

" O N R H E U M A T I S M "

A THESIS FOR THE DEGREE OF M. D.

presented by

GEORGE CLARK CAMERON, M.B.,C.M. Ed. 1888.

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For some years back the writer's attention has been directed to the study of Rheumatism, chiefly from the fact that he has unfortunately had the opportunity of studying it in the case of several of his own relatives, and he has thus endeavoured to form some idea of the ultimate causes which produce the Rheumatic Symptoms, with a view to their prevention and in actual attacks, to clearing the "poison" out of the system.

As an aid to the practical study of the subject the following works have been referred to from time to time as cases occurred :-

"Garrod senior on Gout". Garrod junior on "Rheumatism".
Haig on "Uric Acid". Duckworth on "Gout". Latham's
Croonian Lectures 1886. Roberts's Croonian Lectures
on

Uric Acid and his scattered articles. Levison on "Uric Acid" 1894. Berkham on "Gout", B. M. J. 1895. and since making the notes on Ptomaine Poisoning, Bouchard on "Auto-Intoxication" and Mortimer Granville's articles on "Uric Acid" as well as the various articles in current literature and Medical Annuals together with Landois' and Stirling's Physiology, so far as it was connected with the subject, and the ordinary text-books on Medicine.

Since arriving at the conclusions given below on the need of oxidation in the sub-acute form in the shape of Exercise, etc. I have come across an almost similar line of idea in Dr. Percy Wilde's theory of Asthenoxia (Rheumatism and the Rheumatic State, By P. Wilde, M.D., London. Bale and Son) which however my own observations had led me to from the commonness of clinical experience of Anaemia, etc. as forerunners or predisposers to Rheumatism. For the corroboration of the remarks on climate, and its connection with Rheumatism and for suggestions on the microbic aspect I am indebted

to my brother, Dr James Cameron, who has unfortunately had personal experience of acute and sub-acute attacks, both in the various climates of Great Britain and Ireland, and in hot climates.

For the purpose of a Thesis, which I presume is supposed to be rather of a speculative nature than a dry record of facts, it is not necessary that what is here given as new should be true, but rather that the remarks should be of a general and suggestive nature.

I have here noted some of the Questions that pertinently arise in such a study and the Explanations which to me seem to follow the clinical facts and the indications for rational treatment, checked of course by the working of actual experience, to which the teachings of recent physiological and pathological research must be referred to when used as a priori grounds in clinical work. The short time at disposal has led to the following notes being penned in rather disjointed fashion, the various points in the following pages being

discussed categorically without going into a long discussion as to the definition and demarcation of what is ordinarily understood as Rheumatism, in contra-distinction to Gout and to Rheumatic Arthritis. The recent complete works of the younger Garrod practically renders a table of references and of statistics almost a work of supererogation and I shall only shortly refer to the various authors' special works on Rheumatism in the course of the following pages.

I shall therefore at once take up the following points, a short discussion of which forms the subject of my paper:-

The hereditary tendency as a predisposing factor in Rheumatism, and its bearings.

The state of the individual's general health before an attack, and the main symptoms, other than articular, which are forerunners of the Rheumatic development.

The classes of persons attacked.

The state of the Blood and Lymph in the

Acute Rheumatic And ^{Pre-}_^Rheumatic state.

The various views which explain more or less satisfactorily this supposed morbid state, noting chiefly Haig's Uric Acid theory which is by far the most important contribution to the subject of late years.

The view here advocated that the poison is due to the formation of Fatigue Products formed in the muscles and aided by absorption from the alimentary canal in certain cases, of ptomaine poisons.

The indications for rational treatment based on such a view, more particularly in connection with the need for restoration of the blood and removal of the poison by free oxidation and elimination.

Herédity as a predisposing factor in Rheumatism.

It is a matter of great difficulty in the case of such a common disease to determine how far the element of Heredity is to be considered as a predisposing factor in the development of Rheumatism. Although in many well marked cases of Rheumatism there can be elicited no history of an Arthritic heredity, in other cases now and again we meet with very definite histories of cases of families in which the parents and most of the members have suffered from one or more acute attacks, and as I write I have in my mind the case of a family where the father is a chronic victim of Rheumatism now apparently assuming the Rheumatoid Arthritic form, while two of the sons have had several acute attacks and two of the daughters along with the mother are of a pronounced nervous and hysterical type; and in a case under care at present of a young man with acute Rheumatism, Pericarditis, and Pleurisy, his sister has had several attacks of Rheumatism and Chorea, and the rest of the family are nervous. On the

principal that "one swallow does not make a summer" these isolated examples count for nothing, but are merely quoted as examples of numbers of cases in which however on closer examination one finds the other factors the "nervous" and important factor.

Granted that an Arthritic Heredity is a common though not essential feature in the history of Rheumatic Families, this would lead one to look for other evidences of a tendency to Rheumatism beyond the mere occurrence of the actual attack itself in the case of any one patient and his or her Rheumatic relatives, and here difficulties crop up in the way of accepting the idea of a well defined "Rheumatic Diathesis", which to be of any use should mark out these "Arthritics" with definite physical traits such as one is taught to observe and accustomed often to note in the case of hereditary Syphilis with its Hutchison's teeth, etc., etc., and the general type of physique etc. of the tubercular and included strumous diathesis.

The type of the Sthenic Gouty Diathesis, "Fat Gout" as, it is called in Yorkshire, is fairly definite,

but the gouty ideal has been largely founded on the alleged free eating and drinking habits of the Georgian era, at least in England, and as society at large is becoming more temperate this ideal must fade just as the classic symptoms of an acute attack of gout have become appreciably modified.

But even the Definite Gouty Diathesis is becoming a thing of smaller import now that Gout has been studied by a host of careful observers who tell us that "*Podagra pauperum*" is just as common as "*Podagra divitum*" as we can readily believe on comparing the various irregular forms that occur both in the Gouty (and hence truly "Gout") and those of spare forms and abstemious habits; these irregular forms of this protean disease being often similar in both classes.

In the case of Rheumatism a Rheumatic Diathesis has been described of which the subjects are described as being well built, stout, full-blooded, and complexioned, with good teeth, and of active and mercurial habits. This description closely borders on that of the ideal Sthenic Gouty Diathesis and is

only another evidence of the difficulty of separating Rheumatism from Gout.

My own experience does not lead me to coincide with the view that the Sthenic or Plethoric semi-gouty type is so frequently associated with the more purely Rheumatic Diathesis but that the general characters of the Rheumatic Diathesis, if it can be described as such, are rather such as would be classed by modern writers as descriptive of the Nervous Diathesis.

Thus in a number of cases in which my attention has been drawn to this point, the patients have been of slight build, small boned, with fine hair, dark or fair, of high nervous tension, and with general readiness for the liberation of their entire stock of nervous energy and thus apt to be completely run down. The young naturally exhibit in general these characters more than elderly people and this may in part, as we shall see later, explain the excessive readiness with which young people fall victims to attacks of acute and sub-acute Rheumatism.

In connection with this point I may draw attention to the commonly seen connection of Chorea, a nervous disorder, with Rheumatism. Altho' of course in explaining thus the connection of those two diseases or forms of the same disease, a great many other factors have to be taken into account, still, the fact remains, Chorea is often a sequel or concomitant of Rheumatism. The mere fact that several members of a family have had attacks of Rheumatism (acute) neither proves nor disproves the view that there is a special tendency for the "body chemistry" of individuals of such a family to deviate from the normal in respect to the excessive formation of certain more or less poisonous products such as Uric or Lactic acid, certain alkaloids, ptomaines, and leucomaines, poisonous or not poisonous, and toxalbumins, or poisonous albumosés, or the Rheumatic poison whatever it is and which is supposed to give rise to the acute, sub-acute, and probably to in lesser degree, the chronic forms of Rheumatism.

There is a tendency at present on the part

of writers on the subject of heredity and the so-called diathesis to endeavour to connect the occurrence of Rheumatic phenomena with what for want of a more definite term are called the "Neuroses" a convenient limbo to which to relegate, at least temporarily, a number of symptoms depending on ill-understood states of morbid nutrition. In fact, Bouchard (Lecture 1). defines "Diathesis" as a chronic state of malnutrition of certain body arrangements of which the "nervous" is specially concerned, with consequent morbid functioning. Looked at in this light, the subjects of Rheumatism, especially when it can be called hereditary, find themselves in the same category as patients suffering from the various forms of nervous and mental breakdown.

From actual observation I am inclined to agree with the nervous relation of the predisposition to Rheumatism; but would explain the actual incidence of the disease not to the nervous breakdown of itself, but rather to the rapidity with which many of these high pressure, nervously organised individuals are able to go on working, bodily and mentally until they are completely exhausted, or to use an analogy, until their nerve force is at Zero and the waste fatigue products at a point of maximum accumulation, whence they pass into the dyscrasic rheumatic state and fall a ready prey to incidental chills etc. All this in such cases can be brought on in a very short time. Of course individuals of a phlegmatic temperament may also become run down but here the process is more gradual as they rarely work at high tension and in general husband their strength more. Practically I cannot recall many examples of really phlegmatic people. .

with hereditary Rheumatism and the coincidence if not effect of well marked acute rheumatic attacks occurring in "nervous" families, makes the combination perhaps disproportionately prominent as compared with ordinary phlegmatic or non-nervous cases. To sum up this paragraph, it may be said that while a large number of "histories" of cases of well marked Rheumatism shew no hereditary history yet when this history is one of Rheumatism it is well marked and the family is an example of what is at present termed the "Neurotic" type.

Haig in his well-known work on "Uric Acid" has endeavoured to connect nervous diseases in general on the one hand with Rheumatism and Gout on the other through the medium of his Uric Acid theory which of late he has so persistently urged so that from this point of view Melancholia, Epilepsy, Migraine, Raynaud's Disease, Neuralgia, and half the ills that flesh is heir to are only varieties of the same class as Rheumatism and Gout; the variety according to him, depending on the solubility of Urates and Uric Acid in various parts and their accidental precipitation following from varying local states

of acidity. In the case of the writer's own family while his mother, grandmother, brother, and sister all of rather nervous build, have had acute attacks with years of chronic misery, he himself from the fact that he is of rather "bilious type" in place of a rheumatic attack, after a chill or overexertion has an attack of slight jaundice and diarrhoea, the bile-eliminating action in his case being prompt and bearing the brunt of the "poisoning" (which will be developed in this study later on) so that he has never had a joint pain in his life and his own experience is distinctly in favour of the old adage "That life depends on the liver" in contra-distinction to Haig's new Epigram that "Life depends on Uric acid".

The next important point I shall discuss is this :-.

"Can an individual in an ordinary state of health succumb to Acute Rheumatism after the amount of exposure, etc. ordinarily credited as the cause of an attack ?

I believe that a person in an ordinary state of "good health" rarely if ever succumbs to an attack of acute rheumatism after exposure to the ordinarily alleged exciting causes, which are apparently followed by an acute attack in those who actually take the disease and who have been more or less ripe for it for some time.

In going over a number of cases of Rheumatism I have never found a case of a person who was in ordinary "good form" at the time of the attack or at the time of the exposure to the over-exertion, chill, wetting, etc. credited as the "cause" of the subsequent acute rheumatism.

For a person to take acute or sub-acute rheumatism he must be in that lowered state of nutrition and vitality which for want of a better name I shall call the pre-rheumatic state (Dyscrasia Rheumatica). This pre-rheumatic state is evident generally in the shape of increasing Anaemia, which I believe is the most common bodily antecedent, with a general run down state of health and nerve

tone along with which there is generally seen a tendency to Eczema and Atonic Dyspepsia. So much has this Anaemia and in the case of young women this atony and flabbiness of stomach, become associated in my mind as one of the commonest antecedents or danger signals of Rheumatism, that I always warn young anaemic patients of the danger of chills in this connection and the danger of over-exertion, cold feet, etc. Thus in the case of a young woman under treatment for anaemia a few years ago, I then remarked about the probability of a chill in her then present state of health developing rheumatism and unfortunately she shortly afterwards succumbed after a preliminary acute tonsillitis, to a long drawn-out attack of acute rheumatism.

Sir A. Clark's theory of Faecal Anaemia in young adults and its cure by purgatives (e.g. Mag. Sulph) may here be referred to for a moment as apropos of the connection of alimentary poisoning with anaemia and as I just said of the connection of the latter with rheumatism and which will be devel-

oped later in the study.

In another case, that of a young man, a run-down state of health with anaemia and troublesome eczema was soon followed by an attack of acute rheumatism subsequent to a very slight chill.

The case of plumber's assistants, young and generally anaemic, is noticeable as furnishing a number of cases of chronic and acute rheumatism. As is well known, workers in lead are very subject to gout, but the tendency to rheumatism here can be quite well explained on other than the Uric Acid argument, for the lead of itself leads to anaemia and as well ^{as} ~~to~~ the retention of Uric Acid, which almost seems specific in lead poisoning, there is a deficient oxidation, from the anaemia generally, and waste products other than Uric Acid are accumulated in the system which go to induce the rheumatic state. In fact any definite cause of Anaemia is one of the leading predisposing causes of rheumatism.

It may next be noted that individuals in ordinary states of health may expose themselves

- even those supposed to be of the hereditary rheumatic diathesis - to cold and wet with impunity and the same applies, altho' in a lesser degree, to those who have had one or more previous acute rheumatic attacks, but who have recovered completely and are not anaemic at the time of the chill. Here the supply of blood, rich in haemoglobin together with the normal functional activity of the excretory organs completely oxidises and excretes the extra waste or fatigue products following the chill, which are thoroughly burnt up or excreted and the system is not poisoned; as in the case where this is imperfectly accomplished with a resulting attack of sub-acute or acute rheumatism.

The Classes of Persons attacked. by acute Rheumatism might, if ascertained correctly enough, lead to a knowledge of the condition which cause or favour the developement of the Rheumatic Attack. The report of the "Collective Investigation Committee" of the British Medical Association assigns the first place to Domestic Servants, Married Women, School-children, and Labourers.

The striking frequency with which domestic servants become rheumatic is notorious, and the same class affords the most well-marked examples of the more severe cases of Gastric Ulcer and, as outpatient hospital clinics, as well as private practice, testify, they practically all suffer from Anaemia and constipation. In fact it may almost be taken as an axiom that more than half of all domestic servants suffer from Rheumatism, Anaemia, and Gastric Atony and in many cases from Gastric Ulcer. The favourite explanation used to be that the above symptoms were due to poisoning by excess of Uric or Lactic Acid in the system, from their supposed large consumption of meat and beer, from the opportunities afforded in a few cases of hacking at roasts and drawing beer ad lib. Practically, however, one finds that rheumatism is just as common among this class in parts of the country where beer and meat are not allowed and in homes of total abstainers as well as cases in the establishments of the wealthy. What, however is a common

factor in both sets of cases is the presence generally of Anaemia, with Gastric Atony and probably dilatation, all induced from being much shut up, from bad habits as regards regularity and quality of meals, stairclimbing, hot kitchens, etc.

Both the anaemic and atonic dyspeptic state pave the way for rheumatism by preventing, from lessened haemoglobin in the blood, the proper and complete oxidation of fatigue products (leucomaines and poisonous albumoses) and by producing ptomaines and certain poisonous albumoses by imperfect gastric and intestinal digestion, with the absorption of the latter into the system, which absorption is much aided in the case of intestinal poisons by the generally concomitant constipation.

Even in the Uric Acid theory of Rheumatism, which I shall discuss later at length, anaemia may be considered as a **causal** factor of the excessive production of Uric Acid (V. Jacksh Quoted by Levison, p. 43.) and hence according to Haig, of Rheumatism for, as Horbaczewski has shewn, Uric

Acid is excreted in large amount in Anaemia and Leucocythaemia, being a product of nuclein, or it (the Anaemia) may be considered on Haig's theory as the effect of destruction of the red blood cells by the alleged excess of the Uric Acid in the blood.

As regards school children, the explanation of the occurrence of the rheumatic state may be considered both from the Uric Acid, and from the fatigue product poisoning which I maintain.

In either case, children ought a priori, to be victims in much higher proportion than adults as their relative secretion of Uric Acid is much higher in proportion than in adults and decreases relatively with age. As in birds, the processes of metabolism are more active than in adults, but Haig's Uric theory, at least from the **dietetic** point of view, does not hold as children are mostly fed on farinaceous and starchy foods and their intake of Urates in the shape of meat is limited. Hence the rheumatic poison in their case, at all events, must be due rather to fatigue products other than Uric Acid from the food, than to Uric Acid per se.

and this point is in favour strongly of what will be considered later in more detail that as Horbaczewski has shown the amount of Urate excreted is dependent rather on the rate of body metabolism of the organism than on the amount of Nitrogenous ingesta which, however, does increase the amount of Urea. According to Haig's line of reasoning, a small ingestion of meat foods lessens the formation of Urates in the system and on this he has built up his "no meat" treatment, whereas in the case of children we see it flatly contradicted and the fact remains that they who excrete a high relative amount of Urates are very liable to rheumatism in spite of their generally not consuming much meat for their regular meals. But as is tried to be proved in this paper, from their general activity and rapidity of metabolism they are able to form poisonous waste products other than Urates rapidly and in excess and hence as a class furnish a large proportion of cases of rheumatism. The same line of argument explains in part the liability of married women and labourers, but in these cases there are many other factors which must also be taken into account.

At this stage I shall pass on to discuss the State of the Blood and Lymph which favours the development of the acute rheumatic attack. Is there an abnormal condition of the blood etc. present in the rheumatic state which will explain the symptoms? The proper and direct answer to this question which is the most important of all in deciding as to the value of the present views on the subject can only be answered satisfactorily by a prolonged and careful examination of the blood both in Rheumatism and in health, at the same time taking into consideration the various side issues which naturally suggest themselves and comparing excretion with the amount of work and kind and quantity of the diet. Unfortunately for the ordinary medical practitioner this practical method of settling the question is not available without all the methods and materials of a laboratory at hand and he has to rely for the solution of such problems on the data furnished by experimenters, or may be fortunate once in a lifetime to observe a natural experiment or set of variations attended with new results on which to found a rational

hypothesis.

I shall here pass shortly in review the various views as to the nature of the supposed morbid states of the blood in Rheumatism, noting how far in my estimation they explain the various phenomena and offering my own explanation as to Rheumatism being due to the presence of waste products, not necessarily Uric Acid, and the rational method of combating the state by treatment consisting of free elimination and oxidation. The views by means of which it has been sought to explain by altered states of the blood the occurrence of Rheumatism include the following as the most notable, viz:—

(A). The old Humoral theory. (b). The lactic Acid theory and its various developments along with the nervous. (c). The Uric Acid theory of Haig.

(d). The Malarial theory of Maclagan. (e). The Bacterial theory of Sahli in Germany. (f). Lucatelli in Italy 1892. (g). The view of the younger Garrod that Rheumatism is a systematic disease distinct from Gout and Rheumatoid Arthritis but the nature of which is unknown. (h). The "Arthritic" theory of

the French school. (i). The writer's own view on the subject.

The old "Humoral" pathology which the pendulum of medical opinion has nearly swung back to but with the addition of the germ theory etc. as explaining the causal factors of the depravity of the Humours had much in its favour looked at in the light of direct observation by the older physicians. In the ordinary course of treatment, by bleeding, the state of the blood was much more readily observed by naked eye inspection than at the present day. The "Buffy" coat and "Cupped" surface of the clot in the bleeding basin was no bad argument that the humours were profoundly "depraved". As the phenomenon came to be more closely studied it was found that in the fever of Rheumatism as in Pneumonia the amount of Fibrin was in excess - Hyperinosis, (Vide Hooper's Physicians' "Vade Mecum"). The only reference I can find that this is a point worth attending to at the present day is given by Garrod, Junior on Rheumatism p.66, where he says that M. Hayem believes "that the increase of Fibrin

affords a valuable diagnostic sign in cases of obscure nature" the blood being examined during coagulation on a glass slip and he further states that with the anaemia the "white" corpuscles are increased.

More recent experimental physiological researches since the time of the old humoral pathologists have shown that "Buffing and Cupping" depend on physical as well as physiological and pathological conditions and the importance previously attached to the phenomena and the amount of which was a guide to the physician as an indication for a repetition of bleeding has now become insignificant. It is perhaps worthy of incidental remark that in ordinary anaemia which I have noted as in my opinion an almost constant antecedent of Rheumatism, the buffing and cupping of the clot has been generally observed, in this case depending on the reduction in the amount of the corpuscles and the more hydraemic state of the blood, both material factors in the physical process of "buffing and cupping" and which in acute Rheumatism may act quite as much as a relatively hyperinotic state of the blood. This anaemic

state may have partly associated with it the phenomenon of apparent hyperinosis in the minds of the older physicians (Hooper mentions that the cupping and buffing increase in Rheumatism after each bleeding). The theory of Cullen attributed Rheumatism to the direct effect of cold on the joints and subsequent inflammatory action.

The Lactic Acid Theory only rests on indirect presumption and on the results on the joints noted by Sir W. B. Foster where the commercial lactic acid had been administered in a few cases. The acid itself has never been separated from the blood in cases of acute rheumatism, and lactic acid administered to healthy individuals (not diabetics as in Foster's cases) is not at all poisonous. In laboratory experiments with contracting muscle the reaction of the fibre which is normally alkaline before work becomes acid from the development of the isomeric sarcolactic and ethene lactic acid. Granting that the presence of Lactic Acid is the cause of symptoms in acute rheumatism it would explain in part the incidence of an acute

attack following excessive exertion as is frequently seen in actual practice.

In Sir W. B. Fosters cases the administration of lactic acid was followed by joint pains, and other experimenters have produced effusion into the joints in the case of the lower animals by its administration. On the other hand Haig declares that in his own case he can produce joint pains by taking small doses of Uric Acid for a few days, but it has been experimentally proved that acids injected have practically ^{no effect} on the reaction of the blood, (Levison, p.86.)

The idea that there might be a relation between the effect of chill on the nerves and the subsequent formation of a poisonous quantity of Uric and Lactic Acid is developed in Latham's theory (Croonian Lectures 1886). A short resume is given by Garrod p. 25, and I need not further refer to it.

It is commonly stated by writers and also believed by rheumatic patients that after drinking acid wines such as new bad claret and champagne, rheumatic pains are liable to follow. This is a

point which requires further discussion as I have practically found in chronic rheumatism cases in which the dilute mineral acids given at times have been followed by the best effects.

It used to be the fashion to prohibit on the Lactic Acid theory, starchy and saccharine articles of diet - potatoes, rice, sugar, etc., on the idea that these give rise to injurious increase of Lactic Acid. The effects of this dieting were pretty much the same as follow Haig's Vegetarian diet. Other upholders of the Lactic Acid theory draw attention to the frequent association of acid (lactic) dyspepsia with rheumatism, lactic acid being one of the products of gastro-intestinal fermentation, its subsequent absorption perhaps occurring. As I shall point out later it is unnecessary to invoke the aid of lactic acid in explaining the connection.

The Nervous Theory has already in part been touched on, but the purely nervous theory is that certain centres in the medulla are disturbed and the symptom of sweating is instanced in its support:

various poisons can however give rise to this symptom (vide post).

Haig's Uric Acid Theory.

Haig's Uric Acid hobby is one of the latest and largest orders on our credulity recently put forward as an explanation of the "poison" of acute rheumatism and its congeners (according to him) Rheumatoid Arthritis and Gout, and the argument is urged with such ingenious persuasiveness that one is apt to be carried away by his short and easy method of setting forth and dealing with the enormities of the Uric Acid fiend. Dr. Haig (vide his book) considers that the modern individual consumes a far larger amount of nitrogenous food than is physiologically necessary and that as a result there is directly introduced into the system the elements of which Uric Acid is a product of lower grade chemical change and that this can be proved by analysing the urine where the diet is regulated and that in Uric Acid cases the acid excreted is higher in relation to the Urea than Haig's regulation proportion of 1:33.

Further, bodies closely allied to Uric Acid such as Creatinin, Thein, Xanthin, Caffeine, etc., which he alleges may readily increase the amount of Urates act as direct rheumatic and gouty poisons, by giving rise to Uric Acid. As is well known, Horbaczewski has lately shown that the amount of Urate excreted is not "practically" affected by the amount of nitrogenous ingesta but is rather the result of increased bodily metabolism and that Haig's ratio of 1:33 may be altered at pleasure, on the Urea side by the amount of diet and on the Uric side by exercise. This is borne out by what one sees every day. After a long ^{ride} or other severe exercise the Urine throws down a dense red deposit of Urates and in the case of one indulging in hard walking or riding as in ordinary medical practice this state of the urine is habitual and the "Silver-nitrate" process is unnecessary in roughly gauging the amount of urate, as there it is precipitated in the pot-de-chambre as the ordinary brick-red urate ! This state of affairs can be readily altered by stopping the exercise and the urine once more returns to its clear transparent character and this on the same diet as before.

Haig further shows that at the same time as the excess of nitrogenous matter is ingested the excretory power of the kidneys is unequal to the task of getting rid of this excessively formed urate and, given an acid condition of the fluids in any locality of the body, say a joint, the Urate introduced as such or formed in situ is precipitated locally, the argument being that a very slight change from alkalinity to acidity causes the precipitation, as urates are only soluble in alkaline media. Practically however it is found that the blood cannot be rendered acid by giving mineral acids and in ordinary practice it is practically impossible to make an alkaline urine acid by giving the ordinary B. P. acids. As I shall note again later, Haig's view does not tally with Robert's remarks on "Biurate" "Quadri-urate" etc.

It is next shown by Haig that Uric Acid itself or any other acid will have exactly the same effect when ingested as any other acid formed in the animal economy has, and hence urates in the body fluids will

be precipitated by a dose of Uric Acid administered, either
 experimentally or in the form of the acid tartrates
 etc. of new claret and bad champagne, while at the
 same time the blood will be "cleared" of Urate at the
 expense of a corresponding deposit in the joints and
 even the viscera, the fibro-serous membranes being
 commonly the seats of election for the Uric deposit.
 The uric acid introduced in meat is said to produce
 the "clearing" effect by robbing the normal sodic-
 phosphate $\text{Na}_3\text{P.O}_4$ of the blood of part of the base
 converting it into acid phosphate $\text{Na}_2\text{H.P.O}_4$ the former
 being a good solvent of urates, the latter rendering
 them insoluble and it follows that as long as the
 body fluids are "acid" it is nearly impossible to
 get rid of the urate by the ordinary excretions and
 it lies "stored" in the joints, liver, kidney,
 spleen, etc. As will be noted later, Roberts by
 actual experiments shews that an "alkaline" state of
 the blood is just as bad for the solution of the
 urates as Haig's alleged "acid" state in Rheumatism,
 Gout, etc.

To return to Haig - He next argues that the excess of Urate in the system being established with a concomitant relative acidity or lowered alkalinity of the body fluids, any slight disturbance in chemical reaction of the fluids of any local area, such as a joint in which it is affirmed that from the relatively slow circulation, augmented by the functional joint activity which may be true, or as I concur in, by an injury, as often actually seen, will be followed by a deposit of urate in the now acid fibrous tissue of the joint with the subsequent train of Rheumatic Symptoms. Similarly, gout is regarded by Haig as the result of excess of urate in the blood and followed by its deposit in the joints the great toe and ankle being functionally more active than the others and especially liable to slight twists which when the blood is teeming with Urate determine the seat of the deposit and give rise to Gout. I may point out in passing that against the view, first clearly enunciated by Garrod, senior, that the urate mechanically or otherwise causes Gout, Dr Berkham has recently tried to

shew (B.M.J. Feb. 2nd. 1895) (On the Pathology of the Gouty Paroxysm) that the deposit of Urate is a mere epiphenomenon. Haig similarly regards Rheumatoid Arthritis as the result of the more gradual deposit of Urate and he describes cases where the urate as in old age from lessened acidity(?) has continued to be excreted with constant diminution of Uratic joint deposits and he finally appeals to the impossibility of differentiating P.M. between a true gouty and a Rheumatoid Arthritic joint.

From all these considerations he urges as the rational line of treatment a practically vegetarian and bread stuff diet, forbids, untill the supposed "stores" of Urate in the system are got rid of, the use of beer, wine etc., the acid of which would "precipitate the Urate in the joints, as also the vegetable alkaloids occurring in foods in the shape of tea, coffee, caffeine, etc., and, with a view to dissolve for excretion the "stored" Urates advises a long course of alkalies and salicylates the latter being alleged to owe their efficacy in rheumatism, etc. to the power of fixing the Uric Acid as

Salicyluric Acid in which form it is excreted.

To criticise Haig's views briefly, as I have pointed out it has been shown that the ordinary high level of nitrogenous ingesta does not materially increase the total quantity of urate excreted in 24 hours by the individual, there being only a slight temporary rise after a meal due to digestive leucocytosis and not to the nitrogenous ingesta themselves (Horbaczewski, quoted by Levison p. 38). That in children who excrete a relatively larger amount of urate than do adults, the food is as a rule of a non-nitrogenous kind, shewing that the increase is due to relatively more rapid metabolism and constant bodily movement. Again country people who do not as a rule indulge in much meat, are often rheumatic (chronic) which tells against Haig's view as to being able to control the Uric Acid by dietary, and the same applies to the rheumatism of children. In all cases the excretion of urates varies with the amount of bodily exercise, the kind of diet being constant. (Here it is presumed the excretory power of the

Kidneys is normal).

The alleged acid state of the blood in rheumatism is only assumed and has never been actually proved and any one can perform the experiment roughly by means of red litimus paper first dipped in Saline Solution to prevent the blood clotting, and then washing off the blood gently. The blue alkaline reaction is quite distinct . I have noted previously that several observers have shown that the various B. P. Acids exhibited produce no diminution of the alkalinity of the blood and Garrod Junior says he has never found the reaction acid in rheumatism, hence there is no reason to believe that an acid state of the blood exists sufficiently to precipitate urates as Haig supposes. Further, as Roberts points out, any uric acid in the blood occurs as Biurate, the most insoluable compound of Uric Acid.

It is true that the Urine and perspiration are acid due to Na_2HPO_4 (and Lactic Acid(?) Fuller) but certain observers have pointed out that even in acute rheumatism the perspiration may be

alkaline and Garrod, Junior (p.p.21 & 65.) says:-

"There is however little evidence of any undue acidity of the sweat of rheumatic patients" , normal sweat if carefully collected being practically neutral. The elder Garrod says positively that he has never been able to find Uric Acid in the sweat even of Gout, and even where it is acid as it often is it does not follow that the blood too should be so, any more than that because the Gastric Juice contains free HCl, the blood as a whole should not be alkaline. Again Uric Acid is practically not a poison and certainly not in the small quantities sufficient for Haig's theory. In Gout the blood may teem with it as readily shewn by Garrod, senior's thread experiment with gouty serum acidulated with acetic acid and where the Uric Acid crystallizes out on the thread.

Bouchard has made direct observations and experiments and finds that it requires the whole of the poisons of at least 24 hours urine to kill a man and that Uric Acid can be injected without toxic effects in doses of 0.64 gramme per Kilogramme of

an animal's body weight (Bouchard p.p. 350. 118.)

Similarly with respect to Creatin he quotes Fritz and Ritter as not being able to kill an animal by injecting ~~into it all~~ at once its total excretion of Creatin Ptomaine for 17 days and similarly in lesser degree with Leucin, Tyrosin, Zanthin, and Hypoxanthine Ptomaines. (Bouchard p. 118.)

Finally ~~even~~ were Uric Acid such a poison as Haig fancies it has never been found in the blood in Rheumatism which fact is accounted for by Haig by assuming that unlike Sir Boyle'Roche's bird "it cannot be in two places at once" in Rheumatism, the acid state of the blood having already "precipitated" it in the joints.

As regards the actual state of the joints during the acute stage there are no very definite observations on the subject, the fluid in them by ~~ing~~ some observers being described as Acid and by others Alkaline. Even were Uric Acid when precipitated the cause of Rheumatic Symptoms it is not necessary as Haig states to postulate an Acid state of the body fluids. This part of the subject has been experiment-

ally investigated by Sir W. Roberts (Croonian Lectures) who says:-

"In the normal state Uric Acid $C_5H_4O_3N_4$ is primarily taken up into the system as Quadriurate and is excreted in the Urine as such, but in the gouty state this tranquil process is interrupted either from defective action of the Kidneys or from excessive introduction of Urates into the circulation and the Quadriurate lingers unduly in the blood and accumulates therein. The detained Quadriurate circulating in a medium which is rich in Sod. Bicarb. takes up an additional atom and is thereby transformed into (and precipitated as) Biurate, a less soluble and less easily excreted compound".

(In tophi the deposit occurs as Biurate, in Urine as Quadriurate, in blood probably intermediary).

Even the success of the ordinary alkaline treatment for acute rheumatism does not prove that the poison is necessarily Uratic in nature as Haig thinks, for Sir W. Roberts holds, although I would not be disposed to accept the practical results which follow his view in the shape of with-holding

alkaline urates in Gout etc. that :-

"It has been conclusively proved that alkal-
esence as such has no influence whatever on the solu-
bility of Sod.Biurate" and that Clinical experience
on the use of "alkalies speaks with a doubtful voice"
that is in Gout which Roberts supposes due to Uric
Acid and which should apply to Haig's Uric Acid
Aetiology of Rheumatism. As I shall point out later
the good effect of Salicylates Etc. can be explained
on other than the Uric Hypothesis. Enough has I
think been said to cut the ground from under the
feet of Haig's theory, which if confirmed, as its
author fondly hopes it may be, will revolutionise
modern medical ideas and treatment on the subject of
Rheumatism, Gout, Nervous Diseases, High arterial
tension, Epilepsy, etc., etc., and will form the
basis of a completely new general pathology, as may
be readily seen from a careful study of Haig's
work on Uric Acid.

Before making my own remarks proper as an
explanation of the "poison" of Rheumatism, I shall say

a word or two as to the "malarial" origin of Rheumatism and the associated Bacterial view on the subject.

The malarial origin of Rheumatism advanced by Mc.Lagan in explaining the action of the Salicylates and which had already been given expression to by the older writers (Garrod p. 33.) naturally suggests itself from a comparison of many of the symptoms of Rheumatism and Ague from the similarity of certain antecedents of both. Thus both Rheumatism and ague may follow residence in a damp low lying piece of country and the more chronic forms of Rheumatism are just as common in damp tropical countries as is Ague, and in this country, damp low lying houses are frequently powerful predisposing causes of Rheumatism. I, however think that the effect is rather an indirect one in these cases by producing Anaemia along with a general lowering of the body tone and its results and the same may be said of those cases of defective drainage when sewer-gas escape has been attributed with apparently reasonable assumption as the cause of a subsequent series of cases of Rheumatism. I have seen one fair instance of this "Drain Rheumatism" in the case of two members of the same family.

Since it has been definitely proved within the past few years that ague is due to the effects of the Plasmodium Malariae of Laveran, an organism allied to our old friend the Gregarine of the Earthworm and probably multiplying in the same way by the detachment of processes which carry on the life-circle of the parasite, the causal connection between Rheumatism and Ague is not now directly supportable. The chief points of resemblance in both are the copious perspiration, the anaemia, the tendency to recurrence, and the similarity of the pain both in the acute and chronic forms.

As I write, the Influenza epidemic furnishes a link between the two, in many cases it being nearly impossible to diagnose Influenza of the Rheumatic type from acute and sub-acute Rheumatism, the appearance of Coryza being a welcome sign in many of the rheumatic cases as a diagnostic aid.

Very probably the above ague symptoms are due to the poison formed by the Plasmodium Malariae, either poisonous alkaloids (or Ptomaines) or poisonous albumoses (tox-albumins) resulting from the decom-

position of the albumen of the blood etc., just as in the case of Phthisis and Septicaemia one sees the profuse perspirations probably due to the same cause, the poisonous chemical products produced by the Tubercle Bacillus. In Rheumatism it is just as reasonable to suppose, that the severe perspiration is due to a similar cause (excluding the agency of a microbe) which is not explicable by the Uric Acid or Lactic Acid theory. Of course toxalbumins and alkaloids (leucomaines) may also be formed without the presence of microbes by the ordinary metabolic changes in the body.

The profound anaemia often noticed in Chronic Ague and after Rheumatism points to a similar destruction of the blood corpuscles, by the plasmodic poison in the one and the probable leucomaine and poisonous albumose in the other. The tendency to recurrence in both is less easily understood. In ague, contracted in the first instance abroad, the organism may be lying dormant or a fresh inoculation of the susceptible patient may occur even by the mild ague germs of the damp parts of this country.

In Rheumatism, as I said in connection with the nervous theory, the periodical recurrences simply mean that the patient has been working at high pressure and the last straw is put to the accumulation of fatigue products in the blood, and the attack is repeated. From these considerations one readily passes to the purely Bacteriological explanation of rheumatism of Sahli and Lucatelli but which has never been taken seriously. That Arthritic inflammation and severe joint pains indistinguishable by themselves from acute rheumatism can occur as the effect of microbic action is seen in the case of Influenza now believed to be due to a specific bacillus (Pfeiffers') and in the case of Gonorrhoeal rheumatism (Bumm's Bacillus) and in the cases of pyaemia (micrococcus and streptococcus Pyogenes Aureus,) with joint implications, and where the heart valves and other fibro-serous membranes may also be effected. However, no two observers are in accordance as regards the exact coccus or bacterium. The prima facie evidence for the bacterial origin

of Rheumatism is not to me so strong as the poisoning by leucomaines etc. and which will shortly be discussed here but before which "the bearings of the effect of general conditions of temperature and hygrometric variations as predisposing and exciting causes of Rheumatism," will be shortly considered.

As far as my observations go I believe that the direct influence of climate in the wide sense and local variations in the degree of temperature and moisture have been exaggerated, although by sudden variations they may readily act as immediately exciting agents of the rheumatic attack in those ripe for the disease. To discuss this part of the subject properly would involve a thorough study of the effect of chill etc. both in health and disease and in turning to Landois and Stirling's Physiology (page 337 3rd Edition) the paragraph on the subject does not aid much in the way of furnishing data. The only way chill in a local neighbourhood, say a joint, can act must be by lowering the full metabolic process and the imperfectly oxidised products resulting may poison

the locality, which may be followed by a true inflammation such as is started by any other chemical, mechanical, or nervous irritant. In cases where the whole body is chilled the consequences are proportionately extensive. As regards actual climate, rheumatism acute and chronic occurs practically all over the world, both in the warm damp climates of the East and certain parts of Australia e.g. Melbourne, Africa e.g. Natal, as well as in the warm dry e.g. California, and in our own dry bracing uplands of the West Riding (grits and sandstones of coal-measures). The warm dry climates seem to be more exempt e.g. Upper Egypt and certain dry parts of India probably due to the more rapid evaporation of perspiration; warding off chills considerably during the day, although as in all dry climates the day and night temperature vary very much, the apparent absence of visible perspiration is explained by people who have lived in those parts by saying that they "never sweat." In the case of the warm damp muggy climates the intense heat and moisture rapidly induces a state of anaemia in which the vulnerability to the effect of chills is very great and it is

a matter of common observation that persons thoroughly anaemic from residence in the tropics can never get or keep warm on their return to this country until the blood is renovated, and how liable they are to rheumatic chills. The analogy of the anaemia which is such a common occurrence in country blacksmiths from the effects of the heat of the forge in this country exemplifies this point. The same occurs from the relaxing effects of certain parts of this country, anaemia etc. and subsequent tendency to rheumatism. In those cases of anaemia where the patient is in the pre-rheumatic state a slight chill or wetting acts as the exciting cause of an acute attack, whereas if the patient is in good form all that may occur is a catarrh of the mucous membranes. Thus in the case of a medical friend who has several times had acute rheumatism, when he is in good form a chill will be simply followed by a catarrhal cold, whereas when anaemic he invariably suffers under the same circumstances from a sub-acute rheumatic attack, so much so, that he has often remarked upon the vicariousness of his joints and mucous membranes in this respect.

In this country a large number of the cases are observed in Winter and Spring especially the latter in exposed upland parts of the country. In Paris on the other hand, it is said that most cases occur in July and August, at least in Hospital practice, and even in this country bad attacks are often noticed in very warm weather. I am credibly informed by a Dutch priest (Rev. Father Hassing) that in Holland, the amount of rheumatism is not remarkably noticeable in spite of the canals and very cold winters. The evidence of the "Collective Investigation" Committee, B.M.J. is even unsatisfactory on this point and I think that climate as a whole only predisposes to rheumatism following if the subject is in the poisoned pre-rheumatic state.

Thus far I have attempted to shew that neither the presence of acids, chiefly Uric, in the system nor the effect of cold in itself is sufficient to explain the aetiology of the Rheumatic State. On looking round at the results of quite recent research one may suppose that some other

* Foot Note to Pages 50 & 55.

Ptomaines (Gautier). B.

A. Non-oxygenated.

Parvoline.

Hydro-collidine.

Collidine.

Neuridine.

Cadaverine.

Putrescine.

Mydaline.

B. Oxygenated.

Neurine.

Choline.

Muscarine.

Gadinine.

Leucomaines.

1st. Uro-leucomaines.

Betaine.

Karnine.

Adenine.

Gaunine. *Qua*

Hypoxanthian. *u*

Xanthine.

Pseudoxanthine.

2nd. Kreatinine.

Xanthokreatinine.

Crusokreatinine.

Amphikreatinine.

Pseudoxanthine.

3rd. Not well defined

but found in Urine

Blood, Saliva,

Intestines.

poison than Uric or Lactic Acids may be at the bottom of the symptoms which the alkalies and salicylates and the water in which they are administered in the current mode of treatment may by their general eliminating effect, rid the system of. The most instructive points in this connection may be gleaned by a study of those cases where acute and sub-acute rheumatism follows violent or long sustained exertion. Here the copious excretion of Urates testifies to the amount of metabolism, the Nuclein of the tissues according to Horbaczewski breaking up into Uric and Phosphoric Acids. As well as these there are also produced certain compound ammonias now called Leucomaines. Some of these are poisonous and others not. The exact toxicity of certain of these, Cholin, Neurin, etc., has been demonstrated by Bouchard as noted above. As well as these * alkaloidal bodies, albuminous matter is probably also partially decomposed into slightly modified forms which unless rapidly broken down into lower grade products may be poisonous (vide Hill's Note

Book on Physiology p. 191.) this partial decomposition, being probably of frequent occurrence where there is insufficient oxidation as in cases of anaemia. Thus in the case of one patient, a gentleman convalescent from rheumatism, a short walk of $\frac{3}{4}$ mile was invariably followed by severe muscle and joint pains and in most persons who suffer from the chronic variety of sub-acute rheumatism, excessive (for them) exercise is frequently followed by stiff back, shoulders etc., Here it seems that the muscular exercise produced a relative excess of fatigue products, acid Sodic Phosphate, Uric and Lactic Acid Leucomaines and altered albumins. The effect of alkalies, e.g. Ammonia, in rendering inert the poisonous albumose of Cobra-bite shews that the poison to be removed or rendered inert, need not be an "acid". It can be experimentally shewn ((Landois and Stirling p. 496. 3rd Edit.) that in a muscle which will no longer re-act from the accumulation of fatigue products, an injection of salt, sodic carbonate or permanganate of potash solution., or of

arterial blood is followed by return of contract-
ility and conversly a healthy contracting muscle
can be rapidly "fatigued" or poisoned by injecting
Phosphoric Acid, Sodic Phosphate, or extract of meat.
The effect of massage is partly explained by the
removal by expression of the fatigue products into
the lymphatics after exercise by rubbing.

As far as I have been able to observe, as
previously stated, the invariable antecedents of
Rheumatism seem to be general impaired health as
shewn by the anaemia often seen following previous
wear and tear bodily and mental and along with this
frequently Dyspepsia and Eczema. When the patient
is taking the attack, the chill is often probably
an effect, not a cause, of the poisoning and results
from inability to maintain the surface temperature
from clogging of the intracellular and intercellular
networks and lymphatics by fatigue products and by
from the impossibility of maintaining perfect
oxidation which is the cause of the ordinary feel-
ing of warmth in health. The metabolic process in

the muscles which is said to produce 4/5 of the body heat is upset and as a consequence the patient feels chilly for a day or so, or has a more sharp attack of shivering or even a distinct rigor. The pathology of Fever is too complicated and large an order for me to try to discuss, but I think that the leucomaine (and Ptomaine which will be touched on further) and tox-albumin poisons explain the occurrence of the subsequent fever better than the Uric Acid idea. It does not matter much if it be supposed that the thermogenetic centres in the brain are themselves stimulated by the vitiated blood or whether the tissues themselves are stimulated to exaggerated (fever) metabolism by the poison formed in loco. That the severe perspiration of the acute and sub-acute rheumatism is not due to the heat of the fever but to the action of the poison itself is seen from the fact that Rheumatics perspire copiously even when the temperature is not elevated. Here the sweating, as in Phthisis, is probably due to the effect of the leucomaines, ptomaines, and

tox-albumins on the sweat centres or peripheral nerves.

This part of the subject might be cleared up in part by a careful examination of the perspiration (which however is known to contain practically no Uric Acid (Garrod)) and its effect noted as in Bouchard's method with other excretions by injecting into the veins of rabbits. The effect of the elimination of the poison is seen when Rheumatics are got once again into "good form" and when the excessive perspiration on exertion during convalescence gradually disappears (Landois and Stirling p.248.)

As regards the mode of production of the Rheumatic poison or poisons and its effects the following considerations taken in connection with states of Anaemia, Dyspepsia, Constipation, and Eczema are available as explanatory. It is known that Peptones injected into the blood in sufficient quantity are poisonous, probably from contained albumose. Brieger has extracted from Gastric peptones by means of Amyl Alcohol a peptone-free

poison with action like Curarà. The intermediate stage of the conversion of Proteid into Peptone is that of albumose. (poison if injected into blood, Hill, p.193.). Similar poisonous products may also be produced by the action of Bacteria and imperfect Hydrolytic changes in the alimentary canal and these products may be absorbed especially if the Liver is at fault. Leucomaines (alkaloids) (Gautier), * ptomaines and non-poisonous, are produced in the muscles etc. by the animal metabolism and in long continued overwork these will accumulate faster than they can be excreted, particularly in Anaemic states. The effect of Fatigue Stuffs on muscle has already been noted. It has been noted that ptomaines and leucomaines are the agents by which the central nervous system is influenced in such a manner as to effect the temperament in dyspeptic conditions, constipation, jaundice, etc. (Hill p.190)

In ordinary typical anaemia of young people there is with the anaemia generally dyspepsia, constipation, and often eczema and also nervous disturbances.

It is very difficult to say which of the symptoms with its accompanying pathological change is "cause" and which "effect", the nervous element to the anaemia and dyspepsia, or vice versa. Sir Andrew Clark considered the anaemia as due to a poisoning from faecal retention and absorption of the poisonous products found in the bowel. Haig believes the anaemia in these cases to be the result of the Uric Acid poisoning. Bouchard tries to show that in all such cases there is "dilated stomach" with the production of ptomaines and tox-albumins, which are absorbed and give rise to the anaemia etc. and are produced from imperfect gastric digestion and he goes on to evolve a general "Arthritism" from this dilated stomach condition (vide his book).

Practically one finds there is often Dyspepsia with constipation, this is accompanied by increasing anaemia and this again is followed by Rheumatism. The dyspepsia is such a common symptom that many patients attribute their Rheumatism to Dyspepsia. (Haig would say that the dyspepsia was

due to the precipitation of Uric Acid on the surface of the stomach from the blood.) Although it is taught that Peptones during their passage through the stomach wall are instantaneously reconverted into non-diffusible albumen - otherwise poisoning would occur - it has not been shown that the ptomaines and poisonous albumoses which under certain circumstances may be found in the intestine as by Bouchard are deprived of their poisonous effects unless they are absorbed by the liver, and one might conceive that a dyspepsia continued for some time would lead to a gradual accumulation of the poisonous products in the blood and as a consequence to destruction of the red corpuscles. The anaemia next leads to deficient oxidation as seen by the "plump" state of many anaemic girls and a storing up in the blood of imperfectly oxidised metabolic products. The eczema often seen would seem to be an effect of the attempt on the part of the skin to excrete these waste products for one often sees "stomach" rashes and eruptions follow mild poisoning by

shell-fish, certain fruits, cheese, etc., and the rash of Typhoid etc. and in some cases those seen in Diphtheria and Rheumatism and in the recent epidemic of Influenza, may be of this nature.

There are thus two main varieties which may be combined where excess of poisonous fatigue stuffs occurs

^ on the one hand from over-exertion and imperfect oxidation, and on the other from imperfect digestion etc., and these may be the starting point in both series, the anaemia, whether occurring as cause or effect, being a main factor. Although the proof of the nature of the Rheumatic poison along the above lines may be rather feeble yet it seems as reasonable as any to consider it as due to the combined effect of indigestion, improper oxidation from anaemia and sudden accumulation following over-exertion, sudden or more prolonged when the "chemical capacity" of the individual is feeble or where from certain states of the nervous system all the available energy may be suddenly discharged and the system flooded up with an excess of imperfectly oxidised waste products which act as an acute poison

and where if they are not rapidly excreted general poisoning follows in the shape of an attack of Acute Rheumatism.

The Symptoms of Poisoning.

In many cases which "sicken" for Rheumatism, the onset of the joint and general trouble is gradual. The patient for some time back has been out of sorts and getting worse and for a few days all the symptoms are exaggerated, feeling of tiredness, want of appetite, costiveness, inability to cope with ordinary vocation, chilly feeling, and in most cases there is a history of chill from sudden reduction of surface temperature. The "poison" seems to have accumulated to such an extent that as in Diptheria, Ague, and Influenza, when a minimum effective quantity of poison has been introduced the system as a whole has to "re-act" to it, poisoning being a rapid form of stimulation followed by exhaustion and death of the organism.

Why are the joints selected by the poison along with the cardiac valves and certain other

fibro-serous membranes, in rare cases the pia-arachnoid and peritoneum ?

As regards the joints, the most common explanation is that the selection is the result of the Functional Mechanical Activity of the parts. This is insufficient. In Influenza and Ague the joints are similarly though not to such an extent affected. The only practical demonstrable explanation is in the cases where the individual joints have been exposed and then subsequently become the seat of acute inflammation. This does well enough in chronic and recurrent sub-acute attacks and, as Garrod explains it, is a mere inflammatory lighting up of an old inflammatory area but the explanation fails for the cardiac valves and in the "ingravescent" sickening cases.

Works on Physiology (e.g. Landois and Stirling) say little as to the excretory and secretory functions of the joints and I have been able to find little to aid me in conceiving that the poisonous fatigue products are in the first place

"reservoired" in the cavities of the joints before being carried off by the main lymphatics to the Thoracic duct.

Is there a special affinity of the poison for serous surfaces ? . I think the secretory view best explains the case even though not demonstrable on anatomical grounds. The case of the Tubercle bacillus and its poison offers an explanation by analogy. Here either the joints or fibro-serous membranes endeavour to excrete the Bacillus and its poison so as to partly rid the body of its unwelcome guest, and in this connection phagocytic action at least for the Bacillus may come into play, or the fibro-serous membranes are specially vulnerable to the poison of the Bacillus, which has an equal chance for any part of the body, and so they re-act (e.g. the phagocytic action is not powerful enough to destroy the microbe without the cells themselves being damaged in the struggle,) in the form of joint disease and the Bacillus finding itself at home, multiplies, and the case goes on from bad to worse.

Curiously enough Tubercle attacks most of the sites selected by rheumatism with the exception of the valves of the heart, but here the mechanical effect of the blood current may explain this and the difficulty of the microbe in thus effecting a hold. That poisons are excreted by the various serous cavities is shewn by the excretion of alcohol into the arachnoid in Delirium Tremens, which is similarly selected in cerebral rheumatism and the virus of hydrophobia, which is however not "proved" to be causally connected with a microbe.

Looked at in the tubercular -analogy - light there is much to favour the idea of a rheumatic microbe. Burney Yeo noting the difference between the relative frequency of the left side of the heart being attacked in Rheumatism as compared with the right, thinks (Clinical Therapeutics) that the poison circulating in the venous blood becomes "oxidised" after contact with the fresh air in the lungs and its virulence thereby becomes increased. This is hardly in keeping with general ideas as to

the effect of the oxidation of poisons, the effect being mostly to reduce them to simpler and less noxious terms, and does not explain the primary - at least in most cases - the effect on the joints.

In Influenza, one sees practically a sub-acute rheumatism along with catarrh etc., and the same is seen in many specific fevers, namely a joint poisoning. Where the patient is ripe for the attack, the exposure of a single joint or limb to chill may be followed by local and general rheumatism. Here the local chill stops metabolism and imperfectly oxidised products are formed and produce local poisoning.

One would almost think that a Ferment (unorganised) was formed which gradually got distributed by the blood and was excreted into and poisoned other joints. The only trouble about the "increase" of the ferment, except in the joint first attacked, is that this would lead to the assumption of a microbe which would produce it (if organised). Or a minimum quantity of the poison in certain individuals may be able to affect a number of joints.

Just as the tox-albumins of the Diphtheria Bacillus separated from the microbe may in certain animals produce deadly effects in relatively minute doses. Were the ferment the result of a microbe in rheumatism its indefinite multiplication would be easy enough.

The pain may be explained in Rheumatism both by the direct action of the poison on the nerves and by the inflammatory swelling produced as a result of the irritation of the poison and by the direct inflammatory swelling when cold has caused local pain in a joint. Stretching of the fibrous tissue of a joint is excruciating as is well known in the case of sprain.

The Reason why Rheumatic Pain is worse at Night and in acute and chronic cases increased by the warmth of the bed has never been satisfactorily explained. It may be due to the inflammatory ^{re}action after exposure to cold, or excretion of the poison, being aided by the dilatation of the vessels by the warmth and the subsequent stretching of the fibrous

tissue. The effect of an injury of a joint in a person in the pre-rheumatic state is explicable on the same lines as in the case of cold causing rheumatism, viz., hyperaemia, formation of waste (inflammatory) products, perhaps the ferment, and subsequent swelling and pressure of the fibrous tissue following the dilatation of the lymphatics which excrete the poison into the joint.

The Rational Indications for Treatment in Rheumatism.

Since its introduction by Buss of Basle in 1875 Salicylate of Soda has come to be considered "the" specific for rheumatism if not the only useful drug for the purpose. In actual practice one finds in many cases it fails and in the recent Influenza epidemic where I have generally seen between forty and fifty cases a day, here in Dewsbury, I have found it of little benefit in alleviating the severe pains and fever of the "rheumatic" variety. In ordinary acute rheumatism one has frequently to ring the changes on the most likely of the host of drugs which have an anti-rheumatic reputation.

Taking them all round one arrives at the conclusion that their action is chiefly eliminant which is aided by the copious draughts of fluids generally given with them in acute and sub-acute attacks. If one believes, as I have tried to point out, that the "poison" to be dealt with is of the nature of a "fatigue" product, then the rational treatment is to wash it out the system, as in the laboratory experiment, or if possible, to neutralise it in loco, generally a more difficult matter. Hence it would not matter greatly except as a slight difference of degree which drug one employed, so long as it would aid in this washing out, the point being to convert the patient for the time being into a Filter at the same time supporting his strength. The success of the "expectant" treatment with good nursing gives point to these remarks. (vide Garrod. p.196)

A rapid and rational mode of treatment in very acute cases e.g. the cerebral and hyper-pyrexial type would be the intravenous and intracellular injection of saline alkaline fluids 9 Na Cl Na_2HCO_3 1 in each

90 parts of boiled water) one or two pints of the filtered fluid being injected every two or three hours. if necessary, and unless the perspiration had been very copious the abstraction of say ³8-10 of blood might be beneficial although generally the sweating in these cases has drained the patient free-
of
ly of fluid and the blood must be [^]considerably elevated Sp. Gr. By this means the blood and tissues would be rapidly "washed" and neutralised and I have a strong presumption that the effect would be highly beneficial. The writer has had recently a case of hyper-pyrexia (T. 108°F.) complicated with pericarditis in which the ordinary available methods were used in vain and the patient died. Should a similar case occur again in his practice he will certainly try the effect of the above treatment which if it do no good can at least do no harm.

Small injections of distilled water etc., have been made hypodermically into the tissue around and also into the joint cavities with reported prompt relief of local pain (vide Garrod) but these

experiments have little bearing on the above radical method which is based on the same mode of treatment of Cholera where there is a general poisoning as well as the local intestinal symptoms, and in what has been done in cases of Septicaemia and Collapse following severe abdominal operations.

The ordinary treatment of Acute Rheumatism.

When called to an ordinary case of Acute Rheumatism with say temp. of 101-103°F. - Perspiration - blankety tongue - joints slightly swollen and with just a shade of pink - constipation and general mental perturbation - after reassuring the patient, set about nursing arrangements and eliminant treatment. In the first place a dose of calomel is given the danger of chill when the bowels are moved is small if there are the proper nursing arrangements. Salicylate of Soda is prescribed in 20 grain doses every 3 hours to commence with and at night a sedative draught is given, at the same time the patient being made to drink freely of aerated soda and potash water. Care should be taken that the flannel night dress when thoroughly drenched with perspiration is changed for a dry hot one as frequently an exacerbation takes place from the rheumatic patient taking a chill in a damp night-dress and the same remarks apply to blankets which should be used instead of sheets if the patient can bear them.

The joints should be carefully wrapped in the first instance with lint wetted with Lin. Belladonnae and Lin. Opii and the whole enveloped in wadding and bandaged.

A few years ago on Davies' Plan I used to use blistering fluid to the joints and the results were excellent as regards removal of pain; but having on one occasion by accident rather extensively blistered the whole front of the leg I have not continued, but can strongly recommend, the plan.

In every moderately severe case it will be well to apply a mustard leaf on the precordium, not to the extent of blistering it but to keep up slight counter irritation, the whole being covered with cotton wadding. As the patient often gets pains in the finger joints from exposing his hands, a thin pair of woollen gloves is recommended.

When the Salicylate does not show signs of being followed by lowering of temperature or relief of pain as in some cases, the patient should be put on Quinine Gr V. every three hours. The quinine dissolves quite sufficiently in the aerated potash

or soda water used from syphons; at the same time a mixture of Potassium Bicarbonate Gr. XX. in gentian infusion and glycerine may be given. Practically it is found out that cases that do not yield to Salicylate in a reasonable time, turn out to be what I generally call Chronic Sub-acute cases which will be touched on later. Celery water is much advocated popularly in this part of the country and acts as a good "Wash out" if taken in sufficient quantity. It contains relatively a larger proportion of saline matter. A seidlitz powder may be given on alternate mornings.

The Diet for the first few days should consist largely of milk diluted with barley water or soda water or the milk may be mixed with an equal quantity of water with a little Bicarbonate of Soda and a few grains of salt. I have seen no bad results produced from allowing the patient a little weak beef-tea. As I have noted in the opening remarks Dr Percy Wilde considers a high temperature favourable by "burning up" (oxidising) the waste products and systematically treats his patients by means of "hot air". I cannot speak of this plan from experience. Gener-

ally speaking, in four days the most acute symptoms, pain and temperature, have subsided and it only remains to follow out in part the old dictum "Bed and six weeks" gradually feeding up the patient and putting him or her on Salicylate of Soda and Citrate of Iron and Ammonia or Quinine which makes a pleasant mixture.

As the patient becomes stronger he may have a few hot baths. Generally it is found that the pains return after these and I have often heard patients at Harrogate remark that this was the effect of the "bringing out" of the poison. It is probably due to the swelling of the recently inflamed tissue of the joints and acts like the warmth of bed in the more chronic cases. Care should be taken that the bedrooms (and especially the beds) at the place selected for a change, if indulged in, are warm enough. Frequently one meets with cases that from inattention to little points like this become chronic as the result of slight recrudescence at Harrogate and similar resorts. The amount of

exercise should be moderate, as in these convalescents fatigue readily brings on pains and they are very liable to chills from the ease with which they perspire on exertion.

The Chronic "Sub-acute" form of Rheumatism.

This is a very troublesome variety of rheumatism to treat. It may follow acute attacks although it is generally seen as a sub-acute form from the first - temperature varying from 99°-101°F. and varying irregularly from day to day. In this form the pains often disappear during the day only to return with great severity at night, gradually reducing the patient's strength. He becomes worn out and mentally depressed. In these cases one often finds the smaller joints of the fingers and toes affected so as almost to suggest Gout. Here the Salicylates and other things seem nearly useless. The whole system is in a chronic state of poisoning, the patient if possible must be got up, in spite of his aches and pains and sent to where he can get abundance of fresh air, the risk of travelling being nothing

to the long drawn out confinement to bed. A six-months stay in the country is strongly recommended in these cases as the only means of getting rid of this form of Rheumatism and if patients can be advised to take a nine months' or year's fresh air cure it will distinctly "pay" and the chances are that the pains and run-down state may be got rid of for good. Otherwise what it means is one or more sub-acute attacks every year with sleepless nights and a miserable existence in the interval.

The patient must be encouraged to take as much quiet exercise ^{as possible}, short of fatigue, keeping to a good plain nutritious diet and, in keeping with the atonic state of the stomach, taking a fairly dry diet at meal times, but when the stomach is empty say an hour before meals taking large draughts of pure fresh water. If the patient is not taking a chalybeate water one may prescribe the alkaline tartrate of Iron with a bitter, and on getting up the ordinary "Magnes. Sulph. Sod. Sulph. and Ginger" in hot water twice a week. In some cases I have tried the pure Tinct. Iodi, as

recommended by Dr. Mortimer Granville (Med. Annual 1887) who fancies it is the only way of "breaking up" the Uric Acid which he considers the cause of the trouble, in doses of M. V to X three times a day. Patients, however, have fancied it was "lowering" and in these cases this effect has to be constantly combatted.

As regards the places to send these patients e.g. a so-called "Watering Place" , Harrogate, Buxton, Bath, etc., or one without baths and waters I consider it a matter of no great importance. Having had good opportunities of studying Harrogate, which is a good specimen of the "English" style of watering places, I believe that the efficacy of a residence there depends not on the special ingredients dissolved in the waters (for which vide Dr. Oliver's Book) and taken internally or as baths, but on the quantity of fresh air taken, the very dry bracing air with its great oxidising and possibly ozonic properties, the very dry soil and the open country around, in fact in the spring Harrogate is rather too cold and bracing for these cases and I generally recommend a less elevated place during the early months of the year.

The patient should have a hot bath every other night and he may add to it salt, soda or a little liver of sulphur to satisfy any hydropathic fancies. The main point in the so called "Aix" treatment is the douching and I have found that there is a great difference between the mere passive effect produced by immersion in a hot bath and the "Aix" plan of douching the joints. This latter as well as having the general effects of the hot bath, increases the local circulation in the rheumatic joints and probably leads to the carrying off of the inflammatory deposits the results of the previous acute and sub-acute attacks.

One of the first effects of the fresh air cure is the disappearance of the anaemia and along with this a return of appetite as the stomach regains its tone. The dragging muscular pains and joints aches gradually disappear and the patient becomes more and more able to stand exposure to wind and weather.

As regards the use of Lithium Salts - In a few cases I found that its effects were very slight

and quite insignificant as compared with the experimental results obtained by adding Lithium Carbonate to Uric Acid in a test-tube. Haig and Burney Yeo mention the same fact and Haig in his Uric theory considers that Lithium Salts form a compound with Sodic Phosphate in the blood and so reduce the alkalinity of the latter. When it is impossible for the convalescent from either acute and sub-acute rheumatism to proceed to the fresh air cure, the administration of Cod Liver Oil with Iron much diluted along with Liq-Arsenicalis and as much quiet exercise as possible forms a moderate substitute. Make the patient a "filter" taking care to let him wash himself out with drinks when his stomach is empty. When Cod Liver Oil disagrees, good streaked bacon does well and attention as above should be given to the bowels on alternate mornings with the sulphate mixture. The tendency to chills after exercise from the free perspiration in convalescence after acute rheumatism or in the chronic sub-acute form should be systematically provided against. In every case the stockings should be changed at mid-day and it will be found far safer when coming

in warm and perspiring from a walk or ride to strip at once and after rubbing down to change into dry things. This is much better than waiting until the body cools as then there is more real danger of chill and although not quite in accordance with the popular idea is practically found the safest if the bedroom is not too cold. I need only add that the clothing should be warm, and during the cure, the patient, to quote Shakespeare should if possible "Let the world slide".

In the above pages a rapid glance has been taken at the various views and possible explanations of the rheumatic process and the more one considers it the less one becomes inclined to be dogmatic as to there being any one definite cause or poison such as Uric or Lactic Acid - at least until there is ocular evidence of the presence of a microbe or microbes which as in Ague may cause the symptoms - which can explain even moderately satisfactorily all the symptoms of Rheumatism. I have attempted to shew that a poisoning may occur by Leucomaines Ptomaines and possibly

by tox-albumins (which Kanthack considers may only be Toxines mixed with albumin and which is not a necessary part of the poisonous compound, B.M.J. March 9th. 1895. p.533.) as the result of lowered nutrition giving rise to imperfect oxidation (from the anaemia) of the materials used in the production of muscular and gland energy, the accumulation and absorption by the joint- and valve-etc.-serous surfaces, of the poison, giving rise to joint and general symptoms, the last straw to the accumulation of poison tolerated being added in the shape of fresh poison following imperfect oxidation after a chill or extra exertion, the general store of rheumatic poison being often augmented by the ptomaine compounds absorbed after imperfect digestion and passing into the general circulation unchecked by the Liver aided by local retention in the shape of constipation or by in some cases the excretory power of the Kidneys being at fault. One is thus led from the consideration of these views as also from actual experience to believe that the idea of a

"specific" in the proper sense of the word for rheumatism is chimerical (until a "specific" cause such as a microbe is discovered which would simply be a more definite *causa causans* in the origination of the Ptomaine etc. poison) and that the only rational method of dealing with the effects of the above poison is by free elimination by actual physical means such as by diuretics etc. ^{and} by more purely chemico-vital as by oxidation. These may be practically carried out by the saline injections, which I suggest for very acute cases and in ordinary cases in the shape of the administration of the salicylates and general alkaline compounds in dilution with plenty of water, the oxidation by the fresh air cure aided by improving the oxidising powers of the blood by treating anaemia by chalybeates together with attention to the stomach and bowels and preventing re-accumulation by warning previous sufferers against the effects of excessive exercise bodily & mental, the whole being summed up by saying "Look after the nutrition and the Rheumatism will take care of itself".

P.S. Since finishing this paper I have seen a further number of cases of Influenza which were practically indistinguishable from Acute Rheumatism and which would seem to be in favour of a similar cause - microbic - in both. The difference, however, is that in the Influenzal Rheumatism I have found Salicylates of little value and now treat all cases by Quinine with very satisfactory results.