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<th>On typhoid fever in India</th>
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<tr>
<td>Author</td>
<td>Bedford, Charles Henry</td>
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<tr>
<td>Qualification</td>
<td>MD</td>
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<td>Year</td>
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ON TYPHOID FEVER IN INDIA:

A Thesis presented to the University of Edinburgh for the Degree of Doctor of Medicine.

By Charles Henry Bedford, M.B., B.Sc., M.R.C.S., Surgeon-Captain, Bengal Army.

For graduation on 14th of April, 1892.
55, George Square, Edinburgh.
9th February, 1892.

I hereby certify, on my honour, that the thesis which I have lodged for the degree of Doctor of Medicine, of Edinburgh University, has been composed by myself, without any aid from any other person. I have complied with the other provisions of the Statutes, for the purpose of graduating as above, in April, 1892.

Charles Henry Sedgford,
M.R.C.S., Surgeon-Captain, Bengal Army.
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The numbers placed above the line refer to the Bibliographical Chapter.
* denotes "vide footnote."
Chapter 1. Introductory.

In this Thesis, it will be my endeavour to consider the large subject of the Typhoid or Enteric Fever of India in as short & complete a way as possible. The questions of its history, causation, & prevention are the points of special interest in connection with this disease, & will receive relatively more attention. This subject has not, up till the present, formed the Thesis of any candidate for the M.D. degree of the University, so far as I can learn. At the outset, I wish to explain that I left India for home very hurriedly, being on sick-leave; I was unable to collect & bring home with me a set of thermometric records I had intended to incorporate in this Thesis.

I had, moreover, hoped to have been able to experimentally investigate some points in the Pathology & Bacteriology of Indian Enteric Fever, also for incorporation here; but, once landing in India, it has been impossible, the circumstances seemed to have
considered against my intentions in this
score. Landing in India in March, 1890, I was never left long at any one
station, as is too often the case with
junior Officers of the Bengal medical
service. The impossibility of carrying
on a connected piece of work; the
frequent difficulties of obtaining, or pre-
serving (when obtained) from Europe, in
India, the necessary research apparatus;
the insanitary conditions of climate with
regard to bacteriological work;
the liquefaction of solid media by
the continuously high temperatures in
the hot season, or by the damp heat
in the rainy season; the absence of
gas for sterilizations, regulation of
incubation apparatus temperature;
the absence of the necessary material
at some stations; the impossibility
of carrying about culture apparatus
with me in an Indian train, where the
heat of which have to be experienced
to be realised; the necessary interruption
to one's work consequent on being ordered
off for a few days here or there on some special duty; the absence of
medical libraries to consult; 
I to "get
up the subject" by: all these have de-
termined me to seize the opportunity of
being once more in Edinburgh, & near
large medical libraries, to write my
Thesis now, not later when, perhaps,
more leisure might come, but not the
opportunity of consulting works from
a large medical library.
In this Thesis, I have dealt with 
fever as seen in stations & cantonments,
not in camps & on active service in
the field.
I have not thought it necessary to advert
particularly to — at least, to the kind of
giving a chapter to — the conditions of life
of the European in India. In the chapter
on etiology, such very brief explanations
of these conditions, as are wholly unfamiliar
to the dweller in Britain, will be given.
In the same chapter, I shall not enter into
the discussion as to Eberth's "Saliclis
Typhosus" being the true pathogenic organism.
of entire fever or not, but will range myself on the side of the majority of distinguished European pathologists, bacteriologists, as practically acknowledging it to be the "vita causa", in the absence of animals which take Typhoid fever, either naturally or by inoculation.

In no case shall I attempt to sketch the European phase of opinion or belief in detail, for purposes of contrast with that obtaining in India.

I need hardly refer to the enormous loss the State suffers from deaths and invaliding from this disease in India, the prevalent opinion there that it is the fever which bulks not largely as a mortality-producer: these are facts well-recognized by all men of medical-literary tastes in this country, I do not require to be mentioned as an apology for this "brochure" on the subject for a Graduation-"thesis".
Chapter 2. Historical.

It was undoubtedly the publication of the observations of Dr. A.P. Stewart, in the "Edinburgh Medical Journal" in 1840, and later in 1846, of those of Sir William Jenner that gave an impulse to British physicians in India in the work of differentiating the fevers of that country. Up till 1853, the general impression in India was that no such disease as the Parisian Fever of Louis, "Toxinentritis" of Bretonneau, or the Typhoid Fever of Jenner, existed there.

The writers on Indian diseases, before the authors of the early classical works on Indian medicine, were chiefly naval surgeons, — of whom Johnson heads the list. The observations of these writers were necessarily restricted to descriptions of diseases encountered by them in port, or within a comparatively short radius of the port where their ships were lying. Yet even they noted cases of "fever with bowel complication," many of which must have been cases of true typhoid fever. Dr. John Clark gave an interesting account of the fevers which occurred on the H. E. I. C."
ships from 1770-85. In the ship "Liton", after leaving Bengal, there occurred so much fever that the sick list numbered sixty, when they arrived at the Cape of Good Hope. In the beginning of the fever, inflammatory symptoms chiefly prevailed, frequently with brilliant vomiting, "but in its course it "changed into a typhus, or was succeeded "by a flux." Now, it is not too much to suppose that many of these cases were veritable cases of Typhoid Fever. Remitting fevers of such severe type would not present under the healthful malaria-destroying influences of a sea-voyage. It is possible that a few of these cases were malari"al in origin, as it is by no means uncommon for the change of air, diet, and nervous disturbances consequent on a sea-voyage to induce an attack of typhus in a malaria-saturated subject, but such attack would be but transitory. Again, malari"al diarrhoea, as a sequel, is common enough, especially when a putridic taint is present; this would seldom be absent on a long sea-voyage where salt water would be
largely used, in addition it is well-known that malaria helps to a very considerable extent in the production of cancer by the blood-changes it induces.

The symptom of bilious vomiting is common to both Enceph醚 Fever, Malarial fevers in their early stages. But the severity of type, the change to a "typhus" kind gives probability to the idea that many of these cases were neither exclusively malarial in origin nor true typhus from overcrowding on board ship, for our author would have been, it is presumed, able readily to diagnose such a common condition then as "foul-fever." In 1784, seven ships, stationed at Redjorce, at the mouth of the Hooghly, lost 170 men there, the diseases which prevailed were Remittents, Intermittents, a continued fever probably identical with Enceph醚 Foe. In fact, "ship-fever," as it was then called, was undoubtedly Typhoid Fever, as has been demonstrated frequently.

Dr. Miers, of the Bengal Medical Service, argues that Charles Carter, Surgeon of the
"Indea, frijat, described the true febric of India under the designation of "bilious fever & flux."

The general teaching of the older school of Indian physicians was to describe fevers with reference to the seasons—"artful fever," in the hot weather; "bilious fever," in the rainy season; & "infective fever," of the cold season. The first & last were called Continued Fevers, & "bilious fever" was termed Remittent, although all three were presumed to have the same cause.

Sir J. Ameesley, of the Bengal Medical Service, published, in 1828, a work on Indian Diseases, in which he notes "marks of "Disease of the small & large intestines," which were "confined to their inner linings," that "especially the duodenum & termination of "the ileum are very frequently diseased" "in their mucous surfaces, which are "inflamed in patches ..... loaded with "small ulcerations, particularly the termination "of the ileum." "In several cases, the ulcerations which are, sometimes, large & far apart,"
"at other times, small ulcerated, have"  
"nearly penetrated the tunics of the intestines,"  
"in a very few cases we have observed"  
"the occurrence actually to have supervened,"  
"the contents of the bowel being partly"  
"effused into the peritoneal cavity."

Again, Sir Ronald Martin, of the Bengal Service, describes a condition which he  
terms "infective continued fever of the cold season in Bengal," noting its dangerous  
insidiousness; its frequency; the fact that  
even the most carefully living Europeans  
are attacked — of all ages, sexes,  
men, however, being most liable; the very  
gradual onset; the fulness of the belly;  
intense headache, delirium. He  
attributes the cause to "exhaustion of the  
liver by the hot season trains," noting  
the frequency of inflammation, ulceration  
of the intestinal mucosa. The patients  
generally recovered if there were no severe  
abdominal complications, but when a  
"generally typhoid condition" supervened  
there was "great peril." The treatment  
was bleeding, Colonel Paul Jacob, followed
by active pulizes; along with "toal ab-
"stinance from food, & coal drink!"

Dr. Turner, of the Royal Service, wrote, in 1825,
in the "Fowers of Mezpel", was well acquainted
with "Glydrot" fever as to "typhus with abdom-
"inal complications" of Richard Bright,
or the "Dolhierterik" described by Bret-
onneau, in 1825. He asserts that
"typhus is rare in India"; to speak of convulsive fever of the cold season, etc.
In a few rare instances where patients
have died of protracted fever of this sort,
"superficial ulceration of the mucous
membrane of the small intestine were
found..." In reason for not dismissing that
"pathological condition a primary condition,
existing at an early period of the disease,
"is that active purgatives may be repeated
"daily for a long time at the commence-
ment" without producing irritation. In fact,
"they almost always afford relief, whereas
"we do sometimes find that active purgatives
produce a degree of irritation at a later
period, i.e., when a fatal termination takes
place afterwards, ulcerations of the small"
"intestinal fistulae generally in these subjects."

"more studied observations should prove that"

"these ulcerations of the small intestine exist"

"generally in the cases which terminate fatally,

"that such a pathological condition is met with in the inspection of subjects that had"

"died of other descriptions of fever in Bengal;"

"he would be inclined to adopt Dr. Bödeker's opinion

"that a peculiarity of this disease would be

"ascertained, which 'combined with the

"exclusive (sic) prevalence of the fever in the

"cold season, its insidious invasion, obscure

"symptoms, slow progress, protracted course,

"attended with protracted delirium + delirium;

"the organic change at its latter stage right

"establish a resemblance to some cases of

"European typhus, although the resemblance

"be not strictly correct in all its details."

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"Footnote. Richard Bödeker held to think that "in

"recent cases of typhus there is generally constipation

"the stools should be opened. But in this disease (typhus

"with abdominal bowel complication) this is impossible, since

"only in 3/4 of cases did we see the disease with

"one of marked typhus with bowel complication, which is a

"falsely serious condition"
Dr. Edward Longe, of the Bengal Service, writing later recognised the fact that poisoning had met with true typhoid fever, from the above account.

Surgeon-General Maclean, of the Indian Service, later Professor of military medicine at Kellog, saw true typhoid fever in 1838 at Madras City, & afterwards at Secunderabad, in India. He treated them then as cases of "continued fever with bowel complications," & says, "I saw such cases, & I would unhesitatingly diagnose them as typhoid fever." Dr. Macleod, of the Bengal Service, writing in 1843, says: "A state of ulceration of the glands of Peyer at the end of the ileum also occurs in cases of typhoid remitted with fatal outcome symptoms, just as it occurs in European Continued Fever with typhoid symptoms, with similar bowel complications."

In the first edition of his book, Macleod also stated that the disease was unknown in the clinical study of fever.
But in his second edition he, though admitting the existence of the disease in India (as was demonstrated in the India Office Sanitary Report for 1861), steadily maintained that the malady was long of rare occurrence in India. This is probably, so to a large extent, as the number of the men serving in India then was very considerably greater than it is now.

Sir W. Moorty states that Allan Webb, of Simla, was the first to mention typhoid fever as existing in India, in 1862. But he probably referred to the typhoid state, as did also Kirk, who, in 1848, wrote: “At the termination of the rainy season, the beginning of the hot season, congestive typhoid fever is abundant all over the country.”

It was, however, Assistant Surgeon Serjeant, of the Bengal Service, who did for India what Osmundair & Jenner did for Britain. In 1854, he published an account of three cases observed by him in Burnaham, and, again, in 1857, he describes the details of some cases, of which two only were recovered.
The others were found, at the autopsy, to have Peyerian ulceration, especially in the lower part of the ileum, in cases partially separated clumps. The solitary glands were inflamed. Most died about the end of the third week; all these cases came from the same regiment.

Thus to Serevo must be accorded the honour of identifying the typhoid fever of India with that of Europe.

In 1855, Dr. Arbuthnot Lane, of the Bengal Army, described Paratice first among the native prisoners in the Jemere Ghat, in Rajputana. He was the first to recognize the existence of true typhoid fever in Indian natives—a discovery made quite independently of Serevo's first discovery of the disease among Europeans in India.

Prof. Edward Jowett, of the Bengal Service, described the disease admirably in a clinical lecture given at Calcutta in 1858, noting the mild onset, long duration, and obdurate progressive and intransient character of the symptoms. He cites seven cases,
all of considerable duration, & presenting the features of nervous disorders, typical typhoidal stool, & adynamic signs. He notes that Roscoola was rare.

by ens, Moffatt, Deasey, O'Driscoll, Barclay, Clyhorn, Greene, & others of the Bengal Service; Cornish, Room, & many general Jordon, in Madras; they, & the late Sir James A. Stansbury, K.C.B., R.E., in Bombay, have severally enriched our clinical & pathological knowledge of the disease since then.

In 1861, Typhus Fever first appears in the official returns, 24 cases are reported from Bengal, 2 from Madras, & 1 from Bombay Presidencies. From this till the present year there is no year in which the disease has been absent from the Returns, though the amount fluctuates considerably. It was in a paper by S. T. N. Dryden entitled "An age of anxiety of disease as affecting the sickness, mortality, & invaliding..."
of the European Army in India," that the recognition of the disease to which the disease prevailed was brought about, consequently the occurrence of the disease leading to the official returns.

Brigade-Brigadier Baron, on the other hand, formulated his experiences, having 15 years in large European military hospitals & large barracks in India, stating that he had only seen one true case of bubonic plague at Jumurr, in the Punjab.

And Prof. Chevers, of the Bengal Service, stated that, in 1866, the splendid pathological museum at Calcutta only contained 9 preparations of bubonic fever lesions—two from Dr. Bonsor, 1 by Lastwell, 3 by himself; & for a day, "Thus 120 beds in the largest hospital in India, from 1858-76, only nine cases." I should here point out the very obvious fallacy here.

(1) that many true cases of the disease treated no doubt recovered; (2) that many true cases of bubonic from were treated as Material Remittent Fever of malignant or lymphoidal type; (3) that many cases
of such habit fevers died in hospital, but were not allowed to be examined "post mortem".

Cheers for us to say that, "if this disease has long been common in India, it is a very extraordinary fact that several of our best and most careful observers should have met with so little of it, although they sought it diligently in the light of some experience." Here he introduces the question of the "personal equation" always dangerous. No one would question that Richard Bright, for example, was one of our best and most careful observers" yet he treated many cases of "typhus with bowel complication" as he called them, and failed to differentiate it as a separate disease entity. His disease from phthisical typhus. And in the history of Indian medicine, Swinson, morehead, Amers, Martin failed to identify the Indian with the European Typhoid Fever. Why these last named observers met with so little of the disease was that they diagnosed true cases of it with "as declarative recurrent fever; besides
there was not then, as now, the very large proportion of men at the most susceptible age for acquiring typhoid fever.

In 1869, Surgeon-Major De Rozzy, of the Bengal Service, notes a large number (p. 121) of cases of Native fever among the native prisoners in Ounjap Jails, where it began in the native villages. He goes on to say that its severity did not vary constantly with that of the famine, thus distinguishing it from Relapsing Fever. It affected more the native women than men (p. 187), because they spent most time in the homes, and larger doses of the poison from the contaminated air and water in the vicinity of their dwellings. And he states that out of a total of 91 deaths from fever, more than a third, i.e., 31, are ascribed to Native fever (p. 60).

In 1880, Deputy-Surgeon-Major Pinkerton, Bombay, states that Native Fever has existed there for more than fifty years as

"Austic says, "Native Fever is the special epidemic of humbrous, conservative, rural districts."

in India
"twenty-one days' had Bombay Fever"; and, among the natives, under the name of Remittent, simply as "fever," it causes great mortality all over Bombay Presidency.

We must now shortly consider the history of the Prevalence of the Disease in India. Upon first drawing attention to the great amount of sickness and mortality the disease gave rise to, points out the significant fact that the ratio of "fever" mortality (in the gross) of recent years, if taken in relation to months newly arrived recruits, is nearly absolutely identical with that of "native fever," at the present time. That is to say, that the bulk of fatal fevers in India is, then, been from the first, native in nature.

Since first the disease was allowed a place in the official returns in India, there is to be noticed a marked excess of the number of cases during the later as compared with the earlier years. This results from the gradually increasing improvement in the diagnosis & differentiation of fevers.
into their proper categories, rather than to an actual increase.
It is to be regretted that the returns in many instances, have been vitiated by a spirit prevalent among the authorities practically insisting on the adoption of their personal views by their subordinates. For instance, a few years ago, in one of the Presidencies, all medical officers were hindered from returning any cases as Enteric Fever, inasmuch as the Principal Medical Officer of the Presidency held that no such disease as Enteric Fever existed in India.
"As contraire," another P. H. O. ordered all cases of fever, which, at the autopsy, showed ulceration of the bowels to be returned as Enteric Fever. And I know of a case where the Junior Medical Officer at a station ordered his junior officers to return all cases of fever continuing more than three days to be returned as Enteric Fever! Up till 1868, Enteric Fever is given in bulk for each Presidency, with no information as to its prevalence, or otherwise.
at stations or in camps, as a rule.

Between 1859-1868, the main defects of sanitation noted were badly ventilated barracks, existence of cess-pits, bad water-supply, bad drainage, the dangers consequent on the proximity of large, crowded native communities, in villages. The main improvements of this period were new barracks, the purchase of land for conservancy for Nagpur & Bombay—earth at all in hadmus—improved drainage of several stations.

The water-supply, chiefly from wells, with the dangers inherent to that system, and the danger from the surrounding native community still remain unaltered.

In his Report on the prevalence of Intermittent Fever in India, in The A. M. D. Blue Books up to 1876, Brijson stated that it was "a matter of popular observation" that no regiment or battery escapes "Intermittent Fever in its first year's service in India," whatever cantonment be selected. Of 73 Regiments & batteries which arrived in India between 1871-7, nine only
remained free from the disease in the first year after landing. He compares the prevalence of the disease at various stations, per 1000 of average strength:

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<tr>
<td>Home</td>
<td>.99</td>
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</tr>
<tr>
<td>Gibraltor</td>
<td>4.04</td>
<td>1.89</td>
</tr>
<tr>
<td>Naples</td>
<td>3.75</td>
<td>1.53</td>
</tr>
<tr>
<td>Malta</td>
<td>4.72</td>
<td>1.57</td>
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In the Sanitary Commissioner's Report for 1877, 233 cases of Typhoid Fever were reported of which 92 (i.e., 39 p.c.) were fatal, the admission rate being 4.1 per 1000; of these, 2.45 p.c. occurred at or under 24 years of age; 1.55 p.c. from 25-29; 0.99 p.c. from 30-34; 0.2% or none after.

Previous to this, from 1870-71, the admission rate fluctuated between 2.86 and 4.6, the mortality per cent. of those attacked varied from 60.8% to 30-49, or a mean of 43.73.

In 1878, 132 deaths from Typhoid Fever were reported, 90 occurred in men under 22 months' service in India.
The admission rate rose to 8.5 per mille, and during 1879 it continued high (8.0 per m.) of 1880 (7.9 per m.). The death rate of those attacked remained much the same as it was during the period 1870-77, for it varied from 43.01 to 46.17, or a mean of 44.72.

During 1881, the admission rate again fell to 5.6, but the percentage of deaths somewhat increased (47.28).

In the Report for 1883, the Surgeon-General with the Government of India says: "The tendency to return an increasing number of 'Continued Fever' as 'Interruption Fever' still continues in the Army of India."

"429 cases of fever were recorded as "Interruption Fever" as against 357 in 1882," i.e., 7.7 per mille of admissions for 1883, as against 6.2 per mille for 1882. There were 133 deaths from 'Interruption Fever' in 1883, or 2.4 per mille, as against 2.55 in 1882. Hence, 'Interruption Fever' was more frequent but less fatal in 1883 than in 1882.

In 1884, it heads the list as being the
to the largest proportion of deaths in the Anglo-Indian Army alone. The mortality was 3.31 per mille in Delhi; 2.05, in Bombay; 1.67, in Madras,—all of which exceed in ratio of deaths the decennial average. In 1885, the percentage of admissions for Ague remains unchanged, but the increase from Intermittent Fever is partly compensated by a decrease in that of Remittent + Simple Continued FEVER.

In 1886, there occurs in the Reports a summary of the views of medical officers as to the frequency of Intermittent Fever in India:

1. Intermittent Fever is the principal fever of India along with Ague + Simple Continued Fever, & some of the cases returned as Simple Continued Fever are in reality mild cases of Intermittent Fever.

2. There are separate Diseases called Intermittent, Remittent, Simple Continued FEVER, & Ague, the Differential Diagnosis of which is, as will be, possible through patience, perseverance, increasing Knowledge.

3. There are cases which can easily
### Annual Admission- & Death-Rates, per mille, from Enteric & other Fevers in India.

Compiled from the Official Returns, inclusive of the last issued report, i.e., for 1889, published in 1891.

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<th>Deaths per 1000</th>
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<td>Other FEVER.</td>
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<td>7.3</td>
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<td>437.1</td>
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<td>424.3</td>
<td>5.93</td>
<td>0.7</td>
<td>6.63</td>
</tr>
</tbody>
</table>
be recognized respectively as Ague, Intoxic, Remittent, and Simple Continuous Fevers; but difficulty of diagnoses, when it exists, corresponds to the reality that these affections shade off into one another, being due to one "cause constans," influenced by different circumstances.

(4) There is little or no European Subtive Fever, but a fever with catarhal follicular inflammation & cataract of the bowel is common, — an Intoxic Fever, but not the Intoxic Fever.

In 1889, the death rate from Intoxic Fever increased by 2 per 1000 over that of 1888 (6:11), the admissions by over 9 from 1888 (22:9). Bengal comes highest. Madras lowest for both admission & death-rates. Bareilly — the station at which I was last posted with the 7th Bengal Cavalry before leaving for home — coming highest: 77:9 admissions, 23:77 deaths; 75:4 admissions, 17:89 deaths respectively.

The last published Report of the Indian Government (that for 1889, published 1891)
shows that we have entered on a new and most important epoch in the Indian history of the disease. In the beginning of 1889, specimens of pleural mesothelial glands from two typical cases of Indian typhoid fever were sent to Prof. Bernhard Ficher, of Heil, a distinguished bacteriologist—who reported that he found the specific bacillus of European typhoid fever in all the specimens sent. How this proves that at least some of the cases treated in India as typhoid fever are such in reality, for the general consensus of opinion of the most able and conscientious bacteriologists and pathologists is that Shattel's bacillus is only to be found in true cases of typhoid fever, it is to be found in all such, except a few in which the failure to find it is either due to technical difficulties, or from the fact that the bacillus does not always last as long as the disease it produces.

Moreover, Surgeon-Major Rankin, of the
5th Bengal Cavalry, isolated, in 1889, from the albuminous urine of a patient suffering from enteric fever, a bacillus which, when cultivated in the nutrient agar-agar or on potatoes, showed the characteristics of the Bacillus typhosus, whose constant association with the disease - with it alone - makes it all but certain, in the absence of animals which take enteric fever, that it is the "vera causa" of this disease.
Chapter 3. Etiological.

In this chapter it will perhaps be best to consider the theories which have been advanced to explain the causation of Indian Typhoid Fever in three groups:
2. The Climatic Theory.
3. The Pyrogenic Specific Theories.

The first of the Humic Theories is that of (2) Stiehr: the Self-inpoisonment Theory. It asserts that the disease may arise by chemical & bacterial changes induced in the contents of the alimentary canal "under the influence of disturbing causes." It is certainly, as I have myself observed, frequent to find among newly-arrived men in India, the following conjunction of symptoms: foul breath; slight abdominal cramp; diuresis; headache; diarrhoea. When a surge is given much undigested decomposing animal infestas come away. How this may predispose to retoric fever by producing
intestinal catarrh, but it requires the presence of the specific germ to produce the specific disease.

Laveran has indeed pointed out that this theory has no precise facts to rest upon, hence I shall more fully mention it.

W. E. Porter's theory that it is due to the decomposition of the individual's feces in his intestinal canal is another example of a hypothesis unsupported by anything resembling a scientific observation of facts.

(B) Sir William Moor's theory of need not mention. He states his opinion that Ziyadid fever is not a specific disease at all, but simply a "phase of fever"; that all fevers belong to one genus—a view long since exploded.

He, moreover, describes the eruption of Scurvy Fever as only a "petechial eruption," i.e., it is identical with those of typhoid, dysentery, &c. It is sufficient to point out that while the typhoid resolves itself disappears or pressure, the last
true do not. New facts such as these are stated, it is surely scarcely worth while discussing this theory, of which I believe Sir W. Moor to be the chief disciple.

MARTIN'S VICARIUS THEORY

Surgeon-Major Martin, Judicial Staff, has brought forward a theory, which has attracted a good deal of attention. He ascribes Indian failure to the exhaustion consequent, in a larger or shorter time, — according to the individual's tissue-readiness to such stimulants — on the prolonged stimulation of the hepatic functions by thermal heat; in the excessive work thrown upon the liver by the increased amount of oxygen in heated air, — the consequent respiratory modifications. He thinks that the "intestinal glands" assume a "vicarious" and abnormal activity, supplemental to the hepatic insufficiency. This eliminatory function of the intestinal glands at length induces a "suppurative interstitia," he thinks. The reason the intestinal glands assume this vicarious function he states as due to an idiosyncrasy or weakness of the glands to this
abnormal function."
This theory would, he thinks, explain the "occasional spontaneous origin (of future fever), also its non-occurrence or rarity among the natives of tropical climates. But here the premises are incorrect, where, I would ask, is there any proof to be found of the assumption, by the intestinal glands, of a vicarious function either in this or any other condition? Budd held that the Peyerian lesions were only the local expressions of a general state; yet it is certain they do not occur till the fever has been in progress for some days, but Martin would make the Peyerian process the starting-point of the whole pathological condition. Again, the longer a man lives in India the more his liver becomes deranged, as Sir Ronald Martin demonstrated to give a satisfactory physiological explanation of theshould effect to find more vicarious action of the intestinal glands, a consequence future fever as age increased, but this is just what we do not find.
for is functional hepatic derangement a profound symptom of enteric fever.

Martin says the "intestinal glands" take on this "abnormal function", but it is

the solitary Peyrier's glands which are alone affected in enteric fever. Stammer's
glands + Lieberkuhn's follicles are in
easily more than a condition of "Cloudy swelling", which may occur in
any febrile state.

Again, the functions of Peyrier's + the
pollitory glands are absorptive not elimi-
natory, unless we are to believe a
most improbable contingency, the
direct reversal of their function.

And, moreover, natives suffer very fre-
quently from enteric fever, so I hope to
show later on; + the enteric process is
by no means a supplicative-enteritis.


Prof. Leon Colin, of the Vat de Grâce, an
eminent French Army Medical Officer,
holds that "saludal typhoid" exists, it
is the result of the combined action of
saludal + typhoid elements, (well described)
by Surf.-Surg. Woodward (U.S. Army) term, "typho-malarial" — that many cases of malarial fever merge into typhoid fever. It also holds that "all acute febrile conditions, accompanied by a marked alteration in the secretion of the gastro-intestinal complications, may induce the spontaneous development of typhoid fever (op. cit., p. 276);" that, in such cases, it would be impossible during life to recognize the affection, for the two diseases having ceased to be distinct — the remittent being transformed into intense fever.

While admitting the etiological identity of certain epidemics of typho-malarial fever, "il establit qu'il ne faut pas chercher en" "d'ahors de l'organisme malade les conditions" "pathogéniques de l'alteration des flèvus de" "Peyr dans une fièvre paludéen d'origine" "des arguments invoqués ont la lâchereté" "des lecons de l'interesse folliacéule, et" "l'identité des circonstances étiologiques au" "milieu desquelles apparaissent les fièvres" "pravo avec lecons utéritale (p. 366)"

"Quelques observateurs se refusant à voir"
"Dans la typho-malaria, une maladie con-
plexe, l'est considérée soit comme une"
"modalité spéciale de la fièvre typhoïde"
"(Borelli), soit comme une forme grave"
"de la fièvre paludique (Alton Medcalf).
"On considère une telle morbidité distincte (Hann)"
"which, he goes on to say, is due to a con-
fusion of processes which are essentially
distinct. He thinks he has proved
that the body can receive, at the same
time, two "miasmatic agents." And it
is held by many other observers that people
"sucked" with malaria are more prone
to receive "miasmatic" poison.
"But, on the other hand, there is no much
evidence of a positive nature against this theory.
And, although it is true that certain fevers
malaria are in no respect antagonistic
to the other, yet there is in reality no
evidence to indicate any causal or
essential connection between them.
Entire fever is certainly not a form of
malaria fever, for it occurs anywhere
in the plains of India, even where Ague is
unknown; when the two diseases coexist, they do not prevail contemporaneously, nor are those stations which are notoriously malarious especially remarkable for the prevalence of Subtropical Fever; in fact, the reverse very often appears to be the case.

Again, Ague attacks men of all ages while Subtropical Fever does not, nor do any number of attacks of Ague (except as those simply length of residence in India) confer any immunity from Subtropical Fever. Moreover, Subtropical Fever does not respond at all to quinine, which, however, cures Ague.

In South Africa, there exists a state of affairs which practically negates the idea of the existence of a highly malarial fever. Here, Ague or allied malarial diseases were observed to be almost by medical officers of wide African experience. The only attacks of Ague which were observed might equally have occurred in non-malarial Edinburgh, for they were in soldiers from India, in whom the latent malarial
poison (received in India) was provoked into a display of fever by chills, etc. All these cases — very few in number — were in old men who had all had long periods of Indian service. Dr. Porter, at Barar in India, only saw one case of enteric fever there, which nineteen of the admissions were from malaria. There is indeed no more sound for allowing that enteric fever can be caused into action by two or more sets of poisons than there would be for proclaiming the theory that any of the fevers of malaria can be affected by more than one specific cause. Finally, Colin, in his remarks on the alteration of the secretion of the intestinal glands affected in enteric fever, falls into the error made by Pasteur in supposing their to be ample but absorbing structures.

(3) Pfeiffer's lymph-water theory may be dismissed in a few words. Ranke, Roth, Fodor, and Buchanan have all shown its fallacies. And in India, it has been found that both cholera and enteric fever prevail whether the ground-water
is 8 or 10 ft. from the surface (p. 286). And the conditions of soil, impure from animal impregnation, a certain temperature, the influence of specific forms, which Pettenkofer says must also be present, in addition to the level of the ground water, are, in themselves, all-sufficient for the production of a typhoid epidemic, quite irrespective of any help from such an accidental condition as the level of the ground water.

We now come to (2) the Climatic Theory. Here there are two schools: (a) Farrar's; (b) Brydges.

(a) Sir Joseph Fayrer, of the Bengal Service, states, in his Romanic Lecture for 1882, at the College of Physicians of London, that there is a large number of cases of typhoid fever which are strictly caused by the inversion of the physiological functions, consequent on the European's landing in the tropics—"geographical position + "climatic influence, heat + moisture, organic "composition, microcosmata, + a variety of "aerial + telluric conditions, which are "more likely than a specific cause in India. But he does not deny that cases of true
Interci Fev (European) do occur in India. He would distinguish the two (p. 224) as "specific Interci Fev" or "Climateic Interci Fev," i.e., that the diagnosis between them is very difficult.

Surgeon-Major Duncan, of the Royal Army, criticises these views very ably: "In the collection of opinions forming the basis of the theory, we find scarcely any definite post-mortem accounts; cases are described which I consider to be utterly irrelevant to the point at issue; whilst, finally, there is an absence of proof that the cases brought forward to illustrate it were not due to a specific or putridgenic cause."

From an examination of the cases, the argument reduced to logical expression is the following: "Cases of ulceration of the intestines are caused, in warm climates, by a variety of aerial and telluric conditions. But interci Fev is accompanied by ulceration of the intestines. Therefore, interci Fev is caused by a variety of aerial and telluric conditions. Finally, Sir J. Fayrer himself considers his whole infrastructure for the proposition to
call the fever 'subacute' as distinguished from "the "specific" subacute fever, but Burdick "remarks that it is the subacute disease of "England, as it is of France & America."

"It is a peculiarity of the disease that it "is subacute everywhere... Two conditions are necessary... "to demonstrate as distinctly before" "they can establish these peculiar doctrines" on a scientific basis. The first is, that the fever "in any given case is actually subacute fever; "the second is, that all specific or pyhogenic "causation is absolutely excluded. But at "present these two conditions often have not "been established. I shall presently "show that the conditions for subacute fever, "on the specific or pyhogenic theory alike "are "a priori" in India.

The logical & philosophical rule to have "because of unknown laws where they "seem capable of explaining phenomena, "rather than to seek in some new & "totally unknown direction is sound. "And we must have these conditions of heat, "moisture, soil explained first, which are "said to be capable of producing this disease;"
defined as existing before we can make any progress.

In India, it will be shown, how unnecessary a climatic cause is in the presence of an overwhelming abundance of those fitch causes which elsewhere produce the disease. Rankeilour says, "There is no station in India, however carefully the sanitary conditions may be attended to, where local contamination of water easily accessible for drinking purposes—but not intended for such—do not exist in profusion."

I consider the case of Fort Asigarah, in Central India, to be one of the most powerful arguments against the adoption of either fitches, Dryden's, or other climatic theory. The conditions there existing for protection against Interic-Fever's introduction are practically perfect. The fort is situated on a rock much like that on which Winchester Castle is built. There are no native villages near it, and it is far distant from a railway-station. The water is rain-well, collected in care-
fully looked after & well-conducted tanks.
The conservancy arrangements consist of the
horizontal system, the ordure being lowered
from the Fort walls to the plains below,
where the animals from the adjacent
jungle room eat it up entirely.
The food, milk, & supplies are closely
supervised.
The consequence has been that a large
European garrison, at the Native Fever
age, a man almost, had no cases
for many years of Native Fever, though
all the conditions of a tropical climate
were present, & Malaria evidenced
its presence by attacking the men
in the form of Ague. Here the
specific cause was excluded, hence the
Disease was about:
how if Native Fever is due to the effects
of heat, & other tropical climatic conditions,
or to Malaria, or to self-imprisonment,
&c., here were the climatic & telluric
conditions, but where was the Native Fever?

(3) Bryden's theory states that Native Fever
is subdued by the exposure of immature
Constitutions to the conditions of tropical life. 

In dealing with this theory, we must take into account the (1) seasonal prevalence, (2) distribution of the disease; (3) the physical factors in the soldier which are said to predispose him to fevers. Fever in the tropics.

(1) Seasonal prevalence. This varies to a great extent, but we are safe in saying that the two maximum periods of prevalence in Bengal are (1) from April to May; (2) from August to October.

The second quarter of the year generally yields most cases, but in third quarter next, in places like Madras Presidency, the third quarter is the official season of prevalence.

One may here remark on the strangeness of the fact that Intermittent Fever, which is so closely related to local conditions, should have two annual periods of excessive prevalence in one or the same place at times when the local conditions differ so greatly as they do during the hot and rainy seasons. Malarial fevers have a distinct maximum in the rainy
ocean, on the other hand, this does not tend
to strengthen the identity of these fevers with
funicular fever, but rather to indicate the
outcome of imperfect discrimination of
distinct forms, one group (funicular) finding
the local conditions prevailing during the
hot weather, the other (malarial group)
there during the latter part of the rainy
season especially favourable to its development.
Again, imperfect diagnosis (for increase of
prevalence of malarial fevers during the
hot weather) should to some measure be
credited to funicular fever, "vice versa."
If this is so, then funicular fever is especially
suitable suited to flourish in the hot
seasonal condition, and again malarial
fevers in the rains.

Now, heat, moisture are the two prominent
features of the tropical climate, but both are
universal except in hill-stations, but we
find it flourishing in all climates in India.
— in the hills of plains of itself.

Another point to be laid great stress on
is that the disease is present in every
month of the year, as even when at
its maximum intensity this is found not to coincide with the heat maximum, but to be attained prior to its culmination; it is receding when the heat maximum has been attained.

Again, Surgeon-General W.A. Kinsler notes that, at Chelsea, where there was a garrison of young soldiers, no cases of enteric fever occurred from April, 1887, till June, 1888, although the weather was extremely hot. Here, again, a garrison composed of men at the susceptible age, must have suffered had the cause been climatic.

(ii) Distribution of the Disease. No station is exempt. It affects equally the hill-station with their almost British climate; where heat and moisture coexist to a very marked extent, as in sea-coast stations, plains' stations, as well as in the very dry hot Central Plain stations of plateau, as in Central India and the Punjab.

It is universal in distribution, it is quite independent of the degree of prevalence of heat, either combined with dryness or moisture, or of the cool hill-climates.
Rather than this disease prevailing in ratios to the heat and moisture, the tendency of the station or regional statistics is in support of the inference that the disease has existed proportionately more where their climatic features are less pronounced — in tablelands, true hill-stations especially, than in those stations of little elevation or on the coast.

Another important argument, which offers itself from a consideration of the distribution of the disease in India where we find all degrees of climate present, against the climatic theory is that we have three different climates arranged in zones according to their altitude. But if we believe the climatic theory, we must also believe that at some level — as we enter a climate similar to that of the temperate or even arctic zones — we should have behind us climatic tertie fever. But this is just what we do not do, for it occurs at the highest of the lowest altitudes alike.

(1) The physical factors predisposing to the disease are (a) youth; (b) recent arrival at the
Table to illustrate the relation between Enteric Fever, and the Death - Liability - Ratios at different ages, in the European Army in India.

<table>
<thead>
<tr>
<th>Year of Off. return quoted</th>
<th>Under 25 years</th>
<th>From 25-29</th>
<th>From 30-34</th>
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<td></td>
<td>Deaths per mille</td>
<td>Percentage of liability</td>
<td>Deaths per mille</td>
</tr>
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<td>1879</td>
<td>6.17</td>
<td>54.1</td>
<td>2.73</td>
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<td>1880</td>
<td>6.25</td>
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<td>3.15</td>
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<td>4.56</td>
<td>59.8</td>
<td>1.57</td>
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<tr>
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<td>5.37</td>
<td>54.2</td>
<td>2.63</td>
</tr>
<tr>
<td>1888</td>
<td>5.46</td>
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<td>2.36</td>
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<tr>
<td>1889</td>
<td>9.81</td>
<td>64.5</td>
<td>3.25</td>
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</table>
country which occurs with "want of acclimatization"; (v) alimentary conditions.

(2) Youth. This predisposing cause has been so fully established in Europe by Burnetson, Collee & other observers that it need only be amplified, as the case of the Indian disease, by the table opposite, which I have compiled from the official returns. We have thus one condition for a great prevalence of the disease, for our young army is composed of men not only at the most susceptible age but of strong healthy physique; & it has been repeatedly pointed out that enteric fever spare the weaklings of the sufferers from chronic diseases & attacks the robust. Between 23 & 30 years of age, men are, generally speaking, in their highest physical condition; under 23, they are undeveloped & very liable to the disease; above 30, the activity of the heart, liver & the "jack"—so necessary to the rapid campaigns of today—have begun to deteriorate, especially in India. 30

(3) Recent arrival. Andral long ago showed how especially prone medical students coming...
Table to show the relation existing between Shortness of Service in India and death- v liability - ratios.

<table>
<thead>
<tr>
<th>Year of Official Return quoted</th>
<th>1st &amp; 2nd years</th>
<th>3rd - 6th year</th>
<th>7th - 10th year</th>
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<tr>
<td></td>
<td>Deaths per mille</td>
<td>Percentage of liability</td>
<td>Deaths per mille</td>
</tr>
<tr>
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<td>7.99</td>
<td>67.2</td>
<td>2.18</td>
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<td>1880</td>
<td>9.08</td>
<td>75.9</td>
<td>1.78</td>
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<td>4.55</td>
<td>60.0</td>
<td>2.07</td>
</tr>
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<td>1882</td>
<td>4.68</td>
<td>61.1</td>
<td>1.93</td>
</tr>
<tr>
<td>1883</td>
<td>4.98</td>
<td>70.3</td>
<td>1.55</td>
</tr>
<tr>
<td>1884</td>
<td>5.47</td>
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<td>2.37</td>
</tr>
<tr>
<td>1888</td>
<td>6.39</td>
<td>62.4</td>
<td>2.77</td>
</tr>
<tr>
<td>1889</td>
<td>11.65</td>
<td>57.02</td>
<td>4.2</td>
</tr>
</tbody>
</table>
from the provinces to Paris to study were to be attacked by Typhoid Fever; 
has shown the same thing in connection with the liability of peasants coming to reside in a large city; Thrushes in Thuringia proved the fact by statistics based on an enormous number of cases. 

The chance of an arrival, nearly in an endemic centre of the disease, to acquire Typhoid Fever I look on as, with age, being one of the most important determining conditions. It is this, rather than to altered environment, the physiological changes thereby induced, that I look very largely for an explanation of the excessive prevalence of the disease in India, for I hope to show that there are scarcely any Indian stations which are not more or less active endemic centres of the disease. Conversely, those inhabitants of a locality who have resided some time there enjoy a relative immunity.

The table opposite shows the death prevalence ratios at the different periods of service. An average percentage of liability would be
64.12, during the first two years of service; 24.59, during the third and fourth years; and 8.52, during the seventh to tenth years.

Therefore, the first two years are shown to be, as they actually are, the most dangerous or fatal periods of the soldier's Indian service. I should also add that the young soldier is compensated for his age-proneness to disease by his lesser chance of suffering and dying from other diseases over older soldiers, for the average expectation of life diminishes much more rapidly in India than at home.

These two factors, then, of youth and recent arrival do not predispose to any greater degree in hot than in temperate climates. It is the arrival in Indian stations, which are (except the hill stations) actual suicide centres of this disease—active from the more rapid decomposition and distribution of faecal particles, with or without the specific germ,—that brings about such an excessive number of cases of the disease in India, just as Andreae, Cimel, and Louis showed was the case in Paris. "Want of acclimatization" is by some read...
- it very properly so - as meaning that the new-comers have not learnt to guard their health in the tropics. And it has again and again been shown that, in India, "acclimatization" does not confer immunity, nor lessen the influence of sultry heat on the human frame. If the cause was climatic, it "acclimatization" would confer immunity. Moreover, rapid subjection to tropical heat does not augment the disease-ratio, as it is immaterial whether a corps arrives at the beginning of the cold season or not, as far as the prevalence of malarial fever is concerned.

The only condition comparable to acclimatization is the immunity obtained by long residence in an endemic area, but this is quite irrespective of climate, as it obtains in temperate climates equally. The lessened prevalence of the disease in relation to length of residence in India is due to this, of the increasing age of the individual rendering him year by year less susceptible, from the lessened functional activity of his pyrogenic, solitary glands.
(V) Alimentary condition of the individual predisposing him to the disease: incidence.

It appears that meat-eating & spirit-drinking races are specially liable to hectic fever. The hill-tribes of India & the Parsees from Bepal are great sufferers from the disease, & both these classes conform to this description. Surgeon-Major O'Brien, of the 43rd Foot, reported many cases of typical hectic fever in his regiment; & also had similar experiences in his late regiment, the 2nd Battalion, 3rd Foot. The European soldier is well known to consume a great deal too much meat, of often, beer, when serving in India. Beer is cheap, & also tough because recently killed! There is a drink familiar to him at home, readily accessible in the canton, where no spirits are sold. It is a "bany drunk" which in a "thirsty country" like India in the hot weather, is apt to be abused rather than used for thirst-alleviation.

It has been stated that the use of meat & alcohol produces, in the European soldier,
inspection of Ripper’s hand, but I can find no positive evidence for this hypothesis. It has even been stated that total abstainers are more liable to bubonic fever than moderate drinkers. This is a comforting creed for the latter class, and doubt is explained by the fact that beer is either imported from home or from hill-breweries in India, in both of which cases the probability is strong that the water used in its manufacture is of good quality, and innocent of any specific tainted of bubonic fever.

On the other hand, abstainers drink aerated waters made from well-water in the Plains, generally more than suspicious in quality, which may not have been boiled before, and in the manufacture of soda-water, lemonade, etc., for instance at Kampfortee, 27 men were attacked by bubonic fever, all from different barracks. The latrines were in good order; the drinking water was from a carefully-guarded well; it was passed through a filtering tank, and then refiltered in barracks; the food supply seemed good.
The climatists would have triumphantly asked what it was due to, if not to climatic acting on young, unacclimatised men, &c.

But it was found that all the cases were total abstainers, that the water used for their aerated drinks was obtained from a well which was thought good enough for cooking purposes, which was situated close to the site of a former latrine, it had the surface-drain from the wash-house passing within four feet of it, there were signs of percolation from this drain into the well. Analysing the tests of organic matter was found the well was closed, or the power ephedrine effused. If these physical factors, rendering the person subject to the disease, are about in the acclimatized European, then will they also be in the native when the climatists hold, no doubt, to be evolutionally fitted to withstand the disease, as also by sickly and other constitutional habits & conditions.

The climatists assert that this is so, but this is luckily a mere question of fact, if there are decided against the climatists.
At p. 14, I have alluded to swarts evidence on this point. It is amply corroborated in his statements by Sir Jagger Hacker, late Professor of Medicine at the Grant Medical College, Bombay, who states that, in his long and ample experience, he has observed that no race or sect is exempt from the disease, that he has very frequently treated undoubted cases among Hindus, Mahommedans, Parsees, &c. Dr. Swinburne amply confirms this, & Dr. Runy, in his Sanitary Report on the Punjab in 1869, draws attention to the prevalence of the disease in the Rawal Pindi Jail, and gives perfect histories of pathological reports thereof. It has also been reported, on the strength of symptoms & post-mortem appearances, by Chevers for Nusayl, Anmbo for Barmah, to Burn for Roosam, as also by observers in Madras. Thus, indeed, there that there is no racial complexion throughout the world, from dysoid fever.

Syrjon - Major Rankey, of the Royal Army, indeed states that natives suffer from it.
far more than Europeans who boil their
milk-water, to avoid all source of fecal con-
tamination. The native community is
notoriously careless about their
cooking and drinking water's source, so this is
just what we would expect.
Dr. Clough and Holmes, of the Royal Army,
both officers of great experience and ability,
state their conviction that most Indian
natives acquire immunity during childhood
by a more or less mild attack of the
disease which passes unheeded by
the parents as "feverish diarrhoea". We
know that, in Europe, bacille Javie is
not at all a fatal disease among children
(Annie Smith), so the same might be
expected to hold in India, a priori.
I think I have adduced sufficient evidence
to show that we can place no reliance
on climate as a cause of Typhoid Fever in
India. We must now proceed to consider
3. The Pyrogenic Specific Theories, either
or both of which serve to explain the
pathology of the disease in India best, or
are most in accordanca not only with
observed facts but with our most advanced successful means of preventing filth disease. The climatic cause is more or less fatalistic in its tendency, for we cannot modify climate, whereas we can alter environment. The Pythogenic + Specific theories luckily have the same practical outcome,—the rendering innocuous of human excreta, which are the undoubtedly media which spread the disease. Pythogenic Theory. I shall content myself with a statement of the essentials of this theory. This is the theory of Dr. Charles Burnet, late of the Bengal Army, 1 of Sir W. Jenner. Burnet in fact calls Enteric Fever "Pythogenic Fever." It asserts that the zymotic exhalations from normal human or vivisection animals' stools produce, after reception in the human body, enteric fever. The term "Pythogenic" signifies "born of putrefaction," Burnet considered the point to be some degenerating animal product. One might very coin an aphorism, "Malaria from rotting vegetable, Pythogenic from rotting animal, matter." This theory practically asserts the "Pythogenic theory of fever."
new origin of a specific disease from either 
the genesis of a specific microbe, or 
from the continued action of the products 
of fermentation of the fecal matter 
inhaled. The "de novo" theory should 
be disproved by the arguments used 
in the refutation of Liebig's theory of 
"spontaneous generation," for "de nihil, 
nihil fit."
The zymotic theory is not so easily disposed 
of, it is at present certainly not disproved, 
but there is an immense superiority in 
the case of the specific theory, in the form 
of fact derived from bacteriological 
and clinical research.
The fermentation of fecal matter, derived 
from the external surroundings of the 
individual, may be noxious either from 
chemical products produced thereby, or by 
preparing the way of providing suitable 
substrate for either saprophytes or for 
pathogenic organisms.
Just animal alkaloids which are 
produced by albuminous decomposition 
belong to the group of leucamines of
the muscarinic type, I tend to cause diarrhoea. But, on the contrary, some apparently belong to atropinic group, which, to a certain extent, counteracts the effects of the muscarinic group. But suppose the muscarinic group to predominate in most cases, we have then the conditions favourable for the invasion and development of the specific agent—\( \text{the Bacillus typhosus} \)—for intestinal cataract will render the gut temporarily the "locus resistentiae minoris."

Bouchard has shown that the phenomena normally present in urine are notably increased in Scarlet Fever,—three of the bacillae group, i.e., derived by the action of microbes on albuminous tissue. It is not too much to suppose that a portion of this increase came from the increased production of animal alkaloids in the gut, as well as from the increased tissue changes in fever itself, produced by the action of micro-organisms on the tissues generally to which they had gained access by the blood-current.
But this is all pure hypothesis, there
exist no facts, which will bear critical
examination, to show that bacilli fever
are produced in the absence of the
Bacillus typhosus. And there is a good
deal of evidence to show that the
action of decomposing organic matter—
whether or otherwise—on the system would
lead more to predisposing toward by
losing the "vis resistentiae" by a neural
poisoning of the higher nervous centres,
by a blocking of the intestines of
the lymphatic fluids of the gut.
Dr. Carpenter long ago showed experimentally
that collections of decomposing material
were special food "foci" for the develop-
ment of specific germs; Dr. Barker
conducted a series of experiments on the
effects of the prolonged inhalation of
gases evolved by such collections of de-
composing organic matter, I found that
hyperaemia, irregular feeble muscular
contractions, diarrhoea were produced,
which ceased when the cause was removed.
no specific disease followed this inhalation,
as was to be expected in Kushner’s theory. Emmerich of Munich drank from the filthiest ditches with impunity, and Indian scavengers do not suffer more than other people from enteric fever in their occupation. fashionable night-soil, etc.,

while the stools are those of a case of enteric fever. As to the apparently spontaneous origin of the disease in solitary country-homes or in desert-places, there are more facts against these cases as supporting the “de novo” origin of the disease than otherwise. In the desert, the disease may only show itself after having been receieved four to six six weeks before in some endemic centre of the disease. The universal prevalence of the disease all over the world among all races makes it probable that a case of the disease may have defecated near the desert spot encamped on; for epidemiological has shown that typhoid stools retain their infective properties for at least nine months, probably longer.
And in the case of a large country house, it is impossible to exclude all possible sources of infection, as tramps, visitors, new servants may produce the disease which they may be convalescing from, or may have very mildly, or in the ambulant form. These are just as competent to spread the disease as well-marked cases of great acuteness. Indeed, all the evidence for spontaneous generation is entirely negative, consists solely in our inability to trace with the eye the continuity of the chain whose connecting links are known to be invisible. To conclude from this that no chain links would be ridiculous. As Budd says: "If the same evidence were true, it would, prove the spontaneous generation of plants and animals whose lineage is very often impossible to trace, but this impossibility is no force whatever as an argument against their propagation by the law of continuous succession. We have now come to the specific theory of a process of selection, have attempted..."
to demonstrate its claims to be considered the best working hypothesis till we can make it yet more certain than it is at present. The difficulty here is that Koch's postulates have not yet been

cannot be, fulfilled, in the absence of animals which take pandemic fever.

The Specific theory, therefore, asserts that the disease is originated by a specific poison

derived from a previous case of the disease.

With this I would also include the belief—held by so many of our ablest pathologists—

that the Bacillus typhosus, (discovered in 1880 by Gerth and Klebs) is the specific agent which
directly produces the disease.

In support of my acceptance tentatively of the Bacillus typhosus as the "Causa constans" of the disease, it will be as well to quote

one of the most recent and weighty utterances on the position taken up by the principal

pathologists of the world on this subject: Burdon Sanderson, in the British Medical

Journal, for Nov., 1891, states that.
observed.

"The constancy of the relation between the occurrence and distribution of the bacillus seems to leave no doubt as to its etiological significance."

Let us now apply the specific theory to the conditions of military life in India.

Firstly, with regard to the water-carrying. In the plains of Upper India, it is generally taken from wells sunk in the alluvial clay to a sandy water-bearing substratum, which lies where underlies the surface soil at a depth varying, in different locations, from 20 to 120 feet. The water is raised out of the well by small leather buckets, and then emptied into a "mussuck," a goatskin stitched together so as to form a pouch with a very narrow mouth, which is carried by the "kisti," a water-carrier, on his back. Neither of these can be cleaned after use, and the "mussuck" is almost invariably used to convey water from different sources intended for separate purposes, cooking, drinking, washing, watering horses, &c.
Drinking-water must suffer contamination from the receptacle which has carried bad water as well as good. The wells are generally uncovered, and hence all sorts of filth may mix in. On recovery from an illness, a native takes "the bath of recovery" — a thanksgiving ceremonial. This is performed for him, most conveniently, usually by the well-mouth. Here he leaves his body and clothes, and has the water dripping back into the wells which were subsequently to have been used for drinking-water supplies.

Dead dogs, cats, and carrion are frequently found in wells used for drinking-supplies when cleaned out, as it is a favourite mode of disposing of the murdered, e.g., in the Lahore massacre by Khan Daulat, in the Mutiny of 1857.

It is usual for these wells to be examined chemically twice a year, but such an examination is valueless if not also bacteriological, for it has been shown that it does not at all
follow that even an appreciable increase of any chemical ingredient need be found for the introduction of the pathogenic organisms is sufficient. The water is rarely boiled but generally filtered. This process is quite futile, if not harmful, in India. The common filter thus consists of three pots of earthenware, placed one above the other, on a tripod frame. The top jar contains the water to be filtered and, the water trickles into the middle jar by a small hole in the bottom. In the second jar is placed a mixture of sand (sieved from the nearest river bed, which is a favourite place for natives & animals to defecate, & which is imperfectly, if at all, cleansed) & wood-charcoal in lumps. From this jar, the water escapes into the third jar which is simply a receptacle for the (? fil...
derived from the sand. Personally, I never allow my drinking-water to be filtered, but have it boiled, cooled, and reboiled, and aerated by pouring from one jug into the other. I also see that the receptacles are scrupulously clean and efficiently covered, when they are standing full of water ready for use. Milk may be contaminated either by being watered with tainted water, or by standing in a house in which the atmosphere is contaminated by effluvia from a cesspit, ordure-bench, or other place where putrefying stools are present, or by the clothes of an infective human patient. Food may be similarly infected, especially so by flies, coming from ordure-benches, with the fine hairs of their legs coaxed in loose putrid stool, landing on the articles of food. The disease may also be obtained by inhalation of air, laden with particles of feces on which the specific poison exists.

The prevalence of this disease in the hot
reason is rendered intelligible when we reflect on the presence of the following conditions. Heat produces rapid subrefraction of most animal substances, hence local emanations quickly follow. Heat heat also dissolves, order, causes its reduction to a fine powder, on which the wind can easily act and diffuse into the atmosphere. Dust storms are very frequent in the hot weather; during one of these, one cannot fail to inhale a very large amount of dust, perhaps local particles. Again, the native deposits everywhere about a station—a fact which the absence of slight smell renders all too evident. In the hot weather, there is no rainfall to dissolve and carry below the surface this order, hence aerial contamination ensues.

Coincident with the great heat, the water supply becomes more concentrated. If a well, for instance, is contaminated by enteric poison the dose of the poison inhaled is relatively greater during the hot season; it is more likely to incite it from the terrible thirst which prevails then. Besides, heat (tropical especially) makes
The European, more careless and indifferent by exhausting his nervous system, makes the native water-carrier dissuaded for several journeys to a source of supply of better quality but further off than a nearer and much worse one. Later, when the rainfall occurs, moisture is added to heat in assisting saturation of surface impurities, and assisting also the development of the specific organisms up with them, perhaps, more or less latent. The rainfall also mechanically conveys the cause into the water-storage places, and brings it by this medium, into contact with its favourite "nidi" - the human Persian Solitary glands. At such stations as Rawal Pindi, the stench, in cantonments, in the hot season "Rains" is very perceptible even to the most anaesthetic Schneiderian membrane.

One of the most widespread causes of the disease, spread of production is the habit the British soldier has of visiting the native bazaars, drinking much.
of his time there. The bazaars are built on sites cocked for generations by the secretion - lymphoid & otherwise - of nates, & there always exist a large number of cases of the disease in every large native bazaar or village. Here, the soldier becomes infected by the exhalations rising from the filthy-repentant soil, or floors of the houses, from soiled clothes of entire patients, from food contaminated by excreta from, but mainly by native aerated waters made from the nearest pool or tank of water, whose condition will readily be appreciated from what has been said of the surface & subterraneous. Officers, ladies, women, and children all suffer less from the disease because they avoid the native quarters, as do all their shopping by deputy generally. Another common cause, I am certain, is the scant supervision accorded to native servants' conservancy arrangements. The

*x"ladies" (i.e., officers' & civil service officials' wives)
*y"women" (soldiers' wives) are reckoned two classes, officially in all Returns.
consequence is that a neglected night-soil trench, giving forth poisonous exhalations to contaminate air, food, water, and clothing, may exist within half a dozen yards of a European's dwelling. This is a point no one troubles about, but in a recent case at Rawal Pindi, two young medical officers suffered from bilious fever through a neglected trench in the garden of their dwelling, for they shared a house.

The Conservancy arrangements of the troops are practically perfect, if the system laid down is carefully carried out, no danger should arise from this source. The system authorised is the dry earth system. The latrines are inspected every week by a medical officer and the Regimental Artisan-master, at different times. The points have observed defects were: (1) leaving unlined the airtight iron receptacles, into which the stools were placed after removal from the privy pans; (2) bringing back earth from the trenches (where the defecates are buried), in the same cart.
as the deposits are removed, for use in the latrines — to throw on the grounds when passed; (3) using water for cleaning the privy pan after use, for this operation is carried on upon the ground immediately adjoining either a barrack or a latrine, it cannot fail to defile the soil by percolation, perhaps the water-supply. Again the clothes may become media for the spread of the disease. The native washerman ("hobi") washes clothes from different sources — native and European alike — in one pool by the river side, but not necessary of running water. Hence clothes soiled by typhoid deposita may, if left do, come to be washed along with clothes from unaffected houses. The clothes again may suffer from being stored in a room in the native quarters, previous to being returned to their owners, it may become contaminated by effluvia arising from a floor which has soaked in the loose stools of an infected fever patient, such are very common in native quarters.
The disease is often contracted on railway journeys, in rest camps, on the line of march, from the men incautiously drinking polluted water; aerated waters of native manufacture, made from foul water very currently; from articles of food, milk, butter, and even ice.

In conclusion, it would indeed be more than remarkable if India had alone suffered from a form of malarial fever etiologically distinct from that prevailing throughout the world. The absence elsewhere of the connection of malarial fever with local filth causes has been constant, and the sequence of facts so unmistakable as to render it wholly unjustifiable except on the oyster evidence, which, as yet, is absent) for us to set aside such experience in favour of the view that the disease in India was attributable to no assignable cause, other than filth, climate, etc., that it was, in fact, a tropical fever. I hold that in the existence of the ever present filth conditions in every cantonment in India, a climatic theory of causation is quite unsatisfactory.

And, moreover, all the facts are for a specific cause.
We know that very close personal intercourse does go on between the soldier and native or other civil community around. That the soldier visits the bazaars or native haunts for various reasons, that natives came in close contact with the military in the barracks itself, that the asserted means of transfer exist widely in India. We have seen themata existing among the soldiers ascribed to communication from the native population, or native fever memory, as regards its prevalence in parallel lines with time. Why then should we stop short in the natural reasonable deduction that the source of native fever cause is the same as those of the seamen? Where a filled history is absent, we may feel certain the cause has not been traced; if this is not to be wondered at considering (1) the wide area over which the men contract the disease; (2) the many ways of communication, (3) the practical impossibility to trace any one after a period of 2 or 3 weeks has elapsed, during which the disease has been in-
outbreak in the patient. Indeed, as Corfield says on the European disease's etiology, "it would be very wonderful if we could trace every case of fatric fever to a previous case."

Officers, women, ladies, & children suffered much less from the disease, for they are more careful, as a rule, as to what they eat & drink, & do not go so much as the men into the bazaars [sic]. In support of this, an epidemic of the disease occurred at Rampur, it was confined to the single men of the 33rd Regiment alone. No officers, ladies, women, or children—not, indeed, married men—were attacked. Here the disease occurred in precisely that class which would be most likely to subject itself to the danger of contamination outside barracks, generally, for Hindu cantonments are cases of cleanliness situated in desert of filth.
Chapter 14. Pathological.

The conditions generally present due to the evolution of the introduced virus are:

1. General aspect of blood-poisoning; congestion of brain, liver, kidneys; abnormal condition of the lymphatic spaces of the foot; vesicle on the skin; splenic congestion and multiplication; pulmonary congestion, possibly broncho-pneumonia. These, along with brain exhaustion with delirium, functional cardiac weakness, epistaxis, abdominal "irritative" symptoms, occupy the first ten days of the illness.

In the fifth week, a period of retrogressive changes in the lymphatic glands starts; first of these is an inflammation of the mesenteric glands from absorption of poisons irritative material in the "fossa".

Is the action of the specific poison may also be ascribed the pulmonary oedema, hepatic oedema, congestions.

Let us glance at the "anatomical sign", considered pathognomonic of the disease. Liebermeister tells us that the first week of the disease is occupied by swelling of infiltration of jugular, auricular veins.
During the second week, they either slough or
subside; during the third week, any sloughs
that may have formed are detached, so
that, by the end of it, all the ulcers have
clean floors; during the fourth week,
they begin to heal.

The lesions of the solitary or Pyranean glands
appear to be remarkably similar.

Rarely more than ten of the latter are affected.

The size and degree of severity of the lesions
depend on the distance between the ileo-caecal
valve and the ileum, increased, at least, in India.
It is the rule for the entire gland substance
to be implicated in the acrid chyme changes.

The gland generally is elevated above the
mucous surface of the gut; it is thickened
to many times its original thickness.
The surface is uniformly flattened, and occas-
ionally umbilicated in the centre, and often
is pitted all over. Its colour is generally
tawny or dark brown, often from bile-tinging
of the necrotic tissue. Distended vessels
surround it on all sides, as a rule, to
the serous-coellic base of the gland is often
highly congested and even inflamed.
The process seems to me to be essentially a strangulation of the blood-supply by excessive hypertrophy of the cell-units of the gland, as well as by diapedesis from the congested capillaries in the neighbourhood of the gland. This strangulation results in the death of the gland-mass, its elimination as a slough, this process is more often than the process of ulceration, a molecular disintegration of the gland-substance.

When the ophiodes has been shed, the bare muscular fibres frequently present themselves to view, and often these have also been involved, the peritoneal and subcapsal coat alone remain.

There is often some degree of separation of the individual coats of the fat from one another,—the result of effused "capable lymph" which has broken through the adhered wall, or soon after, the shed slough. After the separation of the slough, or during the process more generally, hemorrhage may occur from the opening up of the lumen, generally of some small vein,
After its separation, the calaminity of perforation of the Jut may occur from the insufficient mechanical strength of or vitality of the thinned base of the "calc." to play its part in maintaining the calibre of the Jut intact. Again, but more rarely, the Jut may slough from impaired vitality, in its entire thickness, hence perforation occurs.

The localized peritonitis of the base is a frequent preventive of perforation. "Pari passu" the cloudy swelling, a frequent degeneration of a portion of the cells of Reuber's follicles occurs.

Cases have been reported in which the solitary glands have been mainly affected, for the reason that Oger's glands were "not available" for the entire person's reception, either from cicatrization from a previous attack, or from atrophy in the case of atrophiant chronic lung disease, for especially in destructive lung lesions, the Ogerian glands atrophy. In chronic disease of the liver or kidney, or in affections of material...
cachexia, even in ample observation (as was observed frequently in nature of India, над a. observation in the famine during 1876-7), marked pyogenic atrophy occurs. The cause, at least in chronic lung disease, is not a mere atrophy but this is generally preceded by enlargement & caseation, accompanied by catarrhal inflammation of the mucous membrane; one of the forms of phthisical diarrhea.

We thus see the crosstination which the observed relative immunity of weakly persons & those suffering from chronic diseases, receives from observed facts. In these rare instances of an attack of future ever dating in the same person, at an interval of years (so as to exclude recrudescence, or relapse) we must seek the explanation. Notive, either in the non-obliteration by scar tissue of the pyogenic in a gland affected previously; or by these glands which did not formerly partake in the diseased process,—for we have seen that all the pyogenic patches do not suffer, becoming affected later;
or by the solitary hand suffering mainly,
not the Persian patches.
Again, there seems to be no relation between
the extent of the industrial lesions, or also
their severity and, in the degree
of the pyrexia, for ambulatory cases
not infrequently succumbed to perforation,
or bowel-hemorrhage, who have
suffered very slight febrile symptoms.
Indeed, if the fever had been very
pronounced, it would have been
impossible for them to go about as they.
The conditions of chronic, pulmonary, central
and hepatic infections found exist equally
in other specific fevers, and are not
peculiar to Dyspnoic Fever. It is the
prolonged nature of this form the im-
portant-physical impairment that brings
the visceral complications into greater
prominence, in Dyspnoic Fever.
The absorption of animal alkaloidal
or osphacemic products is another
great cause of the accentuation of
perpetuation of the visceral inflam-
mations, I believe.
But in India, many autolyses reveal the fact that the large gut is occasionally solely or principally affected. And again the lesions recorded in Return oose often too extensive or irregular to be characteristic of acute fever on the one hand; while, on the other, cases have occurred which presented a perfect "clinical picture" of the disease, but there were, it is stated, "no lesions post-mortem which could be called acute fever lesions." 57

I would account for the principal affection of the large gut by the oblation of the solitary glands as the main seat of operation of the typhoid poison (as before discussed).

The extensive lesions recorded are probably due to the general impairment of the nutritional condition of the intestinal mucosa by prolonged or occasionally very acute congestion from malarial causes. In malarial cachexia of mild degree, not only is the nutritional value of the blood impaired, but absorption of food is lessened.
affected induced by the action of the
malarial poison, which is a nervous
poison producing frequent ophrana
oligemia of the stomach, faint-by-arti
tuated to be succeeded by intense hyposensitivite,
without any intermediate state in
which the nutritional defects induced
may be rectified.

Besides there are, in too many cases,
added to by the dietetic
errors of alcoholic
indulgence of the military in India.
It is then, only too probable that tissue
subjected to such a strain as a pro-
longed severe pyrexia, such as tertian fever,
will undergo molecular disintegration
if not a more wholesale destruction.
In the case of the last group mentioned,
the failure to discover the intestinal
baubles of tertian fever—the explanation
suggests itself that the "clinical picture"
were false ones that the disease
was a severe remittent fever, in which
case the specific lesions would be absent.
Again, one must consider the fact
that very few medical men universal.
welly on an added liver autopsy during their course at the Medical Schools. This was especially true of some very few years ago, when the clinical study of fevers was unknown in a medical curriculum in most educational centres. So it is not too much to presume that many of these cases were judged of in the light — or rather from — imperfect conceptions of the intestinal typhoidal fever. So far as I know, any reference of human autopsies abroad, there is a marked similarity in the type, but often an increased in the degree of the visceral involvement. Often specific lesions of the lymphatic structures. The fact that, in typhoid fever, the anatomical sign is not pathognomonic. Many other observers have observed in: (1) Remittent fever with relative symptoms; (2) Hae-morrhagic; (3) "laceral" pneumonia, with elevation of the small intestines; (4) Tuber-culoso; (5) Cholera; (6) protracted diarrhea.
(7) scarlatina; (8) malignant endocarditis; (9) Acute Bacterial Dysentery; (10) Diphtheria; (11) Colchicine poisoning; (12) Case in Dr. Bell, "Lesions of the intestine."

But on close examination of most of these samples I find that it is not specific petrophenic lesions which are recorded, but mere ulceration of the gut; for no one would doubt for an instant but that intestinal ulceration occurs in very many diseases as was, indeed, to be expected.

The only members of the group above cited which it will be necessary to discuss are numbers (11) v (3); for the others cited are not specific ulcerations of petrophenic glands at all.

Remittent fever with satiric symptoms.

This group of cases seems to be really identical with Typhoid Fever itself. I have myself seen a few autopsies from severe Remittent Fever with typhoid symptoms, of nothing at all comparable to the lesion of satiric Fever was ever present. Superficial ulceration, but not in connection
with either the Bogerian or solitary form, was occasionally — but exceptionally — observed. There was no doubt due to toxic changes impairing the vitality of the nerves, for the ulcerations were very superficial, & nothing of the nature of the emaciation of Typhoid fever was present. Indeed, I do not remember to have observed even intumescence of the lymphatic structures of the put in these cases. Malarial pneumonia, "with ulceration of the intestines." It is recognised that when the Typhoid poison enters the system by the lungs, there is generally induced a very intense pneumonia. Moreover, in persons, whose pulmonary tissue is specially susceptible to become inflamed, either as an hereditary peculiarity, or from the presence of perhaps overlooked malarial atoxia, the pneumonia speedily proves fatal in very many cases. I consider that there is no evidence to show that the cause is malarial.
in these cases, but believe it to be
typhoidal for here are found typical
intra-hepatic lesions. These, then,
are probably cases where the
chief violence of the intra-hepatic
lesions has been spent on the lungs, but,
as the specific disease was induced,
the typical bowel lesions have also
been induced.

Prof. Sir William Arthur, F.R.S., of kettle,
in discussing the question of ulceration of
the bowels in relation to intra-hepatic
state as the outcome of an exceptionally
large pathological experience, his views
in these words: "My experience leads me"
"to maintain that the lesions of the bowel",
"in typical cases of this specific disease",
"are so very different from each other, so
"characteristic of each disease, that the"
"one must not be mistaken for the other,"
"not misinterpreted."
Chapter 5. Symptomatological.

In this chapter I will briefly indicate the modifications & peculiarities of the Indian disease as compared with the European.

1. As to onset, it is not at all unusual for a distinct attack of ague to usher in the disease. This arises from the readiness the system, exposed to malaria & saturated therewith, has to suffer an attack from even the most trivial causes. I have seen a violent ague cold, stage induced after a tooth extraction, after mental disturbance most or less sudden & severe, after a chill, after a fall from a horse, etc. Experience tells me that it is not a mere coincidence, but is due to the lowering of the inhibitory functions of the central nervous system by sensorial disturbances.

In the case of native drivers onset it is due to the rousing into activity of the malarial poison's residuum in the system by the new developing disease. Thus has arisen confusion of the
two distinct conditions of malaria: &

Malaria, for observers occuring a disease

of which manifestation concluded

too readily that there was a causal

connection; in fact, fell into the fallacy

of "post hoc, ergo propter hoc."

Again, the converse holds good, for

in cases of malarial cachexia attacked

with Spurie Fever we have no such symptoms.

Surgeon-Major Ranking, Royal Army, has

stated that a temperature rising in the

first two days of most 6 104°F. is

indicative of the disease not being "Hilalic,

but Remittent Fever. Now true this is

I know from my own case. At recruit

in 1890, I was attacked with

a febrifurish condition, with diarrhea

close resembling typhoidal diarrhea;

abdominal fulness & discomfort;

headache; intense prostration; etc., but

my temperature ranged between 104°

or 105°F. for the first 3 or 4 days.

I had had Remittent Fever, not Hilalic.

Another sign of value is the appearance
ation of the temperature to normal on
the 3rd or 4th evening after the attack:
not probable the disease is not febrile then.

2. Course of temperature. Remittance of
the temperature is extremely common,
but generally the rise of temperature is
gradual, the maximum being attained
on the fourth or fifth day. The evening
rise preceding remission is also to be
observed, whereaugmentation that it
is a bad prognostic if these conditions
are not well marked. The more
straight
the temperature line, the worse the attack,
as a rule. If the temperature, for several
days, is between 103°-104°, or especially
if it is higher in morning than at night,
then the attack is a severe one.

In India, one observes that cases which
attain a temperature of 105° are generally
fatal. I would take 105° as "hyper-
pyrexia" in stature India, instead of the
106° which we used to be taught in
Britain was hyperpyrexia. Have also
observed how "relative" hyperpyrexia is,
for many cases suffer much more from
a temperature of 104° than others who will tolerate 106° for perhaps hours, with comparative immunity. Would you wish to emphasize the danger of taking any particular temperature to denote the condition of hyperpyrexia, for many cases with temperatures of 104° require the most active means for its reduction, or the consequences will be most serious. If one does not, for instance, apply the cold pack or bath till a case shall have attained a temperature of 105°, a certain proportion of cases will suffer from 105° blind adherence to European „general principle.” Again, in India a temperature which approaches to normal at the end of the first week must not be interpreted as indicating that tertian fever is not the disease, for such a remittance is not uncommon, is due to the coexistence of malaria which modifies tertian fever as it does almost all the other fevers in the East.
Indeed, variations in the temperature-chart are the rule in India, due to the climatic heats' direct influence on the thermotactic curves of the operation of malaria.

I have met with antico fever in India show that the severity of the disease can rarely be judged of from the temperature-chart, for exceptions are so numerous to the rules laid down by Wunderlich and others, that the possibility of differentiating the disease by the thermometer from other diseases is negatived.

It is also difficult to judge the temperature by the hand in antico fever, but easier in Remittent Fever where the skin is hot or cold.

Summing up the thermometry of the disease in India, then, we may say, that the ascent from health is probably reached between the 326 and 5th day of pyrexia; that the next period is irregular, from first to last, with considerable amnial fluctuations;
that the later thermal fluctuations are occasionally supplanted by a gradual decline, which fastens on to the case terminally; and that, as a rule, Indian cases do not attain the same height as European cases of equal symptomatic severity; or, lower temperatures than obtain in European cases of the same more fatal. It is not at all uncommon for the maximum temperature to occur at or about noon, and this has a relation to the high temperature generally prevailing there as well as to periodic influences.

In the hot season when the ward-temperature is 85°–90°, there is always a hyperpyrexic tendency. Their observed the same thing in cases of variola and pneumonia frequently. The rash is quite unreliable as a sign of disease. Generally, as mosquito-bites closely simulate it, as do roscolar eruptions from heat, the irritation induced by excessive sweating. 

Tropical is generally easily distinguished. They have been referred to the variation.
solid by Jenner in this matter. Personally, I attach much more value to the "taches bleutées" — slate-colored spots of a diameter of \( \frac{1}{4} - \frac{1}{2} \) inch, of round or irregular outline, which are not elevated, but disappear on pressure. They occur mainly along the course of the superficial veins. These appear to be first observable shortly after the invasion period, last as long as the pyrexia does, fading during defervescence.

As to the roseola, have observed it apparently distinctly, but it is always impossible to exclude the possibility of its being of insectivorous or other origin. The puncture by the proboscis of such insects cannot always be seen, especially in cachectic subjects.

The abdominal surface may, too, very readily become uncovered in the case of a restless fever-patient in India, hence the site does not help us.

The face is generally pale and bedewed by sweat, not, however, air "pale"
Flush.

Profuse sweatings occur, but bring no reduction of temperature with them, even when the temperature is highest.

"Washerwoman's sweat" is stated to be a symptom of fatal apoplexy in Europe, believe; but I have not observed this to be true for India.

Diarrhoea is very much less common, most probably from the excessive sweating draining off so much of the fluid portion of the blood. In other cases it is late in occurrence or even rare.

On the other hand, these occur in a two cases with copious, frequent, liquid motions.

The characters are as in Europe, but in India the stools are not uncommon of bilious characters for the first two or three days.

Pulmonary Cardiac complication: The former

* These sweats are distinct from the "Washerwoman's sweat" which generally occurs later in the course of the disease, and also from those during delirium.
clear, fairly frequently, to be very marked in its severity. It generally appears about the tenth day of the fever, and is very often of tubular form. In hypostatic basal congestion is a condition common to many other fevers, and calls for no particular mention here. I have observed a tenacity for cases of hypothyroid pneumonia to end fatally from hypothermia. This appears to be due to the poisoning of the heart centres mainly, for there is lessened respiratory capacity, consequent tone-on-tone, because of the pneumonia state; hence the poisons absorbed from the cut circulating in the blood are not destroyed by oxygenation to the extent they would be were the lungs less affected. The cardiac signs are also very marked. From the first there is a diminution of the first sound of the heart, a troubled or tumultuous or, worse still, syncopeal reaction of the organ.
to any stress thrown on it; as by any
sudden motion or shock. This has,
therefore, to be guarded against.
The pulse condition is unimportant
in the tropics, where an enlarged omen
from malaria is so common.

The general dilatation of the colon gener-
ally renders it hard to exclude it among
its condition in Indian febrile fever.
Epistaxis is very common, it is not un-
favourable, as it lowers the temperature
very often, relieves the headache.
(If course, this is understood to be when in
moderation.)

The tongue, in mild cases, has a creamy
colour, with bright red tips and
margins.
In cases of greater severity, it is bright;
red and papilled; while, in the worst cases,
it is brown, dry, coated, fissured,
blanched, and along with the teeth &
his, covered with cords.

Aperitive medicine is better than
at home, but in severe cases, food
is freely taken. There is Aperitive
during Defervescence, & convalescence.
Meteorism is slightly more uncommon than in Europe; vomiting in the flanks is more easily obtained than in Europe, especially during the first week. Intestinal haemorrhage seems to vary in occurrence, for in some epidemics, it is very common, while in others, it is rare. Headache is invariably present, it is frontal or general. It is much less severe than in Remittent Fever, but this is a point of differentiation which it is hard for the physician to gauge precisely. Vertigo, back, and loin pains are frequent. Delirium is not of frequent occurrence, but it is of grave import when present. Insomnia is easily combated by such mild measures as aromatic beverages. Decubitus dorsal, but I always try to persuade the patient to lie as much as possible on his sides, well supported by pillows, so as to lessen pulmonary hypertension, etc.

Prostration is great, as also is the association with dehydration, both of course, especially towards the end of the disease.
Involuntary vacuations generally usher in the red more or less speedily. Deingitio appears from what I can learn from the experience of other medical officers to be commoner in India than in Europe.

There is often temporary deafness during the attack, almost invariably transitory. This is probably from anaemia of the auditory end of the cranial cerebral appertaining to which Maclean attributes all the ear cerebral symptoms in dyspepsia. Finally, the pulse may be said to be the symptom most closely resembling the corresponding European symptom: it is, perhaps, slightly faster, but generally the other features are the same.
Chapter 6. Therapeutical.

The indications here are much the same as elsewhere. A well-ventilated room or ward; careful nursing night and day by attendants, who have had the disease preferably, for this renders them immune from the chance of infection — for汇报 (e.g. cit., p. 463) mentions several cases of direct infection in typhoid cases — besides men who have had the disease make more sympathetic and careful nurses, as a rule. But in European military hospitals in India, female European nurses are generally to be found. The disinfection of the bed will be treated of in the next chapter. The patient's linen should be frequently changed — both body sheet — with the minimum of disturbance to him. A bed for the day time, one for night are great necessities. The most perfect quiet must be enforced, the patient must not be allowed to get up to empty his bowels or bladder 

*that is, in Indian Scarlet Fever.*
Dietetic treatment: Milk, diluted to half or one third of its bulk with barley-water or white-water if constipation is desired to be encouraged; with Disceford's fluid hyperia if mild action of the bowels is required, should be the staple article of diet. Four pints of it should be consumed daily (this is a most liberal average); if milk only is given, Beef-tea, broths (fully strained), meat juices or jellies — salted sufficiently — are also articles of diet which are safe will be readily absorbed.

Water, iced or cooled; (aerated drink should be avoided) lime-juice as Camomile, or sweets slightly acidulated with dilute muriatic acid (which may assist the stomach-digestion of meat elements) are also necessary. The soluble material extracted from 1/2 lbs. of good lean beef or mutton, 1 pint as soup or jelly, along with 1/2 to 2 pints of milk is about as much as the average adult will assimilate in 24 hours.

Beef-tea has been blamed worth for inducing...
Diarrhea, but this is due to too much being given at one time: it is the quantity, not the quality, of the nutrient which is at fault. A very minor, no doubt, but very important practical hint is to cleanse the mouth, with a weak solution of potash before giving food. As the case advances, quinine may be added raw to the milk, or later on, perhaps arrowroot, corn flour, but caution has to be observed with starchy, vegetable, and flourous foods, because of the risk of fermentation and consequent gaseous distension of the gut. The stools will here often indicate how these latter are being absorbed. It is better not to run the risk of producing bulky motions or gaseous distension by withholding farinaceous of such characters. Animal food should form the main portion of the diet in initial fevers, in the forms above mentioned.

Alcohol. Prior use of this agent to anything beyond moderation nullifies its stimulating properties; this must be considered in every case we treat.
Intric fever is marked by an asthenic disease, with impaired cerebral and cardiac functions. Alcohol is primarily a stimulant of these organs, hence reduction of the force and frequency of the pulse, check of fall of temperature, cessation or modification of delirium, may ensue when it is exhibited. It also checks oxidation, controls passive metabolism, thus conserves the tissues to some extent in the wasteful process of fever.

In the first stage of the fever, it should only be given if delirium is marked, muscular tremors exist to any degree; or if signs of cardiac failure are shown by increased frequency, weakness of the pulse, especially if the beats run into one another.

In the last half of the disease, when the vital powers are shown nearly, a strong stimulus is very necessary, alcohol is most of all needed. And if the typhoid state supervenes, 6 to 8 2dgs. of alcohol, fresh diluted, near the gruel, from day to day. But
It appears best to keep alcohol as much as possible in reserve for septicaemic cases. 
Good whisky or brandy seems to be the best forms of administering it. 
No solids should, on any account, be allowed till the temperature has been normal for at least 10 days.

Medicinal Treatment.

In the early stages, antipyretics & especially quinine in 3-4 doses in the 24 hours, should be used. In mild cases, in men newly arrived in India, with low temperature, it is unnecessary altogether. 
But in those cases which attain their maximum temperature at 1 p.m. or noon, it should be used, administered preferably in gelatine capsules, or in solution, if the patient is not nauseated by the drug's taste.
Although quinine has no influence on the septic virus, it does not moderate the course of the disease process materially, it often exerts a sedative effect on the brain, of blood vessels, etc.
control temperature, especially as regards prolonging the morbid remission.
In constitutions "paralysed with malaria," it controls paludal manifestations,
serve to control periodic pyrexia.
To obtain these results, three (30. y.)
doses in the 24 hours generally suffice.
Pyrexia is best controlled by cold
applications, preferably of spongy, to
the wet back. The latter is much
safer, more convenient, I am grateful to the
patient than the bath in my experience.
I can be longer applied at one time.
Antipyreis, antifebrin, or other
febrifuge may be necessary to control
a rising temperature, but should be
had recourse to as little as
possible, because of their depressant
action on the heart.
The malaria appears to be modified
by the administration of mercury,
although the rationale is not clear,
further than that this drug is our
most powerful antifebrifuge agent.
Combined with a little chalk or sodium
bicarbonate, calcium in doses of 16 to 18 has a remarkably sedative action on the abdominal viscera, relieving the biliary disturbance which so frequently occurs during the first few days of the disease. It actually seems to lessen the severity of the case and to cause the gland lesion to abort more frequently under its continued administration in small doses, causing no doubt molecular disintegration of the gland substance, the "debris" being removed by the lymphatics.

It is specially valuable in ankylostomiasis and is contraindicated in dyspepsia. It also, as Hay has shown, lowers arterial tension by reducing the amount of uric acid in the blood. This is a vascular sedative. It should never be pushed so as to produce the slightest "constitutional" sign.

Brigade Surgeon Lawrie, of the Bengal Army, has recently had remarkably good results with tartarated calcium in doses of 1/16.
To a grain, in one tumbler of water, every second hour, till 1 grain in the 24 hours has been taken. The solution should be made freshly every day. Its administration should be continued till the temperature is normal. No depression occurs from it; it relieves abdominal pain, headaches, checks diarrhoea, prevents cerebral symptoms. It is said to check the inflammation in the small gut, to shorten the disease. This is now being tried in India, with what result remains to be seen.

To control diarrhoea—i.e., more than 3 stools a day—Resinumth is "facile ministris," and is most useful by allaying the catarhal inflammation, limiting the formation of stools. Compressed chalk powder, opium are very inferior, more disadvantageous than Resinumth in this connection.

Of the means directed to rendering the pulse ascitic, or to lessen its severity, rather, Hydronaphthol in 5s. doses in gelatin capsule, or Hydronaphtol is an impure form of Resinumth B.
that Beta (B) every two hours is perhaps preferable. Donchard finds the latter lessens the diarrhoea & makes offensive motions, by its internal administration.

Perchloride of mercury, iodofurin, carbolic acid, many other antiseptic substances have been administered for a like purpose, but with very doubtful result.

Perfumetin, in capsules, has been frequently given during the second & third weeks with the view of preventing hemorrhage. There seems no definite information obtainable on this point, but I hardly consider this practice one to be recommended generally.

There is no need to discuss the treatment of the complications which may occur in this disease, as there are practically no differences between the modes of treatment in India & in Europe.

The death tendency is through shock from 011, hypophysitis, or general asthenia.

* De Simone (in Rif. Med. Dec. 12th, 1891) recommends calomel for this purpose, after the tenth day of the disease.
Your attempt must be directed to prevent the onset of these conditions, as the possibility of combating them successfully, when pronounced, is but small. In the case of Europeans in India, as every physician's experience tells us, painfully or bitterly received, them. We must particularly bear in mind the strenuous need of conserving to the utmost the vital force in the last half of the illness; at the tropics, we are handicapped by the previous vital depression from heat or "climate"; it also against heat moisture, should the case occur during the seasons when these conditions are paramount.

In convalescence, we have to deal with an inanition, weakened, traumatized individual; functionally weakened heart and brain; an enlarged spleen, perhaps liver, a damaged gut. Several months' rest, generally change of climate—especially a sea voyage—and iron and hypophosphite medicinally, or perhaps ammonium carbonate, to combat...
the hyperkinetic condition of the blood, which predisposes to venous thrombosis. For the latter, should it occur, friction, stimulation, mechanical support, as a rule, will prove serviceable.
Chapter 7. Prophylactic.

First let us consider the disinfection of the deposits in cases of the disease. The ideal plan would be to pass the deposits into any receptacle containing sawdust, preferably impregnated with corrosive sublimate, and then to confine the whole to a clear-burning fire, placing the receptacle mouth downwards on the fire to avoid the risk of sublimation of any infective particles, which might subsequently contaminate the food, water, &c., by falling on them. For this purpose, receptacles made of thin mill-board with a glazed interior, 9 inches x 9 inches, having a rim 3 inches high, would seem preferable because of their cheapness, lightness, & suitability for the surface. Such could be very cheaply affixed, if the demand was created. Failing this, the stools or urine should be passed into a pot containing two or three tea-cupsful of fire-hydrate of mercury solution, which should be acidulated, or coloured by aniline.

*This, after all, is hardly necessary as infective stools do not become infectious for 9-12 hours after being passed.
blue to prevent accidents, according to the formula: Hydroxy hydrochloric, 3 lbs.; Acid. Murial, 3; Aurilide blue, gr. 5; Water, 3 gallons.

After the motion has been passed the disinfectant should be again freely added, then the whole stirred so as to mix the Dejecta & disinfectant thoroughly. The chief of wood used in the ordinary should then be burnt; the stools buried, after standing covered for at least an hour to allow the disinfectant to act well (for Wynter-Dyott has experimentally shown that the degree of disinfection is in direct ratio to the period of subjection of the infective material to the disinfectant's action), it should then be buried in a separate trench, not consigned to the trenches used for burying healthy human ordure.

Carbolic Acid in 5 per cent. solution may be used, if corrosive sublimate is not available.

The directions here given are those I actually use in typhoid cases, which I have found by experience to be most useful.
such other disinfectants as the sulphates of copper, or iron, or zine; the chlorides of aluminium, or zine; lead nitrate have all been found unreliable, as the result of both clinical laboratory reference. In addition, must be enforced. The usual four-room precautions as to placing all linen in pairs of water solutions of disinfectants, subsequent boiling; or for aerial disinfection of the room I would recommend boxes, with very freely perforated lids, containing metallic iodine; or a solution of carbolic acid, 1 part, and ether, 2 parts, placed in saucers about the room; or even in Dufoulin's powder, in saucers.

There are no water-closets in India (except in Bombay & Calcutta), so no mention need be made as to measures to be taken with regard to them, or the drains & sewers.

All the drinking-water used in the house should be boiled, cooled, or reboiled, placed in receptacles pre-

* In imitation of Tyndall & Koch's "Discontinuous Sterilization."
viously washed out with boiling water, kept scrupulously clean, as well as having efficient covers. If the patient dies, his coffin should be thickly sprinkled with Chloride of Lime, & of course, he will be buried the same day - in India. The nurse should wear - if a female - light-colored washing dresses, which should be treated like the sick-room linen; should not mix with the family. An important point is, in washing her hands (which should be frequently done never to wash her face at the same time in the water just used for her hands), to take care not to splash the water she is washing her hands in up on her face. This, I am certain, is often the cause of the infection of typhoid fever nurses. Another important detail is, never to "flick" up the bed-clothes, or patients night-dress when he is going to be examined or attended to; for, by the outdoor jerk, dirt particle
from a soilcd spot of the linen, may be detached from bed.

Finally, the nurse should neither sleep, nor take any food or liquid in the sick-room, for fear of infection.

After the termination of the illness, the room should be thoroughly disinfected, all mattresses should be subjected to a moist temperature of 200-250°F for some hours; all bedsteads should be carefully and thoroughly disinfected, should they be wooden & saturated with fluid excreta, which should always be prevented by placing a waterproof sheet under the patient— this should be used daily and burnt.

Finally, it would be desirable to closely supervise for at least 2 or 3 weeks the other inmates of the house for the poison may be incubating in them, danger may result if they should happen to have the disease in an ambulant form.

So much for the sick-room prophylaxis.
Journal Prophylaxis in India.

Soldiers, on landing in India, should be most carefully shielded from the enormous dangers of contracting the disease on their railway journey "up country." The chief risk is their drinking any water offered them at railway stations, drawn from very dangerous sources of supply, especially, by the "thieves" (native water-carriers) whose "mussucks," or water bottles, have before been advertised as a great source of the disease. To avoid this danger, that of the men drinking native aerated waters, eating food, fruit, etc., probably brought direct from the filthy native quarter, I would suggest that the experiment should be tried of carrying, in troop-trains, a supply of carefully boiled drinking-water in proper receptacles, a stock of aerated waters made under close European supervision (as at many messes in India) from
carefully boiled water; that food, ice, fruit, etc., of good quality should also be carried in the trains. There are only too many European trade men at the various centres who would heartily cooperate in the carrying out of these measures, for the contract would prove a highly lucrative affair to them.

The distribution of food, drinks, etc., could be carried out under regimental arrangements very easily. Troops should be put on arrival to a hill-station for their first two years in India, for these stations are generally not endemic centres of bilious fever, & the barrack sites there are far safer from contamination by faecal & organic impurities than those in the plains. The water supply in the hill-stations is generally very good; so much more hygiene could be taken during this time. More military manoeuvres were as performed — hence a higher

*This would be economical in every way for Government. Smaller barracks (because of smaller cubic capacity) than in the Plains could suffice.
state of efficiency maintained, than is ever possible in the hot season in the Plains. And the troops would then gradually learn to take care as to what they ate, drank; generally how would learn how to live in the tropics. Regiments, proceeding on active service, after being stationed in the hills, have always proved to be the most healthy, rigorous and generally useful of all others.

Moderation in diet & alcoholic indulgence showed, as far as possible, be advised, though it is a very hard thing to influence the soldier in these matters.

The conservancy arrangements should be carefully attended to, the night soil conveyed to trenches 2 or 3 miles from hill.

But if a regiment cannot be set to a hill station, how must we guard the soldier from hiccups and in a Plains station?

I saw, I saw, that Hirsch considers that few fruits in the history of...
are so certainly proved as that it is
most perversely conveyed by drinking water.
And this applies equally to India.
Again bad water - though not tainted
with Salizic Fever poison - predisposes
to the disease by irritating the gut, as has
been shown before.
We must try to educate the troops, or
people generally, in India, to be, at all
costs, for their drinking water supplies,
to a source where the water is proved
by careful, skilled, chemical & bacteriological
analysis, to be absolutely pure.
I should see that it is invariably
boiled, cooled & boiled when taken
from any other source.
Aerated water should alone be obtained
from a source where the water is,
under European supervision, taken from
a good source, & carefully boiled before
the manufacture of the aerated water.
Ice should be made from previously
boiled water.
The milk supply should receive constant
attention. The cows should belong to
some responsible body—such as the
Dress Committee or the Coffee-shop
Committee of a regiment—should
be fed carefully, wel-fed, stabled;
should be milked under the close
supervision of orderlies selected for
intelligence and experience of the devices
by which the milkman defrauds
the buyer, principally by starting
his milking with his milk-can
containing some water, or by concealing
a “mussuck” full of water under his
clothes from which runs a thin rubber
pipe down his arm and along his palm
by which means he runs in as much,
or more water than milk.
The milk-cans should be carefully
raised with boiling water; the cow’s
udder, the milkman’s hands washed
well; if it should be seen that, if
the cow has a calf, there is nothing
of the nature of a specific activity
affecting the calf, for it is uncertain
whether this disease is not closely
related to human typhoid fever.
After the cans are filled with milk, they should be placed in the ice-house (where one exists in the station); it is distributed under close supervision.

As to the food, the meat supplied by the Commissariat is generally good and is supplied under strict European supervision. Vegetables should be carefully washed if to be eaten raw, as tomatoes, lettuce, etc. Fruit should be carefully supervised, and should not be eaten when bruised.

Great care should be taken to cover carefully all meat, as other articles of food, when standing on the table, to prevent the access of flies, with legs probably soaked in liquid excreta, or other filth, as before described. The aerial propagation should be prevented as far as possible by enforcing strict cleanliness in the neighborhood of as well as within Cantonment limits. Latrines and others caught defecating in the open in Cantonments should be punished, etc., public latrines
might be advantageously supplied, it would cut but little if constructed of
fluted iron sheeting.
Surface subsoil drainage should be
strongly efficient so as to prevent
the putrefaction or decomposition of animal
or vegetable matter, thus lessening the
number of "forcing" beds of the disease.
A high standard of personal cleanliness
should be rigidly enforced in all men;
outdoor sports & exercise, in the morning
& evening, should be encouraged as much
as possible so as to keep them away
from the native bazaars, & villages.
The state of the native bazaars, villages,
& towns should be attacked as far
as possible. This is a matter which
the native, unreflecting ignorant, fatalist,
utterly indifferent as he is,—
will probably resist, & to one must
feet that way with the utmost circumspection.
We must begin by explanation
of the necessity for & advantages of,
sanitary reform to educated influential
natives, who should be induced to the
almost to use their influence with their co-religionists to further improvements in this respect. Again native doctors, hospital assistants, 
&c., may be of use in diffusing some knowledge on these
points among the people themselves in their capacity of medical attendants.
Public explanations, or lectures, from time to time on the subject may enlist a
certain amount of sympathy & cooperation among the more intelligent members of the community. And Government
ought to enforce such obvious sanitary measures as good Conservancy arrangements, a pure water-supply preferably led in from a distance, so as to escape the endemic influences of the place supplied; & should aim at modifying over-crowding, & other
sanitary conditions.
Such simple changes as these even
will take time to effect in conservative
aphlectic India; so it will be many
years before even an approximate
sanitary condition can be attained,
as all who know the country the people must respectfully admit. To sum up shortly, the desiderata which must be constantly before the Medical Officer in India are: the elimination of filth as the fostering bed of the virus; the severance, so far as is possible, of the links of transfer between the source of production, the young, susceptible soldier; the increasing of the age of soldiers serving in India, so as to bring them to that country when the age susceptibility shall have assayed materially; to reduce the frequency of change between India and England, by lengthening the service period for India; or by offering greater inducements for twice-joined men to re-engage for a further period of service in that country; to locate every man it is possible to in hill stations; to attack vigorously the hot-beds of the disease—the native quarters with their terribly insanitary conditions.
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FINIS.