I

A Thesis

8 1890

on

Clinical Observations relating to the Parasitic Origin of the Commoner Infectious Diseases

for the Approval of the Senatus Academicus of the University of Glasgow. by.

John A Jackson. M.B. C.M. L.S.S.E. a Candidate for the higher degree of M.D.

The Consideration of the treatment of any disease, must always commence with a study of the causes, which produces it & under which, it generally arises. In proportion as we can discover & remove the factors promoting disease, shall we be enabled to cure or at least influence it, both in course and result. Unfortunately there are numerous classes of diseases, where the cause is, at present at any rate, beyond our comprehension, and success is unlikely to attend our efforts, whilst this is so. It becomes, therefore, a matter of the greatest importance to consider, whether our opinions regarding the origin of disease is correct & will agree with the results of clinical observation. Undoubtedly great advances have been made of late, both in the discovery and prevention of many causes of suffering, though much still remains to be done, before we can have an intelligent appreciation of the circumstances, under which, many of our most fatal maladies arise

Continued

" Dr Murchison expresses himself thus,

" Among the greatest benefits, that medicine has conferred on the human race is the discovery of the causes of disease and of the

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

"the measures by which they may be prevented"

Now far the discoveries of recent years relating to specific microbes being the only source of origin of many ~~of these~~ of our commonest & most fatal cases has tended to enlighten the general practitioner is a grave question, probably a too rigid acceptance of the views advanced has had a retarding influence

Although my observations are similar to what has often ~~been~~ before this, been recorded, & though from the inherent difficulties of the subject & the time & facts at my disposal are both imperfect & insufficient, still they may prove of some little interest, as, owing to the widely scattered and thinly populated district in which I practice, it is easier to follow out my cases with fewer sources of error, than is possible in more densely populated districts.

The first two important questions arising are.

I. How far are we to accept a parasitic theory of origin of many diseases occurring in practice?

II Admitting the probability of such an origin, does it necessarily follow, that all cases are the result of infection, with this specific germ, which in its turn arose from a previous case.? in other words, are we to accept as applicable, the doctrine of Squire, whom, referring to Scarlet Fever, he affirmed.

(Fever. dictionary of

"no other origin (i.e. than direct infection) can now be admitted, its extension to a new locality is traceable to an imported ~~origin~~ infection."

cent Essays elation to ndemiology)

Dr Watson Cheyne sums up as follows.

"For the strict proof that the micro organism is the cause of the disease, three things are necessary"

"1st The same species of micro-organism must be constantly present in the affected parts, at any rate during the earlier period of the disease.

2nd The organism must be cultivated apart from the body

and thus separated from all other morbid materials "

"3rd Their reintroduction in a suitable manner into the body of an animal capable of being attacked by the disease, must be followed by its production"

In cases where these three tests are complied with and all known sources of error eradicated, we must, in all fairness, acknowledge a cause to have been demonstrated. The microbe may however stand all these tests & proof still be wanting, that the cause has been discovered, or at any rate, I submit, that these are no proofs that the microbe must arise from some previous case, reasons for this opinion will, I trust be forthcoming in what follows.

The Late Dr Murchison advanced 6 reasons for adhering to his original views concerning the "de novo" origin of the ×Continued fevers. Let these be considered a basis upon which to ground my observations regarding the origin of 3 of the commoner "preventable" diseases viz Enteric Fever, Phthisis. Scarlet Fever. they are as follows.

× I doubt if he [illegible] [illegible] except [illegible] Enteric [illegible] Typhoid Fever

I Admitting the parasitic theory of contagious diseases, does not exclude the possibility of their independent origin. for two reasons

(a) The parasite may arise independently of preexisting case

(b) Bacteria, Vibriones, etc may arise "de novo" in organic fluids.

II The parasitic theory rests solely on analogy & is unsupported by facts. Now so?

III If origin is always by contagia the germs must be omnipresent.

IV The first case must have originated de novo

V Erroneous conclusions have resulted from discussing all zymotic diseases together, the possibility of their differing greatly having been lost sight of.

VI Several Contagious diseases we have the power to originate "de novo."

I shall first refer to a limited outbreak of enteric fever occurring in a terrace of 8 houses, situated on a slope above the town & entirely separated both in water supply and drainage from other dwellings.

On Sunday evening April 15th 1888. I was called in to No 5 to see a little girl aged 7 years, on the 19th in consultation with a Gentleman connected with the teaching staff of the Newcastle School of Medicine, the case was pronounced to be Enteric Fever.

The household consisted of the parents, 2 younger children, the patient & 4 Servants. They had removed to the house on March 25th bringing 3 of their servants with them, the under nurse joining them after their arrival at their new abode.

On April 17th this under nurse consulted me, on the 20th I advised her removal home from whence the case was reported as Enteric fever by the Medical Attendant, (this case died.)

On the 23rd the waiting maid was sent home, she also having contracted the disease.

Now how are these cases to be explained?

No other cases could, or had been, heard of in the neighbourhood.

The milk supply was common to a great number of families, in none of whom illness supervened.

The water supply was for the whole terrace, on the same system afterwards to be alluded to.

The common sewer was for the terrace alone.

I however discovered that the previous occupants of No 5 frequently suffered from "Sore throats."

An examination of the premises resulted as follows. —

The House drain not being visible at any point. I was

unable to determine its exact condition, with however a strong current from the house to the sewer, the fumes, from the smoke & paraffin test, were distinctly noticeable in the kitchen and passage, showing it to be leaky and that the occupants had been breathing foul air.

The connection between soil pipe & drain was quite open having been filled with some loose dry material.

There was no disconnection between house drain and sewer, so that sewer gas would find its way readily through the leaky pipes into the house.

The 4 inch Soil pipe terminated above in a two inch one which ventilated flush with the attic window

In the yard a few ~~feet~~ feet from the house was an uncovered midden-privy about half full

The back kitchen sink was trapped but entered drain direct

The water closet consisted of an old fashioned pan closet which was leaky & foul.

The water to this was supplied from a separate cistern the overflow pipe entering the closet trap.

A main cistern supplied the bath, boiler, & drinking taps. being filled by a ½ inch pipe, which was calculated to fill the cistern in 24 hours.

This pipe was leaking in the yard & lying close to the house drain, so that there is a probability of it being contaminated unfortunately, being myself at that time unable to properly analyse water, this was neglected & left undone.

These cases suggest one or two points

1st The impossibility of accounting for a specific origin

2nd The period of incubation elapsing between entrance to house & outbreak, were more in accord with what has been noted regarding the incubation of Enteric fever, they differ in this respect in a marked manner from the next two cases to which I allude, as if the intensity or method of receiving the infection

a period of incubation bore some relation to each other

3. whether, the same cause as produced ulcerated sore throats in the previous family, who would probably gradually come under the influence of any poison arising from leaky drains, could when received in full force by the new-comers produce the specific disease Enteric fever?

4 In none of these cases did the "Typhoid Spots" appear.

I. Taylor age 18 years a carman living with his Parents & a family of 8 children.

On October 18th 1889, this lad cleaned out an uncovered midden-privy which had remained undisturbed for some months, the smell was so horrible that on returning home he refused to eat as "every thing tasted like the smell & made him sick" On October 23rd he shivered & complained of headache &c Diarrhoea commenced on Oct 26th.

On October 28th the characteristic spots appeared, being so well marked that on the 30th I took two other Medical men to see the case. They confirmed my diagnosis.

No other possible source of infection could be obtained.

R Short age 41 years a Colleyman in Service of North Eastern railway. Residing with his wife & family of 4.

On August 6th 1889 early in the morning he unloaded some animal refuse, which was to be used in the Manufacturing of Tallow. The Stench was so horrible that he vomited.

On August 9th took ill

" August 12th I pronounced the case of Enteric fever & on the 13th the first "spots" were observed.

There also the absence of any traceable Specific infection was the case.

Let us admit that the Short Bacillus described by Eberth, Koch Meyer & others, was the cause of the disease in these cases, They have, I think, established its probable existence in all cases. Where did it come from? To prove a direct negative is

an impossibility, though my investigations were conducted so as to eliminate all sources of error as far as lay in my power. Certainly the most possible explanation is that of Dr Murchison "that the germs may arise "de novo" in organic fluids, or at least such a soil may alter their characteristics & render them specific".

Gaffky admits "that the possibility cannot be contested" of the entrance of the specific germs by the lungs, though Eberth asserts that anatomical investigations have afforded no evidence of this, It is not however easy to explain, at any rate, the last two of the cases recorded in any other way, unless these being inhaled they entered the stomach in the act of swallowing.

I am however of opinion that an aerial source of infection is much more common, than the above observations would lead one to infer, Not only is this the case, but I believe I could also prove, that in diarrhoea at any rate the breath may be infectious

The Epidemic I now report is probably one of those described by Wilson as "Cases of Enteric fever to be met with in all outbreaks of enteric fever, as a rule the scattered ~~outbreaks~~ cases, which crop up in rural & small urban districts are of this description"

On Friday November 8th 1889 I was called to the R — a house situated in its own grounds with no other dwelling nearer than 200 yards

I found Mrs M. A. aged 44 years ill & obtained the following history

At 7 P.M Thursday Nov 7th She had been suddenly attacked with violent diarrhoea, had become hot, restless & slightly inclined to delirium, passing a very bad night, the diarrhoea & tenesmus becoming worse, the stools were of a faecal character, & She was very thirsty, Early on Friday morning sickness had supervened, & was continuing, the vomited matter being

more or less of a bilious character.
At 12 noon Thursday her temperature in the mouth was 103° F.
On my arrival at 3 Pm. I found her very flushed & restless. Skin dry & extremly hot, tongue slightly coated, & inclined to become dry & glazed, pulse 130, Temp 104-2°. Sickness abating the purging Continuing, no chest symptoms were detected.
Saturday 10 Am Temp 101 F. 6 Pm. 99° F.
Sunday Nov 10th " " 97.8.
The diarrhoea continued for a week, the patient being languid & weak for some little time.
In a house of this description it becomes comparatively easy to follow up a case to its source, with fewer possible sources of error than can be done among the poorer class.
The greatest attention was paid to the prompt removal of all effete matters, mixed with deodorants, to the water closet which was one of Tennants flush out description.
The patient was attended upon by her Mother & special maid C. M. whose duty it was to attend to the above details.
On Sunday Nov 10th C. M. age 19 a delicate girl maid to Sup. th: evinced signs of suffering from the same malady, the symptoms were, on the whole, more severe, but in other respects identical, the following are the temperatures taken in every case in the mouth.

			A m	P m
Nov 10th	Sunday		102	104
" 11	Monday		101	101
" 12	Tuesday		101	101.6
" 13	Wednesday		99.6	99
" 14	Thursday		99.6	—
" 15	Friday		—	—
" 16	Saturday		97.8	—

Throughout this time the diarrhoea continued.
On Tuesday Nov 12th Master T H: aged 9, began to suffer from diarrhoea which continued for 4 days, he was hot & restless but not so ill as the other two.

On Friday Nov 15th Inf. D.H.S aged 6 years, became sick, purged, not so restless, as to render it impossible to take the temperature

The Household consists of Father + Mother, the 3 children above mentioned and 6 Servants.

No other person in the establishment suffered except Mrs H.S who during the week under notice complained of diarrhoea, which she attributed to the nursing.

Now how are these cases to be explained?

An investigation of the premises revealed all to be in excellent order and arranged on a thorough Sanitary system.

The water supply, conveyed in galvanic iron pipes from a distance, was used only by the R—g establishment

I collected a sample from the main tap in the house the following is my analysis.

Physical examination in a 2 foot narrow white glass vessel.

Clear. transparent. No deposit. No smell, pleasant taste.

Chemical.

Total Solids not estimated (owing to deficient chemical balance)
Hardness 9.5 grs per gallon (Clarks scale)
" Temporary 6 grs per gallon. " "
Chlorine 1.1 grs per gallon
Ammonia (free) .0035 grs per gallon
" (Albuminoid) .0098 grs per gallon
Nitrogen existing as Nitrites + Nitrates .45 grs per gallon
Poisonous Metals. A trace of Iron.

These figures show that no evidence can be obtained from the water, though as Wanklyn has observed matters potent for harm may be present + yet the water be returned as organically pure.

The milk Supply now received my attention

The household are entirely supplied from their own two Cows neither of which had recently calved, both were healthy, free

from all sigs of eruptions, scabs etc, both had been at the R-g for 3 years.

They are milked each morning at 8 am by Mr. Wilson the daughter of the cartman, who resides near, all the milk is taken at once into the R-g, in one can & from this, a large jug-ful is at once sent to the nursery, the rest is used for domestic purposes but not drunk as milk, the cans are kept at the R-g Mary is calling for them at milking time

The nursery jug was for the Childrens lunch & for C. M, who had been ordered as much milk as possible.

Inferring that somehow or other Mr. W. was connected with the outbreak I made enquiries with the following result: the facts are arranged in Chronological order

On Thursday October 31st Susan W. age 4 years, returned from Acomb School, not Sick & purged, she was kept at home till Monday Nov 18th

On Sunday Nov 3rd Robert W age 2½ years, attacked similarly, but much more severely; the diarrhoea with him being bloody.

On Tuesday Nov 5th Mary W (the Milker) rose in the morning feeling ill, took no breakfast and started off to milk, upon her return she became sick & purged, but went to Heslam, a distance of one mile to shop, here, she became so unwell that her friends had her to convey home, Nellie W. age 12 taking the duties of Milker that evening & continuing to do so until Monday Nov 12th.

Mary was very severly attacked.

Now are we to consider that all the cases at the R-g arose from drinking milk infected by the breath of Mary W. two hours previous to actual symptoms setting in, or are we to infer that the following, which was the only other source of communication detected was to blame. viz &

On Tuesday evening after milking Mr W. assisted Mrs M. to remove her tantrums from one outhouse to another, being in pretty close contact for about 2 hours.

I am inclined to believe & subsequent investigations confirm this that Nellie W. carried about her person infection from her Sister, Miss M.A. succumbing after about 48 hours incubation, C. in the maid contracting the illness with after a similar period of incubation, in the discharge of her duties as nurse,

If Nellie W. to Miss M.A. Tuesday to Thursday } evening to evening
P. Miss M.A. to C in Thursday to Saturday. } night to night

& that Master T.A. & Miss L.A. caught it from their Sister whom they did not see till the Sunday.

In the Wilson family it is significant that 48 hours, separated Susan W. & the Baby, a similar period elapsed between the baby & Mary W. whereas Nellie W. did not contract the illness till November 13th 15 & 8 days later,

Now Susan slept by herself on a chair bed in some room as Nellie, Mary & Annie who, together occupied a separate bed ~~room~~

~~The [illegible] a in some room~~

The Father, Mother & Robert occupied another room, & two boys, the only other members of the family, & who were only at home at night occupied a third room

Susan W. sometimes used the chamber & sometimes went to the privy.

Mary mainly nursed Robert during the earlier part of his illness When Mary took ill, she changed places with Susan & occupied the chair bed, on Monday Nov 11th she resumed her place alongside Nellie, on Wednesday Nellie took ill,

These facts are instructive & agree with what I have noticed that the malignancy of infection seems to vary very greatly in different cases of the same disease.

A common privy being used, under the latter cases less conclusive Under the same roof as the Wilsons, but with quite distinct & separate offices, dwelt a family called Stokoe, Mrs S suspecting an infectious character in the illness next door forbidden all intercourse, this was easy, as only herself, her Mother & two

Children were at home during the day.
On the 10th this rule was relaxed, Mary was spending a large part of the day with the Stokes,
On the 12th two cases occurred
" 14th one " "
(3 three out of 4 at home were attacked, but all much milder later some of the other Children suffered.
Mary was again appears to have spread the disease, her infective power being apparently very great
We may perhaps be allowed to infer that the germs, (if the disease is due to such cause) found in her a soil more suitable for the further development of its infectious properties.
How, where, and how, did Susan Wo contract the disease?
She had returned to Acomb School on Tuesday Oct 29th after an absence of a few days, due to a slight cold, in which however there was no sign of any sickness or purging, On Thursday evening Oct 31 she returned home ill.
The School registers which were kindly placed at my disposal, revealed a few irregularities but on Monday October 28th Alice Scott, who sits next Susan Wo had returned after a weeks illness.
The Epidemic at Scotts The family consists of self, wife & 4 children
On Thursday morning Oct 17th Annie S aged 9 years, who goes to St Mary's Catholic School, Hexham, was sent home ill, Mother described her symptoms with such clearness as to leave no shadow of doubt as to the nature of the attack. The diarrhoea continued till the Sunday.
On Friday night Monica S aged 7 years attacked, she was able however to return to Acomb School on Monday & sat next Annie Wilson of the R – 9 (the only one of the Wilson's who did not afterwards contract the disease)
On Monday Oct 21st Alice Scott aged 5 years took ill, she was much worse than the others, She returned to Acomb School on Monday Oct 28th & on the 29th sat next Susan Wo as

already mentioned

On Tuesday Oct 22nd John Scott age 3 years ill mildly attacked,
On Saturday 26th Mrs Scott also mild,

Sleeping arrangements

Annie, Monica & Alice in one bed in a room by themselves

Mr. Mrs & John in the Kitchen

The diarrhoea described as Slimy, dark coloured & bad smelling a good deal of tenesmus.

The Chambers were used, but after the first day Carbolic acid was added

The ~~fæces~~ Chamber fæces were emptied into a large pail standing in an adjoining loft, and afterwards buried.

I now endeavoured to trace the two branches of the epidemic viz
1st What other manifestations of the disease appeared in Acomb
2nd The source from which Annie Scott received the infection.

Briefly my conclusions are, that previous to the case at the Scotts no such malady had appeared in Acomb, but that after this time, it became very widespread indeed, very few houses where children dwelt escaping, many of these I have traced out in detail but space will not allow of them being recorded. The illness apparently spread mainly by "neighbouring"

2nd Where did Annie Scott contract the disease?

St Mary's Catholic School Wexham is attended by the poorest section of the town, & Cross examination raised fears of having done wrong, as at the time the town was placarded with bills concerning the Compulsory Notification of Infectious disease, again so much time had elapsed as to render my task still more difficult, the following, ~~case~~ as far as I can make out the landmarks in the outbreak.

On Sunday August 4th Mrs Watson Wexham. was delivered of a healthy child, which on Friday August 16th was suddenly seized

with sickness & purging & died on August 21st, only 4 people had seen this baby, two neighbours whose history was quite clear a person named Hope also quite free from suspicion & a woman named Sewich the last named called on Thursday August 16th on her return from a neighbouring town, where she & her two children had been laid up for 3 weeks of what, she said, was called Typhoid Fever. She & her children nursed the baby.

On Tuesday August 20th John Watson aged 21/2 years took ill.

On September 6th Mary L. Watson was sent home from St. Mary's School owing to her being ill of diarrhoea on that & previous days, she returned to School on ~~August~~ September 16th

From the 9th to the 16th Annie Scott was absent attending to a Sick Grandmother

During these dates several children were absent, from some I obtained a clear history of this illness, in others I could not.

On Oct 9th I was consulted by a boy attending the School and, though at that time quite in ignorance of any epidemic, was so suspicious of the case, that I ordered him not to attend School until further orders.

On Oct 15th Mary L. Watson appears to have had a relapse as on that date she was again ordered to leave School.

On Oct 17th Annie Scott took ill.

Such then is a brief outline of a most extensive series of cases investigated by me with every effort to prevent errors creeping in. We are entitled, I think, to infer that whatever be their exact nature, they were infectious, spreading by fomites & probably by the breath, though possibly in some cases the common fomites may have had a share in their propagation. Presuming them to be due to a bacillus, then the following questions arise.

Not-segmented

How far the infective power of the discharges, which were infectious at the moment of passing, if we are to admit that they held this power at all, might under suitable conditions of cultivation give rise to the specific microbe of Enteric fever e.g. might

not Taylor's case, be produced by some such similar cultivation ~~as th~~ in the midden, under certain conditions of warmth & moisture, which specific bacillus may under one train of circumstances develope true Enteric fever, but in another, take a retrograde step ~~backward~~ producing diarrhoea with high temperature and sickness.

These suppositions are upheld by the knowledge that nearly all outbreaks of Enteric fever are preceded by, accompanied by, & succeeded by, diarrhoea of a more or less typhoid nature. Though investigations have so far failed to develope these intermediate forms, proof is still wanting of this absence, as the pure cultivations adopted are quite a different matter to the natural cultivation in mixed fluids containing numerous other ~~Bacillus~~ microbes, such a method unfortunately cannot yet be followed artificially.

Some such theory as that propounded above would fall in with the results of clinical observation & admit of Murchison's view being accepted, that the Parasitic theory does not preclude a 'de novo origin.'

For the further elaboration of ~~these~~ ideas, we will for a short space study the Etiology of Phthisis

+ Possibly; but I don't find the argument much advanced by these cases, as compared with Murchison's.

Dr Murchison said "The parasitic theory rests solely on analogy & is not supported by facts"

These words applied to the continued fevers – as far as Enteric Fever is concerned, I think, had he lived, he would have withdrawn that statement, from the conclusion of the preface to the second edition of his work in 1873, we may assume that such a possibility was at that time in his mind. The discoveries of the past few years have proved beyond much doubt, that in almost all specific diseases a microbe exists, which after successive cultivations is capable of reproducing the disease, which, in fact, satisfies the three requirements of Cheyne already alluded to.

That the parasitic theory now rests on something more stable than mere analogy. Phthisis to which, I am now going to allude, is a fairly good example

The following questions arise at the outset.

1st Is Phthisis a parasitic disease?

2nd Is it communicable from person to person?

3rd Can it arise de novo, or must each case be accounted for on the theory of direct infection

My conclusions are.

1. Phthisis is parasitic and communicable
2. Its origin is often by far to be accounted for in other ways, than by assuming infection from some previous case.

ys on Bacteria
e disease

Koch has demonstrated.

1st That Tuberculosis never exists without the Tubercle Bacillus.

2nd That these Bacilli, when a suitable soil is used & under certain stringent conditions of temperature, can be cultivated external to the body, but that these conditions are not such as can be obtained naturally in this country at any rate.

3 That after as long a period as 12 months of cultivation through 15 generations, inoculation produced tuberculosis.

Let us then admit that Phthisis is caused by the Bacillus Tuberculosis how are we to account for its presence in so universal a manner

As to be the cause, roughly speaking of one seventh of our death rate?

It appears that Koch seldom if ever failed in producing the desired result, a local inoculation was rapidly followed by the extension to neighbouring organs a general diffusion of the Bacillus, when artificially induced, whether by inoculation or inhalation, the effect was directly proportionate to the number of Bacilli employed, the inhalation of large doses gave rise to a very wide spread dispersion of Tubercle throughout the lungs.

If this certainly exists, how are we able to account clinically for so small a proportion of cases by Contagion, that many authorities still deny its existence altogether?

To account for the cases occurring in practice, more especially in a district like this, on the theory of Contagion, which really means the existence of Sputum dust holding Bacillus spores, is to admit an almost universality of infection, some special circumstance or condition stepping in to render such infection "active & potent" from time to time, in which case we cannot decry the force of Dr Murchison's words when he said relating to Typhus Fever

fevers " If the poison remains passive for years so long as certain conditions are absent, but becomes active or potent, so soon as these conditions come into play, it seems fair to infer, that the appreciable conditions and not an omnipresent & indestructible poison, constitute the primary cause of the disease."

Clinically then it is difficult or impossible to account for many cases, owing to the absence of all ascertainable sources of infection. On the other hand it becomes no easy task to explain the absence of cases, where contagion is so rife, e.g. when we see, as we continually do, Members of the same Consumptive family nursing or even sleeping week by week with a sufferer advanced in the disease & yet come scathless out of the fire, here we have the Soil & the seed yet no fruit.* Even after the Bacillus has

been captured, he appears to be a most unwilling guest, only living a short & variable time, for as Dr Andrews has ~~expressed it~~ pointed out, it cannot thrive unless certain very stringent conditions are continually present. Thus in those very cases, where it is possible to account for the existence of a constant supply of infective material, it appears almost inert so far as healthy people are concerned.

In my own practice I have only met with one case of true infection.

Edward B age 23 a Carman, married with one child, was suffering from Chronic Phthisis.

His wife was a strong healthy woman, during her second pregnancy she began to suffer from dyspeptic symptoms referred to that poverty, which is in such constant attendance upon the downward course of Phthisis in the poor. No trace of disease could be discovered in the lungs, her family history exceptionally good, at the date of her confinement the husband was bedridden, they occupied the same bed, she contracted acute Phthisis & died in 8 months, surviving her husband by 9 (7) months.

The room was clean, large, fairly dry, one stair up, but so situated at the foot of a steep mound, that though facing South, the suns rays never thoroughly penetrated to the interior of the room.

In The Wexham Urban Sanitary ~~Authority~~ District, an essentially agricultural little town of 6000 inhabitants, spread over about 6000 acres, the death rate from Phthisis the last 3 years has averaged 2.1 per 1000, This, comparatively speaking, is larger than that observed in many towns more closely populated. Why should this be?

Mary W age 24 a strong healthy girl, with good family history came from the County to Wexham, to be confined of an illegitimate child, she took a room in the same row of houses as Edward B but 30 or 40 yards to the westward, the same conditions relative to sunlight existing to an even worse degree

Circumstances connected with her position produced a great effect upon her nervous system, she became low & depressed Contracted Phthisis & died in 18 months.

Annie Age 24 daughter of a well-to-do farmer, residing 5 miles from Hexham, the nearest town, the house situated on a level plateau at a considerable elevation; at the back runs a deep, wooded narrow ravine; The house faces South at which aspect is the room mainly used, the girls bedroom windows faces across the ravine is east. Fathers family history Good, Mothers family Good, it is probable that an Aunt died of Phthisis, her own history good till at 19 she began to have a cough, the case was very intermittent exemplifying the self limited course of Phthisis described by Flint, who says

926. 1882 "After each invasion of the tuberculous eruption there is a temporary self limitation of the disease"

It called to mind that Analogy between the Clinical aspects of Phthisis and Ague described by D Andrews

693785 B3 (1884)

This patient never left home except on a market day for Hexham, occasionally to stay with an Aunt residing far in the County on the opposite (South side) side of the Tyne Valley. where she usually improved.

How how, except on the assumption of an omnipresent germ is this case to be accounted for, No Poverty, No Overcrowding, No deleterious occupation, No Heredity? See above case

The rooms of the house were low, the windows were very small scarcely two feet by two feet including Sash, & only one for each room, the mists rolled up & hung over the ravine behind, Sunlight though present in abundance could not gain free access to the house, what early morning sun could get ~~at the~~ through the poor girls bedroom window was stopped by the plan of using close Shutters; in fact the place was a typical natural and artificial Malaria ground described by Candler (See Candler on the Etiology of Phthisis)

The patient died at the age of 24 by haemorrhage.

Dr Buchanan, the well known & distinguished medical officer of the Local-Government-Board, in a report to the Privy Council on "The distribution of Phthisis as affected by dampness of soil" has collected a mass of evidence, which is difficult to reconcile to the Sputum dust theory, he found, as a rule, the rate of diminution of Phthisis to correspond with the drying of the subsoil, eg Salisbury 49 per cent. Ely 47 per cent Rugby 43 per cent; A special enquiry following these observations confirmed their accuracy, Dr Bowditch of Boston at the same time, but independently, discovered the similar relationship

Medical officer

How what would be the effect of damp on Sputum dust?
We are told the bacillus cannot reproduce itself at the ordinary temperature existing outside the body.
We must therefore conclude that spores, incapable of reproduction, contained in dry or moist sputum, are present as the "causa malorum".
The effect of dampness would prevent the particles or "ashes" being circulated in the atmosphere of the room, & would cause dust & other particles to adhere & settle so as to efficiently cover up the sputum, & by favouring the development of fungus growths tend to choke the life from the spore.
On the other hand, dampness by producing a tendency to catarrhal conditions of the respiratory passages, would favour the conditions under which the capture of the bacillus could take place.

Altitude. The most notable advance in the treatment of consumption during the present century, has, without doubt been the total change from a course of rest, warm clothing, & medicine, to an out-door life of activity even amid extreme cold: Nothing has tended to bring about this change so much as the results obtained by residence in the High Altitude Sanatoria.

Speaking generally, Phthisis is found to decrease as the altitude increases, That this is not altogether due to rarity of population can be gathered from the fact, that there are large cities both in Mexico & The Andes, where along with density of population above sea level the cleanliness & sanitary excellence can be said to be conspicuous, yet Phthisis is all but unknown. eg Puebla elevation 7.500 feet population 80.000 & Mexico elevation 7.500 feet population 320,000.

That this immunity is not simply due to rarity of air Dr Emilio Muller has shown, for in Switzerland there is no such complete immunity of the mountaineers, the rate above sea level does depend altogether upon the elevation of their dwellings.

Again in many mountain Sanatoria, such as Davos Platz, there must be an abundance of Sputum dust, a great deal of this must congregate inside the dwellings where cold or other Aseptic factors cannot come into force. Artificial methods of warming are in vogue & the cleanliness is at any rate not superior to that practised in England, here there is a soil in abundance of a kind known to be satisfactory to the caprices of the bacillus, here also in abundance is the seed, yet again no fruit.

Enough at any rate has been said to throw considerable doubt on the "Pure Parasitism" of the Bacillus Tuberculosis, & to give rise to a feeling that somehow or other Phthisis can arise de novo, though occasionally the circumstances are such as to allow of personal contagion.

Now is what theory or theories will all the clinical facts observed in the study of Phthisis conform?

1st The cause of Phthisis must often have a very limited local origin

2nd The one factor common to all methods of climatic treatment is apparently Sunlight.

3rd Hereditary influence plays a most important part in all

investigations of the subject.

The first is used by the Contagionists in favour of their views, the same house, in spite of change of tenant, repeats itself in producing the disease, & proximate houses also suffering, two of my cases viz Edward B & Mary W would uphold this view. But may not the house from construction & situation be to blame?

Secondly In one class of cases we prescribe a climate of dry cold in another, as the ocean voyage, an atmosphere saturated with moisture, in another warmth &c &c, apparently the only factor in common is Constant Diffuse Sunlight.

Thirdly, Heredity or Hereditary influence, whichever we may choose to call it, is without question a most potent factor in Phthisis; in what exact proportion it is difficult to determine, various estimates have been made, in which from one to two thirds of all cases are referred to this cause. exactness is impossible, in the former class the tendency is to over estimate its value, in the latter to disguise any such taint. in all cases it becomes impossible to say how far heredity is to blame & that we are not dealing with a independently contracted attack.

Caudle's suggestion that the old family mansion may suppy the heredity, in some few cases, may be correct.

Clinically however no doubt exists, that a Hereditary History largely increases the chances of falling a victim to the disease, influences the prognosis materially for the worse.

Considering the rarity of tuberculosis in the foetus & the comparative infrequency during the earlier years of life, & that the bacillus is known not to reach the blood till an advanced period of the disease we may infer, that except in cases of Tuberculosis of the Genital organs or general tuberculosis it is not the bacillus that is transmitted if it is not true heredity.

It is a common observation that Children resemble their parents both physically & morally, this resemblance extends far beyond the limits of the family circle & sphere of influence

A few years ago it was remarked that the sister of one of our English Dukes bore a strong resemblance to her Ancestress Nell Gwynne; few families possessing portraits of their members for countless generations cannot point out similar effects of heredity

Straw that has been used for litter in Lions or Tigers cages is totally unfit for horses, the smell terrifies them, though countless equine generations have passed, since their Ancestors had cause to fear attack from such enemies (Laycocks Organic Laws of personal & Ancestral Memory

Whilst these effects are well known, their Cause is obscure.

Darwin Summarises the matter when he says

"We must believe that the life germ invisible to the naked eye is crowded with characters proper to both sides of the body & to both Sexes & to a long line of male & female Ancestors.

"The germ of a defect or quality may be transmitted from generation to generation in a latent form & suddenly revive under favourable conditions"

(Darwins Variation of animal & plant life under Domestication)

This last quotation supplies us with an explanation of the Heredity of Phthisis

The "germ of a defect" is transmitted latent, reviving under those conditions, whatever they may prove to be, which give rise to Phthisis; The bacillus, from wherever brought, finds the soil more congenial & suitable for its growth & development, exhibiting that keenness of distinction, which is so remarkable in the life of micro-organisms & which was, perhaps, a cause of the failure of Koch to cultivate the one in question at natural temperatures external to the body.

Briefly then, to my mind, the following theory modified from Candler would fit ~~it~~ in with our Clinical experience, & yet allow us to acknowledge the undoubted [illegible] of the disease

(Life History)

The Bacillus Tuberculosis is a parasitic form of a fungus, which grows on matrices external to the body, this fungus, which under certain circumstances, may grow luxuriantly, without result at length produces its characteristic fruit. This entering the air & being inhaled, may & probably if the continuation of the infection be sufficiently prolonged, does, become lodged in the lungs & there become parasitic, being to some extent a degenerate form of the free parent form. This, which is, of course, the Bacillus Tuberculosis, in its turn produces spores, capable under suitable conditions of reproducing the parent form viz the bacillus, but as D^r Koch allows, this pure parasitism is of the feeblest description, self limited & tending to perish outright, unless they find their way into the blood & lymph streams, when the life cycle from bacillus to spore & spore to bacillus may proceed with the strict regularity of pure parasitism. Whether the spore, on the other hand, can, under any circumstance, reproduce the free growing parent form, is a difficult matter to answer, Cohn suggests this as possibly occurring among the excreted products of a tubercular patient. To accept such a view will demand a most careful investigation, as to the treatment of sputa matters. For my own part, I am inclined to think that the spore cannot reproduce the free growing form, but that its reproduction takes place asexually in one or other of the numerous ways witnessed among the moulds.

This It is a fact worthy of note that Koch found some of the moulds stained with the characteristic staining of the tubercle bacillus

I am not aware that the above theory contains any thing inconsistent with botanical knowledge.

This Bacillary mould grows both naturally outside ~~in the Earth~~ & artificially inside houses, under certain conditions of warmth & moisture, but more especially in the absence of direct Solar radiation.

The acceptance of some such theory will cover all the arguments met with in the consideration of the subject, account for & allow a rational understanding of the importance, of Tuberculosis in cattle (a subject of much interest, which cannot however be discussed here) & yet admit the correctness of Koch's observations in the main. To accept his doctrine in entirety, would, to quote the words of Jaccoud "influence neither the Pharmaceutical Climatic nor Hygienic treatment of Phthisis, its mission would be ~~mainly~~ merely Nosogenic"

The broader aspect, which I have sketched, would, on the other hand, if adopted by Sanitary Authorities, tend to Ameliorate the condition of the poor in many respects, & promote a most thorough system of cleanliness, though, not admitting the extreme views of the Viennese Medical Doctrinem Collegium regarding isolation, would nevertheless appreciate the dangers arising from a close intimacy with a case of Phthisis, especially to one hereditarily predisposed, or one who, from the nature of his or her occupation, was either debilitated & presented raw denuded surfaces in the respiratory tract, the result probably of an inhalation of solid or gaseous irritants or previous inflammatory attack.

The following is an example of conditions which I have frequently found existing in the course of my practice.

Large low rooms 18 feet by 18 by 9 containing only one small window 24 inches by 18 inches, occupied as sleeping apartment by 3 or 4 outdoor workers, no other ventilating shaft than the window (& no fireplace). On Analysis of the air during the night revealed CO_2 to exist in quantities varying from 2.5 to 4 per 1000 volumes. Sunlight could never penetrate those parts of the room where the beds stood, & ventilation even with a constantly open window was impossible

How utterly impossible it is to ventilate a room without other means than a window the following experiment which I conducted will show

A thoroughly cleaned room, nothing in it but the bare necessities for a bed room, window 4 feet by 4 feet & open all day, no impediments to the free current of air, Cubic space 840 feet, was occupied by one adult at night, the air was examined 4 nights, & the lowest impurity was 1.75 CO_2 for 1000 volumes.

Acting upon the ideas suggested above, I can confidently assert, that where my efforts have been appreciated, & acted up to, the results have been sufficiently satisfactory to stimulate me on to further research in the [illegible].

The first case, says Dr Murchison must have arisen de novo The earlier records are of little use to us in investigating this question, it is at a comparatively recent date in the history of the universe, that Small Pox was differentiated from Measles and Scarlet Fever; Still more recent is the time, when we became able to distinguish with clearness between these, last named diseases, whereas we owe it to Sir William Jenner that Typhus & Enteric fever are not considered as one and the same disease. At a time when the very origin of man as a separate species is a subject of much bitter controversy, a study of the first origin of diseases would involve us in a deep religious & Metaphysical study, ending with certainty in Chaos & despair. But if Conditions arose in the past so as to give rise to new specific diseases, & to cause a gradual evolution of one out of the other, then why should not this occur or be occurring at the present date?

Again Error has, according to Murchison, arisen from not properly estimating the enormous differences, which exist between those separate diseases all of which have a parasitic origin.

Probably this is so, I believe also, that the enormous difference existing in the same disease under different conditions, both in period of incubation & infectiveness has not been sufficiently appreciated

I have already drawn attention to a possible connection between diarrhoea & Enteric fever & spurs had allowed a still closer connection could be shown between sore throat with little or no infective power, & scarlet fever, which itself varies from a mildly & slightly infective disease to the greatest possible malignancy.

The chief interest in the subject of the etiology of Scarlet Fever rests at present, with the observations of Power & experiments of Klein, which give strong grounds for assuming that an illness of very slight constitutional disturbance occurring especially, if not entirely, in recently calved cows, & accompanied by characteristic local lesions containing a micrococcus & which satisfied ~~is disturbed~~ all Cheyne's requirements, is one cause of Scarlet Fever, which is distributed chiefly by the agency of milk.

(in Reports L.G.B for years 1886.

In the 1882 report above referred to Mr Power remarks R.
" The cases of scarlatinal outbreaks due to milk, have commonly been characterised by a small mortality, as well as by mildness of the disease induced, in many such outbreaks & notably it was so in the present instance, a considerable proportion of the sufferers have only had sore throat of little severity, & further this milk scarlatina has on this as on other occasions been little prone to spread to other persons."

This statement contrasts strongly with a few cases, to which I now wish to allude, which show a very different infective power.

On June 18th 1888 a case of scarlet fever occurred at Highfield a small dairy farm, for which no cause was assigned, the cows & all pertaining to the sale of milk were removed =

did not return till July 30th, & even now the greatest possible precautions were taken, those in the house not being allowed near the milking utensils, nor any of the milking apparatus being allowed near the house.

On Thursday evening August 2nd R. J. aged 7 years on returning from a large public picnic sickened, growing gradually much worse and died after 36 hours illness at 6 am on the Saturday of this malignant Scarlet Fever.

On Friday morning two other children of same family sickened, both had very severe attacks, with a tendency to profound coma.

By 10 am on the Saturday all the remaining members of this family had been removed except the eldest daughter J. J. who had returned late on the Thursday evening after 2 months absence from home.

On Saturday evening a child was brought back home, having sickened

On Sunday morning ~~another of on this date~~ J. J broke down.

On Tuesday morning another child returned ill.

These last 3 cases were all second attacks, JJ having 6 years ago nursed 5 cases of fever without contracting it: on this occasion, along with the last two mentioned cases, her attack was a well developed & moderately severe one.

On Tuesday a case occurred at Highford, this child, A6, had returned home on the same date as the cows. (July 30th).

On this date (Tuesday) also two cases occurred at the cottage next door to the J's.

Seeing that Highford supplied 96 families & these were all the cases, it is difficult to believe that the milk was to blame.

A6 who took ill on Tuesday August 7th had, on the Saturday August 4th accompanied her Father with the milk for the first time.

The J.J case proves that she at any rate caught the infection on the Thursday from the malignant attack, it is therefore fair to reason, that the following only traceable connection should explain the other cases. I may state that I was at very great trouble to investigate this matter as the public seized at once on the milk theory so persistently that the farmer seemed likely to loose all his trade,

On Saturday morning A.G brought in the milk to the J.s not being observed she had to sit for over an hour before being attended to, during this time she was known to have been in close contact with the child who took ill on the Saturday afternoon, & with other members of the family, who were moving to & fro from the death bed, articles from this bed were brought close to her, to be placed in disinfecting solution, from this house she went straight to the cottage next door, was taken in & made to relate all that had happened & was happening at their neighbours, this curiously cost both the young girls their character, as they both succumbed on the Tuesday (the same date as A.G) A.G then returned home a distance of a mile & a half.

A source of infection of the first 3 cases was to some extent proved, on the previous Monday July 30th these 3 children had been with a little girl, who had been sent into the country because her brother was suffering from Scarlet fever at home, this child, as seen afterwards by me, was to my mind affected with slight post Scarlantinal dropsy.

These cases, whatever their origin, afford a strong contrast to those due to an animal origin described by Power & prove to some extent, what I mentioned 6 years during the Epidemic observed in different attacks of the same disease.

how what produced the disease in the cows described by Power? No doubt a infection from a previous case of Scarlatina might do so, but in the researches alluded to, such an origin was not discovered, and it is at any rate a question not quite decided, whether even this would produce the disease, unless the cows had recently calved, as if the parturient state were, somehow, necessary, so to develope the characters of the microbe as to render it potent.

Unfortunately we cannot deny that Puerperal Fever can arrise de novo, not only is this the case, but very dissimilar causes apparently give rise to this dread disease, which in its turn is so terribly infectious, under certain conditions, as to those who present the suitable soil for the capricious microbe. On June 20 1887 (Jubilee day) Mrs L a lady in good position was confined quite naturally, on the 3rd day "things went wrong" She died of Puerperal Fever on July 2nd.

On July 27th her three children (all except the baby) were brought to me, suffering from undoubted post-Scarlatinal Nephritis

These facts admit of two interpretations, either Mrs L contracted puerperal fever from an infection common to herself & children, which in the latter produced Scarlet fever, & an infection Puerperal fever having been set-up, produced, as in the nearly analagous cases of the cows, though some peculiarity of the puerperal state produced Scarlet fever.

The real explanation is I venture to think, that a common infection produced under one series of circumstances, puerperal fever, under other conditions Scarlet Fever

Much remains to be said, my sketch is of necessity, both on account of the facts & the time at my disposal, imperfect & unfinished, it may however remain unsaid if my object has been attained viz to show that a more extended view of the parasitic origin of disease, though not by any means necessarily on the lines I have laid down, will be found very greatly to the practical value of that theory, by a more intelligent & rational System of preventative treatment

Finally I submit the following as reasonable deductions to be drawn from the premises laid down

1st That the acceptance of a parasitic theory does not prevent the acknowledgment of a diverse origin, because the parasites may arise independently of preexisting cases & may assume specific properties in the course of their development under suitable conditions.

2nd To deny some such theory will compel us in many instances to assume the omnipresence of many of the micro-organisms in question.

3rd That it is difficult to see why conditions, which were sufficient to evolve the disease in the first, should not occasionally, at any rate, be in action now

4th Clinical observations warrant us in believing that different diseases & different attacks of the same disease vary so greatly in their specific powers as to leave only a small bridge between specific & non specific.

5th That just as artificial cultivations have succeeded in producing an attenuated virus of vastly different properties in degree to the original, so a natural & more complex cultivation may be assumed to be even more perfect & extended in this respect.

The Subject I have endeavoured to lay before you is one still shrouded in mystery, & is an ever increasing field of interest & instruction, for those of all moments which come even to the busiest of us, and so long as we guard against ~~allow~~ the danger of our Speculations & theories taking the place of well established facts, will enlighten & relieve the monotony of practice, making the arduous duties of a General Practitioner full of interest & delight.

John Archibald Jackson
M B & C M Univ Glas 1887.
L S, S C Univ Dunelm 1890

Carntyne.
Newham
Northumberland.
June 1890.