, I was led to give some consideration to intermittent and to rheumatic
fever. The more I studied these ailments, the more was I struck with the points of analogy which existed between them. On a detailed consideration of these I shall not now enter. Suffice it to say that they were sufficiently well marked to lead me to regard rheumatic fever as being, in its pathology, more closely allied to intermittent fever than to any other disease; an opinion which, further reflection and extended experience has served only to strengthen.

This rheumatic theory, as I have said, is thought by Dr. Macleagan to explain the success of his saline saline treatment.

He thinks that there are many points of resemblance between malarial fever and rheumatism: notably, that the former is especially apt to occur in low-lying damp localities, in certain climates, at certain seasons of the year. That some people are more liable to be attacked than others. That they have no definite period of duration. That they are not communicable from the sick to the healthy.
All this, he says, applies to rheumatism.
The symptoms too, he adds, are analogous, for
malarial fever is irregular in its type; so is
rheumatism. Also, one attack renders the
system more liable to a recurrence, and that
arrested by treatment they may have an uncertain
and protracted course.

Dr. Macleayan is at great pains to go fully into
the nature of malaria, which in regard as
a minute organism, the bacillus malariae,
which when introduced into the system, produces
fever of the intermittent type.
Klebs and Tommasi-Crucioli have indeed
demonstrated this bacillus, which consists of ovoid
spores, and which if mixed with water and
injected into animals produced a fever of the
intermittent type.

Macleayan holds that the rheumatism is caused
by the malarial organism, if not the bacillus, by
an allied one. He believes that if it be introduced
into the system, it will give rise to symptoms.
of rheumatism. He goes very fully into the analogies between malaria and contagion, as being both agencies introduced from without, and as likewise being analogous in some respects, but likewise differing in some things, notably in the malaria not being communicable from the sick to the healthy.

He believes that these organisms enter the system somehow, and give rise to inflammation in the fibrous tissues of the body. The shifting character of the joint mischief, he explains by the resemblance to malarial fever with its intermissions and remissions, caused, he says, by the poison being reproduced in the system during the course of the malady. "That that which produces the symptoms of today, has tomorrow ceased to be active, but has given rise to an offspring which keeps up the action which its parent set a going, and in its turn hands down to its offspring the same noxious properties which itself received?"
He does not deny the presence of haetic acid in the blood, but thinks it follows the joint inflammation set agog by the germs, or that it may even be the product of the destructive disintegration of these germs.

This then is briefly Dr. MacKay's bacterial theory in regard to the pathology of rheumatism.

We see that it somewhat resembles Dr. Salisbury's fungoid one, and like that one is open to some doubts as to its correctness.

The theory does not fit in with all the facts in the history of acute rheumatism, let Dr. MacKay explain them as he may. For instance the fever does not occur periodically or epidemically like other diseases owning a material origin. The shifting character of the joint affection can, I think, be much better explained by a nervous origin than by Dr. MacKay's hypothesis, which I venture to say is rather far fetched.

In behavior too, of the disease under the salvein treatment hardly warrants so in ac-
Accepting the malarial idea of its origin, one would expect that the salicin, if it can destroy bacteria in rheumatic fever, would also be able to do so in eruptive fevers, undoubtedly due to this cause. But the remedy is powerless. In some cases, too, rheumatic arthritis arises under circumstances which entirely preclude the idea of malarial organisms. That the presence of these organisms in the system can account for the inflammations of the joints and varied phenomena of the disease I can hardly credit. Notwithstanding this, I cannot shut my eyes to the benefit derived from salicin, but I think its success can be explained in a much better way, draw its power to destroy bacteria, whose very presence we can only guess at, whose existence may be a myth. Before discussing the special origin of rheumatism, I may mention that Hunter of Griezendorf holds, that endocarditis, even though undescended till afterwards, is the primary pathological factor in
The production of the disease; embolism follows the endocardial marking and causes the joint pains. More recently however he has hinted that when the body is heated the blood vessels in the skin are dilated, the “microcoeli” in the atmosphere rains into the system causing the chill. Whatever amount of truth there may be in the latter supposition, we can scarcely support the embolic theory; for why should all the organs of the body be exempt from the usual consequences of embolism except the joints?

Spinal Origin of Rheumatic Arthritis.

In the year 1831 Dr. Willard Mitchell of Philadelphia published a treatise on Gunshot injuries to nerves, in which he states that in many cases of wounds of nerves especially spinal nerves, symptoms indistinguishable from acute rheumatic arthritis occur. The joints become stiff, swollen, and painful. He attributes this to the spinal irritation always present in such cases.

Many years after the publication of Dr. Mitchell's book...
Prof. Charcot voiced a work entitled "Léçons sur les maladies du système nerveux, fait à la Salpêtrière." Among other things he says that the disorders of nutrition consequent on lesions of nervous centres not infrequently have their seats in the joints.

The arthritis may be of an acute or subacute form, and is accompanied not only by the outward signs of inflammation, but by changes in the joints themselves of a distinctly inflammatory character; that is to say, nuclear proliferation in the synovial membrane, which increases in size and number of capillaries, and in severe cases exudations into the joints of a serous fluid mixed with leucocytes in various proportions. The cartilages do not usually participate in the changes, but the adjacent synovial sheaths and fibrous tissues of the joint are acutely inflamed. This description at once puts us in mind of acute rheumatic arthritis, the anatomical appearances and physical signs being so strikingly alike. So much so is this the case, that Prof. Charcot is
at considerable pains to draw up the materials for the differential diagnosis, and failing to find an essential difference, grounds his diagnosis on concomitant circumstances such as paralysis in the limbs affected. But surely, a resemblance so very close as this existing in essential characters, as well as in the more superficial appearances can scarcely exist without a similarity at least in the nature and cause of the disease. At any rate here seems to be an important argument in favour of the hypothesis of the nervous origin of rheumatic arthritis. It happens most frequently after traumatic lesions of the cord. In the cases narrated by Charcot and M. Viguis and Joffroy the arthritis occurred in the same side as the paralysis and spinal lesion, an exchange forming on the other.

The experiments of Dr. Brown-Séquard on animals show that after wounds of a lateral half of the spinal cord, there supervenes motor paralysis of the lower extremity on the same side as the lesion. The limb presents a marked eelation
of tactile sensibility, a notable rise in temperature, and vaso-motor paralysis. The opposite limit, on the contrary, preserves its normal temperature and power of motion, whilst the tactile sensibility is much lessened and may even be extinct. These particulars are reproduced in many under analogous circumstances.

Although these remarkable arthropathies are seen most frequently after traumatic lesions of the cord, still Cachor and Gull record cases following myelitis, ataxia, &c.

There are two varieties of these forms of joint inflammation: following spinal lesions—a benign form which terminates in recovery in several weeks or months, and a severe form which causes erosion of the joint surfaces and after a time dislocations. In this latter too the whole fibrous structure of the joint becomes disorganized.

The same phenomena were observed also to follow "ranckioment" and cerebral hemorrhage. The symptoms generally come on 15 days to a month after the motor disorder. These latter cases were joint
described by Dr. Scott Alison in 1846.

Scott Alison attributed the arthritis to the fact that the healthy relations between the living tissues and the materials of the blood were disturbed. Two or both conditions gave rise to the disturbance, viz: - a state of reduced vitality in the paralytic parts, and the presence of motes and exciting agents in the blood. In proof of this he mentions two cases in which the red line of the gums, following the use of mercury, in paralysis on one side of the face, was strictly confined to the paralyzed side of the mouth. The paralyzed parts were in fact more delicate than the person than in parts in a state of health. In proof of the presence of exciting agents in the blood, the sanguine diabetes of the second case and the lithic acid calculi in the pelvis of the kidney of the first case were added. Bawden, however, believes that these arthritis are explained by his pathogenic theory, and believes that Brown-Eyward has the merit of first explaining their organic cause. This latter physician says that
it is most important not to confound the sensations of prickling etc. often in paralysed members, with other and often painful sensations in the joints of paralysed limbs. These last sensations very rarely exist when the limbs are not moved, or when there is no pressure upon them; they appear at once or are increased by any pressure or movement. They depend upon a subcutaneous inflammation of the muscles or joints which is often mistaken for a rheumatic affection. This inflammation in paralysed limbs is often the reason of an irritability of the vasomotor or nutritive nerves of the brain."

It is in the grey matter of the anterior cornua of the cord that Charcot believes these curious complications of ataxia to be found.

In two cases of the reverse form of ataxia he had an opportunity for examination, and says "it is not very rare to find an spinal grey matter affected in locomotor ataxia, but the lesion is then generally found in the anterior cornua. Now, it was quite different in the two cases of locomotor ataxia com-
presented with arthritis in which a careful examination of the cord has been made. The anterior columns in both cases were remarkably wasted and deformed, and a certain number of the great nerve cells, those of the external group especially, had decreased in size and even disappeared altogether, without leaving any vestiges. The alteration besides showed itself exclusively in the anterior columns corresponding to the side on which the articular lesion was situated. It affected the cervical region in the first case, where the arthritis occupied the shoulder. It was observed a little above the lumbar region in the second case, which presented an example of arthritis in the knee. Above and below these points, the gray matter of the anterior columns appeared to be exempt from alteration." From this it is said, Charest, "to have made it appear highly probable that the inflammatory process first developed in the posterior columns, by gradually extending to certain regions of the anterior columns of gray matter was able to occasion the development
of the articular affection in our two patients.
If the results obtained in these two cases are
confirmed by new observations we should naturally
be led to admit that arthritic affections connected
with myelitis, and those observed to follow an
encephal softening, are likewise due to the
invasions of the same regions of the grey matter
of the cord. In cases of brain-softening the
descending reteos of one of the lateral columns
of the cord might be considered as the starting-
point of the diffusion of the inflammatory work.
Such were the facts before us of arthritic affec-
tions occurring in cerebral and spinal lesions.
These facts show us that when certain portions of
the spinal cord are disturbed, functionally or organically,
arthritic symptoms may supervene.
The trophic centres of the muscles and joints, according
to Rosenthal, are in close proximity to one another
in the cells of the anterior horns.
It follows therefore that it must have been a
disturbance of these centres that gave rise to the
arthritis described by Charcot.
Let us apply this knowledge to what we have seen
of the symptoms of rheumatoid arthritis and inquire if
it can throw any light on the hitherto obscure
pathology of the disease.
No theory of rheumatoid arthritis can be perfect without
admitting a "chill" as the initial oratory symptom.
I remember in my own case this was most marked.
I have also said the system is always weakened from
some cause or other before the attack, and explained
that any cause which does this, prepares the way for the arthritis. This weakness also,
we must take into consideration.
The rheumatic constitution too, we must not lose
sight of.
Now, given these three factors, an attack of
acute rheumatoid arthritis can be produced.
How can it be so produced?
The cold, or rather the depressing influence of the
cold, affects directly the spinal cord. No other
portion of the body would give rise to the shivering.
chilling rigor but the cord. The nervous system cannot be excluded from the idea of chill. This chill gives rise to characteristic symptoms proceeding from the spine: dull aching pains, fleeting joint pains chiefly in lower limbs, general feeling of "malaise". If the disturbance of the cord is not of a very severe nature, these symptoms may pass away and the patient recover in a few days. But if the depressing action has been powerful enough, the centres are acutely disturbed, pyrexia and joint pain soon follow. This increases, acid sweats begin to pour from the body, giving rise to what we term rheum or rhutes. We observe that the acid sweating does not occur till the articular pains, and the pyrexia in consequence, are well advanced. This clearly shows that the inflammation of the joints, and their intimate relations, the muscles, by causing a disintegration of the latter will cause increased formation of acid. The order of events would appear therefore to be this:
1. Hyperaemia of trophic centres of joints, in the anterior cornua of gray matter.
2. Inflammation of fibrous textures of joints, muscles, soon following.
3. Acid perspirations as in consequence.

I cannot urge too strongly the belief in the rheumatic constitution. The same chill in a non-rheumatic subject would often give rise to slight pains probably pass away, but in the rheumatic one the centres presiding over fibrous structures of joints become irritated and arthritic results. This would seem to point to the rheumatic taint consisting in a special weakness of these centres and the fibrous structures, which render them more liable to be profoundly affected by the chill.

We know from the experiments of Claude Bernard on animals, and from Chacron's observations on men that paralysis of sympathetic in a healthy strong subject does our cause
trophic changes in a joint; but if the subject be weakened or debilitated from any cause arthritis is most marked.

This might explain the effects of a chill on a non-rheumatic subject, and a rheumatic one. Such then is my idea of the pathology of acute rheumatic arthritis. It gives a reasonable explanation of the phenomena of the disease and can aid us in understanding the wonderful efficiency of salicylic as a remedial agent for its cure. It can only be by a direct influence on the nervous system that its action can be explained. It is useful in diabetes and probably its action is the same in both diseases. A blow on the head will produce diabetes, with sugar in the urine.

A blow on the spine can produce arthritis with lactic acid in the blood if the subject be of the rheumatic habit. An interesting case is related by Dr. Full of concussion of spine producing marked arthritis. The two diseases are probably
related to each other in other ways, sugar and lactic acid being two elements of the system, which go hand in hand. If a needle introduced into the floor of the 4th ventricle will cause diabetes, there is no reason to doubt but that a needle introduced into the anterior cornua (from its disturbance of adjacent sensory parts as posterior cornua or sensory tract) will cause arthritis.

The chill may act directly on the spine, which is a most unprotected part of the body; the front of the body is generally well protected by wraps, but the spine is never so guarded, hence rain, cold, etc., acts on it readily.

Fibrous tissue in acute rheumatism, being throughout the whole body in a state most likely to take on inflammatory disturbance, the "metastasis" from one joint to another, or from joint to heart, is explained by the nervous hypothesis of its origin. The disease being rarely fatal, few opportunities are afforded of studying the structural alteration to which it gives rise. The synovial membrane of
The joints have been found more or less edematous, the inner surface swollen and cloudy. The connective tissue also inflamed and thickened; the soft parts about the joint variably congested and edematous; sheaths of tendons inflamed; serous contents of bursae increased; jaundice very rarely seen. The blood is firmly coagulated after death.

In hyperpyrexia, a swollen condition of the hepatic cells has been found, but seldom any appearance is found on section. The same holds good with other parts of the cord. This is nothing remarkable as very profound disturbances of the cord, often leave no trace whatever in the cadaver.

TREATMENT OF ACUTE RHEUMATISMS

I prepared this thesis with the remark that perhaps no disease since the days of Sydenham has attracted so much attention as rheumatism; I think I may safely add, now, that perhaps no disease has hitherto proved so stubborn to all forms of treatment. Here I would say that nearly
every drug in the pharmacopoeia has been tried for the cure and relief of the symptoms, I do not think I should be guilty of any exaggeration.

From time to time we have had some new remedy extolled by its advocate as being the only one to be relied upon in the treatment of the disease; but on being employed, it has been found alas! to be utterly useless, and in many instances injurious to the patient to whom it was given. For instance in the olden time, when fever was synonymous with inflammation, antiphlogistic measures were of course the order of the day. Large bleedings from the arm, mercury, and calomel, were thought to be the only reliable method of treatment. Among the most enthusiastic advocates of bleeding was Bouilland. This physician used to recommend as much as four pints of blood to be drawn at the commencement of the attack. He thought that by so doing he could lessen the heart's action, and by thus diminishing the inflammation cut short the disease. Such are the errors a faulty pathology may lead us into! Bouilland was at last forced to admit
that inflammation of the heart had been the rule and not the exception in his practice. It was not, however, until the middle of this century, when Todd, Fordyce, Watson, and Fuller, pointed out by unimpeachable statistics that the mortality of the disease was actually increased by such means, that bleeding was abandoned.

Then salmi purgatives with colonel, or colonel in combination with opium, were vaunted as the means of at least modifying the disease. But all these were found through time to be unavailing, and like the bleeding found to do sometimes more harm than good.

For, as suggested by Prof. Grainger Stewart, purgation of any kind in a patient bathed from head to foot in a state of prostrated perspiration, requires to be carefully dealt with. He mentions instances of patients who having to attend to be calls of nature from this purging, had relapses and even heart mischief; besides, every movement of the patient being attended with pain, we urge it to allow him all the rest he can get. A free evacuation of the bowels, however, as the commencement of the disease I believe
to be productive of benefit.

Mercury was next recommended, but Fuller observed that both pericarditis and endocarditis supervened as readily when the patient was under the influence of mercury as when that drug had not been administered.

Antimony was recommended by Larrey as being more efficacious as being most effective in subduing the articular inflammation; but this too was found on the whole to be of no material benefit.

Opium, first used by Cazenove in Paris, and Corrigan in this country, is about the only drug that has stood the test of time in the treatment of the disease. There is no doubt that in the early and most painful stage of the disease, it is invaluable for allaying the agonizing pain and subduing the nervous and vascular excitement. It is now generally employed in the form of morphia, the grain being an immense
boon to the sufferer. In the form of Dover's powder it rather does harm by exhausting the patient still further, by its very copious diaphoresis. Strychnine Bark was next given by Haygarth and latterly by Heberden, Fothergill, etc., but it was never found to be of much benefit in cutting short the disease.

Colchicum has long been looked on as a specific for rheumatism, but now it is seldom used in the acute form at least. In chronic cases, especially those associated with gout, it is a valuable remedy. It is still, however, administered by physicians of the old school in combination with alkalis in the acute cases. They claim for it the power of alleviating the symptoms and shortening the duration of the disease. It undoubtedly promotes the excretion of effete matters, whether rheumatic or gouty, by means of the kidneys and may thus help to arrest the progress of the disease. Fuller has found that it has proved less advantageous
as the fever has exceeded the articulation swelling, and as the urine has been highly charged with urates. Twenty minims of the wine of colchicum may be given in combination with alkalies, twice daily, but its action must be watched. A free evacuation should be obtained every day, but if the pulse become weak, or faintness supervene, it is advisable that the exhibition of the drug be stopped altogether.

Guaiacum is another remedy still used in acute cases, but found to be of much more value in those of a chronic nature. When, however, we have scanty perspiration and secretion, we may expedite measures by guaiacum aided with hot soup or gruel. In combination with the bittersweet of potash, sulphur and rhubarb, it has obtained great repute in the army for the pains of old soldiers and is known among them as the “Chelsea pensioner”.

Fritiate of potash was at one time highly recommended for the relief of the joint pain
but it soon lost ground, having been found when taken alone to exercise no control
whatever on the course of the symptoms.
Alkalies. Next we must notice the treatment
which in modern times has eclipsed the
preceding, and the one which has lately has
perhaps been of more avail than any other.
Then Bonillant's treatment who proved fallacious
physicians began to think that the pathology
on which it was founded must also be faulty,
and they began to investigate more closely into
the nature of the disease. The outcome of this
investigation was Proust's lactic acid theory,
accepted by the teaching of Fuller and Todd.
They firmly believed that lactic acid was
the cause of the disease and not merely one of
the symptoms of it. Of course their rectaction
was the administering of an alkali with the view
of counteracting all the deleterious effects of
this acid. Undoubtedly this alkaline treatment
was a great advance upon all the other
remedies I have enumerated, and is based on their ideas of the pathology. The alkalies do doubt may diminish the fibrin in the blood, and may warm the irritable heart. They also cause an increase in the flow of urine, and an augmentation in its solid constituents. The breakbane of potash is frequently given, but little the nitrate is ever to upset the stomach when given for any length of time. The acetate, is, on the whole, the best form in which to administer the potash salts, in doses of 20 grains every two or three hours, till we obtain some relief. A mixture containing the acetate with some Cinchona and Morphia in some orange flower water, is perhaps the best method of administering it. Through time the alkaline treatment was found to be materially to effect the ordinary duration of the disease. Its efficacy in diminishing heart complications was also called in question. Noting these things Dr. Owen Rees suggested the administration of Lemon juice, as in his opinion
yielding as good results as that of the exhibition of the alkalies. He holds that in virtue of the excess of oxygen it contains, it promotes the conversion of lactic acid into urea and carbonic acid, favouring thus its excretion from the system, while the alkaline citrate it contains keeps in some measure to arrest the progress of the disease. Guild and Sutton found that under the lemon juice treatment the acute stage of the disease was reduced to 6-8 days, whilst with the alkalies it was 6-75 days; under the blistering plan it was 8-14 days, and 9-1 under an expectant regimen. We thus see that lemon juice is of some little service in checking the disease. This I think must be admitted by all who take an interest in the statistics of the disease as observed in the various hospitals. The remedy, however, has however the drawback of being uncertain in its action, and is much less speedy in alleviating suffering than other methods we shall now enumerate.
Torure of the moisture of iron was first recommended by Dr. Russell Reynolds, and his results were as good as any of the preceding. The rash employment of iron, however, as iron in combination with quinin, has been blamed for the production of hyperpyrexia, consequently due caution should be exercised in their employment. Iron as we know causes congestion or increase of blood in the part affected, and both it and quinin have a tendency to check sweating. May they not thus act as aids in the production of hyperpyrexia? I never give iron in acute rheumatism for this reason. In the chronic form of it, however, it is most useful.

Aconit racemosum is useful in some cases after the acute stage is over.

Alumina and vitriol have been suggested, but require great care in their administration on account of their dangerous nature.

Free blistering was recommended by Dr. Samuel Davie. He surrounds the parts with strips
of caustic, plaster, and he claims by this means the pain will be relieved in 24 hours. He believes that the removal of the blisters from the parts blistered, a large quantity of the material, morbi, he also maintains that it prevents earache complications. I have seen, however, in a young patient strangury result from such treatment. Occasional blistering, however, with the acetate of potash treatment has been that generally adopted till Salicin was introduced.

A water bed is advisable for the patient; his limbs should be so placed that all his tendons are relaxed; mourners are also extremely useful in preventing the weight of the bedclothes from pressing on the tender and sensitive joints. We generally recommend the patient to lie between blankets and have the joints enveloped in cotton wool. "Bedding in blanket" says Dr. T. W. Chambers reduces by 16 to 24 or by a good draughtsman, the
Lucas run by patients in acute rheumatism. German physicians, and among them Senator of Berlin, advise the applications of ice-bags to the joints instead of cotton wool. By modern injection of carbolic acid, recommended first by Künze and used by Stüler of Griesvald in 1894, it is useful in allaying the pain. If the patient has been in the habit of taking stimulants, I am of opinion that he should not be entirely deprived of their use, especially after the acute stage is past. I have notes of one case, a drunkard with a fatty heart who died at this time from the consequences of withdrawal of his stimulant.

Quinine may be given to keep down high temperature. Dr. Barclay remarks "the use of quinine is in no way injurious, that it has no influence over the urinary secretion, beyond that of occasionally rendering it less alkaline in reaction, and that an alkali is not neutralised by any excess of either uric acid or phosphoric acid, but by some other acid."

Salicin. Impressed with the benefits derived from
Suspecting in malaria diseases and with a belief in the malarial origin of rheumatism, Dr. J. MacHagan in 1872 began to employ Salicin in the treatment of the disease. Believing, says he, in the miasmatic origin of rheumatism, it seemed to him that a remedy for that disease was most hopefully to be looked for among those plants and trees whose favourite habitat presented conditions analogous to those under which the rheumatic miasm seemed most to prevail. He looked among the willows and from them obtained salicin.

In Germany at the same time Prof. Thiessen was experimenting with it. He gave it in cases of enteritis, pneumonia, dyspepsia, etc., and found that although it reduced the temperature it exercised no beneficial effect and gave no relief to the symptoms. But in acute rheumatism it both reduced the temperature and exercised a decided action in relieving joint pain.

Independently of these observers, MacHagan in 1874
began his experiments with Salicin, and in 1876 published an account of them.

The following are the conclusions he arrived at:

1. We have in Salicin a valuable remedy in the treatment of rheumatism.

2. The more acute the symptoms the more marked the benefit.

3. In acute cases its beneficial action is generally apparent within twenty-four hours; always within forty-eight hours of its administration in sufficient doses.

4. Given thus at the commencement of the attack, it seems sometimes to arrest the course of the malady as effectually as quinine cures ague, or ipecacuana in 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 more of nervous temperament, the pain is proportionately greater than the abnormal rise of temperature.

5. In chronic rheumatism, salicyic occasionally does good where other remedies fail; but it also sometimes fails where others do good.

Such are the conclusions drawn by Macleay from the effects of salicylic in rheumatism. At the same time, as I have said, experimenters in Germany found much the same results from salicylic acid, prepared by them from carbonic acid. When these results were given to the profession of course speculation was ripe, and physicians everywhere gave the new remedy a trial. Nearly everyone was enthusiastic in praise of the drug. A few were disappointed; they said, but I believe the minority consisted of those who had not administered it in sufficient quantity, or had used an impure article. For we must drive as much salicylic into the system as rapidly
as can be done with safety if we wish to cut short the disease. Impurities also, we know now may give rise to unpleasant head symptoms and prevent the further exhibition of the drug, thus throwing discredit on its undoubted virtue as an anti-rheumatic. We may also have exceptional cases in which it seems not to secure its usual good effect; but quinine may do the same thing in Ague, or pleurisy in dysentery. Again, we cannot say that salicin will of a certainty prevent all cardiac complications in rheumatism, any more than we can say that quinine prevents ague-cake, or pleurisy abscess of the liver. These untoward circumstances may occur in spite of all we may do, but the remedies are not the less useful in tending to prevent these complications.

To obtain the full benefit of salicin, it may be given in full and frequently repeated doses, the bowels being first relieved by a
Saline purge, and the patient put on milk diet. The treatment ought to be begun as soon as we are aware of the nature of the case, as in this way the cardiac complications are littlest to be averted.

Twenty grains or even more ought to be given every hour till we see its effect on the patient. It is generally seen that by the time half an ounce has been swallowed, there is marked improvement in the patient's condition. We will tell you that he can move his joints with greater freedom. But we must still go on with the remedy till all the symptoms disappear, and the temperature has reached the normal standard or nearly so. When this has been achieved, which is generally within twenty four to forty-eight hours in bad cases we may diminish the dose, but not entirely omit its use, as salicyin being rapidly eliminated from the system a relapse may occur.

Raclojen states that it takes an ounce of salicyin
or salicylic acid to remove the acute symptoms. This quantity should be given within the first sixteen to twenty-four hours in doses of twenty to forty grains. At first every hour, then every two hours till the acute symptoms begin to decline. A second dose should be consumed in the next forty-eight hours. After that twenty to thirty grains should be taken every four hours for two or three days, and for a week or ten days, that quantity should be taken two or three times a day. The patient should always be in bed for a week at least after the joint pain is abated, as the fibrous tissue of the joint itself requires a time to recover the effect of the inflammation. The fibrous tissue of the heart requires the greatest amount of rest. In my own cases I have never had occasion to push the Salcinein so far as Dr. Maculayan has recommended above, as I have generally found less rufflee.
In fact if you give alkaloids of code to the above extent, a train of very alarming symptoms are sure to appear. Among these headache, tinnitus, auricula, vertigo, nausea & vomiting, and even delirium with an appearance of typhoid depression have been noted.

Flint in his Practice of Medicine says that calciphyllic acid in some instances has produced alarming toxic effects and even death. No such effects have been observed to follow calcine, and its controlling influence over acute rheumatism is probably not less than that of the calciphyllic acid.

In an interesting discussion on the use of the calciphyllic in acute rheumatism held at the Medical Society of London in Dec., 1881, Dr. Melton Fagge brought forward conclusive proof as to the undoubted efficacy of the calcine treatment. He found that in Dr. Parrot's method of treatment by the carbonate of potash, only 18 out of 51 subsided within 6 days, there being 27 from the 5th to the 9th day. In comparison to this
table, he showed one of 355 cases of acute rheumatism treated in Guy's Hospital from 1876 to 1880 inclusive, being the whole experience of that institution, and found that in no fewer than 180 of these cases, exactly 50 per cent, the disease subsided within 5 days after commencement of the salicyl treatment. One fact of subsidence being determined by the complete disappearance of joint pains, and by the temperature falling to and remaining at the normal point. In many instances the disease was checked or greatly modified after one or two doses of the drug had been given.

Relapse, he looked upon as continuation of the original illness, interrupted or postponed by the drug. In regard to relapses in acute rheumatism, I believe that a prolonged course of salicylate will entirely prevent them, but as is often the case, in private practice at least, the patient after getting rid of the pain
imagines himself quite well, sits up in bed, or perhaps gets out of bed, and having stopped taking his medicine gets a chill, or in other words he has cold applied to his spine, which leads to a recurrence of the original malady. But I have seen after these relapses, by merely putting him in bed, and reemploying the drug the same relief was experienced that was primarily afforded.

Dr. Fajze had seldom recourse to stimulants but in a few cases when the pulse became weak, irregular, and intermittent, he employed them with advantage. He is inclined to believe the toxic symptoms of the abscess are exaggerated, and he found they always subsided on the discontinuance of it. What would happen if the patient were made to take it he did not know. He was not prepared to deny her salicylic acid and even salicylic acid in the patient weak and their stay in hospital was not much shorter than before the
any was employed. But then they were far longer than formerly on low diet, and confined to bed after the treatment of the disease for fear of a relapse. These precautionary measures must necessarily tend to protract the period of convalescence.

In regard to complications Dr. Fagge states that he could hardly find one in his numerous cases in which pericarditis set in at a time when the action of the remedy was fully established. But after these cardiac complications have developed themselves before treatment, he did not shrink from salicylic acid and had any power in controlling or arresting their course.

In regard to hyperpyrexia he states the evidence as far as it goes is in favour of the drug. He recognizes, however, in which probably no amount of the remedy would have any effect, and in which the temperature remains high and rises to a fatal point in spite of the administration of the remedy drug.
Such then is the substance of Dr. Milton Fagge's observations and convictions in regard to the incomplete treatment of rheumatism, and they are of value as being the impartial views of one who, besides being a sound physician, admits that at the beginning of his investigation he was biased against the drug, and who even when he saw the first cases improve under the remedy warned his clinical students not to attach too much importance to this fact, because he had seen the administration of other medicines followed by results that appeared equally striking, but when to quote his own words, "I saw case after case recovering with scarcely a failure, I became satisfied that I had a most potent remedy in my hands. All further experience has strengthened this conviction in my mind, and I would now feel that I was accepting a very grave responsibility if I were to withhold a drug which I believe to be so useful, from any patient placed under my
care, unless there were very good reasons for doing so."

Every one I know who has given the drug a fair trial, must come to pretty nearly the same conclusions as Dr Fagge. The immense relief given by its use in the abatement of pain and fever, a relief not to be estimated by statistics alone, renders it by far the most valuable remedy for the disease at present known.

It has now had a fair and extensive trial, and to say that it far excels any other method of treatment would be to give the drug but scanty praise.

It may rather be said that until the application of Salicylic to the treatment of acute rheumatism, there was no remedy which could be relied upon to shorten to any extent its tedious course.

Now, however, making due allowance for exceptional cases, not only can the cessation of the primary phenomena of the disease - pain and fever, be rapidly secured, but we have likewise proof to show that owing to the remarkable power which
The drug possesses of curtailing the duration of the disease, those secondary affections of the heart which make rheumatism so serious, are greatly diminished in number and intensity.

Mode of action of Salicin,

The question of how Salicin increases its beneficial action is one of the debatable points of the day.

Dr. Maclean believes that it acts by destroying the organisms in the blood, which he maintains are the cause of rheumatism, but his view of the pathology never having recommended itself to our minds we cannot admit the correctness of this idea of its mode of action. If it can destroy organisms in the blood in acute rheumatism (if the existence of which organism we have no proof) why is it so utterly powerless to do so in the grip of true fevers? In these latter the presence of organisms is beyond a doubt, but in them as I have already stated it exercises no power whatever in the abatement of their symptoms. The manner in which rheumatism behaves under Salicin does not appear to me to be consistent with this.
idea of its origin. We have likewise seen that it has an
effect in neutralizing the acidolescented from the
patient, so it cannot act in this way. By
a process of exclusion therefore we are compelled
to admit that it must be on the nervous system,
and no other, that the remedy exercises its benefit.
Our idea of its pathology gives as reasonable grounds
for accepting this view. That it possesses undoubted
property in the alleviation of pain, not only in this
but in many nervous affections is unquestioned.
I believe that it acts as a direct anesthetic on
the centers of articulations and muscles, allaying
their disturbed action, the pain, pterygia, and edema
being lessened as a consequence. This sedative
action on the centers will relieve pain and produce
sleep, which directly tends to lower febrile action.
Such then are my views as to the nature and treatment of acute
rheumatistics. They may not be entirely correct, but they at least
give a reasonable explanation of the phenomena of the disease, and
the success of its treatment, and so such are a step from the blank
ignorance which has hitherto surrounded it.