ATIOLOGY

OF

ACUTE RHEUMATISM

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A Thesis
on the
Aetiology of Acute Rheumatism.

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Introductory

Of all the diseases which the physician is called upon to treat those various conditions indiscriminately spoken of as "rheumatic" are amongst the most common. The name rheumatic is of great antiquity, and until two hundred years ago, was used vaguely in connection with a large number of diseases. Poisson, a French physician, was the first to discriminate between gout and rheumatism, and restricted the word to something like its present meaning. Nevertheless even at the present day the word "rheumatic" is at times loosely used and in the minds of many has about as much meaning as has the word "calamity" in chemistry. Doubtless the disease may make its appearance in various degrees of severity; it may be complicated by many circumstances peculiar to the victim or his surroundings.
and thus increase the confusion and uncertainty in its diagnosis. Probably there are many diseases, rheumatism amongst the number which leave the person a prey to indefinite articular pains, uneasy aching of muscles, and structural alterations of tissue which we now, despairing of a more accurate diagnosis, speak of as "rheumatic". There is however one specific febrile disease around which all our ideas of rheumatism converge, and in the symptoms of which they find their best expression. This disease is acute and subacute articular rheumatism or Rheumatic fever.

Indefinite and uncertain as has been our knowledge of this disease the information in our possession is even less capable of explaining the causes and the conditions which bring it about and until recent times it
Treatment has been most unsatisfactory. Rheumatism is a grave, a distressing, and viewed in the light of the amount of discomfort, disablement, and indirect loss of life which it brings about, a terrible disease. So that anything which tends towards the elucidation of the absence of causes of this malady will we may confidently hope contribute greatly to the lessening of the evils it produces. Physicians have looked forward with an explanation of the origin of Rheumatism. The most acute and clinical observers, the most learned of physiologists, and the most inquiring and observant of pathologists, have all laboured to remove the mystery which envelopes the etiology of Rheumatism; and yet despite their labours, an explanation
has not yet been given which,—such is the opinion of the most recent writers—satisfactorily explains the phenomena of this disease.

I venture in this Essay to give some account of these labours and while studiously avoiding probability, not in the opposite direction by failing to narrate clearly and correctly the work which has been accomplished.

I will deal serially with those theories which I conceive to be of consequence either on account of their historical interest or the ingenuity and sincerity of their Exponents, and in concluding I will try and give an account of that belief as to the cause of this disease which has arisen in my mind as a result of my Education, observation, and Enquiry.
Exposure to cold — The Lactic Acid Theory.

The first rational explanation of the symptoms of rheumatism vouchsafed by the early writers arose from a belief that there existed some important connection between the disease and climatic conditions. They concluded that rheumatism was due to 'exposure to cold,' and regarded this as a sufficient explanation. Against this view the experience of physicians, travellers and others was set up as affording negative evidence in this direction. After the experience of travellers in the Arctic regions as well as in the tropics furnished no account of any prevalence of rheumatism (Captain Parry's Voyage for the Discovery of a North-West Passage to Colonels S.J. Mitchell in Tropical Australia quoted by Dr. W. Fuller in "Rheumatism, Gout & Sciatica") as indeed no vague and uncertain appeared
The relationship of acute rheumatism to climatic conditions that it was concluded must lie something more than the mere application of cold. The more the disease was studied and compared with other conditions which manifested symptoms of fever and sweating, the more likely it seemed that in rheumatic as in other fevers there was some initiated state of the blood. Budd, Holland, and Dr. Chomel, all seem to have been forced to this same conclusion—that there was an impaired state of the circulating fluid, and so the question arose: if the blood is poisoned, by what substance is it poisoned? whence comes this poison? It was a commonly received opinion that the touch of the food was intimately concerned in the maintenance
at animal heat and that it had to pass through an intermediate chemical state before it was finally resolved into Carbonic acid and water. This intermediate substance was believed to be lactic acid and so the idea was well received which Dr. Dow had advanced in a lecture before the College of Physicians. He suggested that the poison in Rheumatism was lactic acid. Just as uric acid in excess led to gout so lactic acid was the cause of Rheumatism. This theory has always been a favourite one and has in one form or another been advanced from time to time by many physicians. They believed that lactic acid is a substance actually elaborated within the body and they suppose that it is partly oxidized into Carbonic acid and water to be excreted.
and partly excreted unaltered by the skin; and that when as a result of chill the excretory power of the skin is arrested, lactic acid accumulates in the system and set up the disease which we call acute articular rheumatism. The excessive sweating so characteristic of this disorder was regarded as the efforts of nature to get rid of the poison.

Dr. Todd in his Croonian Lectures elaborated this theory and remarked, "It is no wonder that as lactic acid is imperfectly excreted through its natural channels, in consequence of the influence of cold checking perspiration and is too freely developed in the alimentary canal it should accumulate in the blood and become eliminated at every point." Dr. Headland (On the Action of Medicines pp 85-90) thought
it was more the excessive production than deficient oxidation and elimination, of the lactic acid within the system, and it is this view which Fuller especially favours. Dr. Fuller concluded that the disease does not arise from the local agency of cold (as is p.29) but from the excessive production and accumulation of lactic acid that it directly poisons the system, by arresting the natural secretory power of the skin intensifies the poisoned condition already existing.

The frequency of the disease, the ease with which its identity could be determined, proved it prominently before the notice of careful observers and thinkers. They recognised a relationship between the onset of rheumatic symptoms and cold and damp weather especially the latter and they arrived at the
Conclusion that the primary cause of the condition was chill. The chilling of a sensitive area led to the arrest of the excretory power of the skin and thus the affected matter, ordinarily excreted, were retained and led to the occurrence of the symptoms of acute rheumatism.

 Fuller varied this theory somewhat as I have already indicated, but I do not think that many physicians will be inclined to agree with him in disdaining altogether the connection of climatic conditions with the presence of acute rheumatism. Lactic acid was suggested as the poisonous matter and the sour acid smell surrounding the rheumatic patient encouraged them in this hypothesis.

It is difficult to believe that a chill lasting for a limited time and the consequent arrest of excretion by the skin would
result in flooding the blood with enough of this lactic acid poison to extend the morbid state over many consecutive weeks; and which frequent and repeated sweatings "efforts of nature to get rid of the poison" should quite fail to wash off. Dr. Fuller seems to have appreciated the fact that his original theory was incapable of explaining how lactic acid could possibly to produce acute rheumatism and materially changed the theory by affirming his belief that acute rheumatism was due to lactic acid poisoning, this substance being manufactured in Greece and with difficulty got rid of or not got rid of at all. Up to this point there seems to be no experimental evidence in support of this theory and the clinical evidence alone
yields no definite information for its support.

The evidence of the presence of lactic acid in the sweat is very slight. Faure (Arch. Gén. de Médec. [3], p. 298) has detected lactic acid in the sweat, but many others who have tried to do so have failed. Salamonn denies that lactic acid is ever present in the sweat.

(Charité Annalen (für 1879), 50 p. 159)

Salamonn again made very careful analyses of the venous blood of acute pneumonia and quite failed to detect lactic acid. The detection of lactic acid in these experiments depended upon the characteristics of the crystalline "ginner" salt and it is said that Salamonn might easily have overlooked it. Still the detection is readily enough by his method in cases of "encéphaloclasia".

There are three varieties of lactic acid: 1st. sarco-lactic acid, which
dentrocolatory, 2nd, ordinary lactic acid as formed in the souring of milk which has no action on polarised light, and 3rd, Ethene lactic acid.

Lactic acid is present in the body in exceedingly minute quantities and physiologists are quite at variance as to the source from which it is derived.

Applesauce test for lactic acid is one capable of detecting the merest trace of this acid and we certainly ought to have experimental confirmation of the presence of lactic acid in great areas in the body (as this theory of rheumatism supposes) if we can satisfactorily apply it. The test is as follows:

1. A solution of dilute Ferric Perchloride and Carboxylic acid is made: 10 cc of a 4% potassic of Carboxylic Acid
   20 cc of distilled water
   1 drop of his Ferric Perchlor (P. R.)
On mixing a solution containing a mere trace of lactic acid with this violet solution it is instantly turned yellow.

(Chemical Phys. and Pathology by Dr. W.D. Halliburton p3 409)

In 1858 Dr. Benjamin Ward Richardson published his essay on "The Causes of the Coagulation of the Blood," an essay which had gained the Ashley Cooper Prize awarded by the College of Physicians. In Appendix III to that essay he gives the results of some experiments which he made upon animals. He had been experimenting upon the effect of alkalis and thought that it would try the effect of acids. In consequence of the theory of D. Proust supported aci
was by the arguments of
Revue, Todd, Williams and
Fuller, and also of Headland
and Spencer Wells that lactic
acid is probably the materia
morbi of acute rheumatic
fever he determined to ex-
periment with that acid.
He used a 10% solution of
lactic acid. For some prelim-
inary experiments he first
gave it by the mouth
but the results were
"unsatisfactory" and he
supposes that "The effect
of the acid was destroyed
by the digestive process.
He could not inject the
acid into the veins because
it brought about vagal stim-
ulation". We felt that any effect
upon the endocardium
would be merely looked
upon as the result of
an irritant." It is evident
therefore that he was anticipating "some effect upon the endothelium." He therefore injected the fluid well diluted with water into the peritoneum. In Experiment they attempted to inject dilute lactic acid into the peritoneal veins of the cat but this failed because it caused coagulation. Therefore at once closed the wound in the neck and ligatured the vein and then opened the abdominal cavity and injected seven drachms of a 10% solution.

He states that the animal breathing increased in rapidity so that the cat was left all night, and that the next morning it was found dead. On examining the cat...
loody they found no sign
of inflammation nor could
they detect any trace of the
injected fluid. The liver
was pale externally but
internally it was congested.
The kidneys also, and the
spleen were congested.
The lungs were free from
congestion but were of
a colour too much
approaching red to be
natural. The blood in the
veins and right side of
the heart was of a dark,
jaundiced, cheesy character
like that described when
lactic acid is merely added.
The right side of the heart
was healthy. The mitral
valve was greatly thickened
red and adherent.
On the surface of the
valve there was a
small fibrinous deposit.
which adhered to its position by means of an underlayer of a white glutinous fluid the like of which they afterwards found could be made to exude from the valve on puncturing it with a needle. The surface of the pericardium lining the left ventricle had an intensely vascular appearance resembling in fact bright red velvet. The pericardium was dry and slightly injected. The joints and the brain were normal. In the next experiment 2000 cubic centimetres of a 10 per cent. solution of choline chloride was injected into the peritoneal cavity of a full sized terrier dog. The dog's chest had been examined previously with a stethoscope and found normal. Next morning the dog was languid and seemed to care little for food. There was no pain apparently and no fulness on pressing the abdomen. The heart beat was quick sharp the second sound being apparently accentuated.
the fist obscure. The respiration was natural. At 11 a.m. another half ounce of the acid was administered with an ounce of water. The operation produced no expression of pain. At 3 p.m. Dr. Snow, Dr. Bradly Hewitt, S. Wells, Spencer Wells, and Rogers saw the animal. Dr. Richardson goes on to say - He distinctly made out that the second sound of the heart was prolonged and the first obscure but we could not decide as to the sickness of a bloat. The splanchnic vessels were greatly congested. The animal lived with much the same symptoms till early the following morning when he was found dead but lax and quite warm. On examining the body they found the cadaveric rigidity less marked than in the cat. No injury to the intestines had resulted from the operation. The peritoneal lining
showed no trace of injection or of inflammation but was rather paler than usual. The mucous surface of the vasa terinae was in part vascular. The liver was rather shrunken and pale, dense in structure, but not inflamed. The spleen was normal. The kidneys were rather congested and their capsules were usually injected the vessels being beautifully delineated. The bladder was contracted and empty and its lining membrane was pale. The bronchial membrane was free from congestion but the lungs were vascular dark and condensed except the surface of the upper lobe of the left lung which was of a bright red contrasting strongly with the other parts. The pericardium contained no trace of fluid but was unusually dry and like the capsule of the kidney had its vessels finely injected. Held up to the light the membrane had a bright vermillion tint.
The heart was distended on both sides but most on the left side with clotted blood. The endocardial surface on the right side was natural but one segment of the tricuspid valve was swollen several lines beyond its natural size from oedema and was of a bright red colour. The thickening was irregular. The pulmonary semilunar valves were natural. On the left side the endocardial surface lining the ventricle was throughout of a brilliant red colour having a velvety appearance and contrasting strongly with the lining membrane of the right ventricle. The segments of the mitral valve were also thickened oedematous and of a bright red colour while around their free margins were several beads varying from the size of a pin head to that of a millet seed. The aortic valves were of a deep red colour, their free borders were thickened
and everted. There were no fibrinous coagula. The muscular structure of the heart generally was more than usually red. The external vessels of the brain were congested but the brain structure was natural. Several joints were laid open but presented no abnormal appearance.

In the next experiment (over) (on account of mistakes in the feeding of the animal) no result of consequence was obtained. The animal died in 12 hours. There was intense peritonitis. The lungs were natural, the heart contained coagula, one segment of its tricuspid valve was adherent and red. The pulmonary values were normal and on the left side there was no injection of the endocardial surface nor any lesion of the mitral value but the aortic values were thickened at their free margins. In this case Dr. Richardson says, it will be observed that too
Large a dose of the Acid was injected with the result that the fluid was not absorbed and that rapid peritonitis occurred with but little cardiac mischief. Experiment III. A large dog was experimented upon. Examination prior to the experiment satisfying the operators that both heart sounds and the respiration were perfectly normal. One ounce of acid diluted with two ounces of water was injected. On the second day the animal was very unwell. It was shivering and the skin was very hot. Pressure on the abdomen caused no pain. Heart's impulse was great, the 2nd sound was intense and accentued the 1st sound somewhat obscure. There was no pain in the joints or limping.

On the 3rd day the symptoms were much the same. Heart's impulse was considerabler. The first sound was quite lost, the second
was sharp and accented. There was no pain on pressure over
the abdomen. An ounce and a
half of the acid was now injected
with four ounces of water.
On the 4th day the tremblingness
was very great; there was no
affection of joints nor evidence
of peritoneal pain. The action
of the heart was now irregular.
The second sound was still accentuated
and accompanying the first sound
there was a soft murmur fluttering
as if were the second sound.
On the 5th day the joints continued
unaffected and there was the
same absence of peritoneal pain.
The action of the heart was
more irregular than before
and there was a loud
systolic bruit audible over a
wide surface. The second
sound was accentuated as
before. There was a slight
hacking cough. Once again
an ounce and a half of lactic acid were injected into the peritoneum without any manifestation of pain. During the day, the symptoms continued unaltered and in the course of the night death took place. After death the peritoneal cavity was filled with a brown glutinous fluid having an alkaline reaction which had escaped as was found from a small wound in the intestine opposite a point where the last injection had been made and evidently produced by the needle. There was peritonitis but it was much less intense than in the last case. The abdominal viscera generally were congested. The lungs were natural but in the left pleural cavity there was an exudation of lymph. The pericardium embraced the
heart closely and the vessels were injected. The membrane was generally reddened and on the superior anterior part just over the left ventricle there was an evaporation of plastic lymph of about the size of the surface of a shilling. At this point the membrane was a little thickened. The right side of the heart was filled with dark coagulated blood and all the segments of the tricuspid valve were thickened firm and of a palish white colour. The anterior segment of this valve was thickened many times beyond its normal size and was bound down by recent fibrous adhesions to the side of the heart. The endocardial surface lining the ventricular wall was natural as were also the pulmonary valves. The left ventricle contained a small
quantity of treacly blood. The segments of the mitral valve were thickened several times beyond their usual size and were of a pale red colour and their consistence was firm and their free borders were choked with beads which in colour was pearly. On examination with a common lens the pale appearance was seen in this valve as in the aortic to have arisen from exudations of lymph beneath the lining membrane. The aortic valves were red, thickened on their free margins and lacerated. The brain and its membranes were normal. Some of the larger joints were opened and in the ankle joint the lining surface was a little more vascular than in the others.
Experiment cccxxiv

One ounce of lithic acid diluted with two ounces of water was injected into the peritoneal cavity of a healthy dog. In this animal, on the 3rd day in addition to the symptoms noticed in the previous experiments there appeared to be "pain in all the limbs and shrinking whenever he was touched." On the 4th day the injection of the sclerotex was intense and the pains in the limbs had disappeared except about the shoulders. On the 5th day the animal was greatly improved and seemed much relieved in consequence. On the 6th day the pain in the shoulders had returned and seemed most marked on the left shoulder so that when the animal was led about he rested the left leg entirely and moved about on the other three. He was exceedingly thirsty and the skin was hot. The first round of
The heart was again obscure and the second strongly accented. The animal took his feed freely. By the 7th day the pain in the limbs had passed away and he moved about on all four legs. A fistulous opening in the abdomen had remained where the last injection of the acid had been made and so another ounce was injected. On the 8th day the pain in the left shoulder had returned, the animal again moved on three legs. The first sound of the heart was slow heard as a blowing murmur. The second sound was sharp as before. Another ounce of acid was injected into the peritoneal cavity by the fistulous opening but most of it returned. On the 9th day the dog was better and free from pain in the limbs. The heart sounds were clearer but were not well defined.
The urine was neutral. Again 2 ounces of the diluted lactic acid were injected and on the following day, the 10th, the animal was dull and very feeble. The action of the heart was quick and tumultuous but both sounds could be distinguished. There was a slight hacking cough. On the 11th day the animal remained much the same and passed large quantities of very acid urine. On the 12th day the symptoms remained much the same. On the 13th day the application of the stethoscope produced great pain. The heart beats were quick and audible the first sound was obscure and the second accented. The animal was feverish that evening and winced very much when his shoulders were touched or the stethoscope applied. On the 14th
day the general symptoms were the
same and a loud and pure
systolic bruit was now heard.
On the 15th day the condition was
unchanged and on the 16th day
the systolic bruit was very loud
and harsh. On the 17th day
there was a return of the pain
in the limbs together with considerable
exhaustion. When touched the animal
was angry, but could not stand
or move without great pain or
difficulty but when food and
drink were placed conveniently
near him he ate and drank
greedily. During the whole of
this day he could not move
his limbs and cried when they
were touched. The systolic bruit
was still well marked.
Early in the morning of the 18th
day the tenderness of the limbs
entirely disappeared. He ran
about freely and took his
food and drink as if quite well.
At 10 a.m. Dr. Todd saw him to
confirmed the fact of the supra-
luminal. There was however some
little tenderness over the region of
the heart when the stethoscope
was applied. A few hours later
he suddenly relapsed into his
previous condition becoming
violently enraged whenever his
limbs were touched. He was
freely purged and passed large
quantities of very acid urine
and seemed greatly relieved after
this. On the 19th day the dog was
more lively and ate heartily.
He was frequently purged.
On this and on several days
there was about the animal
a peculiar sour smell which
scented the room very quickly.
By the 21st day the animal was
much better, the purging ceased
and he ran about without any
difficulty. There was still pain
when pressure was made over the
precordial and the loud systolic bruit could still be heard. From this onwards recovery took place rapidly. After the lapse of three weeks the dog was narcotized to death with the fumes of 
lycoperson giganteum and a post-mortem examination made of the body in the presence of several of the gentlemen who had seen him during life. All that remained of the endo-
cardial lesion was a series of fibrinous beads along the borders of the mitral valve. The beads were firm and pearl-like. One of the aortic valves was slightly thickened and quite opaque. The joints showed no pathological evidence of the disease to which they had been subject in Experiment \textit{?????}. The same method was employed with like results. The joint lesions occurred on the 3rd day and the first sound of
The heart was scarcely audible. The second accented - the last beats were sharp and irritable. The experiment number COCOVI the last of the six was made on a puppy and proved a failure.

These then are the six experiments made by Dr. R. W. Richardson and I have quoted them at length and with full details very nearly in the language used by Dr. Richardson in his essay. In order to complete the account of these experiments I will still further quote from this essay for on page 389 Dr. Richardson, disclaiming all bias wishes it to be understood that his remarks ought to be considered as those of a mere listener on and open to the same criticism as those of a stranger. He then goes on to say that the first inference to be deduced from these
Experiments is that lactic acid has the power when existing in an animal body in excess of producing a class of symptoms attaching themselves to the fibro serous textures and which regarded in all points of view are essentially the symptoms of acute rheumatic inflammation. The symptoms are obviously dependent upon the presence of the poison. Experiment which is in fact a series of experiments tells this fact definitely. By giving time for the elimination of the poison the symptoms could be made to disappear and on the supplying of the poison the symptoms returned; the alteration was as marked as night and day and was as clearly in the order of natural sequence. This was also what the old school men would call "metastasis" of
symptoms, now one joint suffered
how another and again the
heart. Thus it is clear that
lactic acid has the powers of
a rheumatism producing poison
but that in the human subject
it is the veritable cause in
every or any case of Rheumatism
is only an inference. As yet
there are wanting many
experiments to make the matter
a demonstration. But this
seems to me clearly proved
that if not lactic acid yet
some acid of an analogous
character is always the cause
it has yet to be learned by
experiment what formic, acetic,
butyric acids will do under
the same circumstances. Again
if by after Experiments it should
be proved that various acids
have the same influence or if
it should be proved that lactic
acid is the one simple representative
of this class of special poisons the experimental argument will have to move back another step towards the primary cause of the disease. The next question will be where in the body and in what manner is the poison generated? 

In Rheumatè Endocarditis occurring from the formation of the poison in the body itself the left side only is affected as a general rule. Hence I infer that the chemical change whereby the material morbis of acute rheumatism is produced is complete in the pulmonic circuit; that in the respiratory and the acid quality of the poison is produced; that thus formed the poison is carried by the arterial circulation to the affected or decomposed or elimination of both; and that it does not
resembles an acid to the
veins but simply as a product
which admits of re-transformation
in the pulmonary circuit into the
acid state. If this view be true
it is easy to connect further
the origin of the Rheumatic
Diseases with the digestive
system or with the arrest of
special secretions. In either
case the basis of the poison
may be generated and carried
into the returning venous
current hence into the
pulmonary circuit and finally
after oxidation into the left
heart and through the arterial
system:—

The original source of this lactic acid
theory of Rheumatism is to be found
in the generally accepted views as to
formation of glycogen by the hepatic
cells and its ultimate fate.
In 1848 Bernard found sugar in
The liver after death. If however an animal's liver was examined immediately after death it contained not sugar but glycogen. This glycogen shortly after death becomes converted into sugar.
The whole subject of the fate of glycogen is at present not clearly determined and according to Professor Fodor remains even now pretty much as Bernard left it: "In the liver glycogen is formed but this again slowly enters the blood stream as sugar. It is then comparatively slowly oxidized into Carbonic acid and water. What the intermediate products are, are uncertain." (Landor's 'Studying Physical Medicine')
It is believed however that one of these intermediate products is Lactic acid. Lactic acid is present in the tissue in health, so that if it produces disease it can only be when it is
present in excess. This is evidently what those believe
who have fixed upon lactic acid as the poison in rheumatism.
Professor Wise H. Porter says in a lecture delivered to the
New York post-graduate school (International Clinics—Pentland,pp.94)
"In all manifestations lactic acid is a
by-product developed from
the incomplete oxidation of
the protid molecule contained
in the food stuffs, introduced
into the system either as a meal
albumin or as a vegetable
protid." And again "the problem
of the tissue assumes a vicarious
action and induces at these local
points the formation of lactic
acid from the protid
molecule."
In all this Professor Porter
advances nothing of the nature
of experimental evidence.
I have noted however that
There is no evidence of the presence of lactic acid in gout.

Professor Halliburton in his book on Pathol. Chem. speaks on the subject of the cause of rheumatism and states that it is a non-bacterial disease. He states that the presence of lactic acid has never been demonstrated and that at any rate we possess the negative information that it is not uric acid, and so rheumatism and rheumatoid arthritis are easily distinguishable from gout, where undoubtedly sodium urate is the poison (Barred).

How to me Dr. Richardson's opinions are very extraordinary. Despite of the fact that the accuracy of his observations has been called in question, by Müller (Königs Med. Jahr. 1869, p. 277)
one cannot overlook the fact of experiment no cccciv. I fail to see that the accuracy of his observations has been impeached and he has most reliable witnesses to confirm the phenomena which he describes. Still as Dr. Richardson himself says the experiments were imperfect. We want many more such experiments to assure us of that. These results are irrevocable and want evidence that the lactic acid was absolutely free from any form of impurity, that it is the only acid capable of producing these effects. Dr. Richardson's experiments are in themselves almost conclusive that lactic acid as such is never present in the blood stream. Further on in this essay I shall have occasion to refer to the experiments of others. Injections of this fluid were just as certainly followed by indi-
Dr. Richardson claims that lactic acid is developed in the pulmonary circulation.

This point Dr. J. Maelagan regards as the crucial point in Richardson's theory and combated at length in order to show that such a statement is in direct opposition to what is known regarding the formation of that acid.

I have repeatedly used Auffmann's test in trying to detect lactic acid in the blood and sweat in rheumatoid patients and have failed, although I have no difficulty in obtaining the change from the violet to a solution that turns yellow when the acid is as dilute as 1 drop in 1 pint of distilled water and in very dilute Liebig's extract of meat.

In 1871 Sir Walter Foster reported two cases he had under observation.
They were cases of diabetes in which he had administered dilute lactic acid medicinally. On March 8th he took 10 minims doses of lactic acid (3% aqueous). One was doubled on morning of 9th, on afternoon the patient suffered from pain in the joints of that day. When no lactic acid was taken the pains gradually left the patient, they returned as soon as he recommenced to take the drug. The joints only were affected, and the condition appeared to be identical with that presented by Rheumatism. According to some people these experiences constitute a striking evidence in support of the theory that Rheumatism is due to the presence of lactic acid in excess. I think we are entitled to demand further confirmatory evidence of these effects of lactic acid. It will be remembered too that Dr. Richardson in his experiments quite failed to obtain any result when the lactic acid was given by the stomach, and lastly, there is the objection that we have no certainty of the purity of the acid administered nor any proof that it is the same acid as that produced in the body by disturbed metabolism.
Neuro-Chemical Theory:

A theory as to the cause of Rheumatism which has attracted a great deal of attention is that propounded by Dr. W. Latham. An account of this will be found in the Lancet Journal for 1885, p. 1119. Dr. Latham quotes the brilliant results obtained in the treatment of Rheumatism by the use of Salicylic Acid and starting from this point he endeavours to trace the action of the drug and at the same time point to a series of morbid processes which bring about the symptoms of Acute Rheumatism. "This beautiful theory," says Dr. A.B. Barrow in his Treatise on Rheumatism, "which Dr. Latham builds up step by step upon the results of physiological experiment, clinical observation and chemical research, constitutes a great advance upon the old lactic acid theory, by no means the least of its advantages being that it allows for the continued formation of the morbid product as long as the attack lasts; a provision that is wanting in them.
in the purely chemical theory, except that form of it held by Dr. Fuller who believed that lactic acid was a product of mal- 
assimilation." Dr. Latham believes that 
salicylic acid is a true antidote to the 
poison of Rheumatism and he asks, 
what is the poison? Now Dr. Latham 
sets out with the view that one of the 
chief features in acute Rheumatism is 
the formation of large quantities of 
lactic acid, but he does not adduce any 
evidence of this, nor does anyone else 
produce evidence of this excessive 
production of Lactic Acid. It is 
suggested certainly because physicians 
cannot bring themselves to believe 
that lactic acid theory of the origin 
of Rheumatism in its simplest 
form except upon the ground of its 
continued excessive production 
within the system. Dr. Latham 
however, while conceding this much that 
lactic acid is greatly concerned 
in the phenomena of Rheumatism 
yet he does not consider that it is
The chief factor concerned. He says, "it is the excessive formation of glycocine and uric acid in the tissues that develops the symptoms of rheumatic fever and salicylic acid cures the disease by combining with the antecedents of these bodies and preventing their formation."

Uric acid he believes results from the conjugation of glycocine with urea or its antecedents forming the compound

\[ \text{CO} \{ \text{NH}_2 \}
\text{CO} \{ \text{NH} \}
\text{CO} \{ \text{NH-CH}_2 - \text{COOH} \} \]

This substance dehydrates would form

\[ \text{CO} \{ \text{NH-CO} \}
\text{CO} \{ \text{NH} \}
\text{CO} \{ \text{NH-CH}_2 \} \]

and this combining (in the kidney) with another molecule of urea \( \text{CO} \{ \text{NH}_2 \} \) forms ammonium urate.

\[ \text{H}_2 \text{O} + \text{C}_5 \text{H}_7 \text{N}_4 \text{O}_3 \cdot \text{NH}_4 \]

Ammonium Urate

This is excreted "but a portion overflowing from the kidney into the general circulation," and
meeting with soda in the blood is converted into sodium urate. Diabetes results he says from the failure of the liver to effect the metabolism of glucose and Gout results from the imperfect metabolism of glycerine. Glycocholic acid is a constituent known by the bile, into the duodenum during digestion. In this it becomes broken up into Glycocholic and Cholic Acid, the glycerine is reabsorbed into the liver, undergoes a "transformation and a corresponding amount of nitrogen is subsequently excreted as urea by the kidneys". Now Dr. Cathcart thinks that any agent which through the influence of the nervous system will depress the functional activity of the liver will interfere with this chemical process. That instead of the glycerine being transformed into urea it becomes transformed into uric acid and this latter product is a poison with a specific power of acting upon supposed...
central in the central nervous system which have under its control the rotation of the joints.

Salicylic Acid is excreted in the urine in the form of salicyluric acid, a substance which when heated with fuming hydrochloric acid is broken up into glucuronic and salicylic acid.

\[
C_9H_7NO_4 + H_2O = C_6H_4\{\text{HO} + C_2H_3\{\text{NH}_2\}\text{COOH} + C_2H_3\{\text{NH}_2\}\text{COOH}
\]

Salicyluric Acid  Salicylic Acid  Glucuronic

Therefore, if salicylic acid is administered it combines with an essential constituent of urine acid and so prevents its formation.

Dr. Latham now passes to show how by the combination of salicylic acid in the liver either with methane cyan-alcohol or by its conjugation with glucuronic, when it is reabsorbed by the liver, the formation of urine acid will be lessened. Referring to a prior publication Dr. Latham explains that he believes that albumen is a compound of cyan-alcohols united to a nitrogen nucleus. These cyan-alcohols have been synthetically prepared with the result of the lowest in the series CH_2\{OH
\[
\text{CN}
\]
From these cyan-alcohols the corresponding amide bodies, bencine alamine and others have been artificially prepared. Judging from analogy he would expect that, the action of ammonia on this the twelfth of the series of the cyan-alcohols a cyanamide would be formed and this hydrated with acids or acetalic would form amido-acid glyco-cine.

\[
\text{CH}_2\left\{\text{OH} \div \text{CN} + \text{NH}_3 = \text{CH}_2\left\{\text{NH}_2 + \text{H}_2\text{O}ight.\right.\text{Cyan-alcohol}
\]

\[
\text{CH}_2\left\{\text{NH}_2 + 2\text{H}_2\text{O} = \text{CH}_2\left\{\text{COOH} + \text{NH}_3\right.\right.\text{Cyanamide}
\]

\[
\text{glyco-cine}
\]

Creatine is derived from this same cyan-alcohol attached to the benzene nucleus and by changes in it and condensation a new is formed and also the next cyan-alcohol higher in the series. \[
\text{CH}_2\left\{\text{OH} \div \text{CN} + 2\text{H}_2\text{O} = \text{CH}_2\left\{\text{COOH} + \text{NH}_3\right.\right.\text{Cyan-ethyl alcohol}
\]

\[
\text{Lactic acid}
\]

By the hydration of this lactic acid would be formed.

\[
\text{C}_2\text{H}_4\left\{\text{OH} \div \text{CN} + 2\text{H}_2\text{O} = \text{C}_2\text{H}_4\left\{\text{COOH} + \text{NH}_3\right.\right.\text{Ethan-ethyl alcohol}
\]

\[
\text{Lactic acid}
\]

It is with this substance, this methene cyan-alcohol that salicylic acid combines.

Methene cyan-alcohol and cholic acid would
form glyco-cholic acid

\[ \text{CH}_2\{\text{OH} \rightleftharpoons \text{CN} + \text{C}_24\text{H}_{14}\text{O}_5 = \text{C}_{26}\text{H}_{43}\text{O}_6 \]

Methane cyanide + cholic acid = glycocholic acid

and cholic acid acted upon by alkalies is converted into cholic acid and glycocine.

Supposing that salicylic acid does combine with the cyan-alcohols we should get a substance having a formula of the same character as albumen

\[ \text{CH}_2\{\text{OH} \rightleftharpoons \text{C}_6\text{H}_4 \{\text{OH} \rightleftharpoons \text{COOH} = \text{CH}_2\{\text{CN} \rightleftharpoons \text{H}_4 \{\text{COOH} \rightleftharpoons \text{COOH} \]

and the CNOH of this albumenoid body would be transformed into CONH amido body and thus salicylic acid would be formed.

\[ \text{CH}_2\{\text{OH} \rightleftharpoons \text{CNOH} + \text{C}_6\text{H}_4\{\text{COOH} = \text{CH}_2(\text{NH} \rightleftharpoons \text{C}_7\text{H}_6\text{O}_2)\text{COOH} \]

Salicylic acid

Dr. Latham now goes on to discuss the physiological effect of cold and heat applied to the skin. How heat in a warm-blooded animal up to a certain point cold increases and heat diminishes bodily metabolism, but that
in the cold-blooded animal, the application of external cold diminishes and heat increases metabolism, and under the influence of urea poison which paralyzes the end-organs of muscular nerves a warm-blooded animal is affected by external heat and cold in the same way as are cold-blooded animals. In order to explain these phenomena he quotes Foster who says we can best explain these results by supposing that under normal conditions the muscles as we have seen, contribute so largely to the total heat of the body, are placed by means of these motor nerves and the central nervous system in some special connection with the skin so that a lowering of the temperature of the skin leads to an increase, while a heightening of temperature leads to a decrease of the muscular metabolism. Further through the matter has not been fully worked out the centre of this thermo tonic mechanism appears to
be placed above the medulla oblongata, possibly in the region of the pons varolii. When water is given the reflex chain is broken at its muscular end, when the spinal cord is broken the breast is nearer to the centre. (Thermotonic Centre see Appendix note p. 92)

Now Dr. Latham's explanation of the phenomena of a feverish cold is this:

Either by cold or wet or both the sensitive cutaneous areas are irritated, and the vascular areas of the muscles are contracted; but at the same time through the vasomotor system the splanchnic vascular areas and vessels of the muscular areas are dilated on account of paucity of their vasomotor controlling mechanism, until the parasites there is a weakening of the power by which the albuminous molecules in the muscles are held together instead of the cyanate alcohol being transformed into cyanamide.
and glycocine, according to the explanation already given, they become detached from the leucine body and from one another and by hydration are changed into glyco-cholic and lactic acid substances which are more easily oxidised than the amido bodies. After the primary contractions due to an application of external cold, wet, or damp, the cutaneous areas would have their blood vessels dilated and the vessels in the muscles would become contracted and this contraction would be increased by the glycocine or resulting ureic acid, which latter substance would so in gout stimulate the thermo-tropic centre, so that less blood would now go to the muscular areas, and the albumenized particles would be no longer specially apt to split up as heretofore. It would undergo the natural changes, the
acid being excited by the kidneys and the lactic acid by the skin. This would be the state of affairs in a person in good health who was chilled or greatly exposed but if from any cause, debility or exhaustion the vasomotor system is weakened and its vasoconstrictor tone impaired there then would be a more complete dilatation of the reflexly influenced vessels of the muscles. The vessels of the muscles would be paralyzed and would very slowly and sluggishly recover their tone, and in addition to this the vasomotor centres in the central nervous organs being weakened, continuously the stimulation of the forming glycogen and uric acid failing to produce contraction renders the exhaustion still greater. Owing to the loss of tone in the vasomotor system the engorgement
of the deep parts and the muscles of the body continues. The open alcohols of the albuminous molecule loosen from the benzene nucleus, undergoing hydration and oxidation and continuously forming urine and lactic acid as the final product in the disturbed metabolism. The urine and the lactic acid accumulate in the system and produce injurious effects. The lactic acid especially dilates the smaller arterioles and stimulating the sweat centers. At least as the centers controlling the vessels of the muscular areas continue to be stimulated in this paralytic condition the vessels will dilate to their utmost limit. The molecules of albumen will split up into glycoeholic and lactic acids from which rapid production of heat results to bring about the hyperpyrexia of rheumatic fever. Not only this
but the uric acid will in this state of matters affect the joints by irritating the thermolanic centres just as Dr. Latham believes it does in gout. In the young the vasomotor system is peculiarly susceptible to irritation, as from cold, and reacts in the manner above described. If a similar attack occurred in an elderly person it would set up not paralysis in the vessels of the muscular areas, and the consequences of great action and internal congections.

In gout says Dr. Latham the nervous mechanism of the liver is at fault and glycocenic is converted into uric acid. In rheumatism the vasomotor system controlling the muscles is weak, and therefore glycocenic is formed, and later lactic acid from the cyan alcohol of the albuminous molecules in the muscles.
The joints are affected by uric acid irritating the centre for the nutrition of the joints and also by the local effect of lactic acid dilating the capillary blood vessels.

Salicylic Acid then according to Dr. Latham combines with methyl cyan alcohol a substance which results from the splitting up of albuminous molecules and which if not interfered with by salicylic acid would go on to form glyceral and lactic acids.

It is therefore necessary, if we are to accept this theory, to believe that the two diseases gout and rheumatism, presenting very different clinical and pathological features, arise essentially from the same cause. That in rheumatism, as admittedly so in gout, uric acid is the poison, but that it is supplemented, in the case of rheumatism, by the presence of lactic acid which has a slight
action on the tissues where we are to suppose it is manufactured. We are asked to believe that in the guity attack the nervous system controlling the liver is weakened in its power, and that as consequence the changes which should occur in connection with the substance glycine are modified: that instead of being excreted as urea, uric acid is produced and as poison the system. It doubt in great the uric acid is formed in excess and poisons the system and the excessive uric acid formation might with more reason be attributed to the excessive supply of nitrogenous material and to the impairment of function of the hepatic cells consequent on this. If the chilling at the surface is itself sufficient to account for the loss of vasomotor control in the muscles, and subsequent series of events as described by Dr. Latham, it should surely be possible to
excite this condition of rheumatism artificially (Chill see Appendix Note I p 1.)

In the beginning of last winter little crediting the statement that rheumatism
was the result of chill or cold I ventured to expose six attendants
young active healthy men in what I
conceived would be the most favorable conditions to bring about rheumatism
according to the idea that this disease
was the result of a chill.

I placed my six men, at various times
extending over several days, in a small
room heated by a gas stove and
by one gas light. An opening was
made in the outside door six inches
by two, and the door opposite
was allowed to remain open
in such a way as to create a
very rapid current of cold air.
Through the opening in the door
the temperature of the room varied
from 70 - 80° F and the outside
temperature was 52° F, and the
weather wet, foggy, and muggy.
Two of these young men were almost types of that build and temperament or diathesis generally supposed to favour rheumatism. They agreed with Laycock's description of being well built and well nourished, having round, clear, complexions, sound strong, healthy, abundant hair, digestion and nutrition good, pulse hard, blood pressure high. The attendant was comfortably seated and his bare arms and shoulders, some needless, and foot and ankle upon, placed comfortably supported opposite to the hole in the door, with the cold current of air blowing directly against the exposed part. They remained in this position no more than less than an hour and in several instances considerably over an hour. The results were entirely negative and in none of these young men as far as I could ascertain was there and subsequent occurrence...
of pain or unpleasant after effect whatever. Now on the other hand when myself a person apparently in robust health but unhappily the frequent victim of rheumatism and with a very marked rheumatic inheritance, was exposed in a like manner for half an hour suffered from a sharp attack of rheumatism affecting the exposed arm and shoulders and confining me to bed for some 48 hours, and in like manner a young patient a rheumatic subject whom I had induced to submit to the experiment also suffered but in a less degree. I cannot but think that if Dr. Latham's theory was a correct account of how the phenomena of rheumatism are brought about, it ought to be easily verified by such simple although perhaps dangerous experiments. Indeed if such a simple explanation as
his really is more sufficient to account for even a "feverish cold," apart from rheumatism; the former condition as well as the latter very serious affection would be infinitely more common than is even the case now. We should expect to find also that young persons especially those debilitated by exhausting disease or by overwork or other like causes would at all times if exposed to cold and wet fall victims to acute rheumatism. Indeed so manifold are the circumstances under which this chill would arise, (and in those very persons to who are mostly debilitated), that the cases of acute rheumatism frequent as they are, compared with other forms of acute disease, would be infinitely more common. Moreover we would expect to find that in many
of the cases of the incomplete rheumatism — what we can only call the "feverish cold" state of acute rheumatism according to Dr. Latham's theory — the reaction whereat commences the diverging point of "feverish cold" and acute rheumatism would occasionally fail to be established and we should find that condition which at first was a "feverish cold" rapidly assuming the character of acute rheumatism. But do we find this so? Do we not rather find that acute rheumatism is from the outset a well defined disease readily distinguishable from a "feverish cold" with strikingly specific features which are unmistakable. A person who has suffered from rheumatism may get a "feverish cold" which may be accompanied
by pains, feelings of soreness and acheing which he recognizes as like to those of rheumatism or may not. In the same way a person who has suffered from aigue may get cold and may or may not suffer in addition from symptoms different to his neighbours and which he attributes to the influence of the aigue person still remaining in his body.

With our present knowledge of pathology it certainly is difficult to believe that a disease so striking in its symptoms as is acute rheumatism so sharply differentiated from all other affections should have its origin in the imperfect reaction or failure of the vasomotor nerves controlling the arterioles in the muscles.
to react, after the application of one external chill.
The articular lesion is only one point in the acute rheumatic attack. The great source of danger in inflammatory affections of the heart. This Dr. Latham refers to as the acute but beyond this reference from beginning to end of his paper he makes no allusion to the implication of the heart and does not offer the slightest indication of the manner in which the heart is implicated.
If it should be suggested that the heart condition is due to the influence of a poisonous acid in the blood stream, as the lachrymal acid theory pure and simple would suggest why need we imagine a nervous toxic centre to be influenced by this disease in order to satis...
factorably explain the articular lesion and if on the other hand we adopt the idea of a thermodaphic centre irritated by uric acid so as to interfere with the nutrition of the joints would they further suggest that there is some irritation of a portion of the central nervous system to account for the altered condition of the heart and if not, why not?

What is the actual pathological change in endocarditis?

Dr. Sims Woodhead in his manual of Practical Pathology p. 146 thus describes the appearance of a section through one of the vegetations met with in endocarditis: On examining a section with the low power (50X) the base of the growth appears to be made up of a great number of small round cells which are deeply stained.
with most of the reagents
that may be used. This mass
of small round cells is continuous
with a similar layer found
infiltrating the endothelium in
the neighbourhood of the growth
On the surface of this mass
of granulation tissue (sporid
is nothing more or less than this)
is found some granular
looking debris which with picric
carmine takes on a yellow
colour (or in some parts pink)
and scattered through it
are a few pink stained cells
whilst further out still is a
very thin layer of fibrin
which is usually granular
and somewhat opaque, and
stains a pinkish colour
with picric carmine. (Under 14)
High Power (300) the cells of the
endothelial tissue are seen
to have undergone very
rapid proliferation and it
is from this source that the tissue composing the greater part of the growth arises. The proliferation is so rapid and so great that just as in the case of a granulating wound the mass of cells press beyond the level of the endocardium and a small projection is the result. The yellowish mass on the surface is made up of two factors degenerating cells and coagulated fibrin both of which may be readily distinguished by the picric carmine and other staining methods.

Of the microscopic appearances of the joints Dr. Cornil and Ranvier in their Manual of Pathological Histology 2nd edition 1881 p. 463 — as quoted by Dr. A. B. Garrod say, — "When a section is made per-
perpendicular to the surface of the cartilage in the parts where it is possible, a microscopical preparation is obtained in which is seen an active proliferation of the deep and even of the calcified layers of the cartilage. The multiplication of cells and formation of secondary capsules is in no way different from that which has been described in the superficial layers only as the primitive capsules of the middle zones are disposed in lines and are compressed one against the other. They become elongated and form cylinders perpendicular to the surfaces of the cartilage whereas the superficial lenticular capsules become filled with secondary capsules, form rows which have a direction parallel to the surface. The cellular
proliferation is constantly accompanied by segmentation of the ground substance lying between the primitive capsule and this segmentation causes strie in the preparations parallel to the long axis of the primitive capsule. It results from this that in the deep layers this segmentation is perpendicular whereas in the superficial layers it is parallel to the surface. There appears to be some confusion about the condition of the synovial membrane some observers merely recording a hyperaemia, others such processes as lead to the formation known as pus. Probably those who have made this latter statement are in error. Nevertheless the description of the histological appearance presented are sufficiently
Distinct from the joint affection in locomotor ataxia, Dr. Byrom Bramwell (Diseases of the Spinal Cord) says that the pathological changes in locomotor ataxia are very similar to those met with in chronic rheumatism (rheumatic arthritis) but that they differ in certain important particulars. How the changes which are described by Dr. Woodhead are being the precise histological alterations in Rheumatic Endocarditis and by Mr. Rennett Cornil in the joints are as inflammatory character and point to the presence of some local irritation I cannot find any analogous condition arising from change in the central nervous system alone. These latter, when they occur, are of a necrotic and atrophic character, but it is more than probable that many such.
lesions of joints described to the nervous system and known as tropho-neuroses are not entirely due to the loss of neurotrophic influences. For example conjunctivi-
may result from section of the 5th nerve and hence the inflammation is manifestly set up by External irritants. In its state of lowered vitality a tissue may be no longer capable of resisting those External irritants which may reach it through the blood or lymph stream. As D'Coats mentions (Manual of Pathology p. 73) a simple inflammation of the bone marrow set up by Caustics may be changed to a very severe one by simply feeding the animal on pointed food. These tropho-neuroses are said to be of three distinct kinds. One effect of the loss of this trophic influence is to retard or arrest development of
seen in infantile paralysis. A second class of consequences resulting from decay or atresia of atrophic influences may be spoken of as degenerative and atrophic changes e.g. the changes in the joints in locomotor ataxia, and bedsores in many nervous diseases. — I would like to note in regard to this latter that to my mind the occurrence of these bedsores is a parallel condition to the conjunctivitis I have referred to, for it is within my own knowledge that scrupulous care and the most careful precautions will greatly lessen the number of bedsores in an asylum.

It is very evident also that the Commissioners in Lunacy are of the same opinion since they are careful to enquire into these cases. The slightest crease in the sheets, the irritation
of a bread-crumb against his arm, or a drop of water is insufficient to set up this destroying process. But after nine years' experience in the Wellington Asylum in the East Riding of Yorkshire and in most of the County of Norfolk I cannot help having the fact impressed upon my mind that when bedsores occur, in spite of the most careful and experienced nursing, some apparently trifling unilateral being overlooked, has acted upon the arm and tissues of a lowered vitality to set up a process of rapid destruction.

Dr. Alexis Thompson showed before the Edinburgh Med. Chirurg. Society a specimen of "Osteomata of the Skull following the Distribution of the 5th Nerve" (Transactions Edinburgh Med. Chirurg. Society Vol 5, 1841) in his account of the
Specimen the states it is that of a lunatic in the Norfolk County Asylum and he said: "The association of bony overgrowth in the area of distribution of a nerve with an enlargement of its various branches naturally suggest altered nerve influence as the cause of the overgrowth and one would therefore appear to be justified in classing the morbid condition which the specimen presents with other so called trophic neurones. Dr. Jonathan Hutchinson to whom the skull was sent for comparison with one in his possession, (with his characteristic scientific insight), was of opinion that the examination of one such specimen afforded sufficient grounds for the inference that there exist certain fibres in the 5th nerve which provide over the
innervation of the bones to which
they are supplied and which
when their function is "galled"
may cause overgrowth and
lumour formation throughout
their area of distribution.
Dr. Alexis Thompson then
concludes that we have a
3rd class of tropho-neuromes
in which we find overgrowth
and new formation of bone
resulting from an "Elation"
of the tropho-neuromes influence,
and although he does not
say so it is of such a nature
that Dr. Latham maintains it
would have us believe, are the
articular lesions in rheumatism.
I venture to suggest that the
evidence of this third class
of tropho-neuromes is really very
slight, and it seems to me
that to arrive at such a lofty
conclusion on such very
scanty evidence is quite
injustifiable. The man from the skull was taken was insane, his father was insane; — he was an epileptic and a lunatic. I do not think it would be a very extravagant idea to suggest that a ‘poison’ acting on the nervous system so as to produce changes in the great nerve centres might also aid in producing changes in those parts to which the nerves from such centres were distributed. An altered condition of the blood and lymph might act on the nerve centres as well as on other parts, and by joint anaesthetics injuring the nerves going to a part might render the part especially liable to be influenced injuriously by the same ‘poison.’ In other words that it is quite as reasonable...
suggest that the explanation of the cause of the secondary lesion (if indeed it be a secondary lesion) in such cases will be found in a study of the etiology of what for the time being we regard as the primary cause. The etiology of such changes in the nervous system as lead to epilepsy, locomotor ataxia, and so on, is by no means as definitely known as we can ignore the origin of the nerve lesion itself and starting from this as the primary condition build upon it a hypothesis as to how the lesion can bring about the secondary one. Indeed in conclusion the very existence of trophic fibres and centres is not at all so clearly established that we can allow D'habans
in his theory to assume their existence and proceed without pointing out that after all it is an assumption.

It is beyond my purpose to do more than this and to point out that even so recently as the last International Congress at Rome Professor Bizzozero of Turin (British Med. Journ. April 7, 1894) in his address on the "Growth and Regeneration of the Organism" in regard to this very subject of trophic fibres said: "The existence of such fibres has been doubted by many and after closely following the experiments and discussions on the subject for more than 30 years, I am still unable to convince myself that they really exist nor does such hypothesis appear
to me to be required to explain the facts which fall under observation."

If this be the state of our knowledge it is unfair, and it is unwise, to build hypotheses upon hypotheses in this fashion. I would contend therefore, despite the ingenuity displayed by Dr. Latham, that his theory as to the origin of Acute Rheumatism rests upon no clear and satisfactory basis, either as regards the initiative and final stages of its 'neurotic element' or in respect of the chemical element. There is so much that is purely hypothetical, and so little that is established truth, that while it will ever remain an evidence of Dr. Latham's ingenuity and scientific attainments, it cannot be regarded as helping towards a satisfactory explanation of the
The Micromatic Theory.

This theory of the origin of Rheumatism has of late years received special attention at the hands of Bichat in France, and of Dr. Thomas John MacLagan in this country. Dr. John Haggart of Bath, whose genius led him intuitively to a true conception of zymotic diseases and their treatment, described in a paper entitled "Observations on the Population and Diseases in Chester in 1794," in 1809 is reported to have written that he "thought there were several analogies between anague and a rheumatic fever." Dr. J. MacLagan in his book (Rheumatism: Pickering & Co., London) after rejecting that theory which attributes Rheumatism to some disturbance of the metabolism, or...
excessive formation of lactic acid and the consequent poisoning of the system. Concludes that if the poison is not formed within the body, then it must come from without. These poisons which attack the body from without are of two kinds—contagia and university (page 60). Dr. Maclagan says the essential properties of the contagia are:

1. They enter the system from without.
2. They are reproduced in and given off from the system during the course of the maladies to which they gave rise.
3. The maladies are communicable from the sick to the healthy.
4. They have a fixed and definite period of duration.

But says Dr. Maclagan that no one of these things, and there is no evidence that its poison is reproduced in and given off from the system.
As the pneumatic poison does not possess any of these properties of a contagious there remains only the miasmata. But of the actual nature of these miasmatic poisons Dr. Maclagan affirms that we have no knowledge and can only guess at their nature from the effects they produce.

I do not consider however that we can permit Dr. Macclagan's assertion that "examination of soil, water, and air supposed to contain it" (the poison) "has given only negative results" to hold good to the same extent now as formerly. The investigations of Schlesing and Maury in 1877-78 and of Warington demonstrated in the soil the presence of microorganisms belonging to the Schizomycetes and other allied groups of the lower Fungi. Miguel, Rock, Koenkel, Arnold,
Pasteur and others have, by their researches, thrown so much light upon this subject of microorganisms in the soil that we cannot at all events assert that examination of the soil, air, and water has yielded negative results.

The special characteristics of the malariaal diseases Dr. Macleayan enumerates as follows:—

"1. They are specially apt to occur in low-lying damp localities, in certain climates, and at certain seasons of the year."

"2. Some people are more liable to be attacked than others."

"3. They have no definite period of duration."

"4. They are not communicable from the sick to the healthy.

And he further says "for we cannot fail to see that these are quite the attributes of acute rheumatism, Common in low-lying damp..."
localities, with especial partiality to certain climates, and showing itself more particularly at certain seasons of the year. "But it has no fixed period of duration; some people are more liable to be attacked than others, and its poison, we have seen reason to believe, enters the system from without." "But the analogy between rheumatism and malaria does not end here; a still further analogy may be traced in the symptoms." Here Dr. Maclagan goes on to say that malarial fevers are irregular in type and vary in their course, leave an impression on the system so that we get recurrence, and unless arrested may have a protracted course, in all of which features rheumatism bears a resemblance to the malarial fevers.
The origin and significance of the "maculifer" theory of the aetiology of rheumatism will be clearly understood from the conclusion of Chapter VIII of this book of Dr. Maclagan's which is headed "The nature of malaria": "Of the nature of malaria we have no certain knowledge. Its existence is known to us only by its effects. The diseases which constitute the most common and most striking of these effects are the various forms of intermittent and intermittent fever. How all these attributes of malarial diseases which Dr. Maclagan narrates are not unquestionably characteristic of malaria alone. Malarial diseases are especially apt to occur in lowlying camps. Localities says Dr. Maclagan, but this although true is not the whole truth. "In some cases, especially in sandy
plains at the foot of hills, the rain brings down very finely divided debris, and is filtered as it passes through the soil, so that each particle of sand becomes coated over or encrusted with a film of vegetable matter. If such a soil be subjected to alternate wettings and dryings and to the conditions of development of malaria may be present in great intensity, although there is not only no marsh but the sand is to all appearance dry and pure. (Parke’s Practical Hygiene 8th Ed. p. 15)

And again "Malarious marshes may occur at great elevations even 6000ft (Ecuador and Mexico)." (Ibid. p. 12).

1. That some people are more liable to be attacked than others is equally true of many diseases which Dr. Macalayan would include in the group contagia minuens of which let me quote from
Wm. B. Carpenter (Quain’s Diet. Med. Art. Predisposition to Disease p. 125-1) that "Of several persons equally exposed to the poison of cholera, some shall escape altogether, while others shall be attacked by choleraic disease; and of the latter, some may suffer only from diarrhoea; in others nothing more may be induced than vomiting, cramps, and acute watery evacuations, whilst in others the disease may develop itself in its full intensity, and rapidly proceed to a fatal termination."

3. It may be noted that in regard to pneumatome, to which Dr. Macleagane’s argument would lead us, how various writers have formed different, but distinct, ideas as to its duration when left without treatment, or with expected treatment. Sendamore thought three weeks, whilst the experience of Chomel was "De pneumatisme articulaire
eign quelque soit la medication
employee ne se termine jamais avant
le vingtieme jour ; and Sir W Gull
quote the duration of uncomplicated
and untreated acute rheumatism as
nineteen days.
4 Whether or not malarial diseases
can be communicated from the
sick to the healthy, must I should
think remain at present, an
undecided question. That they are
not virulently contagious or infectious
of course we satisfactorily know, but
that the poison is not communicable
by any way is I fancy not so certain,
and moreover this cannot be
taken as a feature peculiar to
malarial affections. The manner
of the communicability of many
diseases is only now coming to be
properly understood and the
most we know of
for example the more certain
we seem to become that there is
some contact at some
period or other of their existence, of an actual transference of infected particles from the sick to the healthy. Lastly he is not justified in distinguishing the contagia from the miasmata on the ground that the former confer immunity whilst the latter do not as even that most contagious of diseases scarlet fever may be followed by a second or a third attack (Dr. Williams Squire in Quain's Diet. Med. art. Scarlet Fever). Dr. Macalagan refers to the researches of Klebs and Tommasci-Candidi as given in the Alleg. Wien Med. Zeit. 1879 and their account of the bacillus malariae, but one feels in reading this book that to him the idea was a novel one and he does not seem to quite grasp the full significance suggested by such a discovery that if correct the distinguishing characteristics of the contagia and miasmata would with all
in importance, and that these
very terms would in the future only
serve to point out the historical
halting places in the study of
pathogenic organisms, and mark
the progress of our knowledge of
disease.
If the further study of these
poisons, which a former
generation of physicians could
only distinguish as febrile
and irremedial, should establish
the observations of Kebo and
Tommasi - Cordei of a bacillus
malariae or ascertain a definite
place in the etiology of malaria
for the haemoplasmodium
malariae described by
Marchiapapa and Credel, then
these distinguishing attributes
so much insisted upon by
Dr. Maclagan will surely
resolve themselves into differences
of type only and not establish
any real generic difference in
The essential nature of the poison of either splenic, typhoid, cholera, or malarial fevers (Organisms in Malaria, see Appendix, 146).

From such a theory of the origin of acute rheumatism as this propounded by Dr. MacLagan, it is but a step to that which recognizes in rheumatism a disease having analogies to those acute specific diseases of man which have been shown to be closely related to pathogenic bacteria.
The Germ Theory of Origin of Rheumatism:—

We are called to the bedside of a young man, who until some hours previously was in robust health and we find him utterly prostrated, racked with pain, oppression with a joint or joint, which renders him exquisitely sensitive to the slightest interference.

We find his temperature greatly elevated, and his skin pleasantly hot and covered with profuse perspiration. The whole phenomenon of an attack of acute rheumatism at once suggests that it is allied to those poisons whatever they may be which cause the specific fever.

As the rheumatic disease progresses we are astonished to find that the condition of fever passes away, and in a few hours we find the affected one cheerful, free from pain, and feeling comparatively well. But this remission is short-lived, for before long the symptoms return with all their former severity, and it may be
with increased danger to life.
We may search the whole category of diseases and find no condition so closely corresponding to acute arthritic rheumatisms as amongst those diseases where microorganisms are intimately associated with the cause of the malady, e.g. malignant anthrax, tuberculosis, swine plague, and ergotismas (Klein).
Again, not only do we find that rheumatic fever bears a general resemblance to the specific fevers but we occasionally find other affections (peryomia, gonorrhoea, and occasionally also dysentery, typhus, typhoid, and relapsing fever—Staphylococcus) producing pain and swelling in joints, as well as changes in the heart which are clinically identical with the symptoms of acute articular rheumatism.
There appears then to be reasonable grounds for supposing that rheumatism is allied to gyno-otic diseases and ki
leads me down to enquire what is the nature of the germ, as Farr called it, in these diseases. There is no occasion for me to attempt a defence of the germ theory of disease, for in regard to many, this is no longer a mere theory but must be regarded as amongst the unascertained facts of Natural Philosophy.

Syndall has defined a contagious disease as "a conflict between the person written by it, and a specific organism which multiplies at his expense, appropriating his air and moisture, disintegrating his tissues, or poisoning him by the decomposition incident to its growth." (Floating matter in air)

Dr. Hilton Tagge more guardedly says of these diseases "whether the contagious virus be a schizomycese or not, typhus and enteric fever, measles and scarlet fever, small-pox, chicken-pox andague are each caused by the invasion of a specific particulate contagion." — (Principles of Practice of Medicine, Tagge)
These are all general diseases and run a more or less definite course: they occur epidemically or sporadically, and in most of them the poison may be passed from the sick to the healthy; frequently one attack confers immunity from a second and several of them have been proved conclusively to be caused by a micro-organism.

What foundation have we for regarding rheumatism as allied to this group of diseases? All writers seem agreed that in some way or another there is a connection between rheumatic fever and climatic conditions; as Dr. Mitchell Bruce words it, "the disease has an intimate aetiological relation to weather, season and climate." Dr. Gubbett in a paper on "Influence of climate upon rheumatism" (Lancet Oct 26th 1883) has examined into this subject and marshals before
as the chief fact with which he is acquainted. His conclusions have reference more particularly to the admissions into the London Hospital from the annals of which institution he collected 2000 cases. He finds that the disease occurs most frequently in November and is rarest in March.

\begin{tabular}{|c|}
\hline
20.75 \% of the cases occurred in the 1st quarter \\
20.65 \% 2nd \\
25.20 \% 3rd \\
33.20 \% 4th \% \\
\hline
\end{tabular}

He gives a chart which shows at a glance this maximum in the fourth quarter of the year.

The continental writers give the minimum number of cases for the 3rd quarter and most of them find more cases occurring in summer than in spring. Thonessen, and De Briesen (Dielichty) do not believe that Rheumatism is influenced by weather at all. Edlefezen communicated an important
paper to the Westphalen Congress in 1888 setting forth what he alleged to be an intimate relationship between the number of cases of rheumatic fever and heavy rainfall. Professor Pel of Amsterdam has observed from time to time what he regarded as miniature epidemics of rheumatic fever and such occurrences have generally succeeded occasions when there has been a particularly heavy rainfall. Gabbett has also noticed this connection between the rainfall and rheumatic fever but he is not satisfied that "the relation is close enough to point to any necessary connexion." He does not think it most common in the coldest parts of the year, neither does he think it is more in the coldest. He believes it is associated with weather in which there is a low temperature combined with the greatest rainfall.
Now suppose we assume that acute rheumatism is dependent upon a 'specific particular Contagium' in not this relation to climate conditions. Exactly what we would expect. —Moisture and a certain temperature?!
The same thing applies to other diseases of a microbic origin. Measles is commenced in June and December, Scarletina in October, Diptheria in November and December, Typhoid in November (Quain's Hist. Med. p. 1128).

They require certain conditions to throw them into activity. A man may be frozen in the Arctic, or plunge through ice water & be wetted to the skin, and yet he does not become the victim of acute rheumatism. Why? Either because the disease element is not there or the conditions are not favourable for its growth. For though Epidemic Disease requires a special Contagium to produce it, surrounding conditions have a potent influence. Commonly
seeds may be duly sown but the conditions of temperature and moisture may be such as to restrict or altogether prevent the subsequent growth.

Loked at therefore from the point of view of the germ theory, the exceptional energy which epidemic disease from time to time exhibits in harmony with the method of Nature.' (Tyndall)

Such a theory explains satisfactorily the contradictory statements of the relationship which acute rheumatism has to meteorological conditions.

Several observers have noted that seems to be an epidemic form of rheumatic fever, and also that the severity of the disease varies in such different epidemics.

The mode of invasions of this fever and the arthritis changing its seat (metastasis of the older physician) are quite in accordance with the theory that rheumatism is a disease arising its origin to a living atom.
Miasmatic is a most vague term. Does anyone really know what he means by terms such as miasmatic and malarial? Can anyone really attain a clear mental picture of a miasm? I confess that in my mind the image formed is that of a mist and when I attempt to conceive what that mist is composed of it seems it must be molecular and that the molecules must be of the nature of bacteria. I quite fail to gain any conception of miasm other than this. The old notion of a miasm was associated with decay of organic vegetable matter. Yeast was decaying matter which multiplied and decayed. Why should not decaying matter act therein the body in the same way as yeast does? But what came of the idea when the yeast plant was shown to be not a dead rotten substance but a living plant.
It cannot make any great difference in our description or arrangement of specific diseases whether the germ be latent in water, air, or earth. To me the conviction returns with ever increasing confirmation the more I reflect upon the subject that acute rheumatism cannot be aught else than a disease arising from the development, under certain favourable conditions, of a microorganism.

It is said that the conditions favourable to rheumatism are those of cold and damp weather, combined with anything which tends to lower the vigour and vitality of the body. Now in the Furness district of Lancashire, where in the medical practice of some of my relatives, I had abundant opportunities of studying disease amongst a large iron-mining and industrial population, acute rheumatism was unquestionably rare. I had special opportunities
of studying the sickness amongst the 6,000-7,000 workmen in the Company's Naval and Armament Shipyard at Barrow-in-Furness.

Two other classes of workers in the Furness district are of interest in discussing this subject. The iron miners - the ore thereabout is the red hematite - and the men working at the black furnaces.

Where could we find men more exposed to wet and cold than the shipyard artisans and labourers? They endure in exposure of one kind or another and are the reverse of careful lives. The iron furnaces workmen are exposed to the western winds from the Atlantic on one hand and to the heat of the black furnaces on the other. The iron miners work hard below ground all day long enough working for many hours actually standing in puddles of mud and water.
They leave the foot mouth steaming with perspiration and walk miles home in all weathers.

And yet I affirm that amongst all these classes of men acute rheumatism is by no means a common disease. I have made diligent enquiries into this matter and I find that the opinion of medical men in the Furness district is that acute rheumatism is a comparatively rare affection. On first thoughts Barrow would strike one as a likely place for the propagation of a disease of such a nature as I am suggesting acute rheumatism to be, and certainly, if chill were the cause of rheumatism for it is much exposed, but perhaps better lies the explanation, because the town is openly built, its streets are well planned and well made, it is well drained, and lastly it is secured by...
The cleansing strong westerly winds, Dalton-in-Furness is seldom free from infectious diseases. It is the most fever-stricken place in the district but here this would seem to be due not so much to general conditions affecting the locality as to the gross carelessness, insanitary local conditions, and dirty habits of the people. It would seem, however, to be the opinion of medical practitioners in the Furness District that if acute rheumatism occurred more frequently in one part than another it was at Dalton. This fact, if the rarity of acute rheumatism among the red hematite iron miners is interesting, it is vouched for by doctors and also by the mine managers. It would seem that if the germ or poison exists in the soil it does not exist at any great depth or
else that it is only present in certain soils and that ground-water bearing district of Furness is not one of them.

Another matter of interest may be referred to here and this with regard to the mineral constituents of the soil disc has been said that iron bearing soils are malarial (Parker Practical Hygiene, Sir Ronald Martin Gals)

This is probably incorrect, indeed it is more likely that the very reverse is true.

Another point may be noted, that is that bacteria exist especially in loose gravel soil where there is air, moisture, and gential warmth and usually in proportion to the amount of decaying organic matter present in the soil, that they become rarer in the deeper layers, and are not met with so frequently deeper than 15 or 20 feet from the surface.
I have not been long enough
in this district to speak from
experience as to the prevalence
of rheumatism; nevertheless my
experience will be shown to be ap-
plicable. My enquiries amongst
medical men practicing in
the Thames Valley have elicited
the fact that acute or subacute
rheumatism is a common
disease hereabout. Here we
have a typically rheumatic
climate. It is a low lying
district with a loose gravel
soil. The temperature is mild
at all seasons. Vegetation is
luminant and all about the
district there are nursery gardens.
The influence of the river renders
the atmosphere excessively humid
flow if compared with this we
have the fact that the drainage
of the Twickenham district is
and has for long been in a
deplorable condition.
Explaination of the several distinct characters of the district is apparent.
That portion of the Thames Valley to which I have paid most attention is that which extends from Staines on the Middlesex side of the river to the County town of Brentford.
The population in this district is fairly represented as follows:
Brentford district 14,662
Staines 16,536
Hampton 8,374
Morphe 27,310
Ickleworth 26,754

The density of population in the district is about 2 per acre; and the number of persons per house is about 5.2. The number of inhabited houses in Twickenham is 3099. The number of families in these houses is 3,338. These figures then will convey in a general way the population of the district.
Although the cause of Mrs. Armstrong's death is difficult to determine, it appears that she had experienced pain in the head and back. She was found dead in her bedroom, with a single gunshot wound to the head. The bullet entered her brain, causing her immediate death.

Mrs. Armstrong had been known to suffer from migraines, which may have contributed to her death. The fact that she was found with a single bullet in her head suggests that suicide is a possibility. However, there is no evidence of a struggle or physical trauma that might indicate murder.

The medical examiner ruled the cause of death as 'suicide by gunshot'. The manner of death was listed as 'homicide', but there is no evidence to support this classification.

The investigation continues, and the family is left to grieve the loss of a beloved member of the family.
nuisances arise, generally from deep
sluggish flows, and insufficient
ventilation. In Hampton Wick
Dr. Gunther says there are probably
5,000 cesspools in the district and
each of them impregnates the
surrounding soil with its
contents. In consequence of the
Continuous pumping which
would place during the laying
down of drain pipes the subsurface
water was diverted from its former
course towards the pumping stations.
This led to the contamination of
many wells previously free from
impurity. The Infectious Diseases
Notification Act of 1889 has only
been adopted in the Brentford
district since 1892 and in the other
districts since 1890. However, as
yet authoritatively published,
are therefore incomplete.
Such as they are they are given
in Dr. John T. Luke’s report to
Sewerage, but that contains any important particulars.
I have a table curtailed for my purpose,
<table>
<thead>
<tr>
<th>Month</th>
<th>Age</th>
<th>Sex</th>
<th>Cause of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan</td>
<td>1</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Mar</td>
<td>2</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Apr</td>
<td>3</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>May</td>
<td>4</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Jun</td>
<td>5</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Jul</td>
<td>6</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Aug</td>
<td>7</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Sep</td>
<td>8</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Oct</td>
<td>9</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Nov</td>
<td>10</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
<tr>
<td>Dec</td>
<td>11</td>
<td>M</td>
<td>日病, 流行性肺炎</td>
</tr>
</tbody>
</table>

Note: The cause of death is listed as 日病, 流行性肺炎 (Japanese fever, epidemic pneumonia) for all the months.
From this table of the causes of
the deaths, the prevalence of infectious
diseases will be noted, and I will
call attention to the column which
gives the number of deaths
from heart disease.

In regard to statistics on deaths from
heart disease, Dr. Parkes mentions
Practical Hygiene (Ed. H.Thomson 1884) p. 576)

Among soldiers at home:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Per 1000</th>
<th>In 100,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carcinine</td>
<td>.727</td>
<td>8.31</td>
</tr>
<tr>
<td>Uncertain</td>
<td>.735</td>
<td>8.4</td>
</tr>
<tr>
<td>Total</td>
<td>1.462</td>
<td>16.71</td>
</tr>
</tbody>
</table>

This is excessively high, and is probably
the life and habits of the soldier.
It is mentioned that Dr. Lawson considered
the average mortality from heart disease
amongst civilians aged 15 to 44
was .45 per 1000. So that the army
had an excess of .277 per 1000
deaths from heart disease.

But compare even this high army
mortality from heart disease with
the table for Middlesex. The army
figure of deaths from heart disease is 1.462
per 1000. The deaths from heart
disease in the Brentford sanitary district is given at 23 and this is out of a total population of 14005. This would give us then a mortality from heart disease of \( \frac{23}{14005} \times 1000 \) 1.6 per thousand.

Now it will be found upon careful examination that this mortality from heart disease does not affect the whole administrative County of Middlesex. The total deaths from heart disease in this table for 1892 (D. L. R. L. Report) were 432 in a population of 593019 for the whole County which would give us a death rate from heart disease of about 0.7; but the deaths from heart disease in Brentford was 23 Twickenham 18, Hampton 5, Hampton Wick 4, Thames Ditton 17, Beddington 12. Total 76.

Now the total population is 54627.

And this would give us a death rate from heart disease in this particular part of the Thames Valley of 1.3 per 1000.
<table>
<thead>
<tr>
<th>District</th>
<th>Pop.</th>
<th>Deaths from Measles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brentford West</td>
<td>14005</td>
<td>23</td>
</tr>
<tr>
<td>Greenwich</td>
<td>16536</td>
<td>13</td>
</tr>
<tr>
<td>Hampstead</td>
<td>5968</td>
<td>18</td>
</tr>
<tr>
<td>Hampstead W.</td>
<td>2406</td>
<td></td>
</tr>
<tr>
<td>Graves</td>
<td>5117</td>
<td>10</td>
</tr>
<tr>
<td>Paddington</td>
<td>10595</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>54627</td>
<td>76</td>
</tr>
</tbody>
</table>

\[
\frac{76}{54627} \times 1000 = 1.3 \text{ per 1000}
\]

There is no comment made upon this fact in the reports. Great attention appears to be paid to the origin of the deaths under the various columns of zymotic diseases but heart disease so proceeds unnoticed which suggests rather that the dictates of passion may even invade the offices whence emanate such official reports.

It is a matter of common report in this district that Rheumatism is a disease of unusually frequent occurrence. The figures which I have quoted speak for...
Themselves. Well I have already stated that I have made inquiries of other medical men resident in this district.

Dr. Leeson, Bolton, Mr. Smith in Swotterham; Dr. Hardy, Rogers & T.C. Trottell in Wokington on Thames and Slaines districts have all given some attention to this subject on my behalf and are all of one mind as to the prevalence of acute and subacute rheumatism.

Unfortunately however there is absolutely no source from which reliable data as to the nature of the prevalent sickness can be obtained. I had thought over every available source, from which reliable information could be sought, and it struck me that most valuable information with regard to this subject might be obtained from the records of the Friendly Societies in
The district also was not forthcoming. I have however now got the cooperation of Mr. Deeney of Trier, a man who has promised me to keep a record of the causes of sickness to help me in obtaining definite data. Still there was something to be learned. Mr. Deeney is secretary to the popular friendly societies' lodges. They are all branches of the Manchester Unity Oddfellows Friendly Societies:

1st. The Loyal Lady George Lodge has a membership of 511; the average age is 24 yrs. males. The balance sheet for 1893 shows 0.89 sick days. This is an unusually heavy sick list for a lodge of young men.

2nd. Loyal General Wood Lodge
No. Members 118. Average age 34 yrs. males. Age sickness are
69.4% males. This lodge chiefly concerns Shepperton, it is considered a
very healthy lodge. The present is at the end of the Thames Valley and is less enclosed. Gould is said to be common amongst the members.

3rd. The Royal Princess Alexandra Lodge, Cop 124, Average age 24 years.

Now last year they had for their 174 members no less a sick list than is represented by the item 1000 sick days paid in the annual balance sheet. This is a very heavy for a young lodge. This lodge is chiefly at Teddington where in a district with a pop of 12,260 and only 140 deaths, Lt. Sykes' report tells us there were 22 deaths from Phthisis, 19 from 'Bronchitis - Pneumonia Family' and 12 from heart disease.

4th. Juvenile Oddfellows Friendly Society. This is practically representing the young boys of Wicroham.

The balance sheet shows that the number of members on the books on Jan 1st 1914 was 98. The number of sick
since Jan 93. was 88. and the total days sickness 967. This is a very considerable amount of sickness for a young lodge. Now as to causes of sickness Mr. Deneck has given me access to only recent club certificates but these are fragmentary, but they support Mr. Deneck's statements which are from memory. Of the 88 boys he knows they were all cases of 'general cold' & 'asthma'. One case only was scarlet fever. Quite a number of applicants for admission to the Lodges are rejected and invariably it is on account of heart disease. There are no figures, but experienced men say that a large amount of the sickness is from 'general cold and rheumatism'. Another old resident of this district a native of and he thinks 40% of the club cases of sickness are 'rheumatism or cold'. Anyway the evidence seems to me to put beyond all doubt the prevalence of Rheumatism in the Thames Valley.
A glance at the maps will show how we are shaded in & how the ventilation of this valley by the natural winds is prevented.

(See Map of England p 186 Appendix)

I present the following notes respecting the climate of this district. These records are taken from the report of the Committee of the New Observatory for 1892. The Observatory is situated about 1½-2 miles from Tweedmouth and the figures represent the climatic aspects of this district very closely.

Yearly mean temp 48°. Highest temp in Aug 69° & lowest in March 31°. The Nov. temp was 39°. Dec, Jan, Feb, Mar & April are all colder months than November.

Mean Annual Bar. Pressure 29.956.
Mean Vapour Tension .271

Annual rainfall is 24 in. The wettest months are Dec, Aug, & Sept in that order and then June, Nov, July. Compare this with Annual Average for England (30 in) and that of the Cumberland hills at 80 or more.

The prevailing winds are SW (on 56 days)
---W (63 days); NW (55 days); NW (42 days) E (42 days)
Exploration of the joint... I have had a case of subacute rheumatism at High St Stones St Margarets Twickenham which typicall illuatrates what I deem favourable conditions for the development of rheumatism.

W. N., a young man of 27 in fairly good health occupied during a part of the day a particular room on the ground floor. The room is warm and dry. He developed an attack of typical subacute rheumatism with pain swelling and effusion in one wrist and both ankles. He recovered but months afterwards when he returned to occupied this room he had again pain and swelling of ankles and general malaise. My brother also who had occasion to be in this room...
for some days was affected with a most unaccountable swelling and effusion into the right wrist associated with general malaise. My wife and myself frequently suffered from slight attacks of malaise often enough accompanied by severe pain in the joints. The room was dry and warm and comfortable to work in, we paid every attention in order to avoid draughts. Still the room maintained its reputation as unhealthy without any apparent cause. My brother's case was the most remarkable so impressed me that I determined to examine the room thoroughly. I took the floor up and ascertained definitely that there was nothing beneath to account for the danger to health which was caused by living in this room.
On examining one of the house drains which ran along the outer wall I discovered it to be in a most faulty condition. It was a large 9-in drain faulty at every junction. The surface drains were completely blocked and opened into the district drain separate from the sewage. This also was blocked. Since then I have had these drains relaid in concrete and they are now made to open into a common intercepting inspection chamber. Since using them I have occupied that particular room and have all my feet with filth and I noticed when the earth was opened up that quite a cloud of condensed watery vapours arose from the ground. These conditions of bad drainage, a soil saturated with
filth and decaying matter, and generally a warm, humid atmosphere, form the most perfect conditions for the growth of pathogenic organisms with which we are acquainted.

Sepsis arthritis may arise from pyaemia and gonorrhoea and scarlatina is often associated with symptoms which are typically those of acute rheumatism.

Now as our cardinal point as our belief concerning the nature of these micro-organisms is that one specific organism will always reproduce its species and no other; we cannot admit that scarlatina will ever produce anything but scarlet fever.

So we must conclude that if this arthritis is not produced by the scarlet fever poison itself, it must be reproduced by another. How as Rheumatism is clearly not scarlet fever the
The two persons must be present together or they may either of themselves produce similar changes in the tissues. And why should it not be that the two persons are introduced at the same time or that the person of rheumatic fever is actually present in the body and is brought into activity by the disturbance set up by the "separatival" poison? (Possibility of two diseases occurring concurrently. Note V. Appendix.) Frequently it has been observed that rheumatic fever occurs in districts where other zymotic diseases are rife. I had an opportunity of seeing one such case in Barrow-in-Furness. E.T. a young woman of 23 living in Fairfield Cottages in that town presented all the usual phenomena of acute articular rheumatism on the next house a healthy young primipara had died of puerperal fever from the
Some now of cottages several cases of rheumatic fever had been reported to the medical officers of health. (The Association of Rheumatic Disease, Appendix)

Dr. P. N. Dalton mentions a number of cases of acute rheumatism occurring in sewer-polluted atmosphere and one case which he believes was due to sewer emanations (Brit Med Journal, 1875)

In regard to the communicability of acute rheumatism, the idea is very generally rejected but this is most unscientific as we really know very little about it. The British Med. Collective Investigation Committee's Report (Brit. Med. Journal, Feb. 25, 1875) mentions several cases illustrative of a possible communicability but I would not lay much stress upon them as they are wanting in any convincing proof of the fact.
Dr. M. Wakeley (Brit. Med. J., 1870, p. 188) mentions half a dozen cases in which he believes there is some evidence of the disease having been communicated.

Dr. F. Ernest Povey in the Lancet for Nov. 11th, 1902, gives a brief account of a case which he considers was 'congenital acute rheumatism.'

Koch has held that in order to demonstrate that any disease is due to a specific micro-organism we must first find an organism in the tissues of the diseased person; we must cultivate it outside the body, and obtain a pure culture; we must produce the like disease in a healthy animal by inoculating a particle of the culture, and we must again find an identical bacillus in the
incubated animal. Now in the London Medical Recorder for 1888 p 303 there is an account by Dr. E. Popoff of Nicholas who assures us that after many endeavours he has succeeded in finding the microbe of acute articular rheumatism. He took a drop of blood from the finger of a rheumatic patient and introduced it with all due precautions into a tube of meat-peptone bouillon kept at 18° C. By the end of 10 days he found colonies of a bright lemon yellow colour. He grew them also on potato. They appeared to be masses of cocci and streptococci. The cocci were rather larger than those of Erysipelis. He found them also in the synovial fluid and in the heart in a case of rheumatism.
An injection of the cultures into the jugular vein in two rabbits gave rise to acute articular rheumatism with pericarditis and endocarditis. Subsequent examination of the inoculated animals revealed the presence of the same micrococci.

Now all this is just as it should be but it seems to me that the accuracy of his observations is simply discredited by bacteriologists.

That we have micrococci and bacilli present in malignant endocarditis seems quite certain.

Dr. Bitter of Philadelphia in his Galtonian Lecture (Ann. Int. Med. Vol. p. 468) refers to the researches of Klebs, Kocher and others. Klebs he says describes two forms, one form the septic and another the rheumatic. Some were googolca like masses, others in chaplets. Some were uncapules. They found some in the form of short rods, sometimes joined in pairs.
Delafield & Budden (Text-book of Path. Hist New York 1886) mentioned the presence of bacilli in the vegetation of a very acute case of malignant endocarditis. Krocker believes they reach the valve through the coronary artery whereas Klebs on the contrary believes they are carried by the blood entering the cavity of the heart.

Wysockowski found cocci in the internal organs in ulcerative endocarditis and produced the disease in animals but not until the valves had been injured by mechanical or chemical irritants. Werschelbauer found a bacillus which was like Friedländer's pneumococcus, in old infects. He also described a bacillus endocarditis griseus. He describes it as an anaerobic microcococcus which grows on agar in a characteristic way and which when injected
into the circulation. Of late years, cases of endocarditis— but only when the valves had been previously injured. Baumgartner has confirmed Weichselbaum's observations. He found streptococci alone in one case and accompanied by staphylococci aureus in another.

(Cross-Rehan's Manual of Pathology p. 336)

Dr. Alfred Maudsley (Brit. Med. J., 1887, p. 138) found micrococci and bacilli in the blood in cases of acute rheumatism. He made cultures in meat infusions and upon agar agar. He found they grew best in an acid medium. At 30°C the colonies grew rapidly and had a reticulated appearance and a brown colour. If the medium was neutral they grew less rapidly on the nutrient material and had a greyish white colour. The bacteria stained readily with
methyl violet, gentian, and by Gram's method. I present here a tracing of the drawings given by Dr. Mantle in his paper of the microscopical appearances of these bacilli.

Cover-glass preparation of blood of case - Acute Rheumatism (20 oil w.m.)

Cover-glass prep. of serum (20 oil w.m.)

From culture in neutral medium

From culture in acid medium

Tracings from Dr. Mantle's paper.
To Wilson of Edinburgh reported two cases in which he has made similar observations in the *Edin. Med. Journal* (1886 Vol. **XXI** p.403 + 1886 Vol. **XXXI** p.924).

One was the case of a young girl affected with acute rheumatism in which there was pericarditis. In a section of the inflamed pericardium he found very small bacilli which stained well with methy1 violet. In the second case under observation some months later the patient was a young boy affected similarly with acute rheumatism and pericarditis. He examined the serum from the pericardial sac collected with great care and precautions so as to exclude contamination. With a 1/10 oil immersion lens he discovered short non nucleated bacilli exactly similar to those which he had discovered.
previously in the case of the girl. There were no micrococci. He sterilized flasks and half filled them with Brandy Beef Extract and again sterilized them. He inoculated them under a carbonic spray and put them in an incubator exposed to a temp of 80° F. and in 4 days they showed a tuber appearance. Check experiments were also made with non inoculated sterilized flasks of beef extract. All the inoculated flasks contained the same kind of very small short bacilli, in some a new appearance had presented itself, for it was found that some short bacilli had nuclei at each end which at first looked like micrococci and in some there were threads composed of pointed microbacilli many of which
showed active movement in some experiments which he made he found that if a solution of soda were added to the meat extract in the strengths of 5% the microorganisms did not develop; 2% of quinine was equally effective in preventing the growth, and in such flasks no organisms were found.

Tracings of Dr. Wilson's drawings - Bacteria with Rheumatic Pericarditis.

Pericardial Bacilli

Lung Bacilli

Section of Pericardium with Bacilli in Wells

Contract - Diplococcus
Where we find a few observers have succeeded in finding bacteria in cases of acute rheumatism, very many others have failed to do so, and no one has ventured upon a description of an organism of rheumatic fever, by which it can be identified.

It seems that F. Bordes, in *La Médecine Moderne* for 21st May 1890, examined six cases of acute rheumatism in only two of which did he succeed in getting microbe cultures. He describes the bacillus as readily cultivated in peptone gelatine and better still in veal broth. The broth cultures, injected in two instances, then occurred a moderate rise of temperature, and when injected into the carotid artery of a rabbit was followed by endocarditis with vegetations on the valves. In this case, on the other hand, whose opinion in regard to rheumatism must carry weight with us, writing in *La Semaine Médicale* Dec 4th 1889, says, 'The unvarying nature of these observations renders us rather suspicious as we too had searched in the articular and
pleural fluids and often in more serious cases of primary visceral rheumatism and have never been able to discover a single microbe. (American Annals of Medicine 99:171-172)

In trying to arrive at the truth about acute rheumatism one is beset with many difficulties - the many complications arising from the use of the old & comprehensive word 'rheumatic', and the difficulties of distinguishing pyaemic and septic arthritis, and endocarditis, being not the least of them.

Discussion with one's neighbours is not usually helpful in assisting one in this direction, for we find so many men are unable to differentiate between rheumatism and other affections; while others accepting the opinions of those high in authority are primarily prejudiced in their views & try to make their experience of the disease fit in with those views of its ætiology which they favour.
Our knowledge of this subject will advance pari passu with our knowledge of bacteriology and when we have discovered, more perfect methods of isolating and distinguishing bacteria which abound in air, earth, and water, we must find means to collect and cultivate the germs in the soil, and investigate the microphytes which attach themselves to our clothing and our bodies. Better methods must be found for isolating and distinguishing bacteria and inoculation experiments must be made to determine the effect on living animals of these various organisms. (Methods of research in pathology of acute rheumatism. See Appendix: Notes XIV.)

In this we are handicapped by ancient laws. Before permission is granted to pursue an investigation one has to anticipate a discovery, and declare its importance before we can possibly be aware of any such revelation. No doubt some such laws are essential to govern the practice of experimental men.
but such exacting conditions, as are
enforced, seem to me unnecessary, and
over zealous humanitarian enthusiasm
appears to a great extent to oppose
the progress which we ought to make
by experimental pathology.

I know at least of one instance,
which observations of this nature
and pathological inquiries carried
on with secrecy, (paralleled only
by the accounts of inventions in the
middle ages, harrassed by the superstition
belief of the King and the People, while
studying the powers of sleam) —
have borne fruit in the demonstration
of the possibility of beneficial
surgical interference in disease of the
lung.

Death in acute rheumatism is
comparatively rare, and no such
opportunity has hitherto presented
itself to me notwithstanding that
at Charing Cross Hospital, through
the kindness of Dr. T. H. Green How
had opportunities of investigating this disease.

I have had to content myself with the study of the literature of this subject and with collecting evidence bearing upon it, while at the same time pursuing the study of correlative diseases, as well as patiently observing the micro-organisms in the soil and atmosphere, particularly under such meteorological conditions as are associated with the prevalence of rheumatism, and in the vicinity of affected persons.

When our knowledge of this most important subject has been advanced, when we know more of the laws which govern the life and growth of these bacteria, when the affinities of particular organisms, the resistance of certain animals and certain conditions of the healthy tissues, as well as the doctrines of vaccination
and immortality have been satisfactorily explained, when physiologists and chemists have elucidated the intricacies of the processes of nutrition.

Then, and not till then, do I hope to find our knowledge of the etiology of rheumatism complete.

It is only by persevering industry that these ends will ever be attained. The history of men of science has this peculiarity advantage that it shows us the importance of little things in producing great results.

Smeaton learned his principle of constructing a lighthouse by noticing the trunk of a tree to be diminished from a curve to a cylinder. White of Selborne carrying about on his rides and walks a list of birds to be investigated, and Newton burning an old
lion into a water clock, are examples of habits of patient observation which scientific biography attractively recommends.

"The grandest truths appear slowly. They are like the shapes of cloudy light, floating in the utmost loneliness of space. Some the naked eye discerns, others a common glass brings into view. But it was the enormous Reflector of modern skill, in the purity of a Southern Atmosphere that gave to those masses of vapour a form and look of glory and kindled strips of mist into rays of Exquisite Lustre. Thus, the cloud of the weak becomes the star cluster of the strengthened sight.

Many radiant bodies yet remain in their mystic retirements. No glass however endowed with vision compels
These shadows to come within its range and to show their faces. Still there is hope. The discovery of one star is the promise of another. The hand of Science grows more cunning every day and its eye endures a stronger blade. This is the lesson for the Enquirer into the far off and distant things of Truth. Hour by hour some eyes are opened more and more by the Father of Light to behold the wondrous things of this law.
Appendix

To a thesis on
The Aetiology of Acute Rheumatism

By

Douglas H. Anderson
MB & CM (Edin.)
Note I. On the significance of "Chill"

The significance of this so-called "chill" is of course exceedingly doubtful. We are all familiar with a sense of "chilliness" after undue exposure. As a rule nothing more of it so we ascribe it purely to reaction of the nervous system to external cold. But beyond this we have all degrees of chill up to the severest rigors of malaria. Professor J. L. Schenk (Cited from Bulletin, American Jrn.) would have us believe there are two kinds of chill a non-infective and an infective chill. The former that when a man enters a cold room a bacterial stream sets in towards him on account of his higher temperature. This is what gives rise to the chill. There must be two conditions in order to give rise to chill: first his abnormal in the temperatures which sets-up his emotional current and secondly there must be some erosion of skin or mucous membrane to allow the bacteria to enter his body.
Note II  Thermotonic Centre

Dr. Buzzard (Diseases of the Nervous System, p. 366) is evidently inclined to favour the idea of the existence of centres above the medulla oblongata concerned in the nutrition of the joints and probably having an intimate connection with those parts concerned in the innervation of the heart. "One cannot help feeling," he says, "that were the existence of such a centre to be rendered probable we might find in it a valuable clue by which to explain the combination of articular symptoms in acute rheumatism with occasional tendency to high temperature and still more with cardiac complications of such frequency as that disease, an association for which, hitherto, no hypothesis has reasonably accounted. I might be thrown incidentally on the nature of arthritis deformans." I don't understand how we should be much nearer to a true understanding of these complications if such a statement could be proved. Liv
William Broadbent (Lancet July 1832)
"objects to the establishment of another of those centres which are postulated for anything of which we are in want of an explanation."

**Note III.** Organisms in malaria.
It seems to be quite certain from the researches of Klebs, Landauer, Marchiafava, and Celli that there are appearances in the blood that are not to be found in any but malaria-infected persons. Various microscopic appearances have been noticed. Colorless protoplasmic bodies in the blood corpuscles, cyclic, segmented, and various mobile bodies.

I have roughly sketched these appearances on the blood corpuscles.
It is recommended in examining the blood for malaria, and of course the same technique would apply in other circumstances, to spread the blood out on a cover glass in the thinnest possible layer with the technique of corpuscles broken up. A 1/10 in. immersion is sufficient. Stained specimens are prepared as in the simple method in common use in examining agamum in pathological cases, i.e., drying blood and cover glass, staining, washing out, with alcohol, mounting in Canada balsam, Harrisin or Methyl blue and the best stains, it is said.

Note IV. The association of Rheumatism & Pyemia.

As bearing out my opinion in Barrow of the association of these cases of Acute Rheumatism, Puerperal Fever, and Malaria, I would refer to Dr. L. L. Longstaff's work "Studies in Statistics" on that
book on p. 317 he gives maps showing the relation which he alleges.

"... between these three diseases and in the last he remarks: 'Though the pericardial fever curve has great points of resemblance to the scarlatina curve as it is more like erysipelas, it is also extremely like that of rheumatism with diseae of the heart or pericardium. So that we may infer some close relationship to rheumatic fever a point that I must reserve for further investigation but may merely remark that this erysipelas curve is still more like rheumatism.'"
Dr. Longstaff suggest the relationship of these several diseases. He also gives a plate showing the death rate per million living from Typhus, Periperal Fever, and Rheumations in which "the correspondence in nearly every case is far closer than mere chance coincidence would account for."

Note V: Freedom from Rheumatism and Heart Disease in Furness.

The Furness District comprises all that portion of Lancashire which is north of Morecambe Bay. The Bay dips in to cut off this peninsula from the rest of Lancashire. On the south west coast of this peninsula is situated the town of Barrow-in-Furness. The town is quite new—fifty years ago there was one small place, all that existed in those days being a few fishermen's huts. The population now fluctuates
from 50 to 60 thousand persons.
The town is well built on a regular
plan mostly in rectangles with
broad, regular streets,
Haviland, in his book on the
Geographical Distribution of Diseases
by numbers of statistics, there
quoted, confirms the fact
I have stated in regard to the
Comparative rarity of Rheumatism
& Heart Disease in Teessis.
This is peculiarly interesting
now, for I certainly arrived
at these same conclusions long
before Haviland's book was
published. Haviland gives
the annual average death-rate
from heart disease in this part
of the North West of England
as about 13 to 16 per 1000 living.
"The four districts", he says, "exposed to the
direct influences of the prevailing
southerly and south-westerly winds,
are Kendal, Ulverston, Bootle,
and Whitehaven. These are well
air-flushed districts and coincident with this fact is the other that they have an individual mean annual mortality (from heart disease) below the average of England and Wales, as to every 10,000 males or females living."

The next part of Dr. Haviland's work is to deal with the Thames Valley and I shall be interested to see the conclusions he arrives at concerning the prevalence of Rheumatism and Heart Disease in his district and the inferences to be drawn from such facts as he has collected.
Nothing could show more strikingly than does this beautiful "Relief Map" the manner in which the Thames Valley is sheltered from the prevailing westerly flow on the one hand, whereas the Cumberland & Westmorland Ranges are sheltered by Moelwyn & South Lakeland Mds.
Note VI  Possibility of two diseases like scarlatina or pneumonia occurring in same individual concurrently.

We have evidence that the bacteria of two distinct diseases may attack one animal simultaneously. The animal will die and the germ of both diseases be found in the animal's body after death.

This question as well as the even more important one as to the relative influence of these two distinct bacteria one upon another formed the subject of an important series of experiments by Dr. Klein – Local Government Board Reports for 1889.
Chapter VII
Methods of Research on the Aetiology of Rheumatism

In enquiring into the origin of such a disease as Rheumatism, we have first to define accurately the term. But it so happens that this is peculiarly difficult to accomplish in dealing with the disease which is the subject of this essay. We have to study the history of the disease, the bearings which race, age, sex, social condition and habits have upon it. Once, and development.

Much interest attaches to all that has been written on these matters although the fragmentary condition of the frequent way in which writers contradicted each other detracts greatly from one's chance of making any deductions tending the core of this disease. We have to watch and mark down its clinical features, and to note its relationship...
to weather. Such an inquiry requires the study of Chemical, Physiology, and Pathology, and in addition a knowledge of the morbid histology, particularly of the heart and joints, may assist us, and lastly, the study of Bacteriology is of primary importance.

The soil is suggested as a probable field for fruitful inquiry. In setting about examining into the characteristics of a soil we have first to note:

Physical Properties:

1. Temperature of the soil and the influence of climate upon the temperature of soil.
2. The mineral constituents of the soil.
3. The amount of water it retains.
4. The amount of organic matter present in a soil; the hygroscopic power of the soil, as well as its capacity to retain water, is largely the porosity of the soil.

The study of bacteria in the soil
as yet the most promising field for research. Since the investigations of Pasteur and Greenfield into the life history of bacillus anthracis we are encouraged in hoping for great results from this source of enquiry. The earth teems with bacteria, if the earth is turned up at any point an enormous multiplication of bacteria occurs, and one yet has not ascertained exactly why.

In trying to detach bacteria from the soil, cultivate them on artificial nutrient material, many simple methods and cases, many of which are practically the same as in examining the bacteria in water; since we first wash out the soil with water.

But this may be an imperfect way of collecting the bacteria and a good way is simply to shake the Earth up with your
nutrient gelatine; or, we may mix the earth with water first of putting it into some form of centrifugal machine.

Drop Cultures. This is a very convenient way for rapid examinations. Have a slide made with a plate ground out of its surface to receive your drop of material; you can then observe any growth under the microscope.

For more extended examinations we use larger solid or fluid media. Milk, beef broth, blood serum, hydrocele fluid, or potato are all of them used, but without doubt the broth useful and pretty well the most used nutrient media are nutrient gelatine and agar agar.

In preparing nutrient gelatine the material I use is made as follows:

1 lb lean meat, to about a quart of water (distilled) boil; set aside the beef tea. Next make a solution
mix thoroughly with the dissolved gelatin in your tube (previously melted) and then pour the whole over the plate, and cover with a bell jar. Very great care must be taken to prevent contamination. I really don't see what special practical advantage there is in allowing your cultures to remain in the tube. The need in adding water washings note the quantity added. If we are quite in the dark as to what is the nature of the solution, we make three tubes, (with advantage), adding 1 cc. of the water to one; 5 cc. to the second; and 10 cc. to the third. If our plates are large it is necessary to keep the atmosphere within the bell jar moist, and we do this by placing within it a wetted piece of filter paper. The double glass moist-chamber in common use are very convenient.
and we now place them in an incubating chamber at about 20°C. If agar is used it should be about 30°C.

When we have made a plate culture we count the colonies,

of which we very often a troublesome matter, but it is made easier by placing over the plate culture a square of glass, which has been marked with divisions into square centimetres. We take 10, or whatever number we like in each direction, and this gives us the number of colonies in a square centimetre; if now we multiply by cubic cm, we get the number of colonies in the amount of plaster taken.

Someone, I forget who, advises us to 'measure' the part we are gaining rather than to take it by weight, or to break it up, or allow it to pass through a piece of thin paper so precise as to act
like a needle & then breaking
the spot with the nutrient gelatine
very thoroughly. Well when
we have roughly got our plate
outline we have got one step
on towards determining the micro-
organisms present. The next
thing is to make emulsions
by inoculating the nutrient gelatine
in a tube, putting in our
sterilized plug & covering over
with a little India-rubber cap.

For oxygenating the air. Various
forms of apparatus are in use.
Here a tube is a large clean glass
tube placed upon a tripod.
The inner surface is covered
with nutrient gelatine, and
the air is drawn through a fine
aperture at one end,
in definite quantities, and
at such a rate as to allow
of the deposit of organic
particles from the air, on the
nutrient gelatine. A very simple
way of drawing the air in is to utilize a syphon-bottle.
A convenient amount of air to draw into Keene's tube is from one to five metres, and it should be drawn into the tube at a rate of something like 1 litre in 10 minutes. We ascertain how much air has passed through by means of an air meter, & set the tube aside in the cold (2° C) incubating cupboard.
We then count the colonies & reckon the number per cubic foot of air.
Strauss' Bubbling apparatus is a useful method. If we have a small glass tube drawn out to a point, & connected with an aspirator. The air rushes down into glycerine & slowly forms bubbles by which means it is thoroughly washed, & deprived of any germs it contains.
Another method consists in first entangling the bacteria of a given quantity of air in sand, previously sterilized, 0 places in a glass tube. One afterwards removes the sand with sterilized water.

Perhaps a good way as any where we are simply searching for bacteria is to draw the cotton wool from out of several tubes for a few seconds, 0 then replacing them allows the growth to grow.

Such a simple method suggests itself especially in examining the air for bacteria present in particular places or occasions. I have used glass rods passing through the wool plug into the tube 0 covered with sterilized gelatine, 0 exposed the rod in the neighborhood of articles of clothing exposed to the weather, 0 then brought into a warm room.
Having obtained our cultures and made a number of secondary cultures from the colonies we have to examine and our bacteria.

I should here note that some bacteria being aerobic, or others anaerobic, require special apparatus for cultivations of anaerobic organisms. Many contrivances have been used for this purpose, a very simple one is that of Prof. W. Ogata.

It is made from an ordinary test-tube filled with a gelatine tube. The tube is drawn out by directing the flame of a blow-pipe below the cotton wool plug. As it draws out long then when a small glass tube is drawn out with a capillary tube, a few centimeters of the original being left so that the capillary portion is longer than the test tube. The cotton plug is removed from the test tube which is again sterilized. The gelatine is liquefied by placing
The tube in warm water and then inoculated by means of the capillary tube. The wide part of the capillary tube is then connected with an apparatus for developing hydrogen or carbonic acid, whichever be used, and lead qrapped down close to the bottom of the test tube. As the gas enters, the air is driven out or the capillary tube is withdrawn if the test tube is closed up at the narrow part.

Stains: --- An excellent method of staining is fuchsins, which is so commonly used. The bacteria being smeared on the cover glass & dried in a flume. Then the flume is placed in alcohol for a few minutes and then transferred to the mixture of aniline oil & dye (fuchsin, magenta, etc.) and kept there a minute or two, washed with alcohol & then transferred to a solution made of iodine 1 part, iodide of potassium 2 parts.
of distilled water. In this they are kept until their colour has changed, then transferred into alcohol to remove all the appearance of the stain. By this means the bacteria alone are stained. As some microorganisms will not colour by this method Richolle recommends another method. (Pert Nat. Ann., 1893)

This is, to stain in Leuflers or Kühn's blue for 1-3 min then wash in water, immerse in a solution of tannin 1 part in 10, or again wash in water. Dehydrate in absolute alcohol.

Such methods as these are apt to injure the bacteria and Brown (Observer, 1892, p. 298-300) has devised a method to avoid injuring the bacteria especially to avoid injury to any flagella that they may possess. A windlass is used made as follows: - Tannin 30 grams, aqua of alcohol 12 drops, alcohol 1/3. The bacteria to be stained are
placed first in a little water enough to smear over a cover glass. Now this is not dried in a flame, but simply allowed to dry. It is next placed in the alcohol tannin mordant, or allowed to remain in it for from two to five hours or more. The cover glass preparation is now taken out of the mordant, thoroughly washed to remove the excess of the mordant and then the stain is poured over it. The usual stain of Fuchsin is made with aniline oil in water and used. Mr. Brown prefers Fuchsin. He then gently dries the cover glass over a lamp, washed in a stream from a wash bottle, and allows it to drain, and mounts in Balsam. An attempt should be made to declore by oven to pass it through alcohol, or oil of cloves to the Balsam. If the balsam is used dissolved he advises us to put a drop of oil of cloves, or injlet in the center of the cover glass before allowing it to fall on to the balsam.
For staining with a good method (Flocas) they are first placed in a 10% solution of ammoniac mixed with an alcoholic solution of an acid dye. Fuchsin is one of the best. Mix 20 cm.

of ammoniac solution, and then add about 1 cm of the alcoholic solution of the dye. Warm the solution, dip in the cover glasses covered with the film to be stained. Leave to stand 3-5 minutes. Anthers require longer; 10 or 15 minutes. When judged to be sufficiently stained, the cover glasses are placed in a decolorizing fluid which is a 20% solution of sulphuric acid. They are then washed in water and contrast stained.