Manifestations of Rheumatism

in Childhood.

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### Introduction.

The Study of rheumatism in childhood is unquestionably a very important one, because of its so frequently attacking vital organs, especially the heart, and thus crippling and shortening the lives of those affected.

The study of this disease in the young, is not only of importance to the individuals concerned, but also to the State, because if early recognition is made, and appropriate and thorough treatment carried out, many lives will be saved, and many made more fit, or less handicapped, for the battle of life.

The signs of rheumatism as seen in the adult are often absent in the child, or present to a much less degree. In the adult the joints are swollen and painful, there is great sweating, the constitutional symptoms are severe, there is considerable pyrexia, in some cases going the length of hyper-pyrekia: in the child on the other hand, the joints are often hardly swollen at all, if pain is present it is usually not severe, there is no sweating, the constitutional symptoms are slight and the pyrexia very moderate.

As a consequence of the absence, or the presence in only slight degree, of these classical signs of rheumatism, the disease was for a long time supposed to be rare at that stage of life; but for many years now, it has been recognized that the disease has other ways of manifesting itself, and these other ways being very frequent in childhood, it is thus known to be in reality a common disease of early life.

These early manifestations are very varied: Affections of (1) the heart; in the endocardium, pericardium and the heart-muscle itself. Involvement of the heart is very common in the child rheumaticpatient, and it is because of this, that the disease at that stage is so grave and serious.

(2) The pleura

(3) The throat in the form of tonsillitis

(4) The nervous system; principally as chorea, which is usually recognized now as being rheumatic in nature.
(5) The skin; in the form of eruptions of the nature of erythema of different kinds, and purpura.

(6) Subcutaneous fibrous nodules, found in various parts of the body, principally in the neighbourhood of joints.

Whilst the first three are not very infrequently accompaniments of rheumatism in both periods of life, they are more so in the child. The last three are almost altogether confined to childhood.

Then again, whilst the first three are set up by other causes than rheumatism, the last on the list (subcutaneous fibrous nodules) is almosy always caused by rheumatism alone.

Just because the manifestations of rheumatism in child life are so numerous and varied, and the treatment so often difficult, they are well worthy of careful study, hence the reason of my taking up this as the subject of my thesis.

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The etiology of the disease has long been a difficulty. Various theories have been put forward, to be supported by some and condemned by others. Within recent years a microbic causation has been advanced, but although very plausible in many ways, it has not as yet been accepted generally.

The scope of this thesis is to give some account of recent work on the subject, with special reference to the clinical manifestations. I will illustrate from some cases observed by myself. The name "Rheumatism" (  $\delta \epsilon \tilde{\nu} \mu \alpha$  = a fluxion) shews from its derivation that there is the idea of the old humoral pathology in it. As Fagge's Medicine says "The words rheumatism and catarrh are used by the Greek writers from Hippocrates downwards, as having similar meaning. The notion was that of an acrid humour generated in the brain and distributed over the body. In the course of time, diseases of the mucous membranes became known as Catarrhs, while the name of Rheumatism was applied to painful affections of the joints".

Baillou or Ballonius (who died in 1616) is said by Bright and Addison to have given the name that latter meaning. This French physician also differentiated rheumatism from the disease with which it had been so often confused, viz, gout. The first however to accurately describe the definite disease now called rheumatism or rheumatic fever, was the great Sydenham, in 1670. This he did in his Observationem Medicarum, and at the same time he also distinguished it from gout.

Heart disease in patients suffering from rheumatism may have been noted before 1827, but we have no record of any such observations till we find them in Scudamore's writings in that year.

Ten years later Bouilland proclaimed that

in his view cardiac disease in patients suffering from rheumatism was neither more nor less than a true rheumatic manifestation.

Since then it has become more and more the belief that rheumatism is by no means a mere joint disease, but that it is an affection of the system, with manifestations in many and varied parts of the body.

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## Etiology.

As to the etiology of rheumatism, there have been, and there are even now, various theories, but the essential nature of the disease is still uncertain.

One theory (the Lactic Acid theory) was propounded by Prout, and it has been supported by authorities like Todd and Richardson. This acid was supposed to accumulate in the body, and the symptoms of the disease were just the expression of its poisonout effects upon the system. Pichardson in 1853 published the results of a series of experiments upon dogs to shew that the injection of this acid into the peritoneal cavity was capable of setting up endocarditis: but a few years later Reyher pointed out that precisely similar appearances are constantly seen in the cardiác valves of healthy cats and dogs.

Another theory (quoted in Quain's Dictionary of Medicine) and associated with the name of Constatt and Seitz is called the Nervous theory. "Chill of the peripheral parts of the body, especially of the skin and joints, causes disturbance of the corresponding parts of the central nervous system, and this gives rise to pain and vaso-motor or trophic changes of the same peripheral parts; and to fever".

The Malarial theory is associated with the name of Maclagan. He thought that the disease was due to the presence in the system of a poison which is of the nature of a miasm entering from without: he thought this miasm allied to, but distinct from, the miasm of malarial fever.

The theory that the disease is due to an infection caused by the entrance into the system of a micro-organism, is now principally associated with the names of Poynton and Paine, but others before them have advanced the same hypothesis. Quain's Dictionary of Medicine quotes Hueter as having held this idea "Chills abe attended with the entrance of micro-cocci into the system, and endo-carditis is the result. The joint symptoms are secondary and embolic as in pyaemic arthritis".

This infection theory is one which meets with acceptance in many quarters at the present time, but as there is considerable diversity in the bacteriological results obtained, there is uncertainty as to the microbe or microbes involved.

Bulloch, in Allbutt and Polleston's System of Medicine, summarises the different views thus:-

- 1. Rheumatic Fever is the result of an infection with a specific anaerobic bacillus. (Achalme)
- Pheumatic fever owes its origin to staphylococcus and streptococcous, and is merely an attenuated form of pyaemia (G. Singer)
- 3. Rheumatic fever is not due to any particular microbe, but is a particular reaction in predisposed persons to various microbes, especially

streptococci (Menzer)

- Rheumatic fever is the result of an infection with a specific diplococcus (Poynton and Paine, Ainley Walker, Beattie)
- 5. Rheumatic fever is due to a virus still unknown (Pibram, Lenhartz and others).

Povnton and Paine's investigations are probably the most striking. They state they have separated a coccus and they attest that the intravenous inoculation of large doses of this coccus produced in the experiments very definite and constant pathological results, including polyerthritis, bursitis, teno-synovitis, multiple valvulitis, pericarditis, plastic pleurisy and pneumonia, nodules, chorea and even iritia: in the midst of these lesions cocci were found by microscope and by culture. On the other hand others (including Bulloch with Theodore Thompson) have examined cases of characteristic rheumatic fever with negative results. Bulloch concludes thus "In spite of the numerous investigations which have been carried out, it seems to me that the etiology of rheumatic fever still belongs to the arcana of pathology, and altho' what clinicians call rheumatic fever is probably a specific infective disease, the virus is not known".

Reference may be made here to some of the predisposing causes or conditions.

Heredity There is strong evidence that tendency to

the disease is transmitted from parents to children. Dr. Cheadle, from his private case-books, made out these figures:- In 30 consecutive cases, 28 gave a family history; and adding to these the cases with chorea and erythema 31 out of 33 were found hereditary. Combining arthritis, chorea and morbus cordis 103 out of 180 gave a family history.

Poynton and Paine point out that an infant may be born actually suffering from rheumatism, i.e. the mother, having had an attack during the later months of pregnancy, transmitted it to the child in utero, and it shewed the disease at its birth. <u>Sex</u> Up to 20 years of age the incidence is more upon females than males; after that age the reverse holds true. One remarkable fact is the remarkable preponderance of cases in females between the ages of 11 and 15. there being double the number in them, during that period.

<u>Climate</u> It is said that "temperate climates in which there are considerable alterations in humidity favour the infection.\*\*\*\*\* Lack of sun, cold winds and damp must greatly favour inflammation of the throat". (Poynton and Paine) As it is usually surmised now-adays that the rheumatic virus probably enters in most cases by the throat, the infection is thus made easy. <u>House conditions</u> Houses built on damp soils probably predispose the inhabitants to a disease like rheumatism, and more so if there is lack of light and fresh air in the inhabited apartments.

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### Clinical manifestations.

As referred to in the introductory part of this thesis, the clinical manifestations of rheumatism in the child differ considerably from those in the adult. Authorities like Cheadle, and Poynton and Paine, are of the strong opinion that we should not take our essential idea of rheumatism from what we see in the adult, but from what we see in the child.

Cheadle says in his "Lectures on the Rheumatism of Childhood":- "The most complete and comprehensive manifestations of the various phases of rheumatism belongs indeed to the period of childhood. It appears then under the simplest conditions: this presentation of the disease should be regarded as representative, and the changes which take place in the phenomena with advancing age regarded as modifications of the earlier and more perfect form".

Foynton and Paine in "Researches on Rheumatism" state that "In the future all descriptions of acute rheumatism will be probably based upon a survey of the disease in the young, and such peculiarities as occur in the adult life will be looked upon as departures from the classical disease which is seen in its truest character in childhood".

Before discussing the clinical manifestations as seen in the various organs, a general picture may be set forth describing how a child is often attacked with the disease: but at the same time it must be remembered that the symptoms and signs are often very indefinite and irregular. The first complaint is often a sore throat of the nature of a tonsillitis, but with the contiguous parts more or less inflamed also. There will probably be a history of exposure to cold or damp or fatigue, or these more or less combined. There is a general malaise with some feverishness but only of moderate degree, and a general soreness over the body. Some of the larger joints may now become involved shewing some swelling and redness with pain, but often there is simply pain and pain alone. Examination of the precordial area frequently gives signs of a dilatation of the heart: definite inflammation of the internal and external linings does not appear till later. Skin manifestations and subcutaneous nodules may now present themselves, and it is evident that the child is rapidly losing colour, soon a definite anaemia being established.

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Let me now take up these clinical manifestations in the different organs in detail.

<u>Cardiac manifestations</u> These are the most serious, because of the vital organ affected. The anxiety is not so much an immediate one (although <u>that</u> may be so at times), but an ultimate one, the patient's whole after-life being more or less seriously affected. These cardiac manifestations bear no relation to the severity of the attack, i.e. a slight case of rheumatism

may be associated with Cardiac disease as readily as a severe one: and on the other hand a severe case may have no heart involvement at all. Comparing the child with the adult, the heart of the former is more frequently affected in rheumatism than that of the latter. C. O. Hawthorn makes the suggestion that it is probably because the child is not so soon confined to bed, the arthritis in it being absent or insignificant.

(1) Early acute cardiac dilatation. Dr. Lees and Dr. Poynton have drawn special attention to this condition. They say:- "Careful examination will shew the area of cardiac dulness to be considerably increased, extending to the nipple line and outside of it: the impulse is weak and also displaced outwards: the first sound at the apex is short and sometimes murmurish, and the second sound in the pulmonary area is accentuated" Along with these signs you have a quick and feeble pulse. This condition they believe to be the earliest appreciable sign in rheumatic heart disease, and points to some toxic influence on the cardiac muscle, weakening it and causing it to relax before the blood pressure.

It may be mentioned here that later in the disease, grave signs of cardiac dilatation in a child patient, almost always means fresh rheumatic manifestations: and if the cardiac failure is extremely rapid, death may be the outcome.

(2) Pericarditis. Whilst pericarditis may occur at any period of the illness, it is usually a late manifestation, and almost always later than endocarditis.

In the adult the signs of it are very severe, but in the child they are much less so. The onset is often insidious, not accompanied by much distress or much fever. There may be slight pain over the precordial region, or the child may be simply restless. The pulse rate quickens to 120 or 130. Auscultation may reveal slight friction. An interesting feature in the child, is the probable development of subcutaneous nodules along with the pericarditis: these two frequently go together. Sturges points out, that this affection of the heart in the child may be recognised or suspected before friction is heard, by the altered rhythm and the quickened and tumultuous cardiac action.

Pericarditis is the most frequent cause of death, but not usually in first attacks. There is not as a rule much fluid thrown out, but there is usually a fibrinous exudate with the result that adhesion is apt to take place: if so, the heart is compressed, and its movements and development interferred with. As regards this restriction of development, post-mortem examination has shewn before now, a heart like that of a child of 10 years in an adult of 20.

Besides this condition of adhesion, the pericardium itself is usually greatly thickened.

Cheadle points out that the chronic inflammatory process sometimes spreads from the external sac to the anterior mediastinum, so that these are matted together in a thick fibrous mass (indurative mediastino - pericarditis). It has been observed that the inflammatory change in the inflamed pericardium is sometimes of the same nature as is seen in subcutaneous nodules. Dr. Barlow found in one case that the pericardial adhesions were distinctly nodular. <u>Case</u> J.A.S. a boy aged 15 years (hospital case) 17/12/13, Complaint - slight pains about muscles and joints, breathless, feverish, pain over heart and palpitation.

Previous history: has had four attacks of rheumatic fever before, in one of which heart was known to have become affected.

Examination: loud V.S. murmur heard over the pericardium and round to the back: area of cardiac dulness increased to the right and left.

After-history of illness: on the following day signs of pericarditis: area of dulness still further increased laterally and upwards; friction heard: two days afterwards friction could not be made out: in other two days it appeared again and four days afterwards boy became very ill, left lung becoming involved: gradually after this, the pulmonary and pericardial signs cleared up, leaving the boy with his V.S. murmur as before.

(5) <u>Endocarditis</u> "Inflammation of the endocardium is twice as common in childhood as in adults; fully three-quarters of all cases under fifteen years of age develop endocarditis" (Symes) When associated with arthritis or chorea it is not usually overlooked because it is watched for, but when accompanying what seems to be a simple torticollis or simple tonsillitis it may not be examined for, and therefore missed, to be found later when treatment is not of the same avail.

Symes puts the matter very strongly when he says "It should be a matter of routine to examine the heart in all children with febrile attacks, and in a family with known rheumatic tendencies, the condition of the heart should be periodically ascertained whether there have, or have not been recent illness". Endocarditis usually occurs early in the series of rheumatic events, and the mitral valve is the one most frequently attacked. When once the soft blowing murmur at the apex is made out, the diagnosis of the presence of endocarditis is quite easy, but other signs should make us suspect its development, previous to the hearing of the murmur, namely irregularity of the heart's action, alteration of the quality of the first sound, accentuation of the second, and any feverishness or quickening of the pulse, coming on without obvious cause. (Of course it must be remembered that

the murmur may not be due to endocarditis, but to simple dilatation).

The lesion of the value is usually one leading to incompetence and regurgitation. There is first a slight roughening of the first sound, which gradually becomes more impure until a definite systolic murmur is formed. Afterwards accentuation of the pulmonary second sound may be noted.

Instead of incompetence, stenosis may take place. The interesting fact is, that this stenosis is met with mostly in girls, and is probably due to the greater prevalence of the disease in that sex between 11 and 15 years.

Symes says that stenosis is the result of slow, subacute or recurrent endocarditis of long durat-

Before the murmur is properly formed its development may be suspected by a reduplication of the second sound at the apex. This reduplication of the second sound in that region, has been rather difficult of explanation. Dr. Sensom's theory is "The first of the two sounds is the normal one caused by the simultaneous closure of the pulmonary and aortic orifices, and the second by the sudden tension of the mitral flaps as the ventricle relaxes. The second is thus mitral in origin: it is not audible at the base". The aortic valve is much more rarely affected, but when it is, it is always serious. Whilst dilatation, as a result of endocarditis, takes place rapidly in children, the usual sequel@as we see them in adults (dropsy, cyanosis and dysphoea) are not common at the earlier age: the reason being that hypertrophy quickly follows and thus compensation is established.

Cases. M.A.G, girl of 18 (private case) First seen on 15/1/14 complaining of chill, inflamed throat, and soreness throughout body. Thescall passed off, but in a month's time, she began to suffer from pain in several of the joints: there was only the pain at first, but in a few days swelling appeared. The first cardiac sound at apex was now found to be "murmurish", and this soon developed into a definite blowing murmur, which still persists. The arthritis gradually subsided.

M. McL. B, girl of 17 (private case) First attack of rheumatism (Arthritis) in May 1913, second attack (slight) some months after. At examination on 1/5/14 there was found V.S. murmur at apex, with suspicion of A.S: also reduplication of second sound there: otherwise well.

R. McK, boy of 13 (hospital case) Began to complain in October 1913, first of all with sore throat and pains in joints without swelling: presystolic murmur present. Subcutaneous nodules developed, their situation being two knuckle joints of each hand, and one elbow. There was the history of an illness with pains in joints, two years previous.

J. C. male (private case) First attack of illness at 15 years, diagnosed Influenza. Definite rheumatic fever at 16, when cardiac disease (not recent) found. Another attack of rheumatic fever at 24. Mitral systolic murmur still persists, History of "growing pains" when a boy. Also family history of rheumatism. C.C. female (private case) - sister of above. Scarlet fever at 9 years, with no joint manifestations as far as can be remembered by patient's mother: attacked with dyspnoea about 2 years afterwards. A presystolic murmur found when I attended her at 17 with acute bronchitis. History of "growing pains" also.

Ulcerative endocarditis is very rare in the rheumatism of children (Cheadle)

Myocarditis. Besides the affection of the external and internal lining of the heart, its muscle is also very much affected, by the rheumatic poison. Proportion--ate attention has not in the past been given to this part of the cardiac inflammation. Within recent years however several observers have been giving special study to it, amongst whom may be mentioned Carey Coombs.

On account of this myocarditis, the heart is both dilated and hypertrophied, and the careful examination of the signs and symptoms relative to this, from day to day, is as important as the mere recognition of the valvular defects. Persistently increased pulse-rate, in the absence of any other cause, is an evidence of

of weakness of the heart muscle: if this increased pulse-rate fails to respond to treatment, the prognosis becomes a grave one.

The myocarditis is often secondary to pericarditis or endocarditis, but sometimes seems independent. Post mortem examination shews fatty changes in the muscle fibre.

Joint Manifestations As already said, these in the child are not usually marked at all. It is unusual to see the crippled painful condition which one associates with rheumatism in the adult. However, one or more of the larger joints, may be painful, red and swollen, but very often there is just the pain and nothing more. Occasionally one large joint may be alone involved, and when it happens to be either of the hips. it may be mistaken for tuberculosis disease there, or if it is the right hip and the pain referred to the groin, for appendicitis. Sometimes obstinate cervical arthritis may occur and thus simulate spinal caries. One interesting feature in child rheumatism is the presence of pain, not so much in the joint, as in the tendons in the neighbourhood of the joint. This applies especially to the knee joint, where pain is often severe in the ham-string tendons there. On account of this, the child cannot straighten the leg at the knee properly, so in walking it does so with bent knees, which again involves walking on its toes.

This gives a very characteristic gait. Besides the tondons being thus involved the fibrous tissue composing the different fasciae may be affected also. When this occurs in that of the side of the neck, it simulates an ordinary forticollis, and when in that of the abdominal wall it is the cause of some of these very indefinite pains which some children complain of, in that region. Or again, when the pains are in the side due to involvement of the fibrous tissue about the intercostal muscles, it may be mistaken for pleurisy.

<u>Throat Manifestations.</u> Ionsillitis is the usual form which the so-called rheumatic-throat takes. Of course there are many causes of ionsillitis which are not rheumatic in nature, but it is so frequently associated with rheumatism in children, that there can hardly be any doubt that the rheumatic virus does thus manifest itself.

Tonsillitis frequently ushers in the rheumatic illness, but sometimes it is during the course of the illness that it develops. Careful questioning will often reveal that the patient suffering from undoubted rheumatism, has had sore throat frequently before.

Sore throat was calculated by one authority to occur in 24% of the cases. Froussean, the great French physician, recognised a rheumatic sore throat, and shewed how this was apt to alternate at one time

with Torticollis, and at another with joint pains or lumbago. He thought that the rheumatic sore throat had distinctive characters, viz: that it was ephemeral in duration, lasting only from 30 to 48 hours, and that it was more than a tonsillitis, that it involved the pharynx, soft palate and uvula. Dr. Cheadle says that it is probable that tonsillitis may occur as a solitary expression of the rheumatic state.

A case of my own associated with chorea may be noted here.

C H. a girl now aged 14: had an acute tonsillitis in February 1911: two months afterwards she developed chorea and was six weeks off school. Five months after she complained of palpitation on exertion, and examination revealed a mitral systolic murmur. Shortly afterwards the chorea returned, to disappear in the course of a few weeks. The cardiac murmur persisted for some months and then disappeared and has not returned. In February of this year (1914) she again had an attack of tonsillitis, but slight in degree: this was followed by an erythematous and purpuric eruption on feet and legs. She has never had any articular manifestations.

Where the tonsils are left enlarged after an attack of inflammation, or where they are found enlarged in the course of one's examination of a child suffering from other manifestations of rheumatism, the wise procedure would be to have them removed (enucleated

if possible) when the child's condition allows of it, so that they may not afterwards supply a surface for the reception of the virus.

<u>Growing pains</u> These pains which affect some children more or less severely, are no doubt in many cases, of a rheumatic nature. Dr. Cheadle points out that a family history of rheumatism was got by him in 49% of the cases enquired into.

As to whether they, and they alone, are associated with or followed by cardiac manifestations, I cannot say. I know of one man, now in the prime of life, who, when being examined for his first life insurance policy at 26 years of age, was surprised when he was told that he had a mitral systolic murmur. He had absolutely no personal history of rheumatic arthritis: he had had a tonsillitis when a child, but he had also the history of severe growing pains when a boy. At 36 years he was again examined and the murmur had disappeared: repeated examination since shews it still to be absent. His family history is suggestive, his mother having suffered from subacute rheumatism coming and going for years, and his father from lumbago.

In some of the cases already referred to the history of growing pains has been noted. <u>Skin Manifestations</u>. These may occur before or during an attack of arthritic rheumatism, or between separate attacks. They may take the form of erythemate of

various kinds, or of purpura, and all gradations between these. Shewing how the one merges into the other, Poynton and Paine point out that "heat applied to a rheumatic erythema, or the stress of gravity upon the dilated capillaries may convert such an erythema into a purpura". Frythema marginatum is the form which is usually allied to rheumatism.

Erythema nodosum is thought by some to be always rheumatic in nature, but by others that it undoubtedly occurs quite apart from that disease. Cheadle suggests that the different erythemata are due to the presence of irritants in the blood: the rheumatic virus is one of these irritants, and as to whether it is the cause in any given case, would be determined by careful examination of the evidence.

Regarding purpura theumatica, the same authority says "While sometimes concurrent with active articular rheumatism, it occurs also apart from it in rheumatic subjects, and is then probably a minor expression of the rheumatic state although far less common than exudative erythema." I have referred to a case of erythema and purpura, C.H. (at end of throat manifestations).

<u>Subcutaneous Modules</u> To the frequency of these nodules and their great importance as clinical signs in the rheumatism of children, Dr. Barlow and Dr. Warner were the first to draw attention. They are essentially

a manifestation of the rheumatic state in the young. Still found them in 27.5% of 200 severe cases of rheumatism in children under 12 years of age, and in the milder cases 10%. They are subcutaneous swellings connected with the fasciae or tendons, varying in size from a pin's head to an almond: they are not painful or tender, and the skin is not red over them. Their growth is rapid and their duration varies from a few days up to some months. They often come out in crops. The sites where they are usually seen or felt (for sometimes they cannot be seen, or sometimes only seen when skin drawn tight over them), are the back of the elbow, over the malleoli, knuckles, edge of patella, curved line of occiput, spines of vertebrae, borders of scapula and pelvis: sometimes on toes and fingers.

The interesting feature about them is that they are associated with cardiac disease: and according to their size and number they make the prognosis more or less grave. "So far as my experience goes, I regard the eruption of large nodules as almost equivalent to sentence of death. They mean persistent cardiac disease, generally uncontrollable and marching almost invariably to a fatal end". (Cheadle) (see notes of R. McK. at end of section on Endocarditis). <u>Pleurisy</u> In relationship with rheumatism, it may occur in one of two ways. It may take place in a secondary or passive way when the compensation of a rheumatic heart is breaking down, and the pleura along with other organs becomes involved: it may also spread from a pericorditis. On the other hand it may take place in a primary or active way, preceding, accompanying or immediately following, arthritic manifestations: in such a case the cause would appear to be more presumptive of rheumatism. There is always the possibility of it occurring as an independent rheumatic manifestation, but this is difficult to prove. <u>Anaemia</u> This condition is a marked feature in the rheumatism of children, even more so than in that of adults. The rheumatic poison seems to destroy the red corpuscles or interferes with the generation of new ones.

Simon in his "Clinical Diagnosis" says "The colour index is usually about 1 , the loss of haemoglobin being equal to the diminution of the red corpuscles" He also states: "The height of the leucocytosis is roughly the index of the severity of the disease: high counts are found when associated with complications such as endo- or pericarditis etc. The fibrin is much increased during the active stage of the disease, accounting for the tendency to the formation of vegetations upon the valves, and of clots in the heart and arteries".

Whilst anaemia is a striking effect in child rheumatism, it is not so much so, as in diphtheria. Chorea It has long been recognised that there is some association between chorea and rheumatism. Some hold that all chorea is rheumatic in nature, others that very few cases are. Dr. Cheadle's opinion is "I do not think the evidence warrants the assumption that chorea is invariably of rheumatic origin, though recent observations tend to connect it more and more closely with the rheumatic state. I must say of chorea as of tonsillitis, erythema, endo- and pericorditis and arthritis (in fact of the whole of the rheumatic series except subcutaneous nodules) that it is produced by other causes as well as rheumatism. But I am convinced that rheumatism is by far the most common and potent factor". In a series of cases which he minutely examined he found satisfactory evidence of acute rheumatism in patients or immediate relatives in 75%. Dr. Barlow's figure is 60% but he does not take account of the relatives. Its association with rheumatism is quite evident when it is accompanied by definite cardiac manifestations, by nodules or by arthritis.

Chorea varies in severity from cases with slight twitching of the face or hands up to those where the movements are so violent that the patient can scarcely be kept in bed.

The condition is often unilateral in character, and Cautley suggests that the poison (without saying that it is the same as that of rheumatism) is a microbe rather than a toxin, because of this

frequent one-sided distribution, i.e. a local cerebral lesion, rather than a general cerebral toxaemia.

Predisposing causes of chorea are fright and cerebral strain.

A case of moderate degree was that of C. H. already referred to at end of "Throat Manifestations".

Another case was that of an older girl I.F. (private case) who had very intractable chorea lasting more or less for years. She afterwards developed subacute rheumatism in several of her joints.

Girl E. S. now 16, (private case) Sore throat when 4 or 5 years -: chorea 8 years: rheumatism a year after: Often complained of "growing pains". Examined 3/5/14, systolic murmur: at apex, but no increase of cardiac dulness.

Epistaxis It may precede, or come on with an attack of rheumatism: occasionally it is said to alternate with the joint pains, or may take place in the quiescent period of a long rheumatic history. It is sometimes present in Chorea.

# Morbid Anatomy.

According to Poynton and paine, it is in the rheumatic nodule that we find the clue to the structure of the rheumatic lesions. These subcutaneous nodules are to be found along tendons, sheaths and over bony prominences. When exposed by dissection their appearance is that of "oval semi-transparent fibrous bodies like boiled sago grains" (Barlow)

In the nodule there is swollen connective tissue at the periphery, and exudation and tissuenecrosis in the centre. There is also found the proof of the tissue reaction in the distended blood capillaries and leucocytic infiltration. Poynton and Paine say that these conditions are the result of a deposit of the infective agent. They state also that Carey Coombs and others have studied the minute submiliary nodules and have found in them certain multi-nucleated cells which they consider to be peculiar to the disease. These cells "have a chromatin substance, and the nuclei in most cases appear as a sharply defined margin with central mass or masses: the rest of the nucleus takes no stain, and this gives to the cell a vacuolated appearance. They are surrounded by plasma cells and leucocytes chiefly of the mono-nuclear variety. The nodules frequently arise in connection with an arteriole, either around it, or even in the wall itself, and their eventual fate appears to be cloudy swelling or cicatrisation".

Taking now the different parts of the body

which are involved in rheuratism:-

#### The Joints

The two structures mostly involved are the serous and the fibrous, i.e. the synovial membranes of the joints and tendons around, and the ligaments, muscles and tendons in the immediate neighbourhood. The synovial membranes are congested, thickened and infiltrated. There is excess of fluid in the joint sac, and it may be serous, or fibrous in nature and may be blood-stained. In the structures around the joint, there is considerable oedema, swelling of the connective-tissue cells, and infiltration of them.

Cultures from the joint exudations are usually sterile.

In childhood the tendons are sometimes more involved than the joints themselves. When the inflammatory condition is more chronic, fibrosis is apt to take place with resulting adhesion.

#### Pericarditis

It commences with an acute congestion accompanied by small harmorrhages. Then follow exudation shewn by swelling of the serous membrane and the effusion of fluid into the sac. This fluid may be serous, serofibrinous or rarely sero-purulent. It may be absorbed or the fibrinous deposit may cause the two-surfaces to adhere and become organized: these adhesions may be in patches, or so generalized as practically to obliterare the sac. In malignant cases of pericarditis there is great thickening of the pericardium. Poynton and Paine state that it is from the pericardial exudation that the diplococcus is most easily obtained.

# Endocarditis.

The changes are most marked on the mitral and aortic valves. According to Symes the first effect on the valves is a slight swelling along the free borders, and the appearance of little excrescences along the lines of contact. As the disease progresses the new tissue becomes fibrous in character, producing thickening, rigidity and shrinking of the valve. Or increasing in size the new tissue may form large papillary excrescences which undergo further fatty and calcareous change.

According to the same authority a section thro' a value affected with rheumatic endocarditis will shew the following changes:-

- (1) A deposition of fibrin on the surface.
- (2) A proliferation of small cells and collection of leucocytaes in the subendothelial layer.
- (3) Development of blood vessels within the tissue which becomes fibrous.
- (4) A thickened endocardium may become atheromatous and finally calcify.

The diplococaus is only found in the deeper parts. Sometimes the atheromatous patches soften and break down, leaving an ulcer. In malignant endocarditis caused by the rheumatic diplococcus, there are found in one or more of the valves fresh vegetations, abrasions or ulcers leading to perforation or rupture of the valve. Examined by microscope, the structure of the lesions is the same with this difference that the diplococcus may be found in abundance in the free edge of the vegetations, thus rendering dissemination easy.

### Myocarditis

In the earlier stages the muscle appears thicker and softer, due to the congestion present. There is an exudation of small cells between the muscular fibres these losing their transverse strike and becoming granular.

In the later stages the muscle is paler and harder, due to fatty degeneration in the muscle cells and increase of interstitial tissue. These changes may be general through the heart muscle, or in patches.

Carey Coombs has pointed out that the changes are principally in the wall of the left ventricle, particularly in the papillary muscles and beneath the pericardium. The interstitial changes shew nodules, histologically like the subcutaneous nodules: they consist principally of large cells many of which are multinucleated. This nodular formation is also seen in the fibrous lining of the arteries in the muscle. Foci of leucoytosis, chiefly polymorphonuclear, are also seen. It is pointed out that in hearts which have

passed through a state of myocarditis, cicatricial lesions remain to mark the sites of previous nodules. Aschoff and Tawara have shewn the presence of many anaemic or infarct indurations resulting from them. These they attribute to (1) recurring embolic obstruction of branches of the Coronary arteries (2) scar constrictions of vessels caused by rheumatic nodules around them.

#### Pleurisy

In rheumatic pleurisy, the histological characters are the same as those of other pleurisies, the pleura shewing epithelium desquamation, the connective tissue swollen, infiltration by small cells, invasion by leucocytes and deposition of fibrin.

#### Tonsillitis

The condition developed may be an acute general inflammation, or there may be little foci of inflammation with exudation from the follicles, or there may be fibrino-plastic exudation.

Repeated attacks cause enlargement, and in their deeper parts there may be foci of disease, with areas of necrosis. <u>Preventive</u> Owing to the fact that rheumatism in its various manifestations is very apt to involve the heart, and thus weaken and incapacitate more or less the individuals affected for the rest of their lives, it is very important that first symptoms and signs should be recognized as soon as possible. It would be very desirable if the attention of parents, guardians, school teachers and those who have charge of children, could be directed to this important aspect of rheumatism. This might be done by printed notices, which would at the same time enumerate some of these symptoms and signs, recognition of which would entail an immediate examination by a doctor.

In the case of children who have previously suffered from rheumatism in one or more of its manifest--ations, or in children who have a hereditary predisposition to it, the following preventive measures should be taken:- House dry, and on dry soil. Clothing - warm, flannel or wool next skin: limbs to be protected also. Exercise in moderation. "Common colds" to be cured within as short a time as possible, and general health not to be allowed to get below par. School hours and lessons not to be too exacting.

<u>Curative</u> Treatment of rheumatism in one or other of its manifestations, accompanied by fever:- Absolute rest in bed, between blankets, in bright airy bedroom. Clothing loose and warm (flannel) and which can be easily undone for changing and medical examination. Light easily digested food, principally milk and light farinaceous foods. Bed-pan and urine-bottle to be used if possible.

Medicines: It is wise to give a smart purge at once e.g. calonel followed by a saline. For the <u>arthritis</u> when present, the drug mostly relied on now-a-days is Salicylateof Soda. It requires to be given in full doses, at regular intervals, till temperature comes down to normal, and continued thereafter in smaller and less frequent doses for a time, so as to prevent relapses if possible.

My own practice has been to give to a child of about 5 years 3-5 grains each 3-4 hours to a child of about 10 years 6-8 grains each 3-4 hours to a child of about 15 " 10 " " " " Joints affected to be wrapped in warmed cotton wool and fixed with a flannel bandage; if pain severe apply a local anodyne in the form of Lin. Sapons or Tr.Opii. Sleep is a necessity; if it cannot be got naturally, Potass. Bromid, or Dover's powder, in suitable dose should be given.

For <u>heart affection</u> : for pain of pericarditis, anodyne fomentations may allay it, if not too severe: if severe or where effusion, a fly-blister of suitable size gives great relief. For endocarditis, the treatment is just the absolute rest and the general measures used.

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If much distress or restlessness, a sedative such as is mentioned above, would be tried. If signs of heart failure present themselves, Strychnine (by mouth or hypodermically) and alcohol must be given.

For <u>tonsillitis</u>: antiseptic losenges such as Formamint or Formitrol are very useful and efficacious. Gargles are not as a rule advisable, because of the strain on the throat in performing the act, but where they are thought useful a combination of Potass Chlor. and Borax might be used. Swabbing out the throat with Glycerine and Carbolic Acid is often very helpful, and gets at the parts involved more surely.

All these can be combined with application of heat (dry or moist) externally. For <u>pleurisy</u>: Moist hot applications, such as Linseed and mustard poultices, give as a rule great relief, if applications properly made and properly applied. Internally Salicylat of Soda and Pulv. Dover. should be administered.

For the <u>anaemia</u> and debility following these various forms of rheumatism, a tonic line of treatment must be pursued, e.g. Cod Liver Oil in some of its combinations, Strychnine and Iron, and Chemical Food. Great benefit should accrue from a residence in some dry country resort.

Regarding the Salicylate of Soda treatment, some diversity of opinion exists amongst well known authorities as to the extent it should be pushed, and

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and as to whether it has, or has not, a depressing effect upon the heart.

Dr. Lees' method is to give it each two hours from 6 a.m. to 10 p.m. with an additional dose at night. He combines with each dose of the Salicylate of Soda, a double quantity of Bicarb. of Soda. For children between 7 and 12 years Sod. Sal. gr X Sod. Bicarb gr XX. Under 7 years sod. Sal gr V. Sod Bicarb gr X. He increases the dose each day by 31 and 2 grains respectively and double that of the Sod Bicarb. He goes on doing so till evening temperature is and remains normal, after which he gradually lessens the dose and frequency. He says frequent administration is necessary because a large part of the Sod'. Salicyl. is quickly converted into Salicylic Acid which is inert, and the remainder is rapidly excreted. Bowels should be opened twice a day to prevent poisoning, and the urine regularly examined to see that it becomes and keeps alkaline.

If heart affection develops, patient to be kept in bed till all signs of active mischief have subsided. When allowed to get up, the heart's response to exertion to be carefully watched.

Dr. Lees does not think that Sod Selicyl. even in big doses has any depressing effect on heart: the depression when present is probably a rheumatic toxaemia acting on the cardiac muscle and therefore all the more requiring the large doses of the anti-

# rheumatic drug.

Dr. Poynton, Dr. Cheadle and others are of the opinion that the drug has a depressing effect when given beyond moderate doses. Dr. Lees recommends in heart affection, especially pericarditis, application of ice bag. Others recommend leeches. Serum treatment. This is still in the experimental stage. Symes says he has tried Menzer's streptococcic antitoxin, a bacteriolytic serum obtained from cultures made from the throats of persons suffering from rheumatic angina. The effect was evidently indefinite. He says he would only recommend the use of such a serum in the case of those who do not yield to the ordinary remedies,

Vaccine treatment. Poynton and Paine state that the results got are still very unconvincing; sometimes these being good, sometimes megative.

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