

PYREXIA AS THE MOST PROMINENT CLINICAL FEATURE OF  
CERTAIN OF THE INFECTIONS MET WITH IN PRACTICE IN  
INDIA, WITH REFERENCE TO SOME CASES ILLUSTRATING  
DISEASES IN WHICH THE CAUSATION IS LIABLE TO BE  
OBSCURE.

by

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The clinical manifestations of many of the infective diseases met with in practice in India are liable, often, to be represented for a longer or shorter period from onset by the phenomena of fever only, and to fail to present a clinical picture from which the nature of the infection can be recognised readily.

A problem in diagnosis is thus presented to the physician practising in India which differs in some aspects from a similar problem in Great Britain. For in India, in studying cases in which the significance of pyrexia is not clear, the physician has to bear in mind its common causes in temperate climates, as well as the causes peculiar to the country in which he is practising. He must carefully guard himself against a supposition that any pyrexia in a tropical country is necessarily tropical in origin in order that he may not be misled into overlooking or misinterpreting evidence of such infectious as are due, for example, to the Tubercle Bacillus, the Spirochæta Pallidum, the pyogenic cocci or the Bacillus Coli Communis.

In the circumstances of the problem which this thesis attempts to describe, the prominence of pyrexia in the clinical picture begins with the patient's/

patient's description of his symptoms.

It is a frequent experience of every physician practising in India to be confronted with the problem presented by the patient who, in reply to the question "Of what do you complain", answers "Fever". This description of his sensations is given by European and Indian alike and by both is intended to convey an impression of the sensations of bodily heat and discomfort which accompany a rise of the temperature above normal, including such complaints as headache, aching in the back and limbs, rigors, feelings of coldness. The Indian patient, if he has no English, will apply his vernacular word, *bukhar* - to convey such a description as the above. Ignorant and illiterate as he often is, he has little facility in giving a history of his illness or a description of his symptoms, so that more diagnostic possibilities are covered by the vernacular word than by the English one. In the case of the Indian patient, therefore, it often happens that, even in reply to leading questions, a description is not obtained of symptoms such as ordinarily might be expected to be present, and to give a definite direction to the search for causation in certain infections.

Very diverse conditions fall to be included in the/

the consideration of causation in patients whose complaint is "fever", and in dealing with such cases, I have found it of value to bear in mind a list of causes arranged in a way which will fit a systematic method of investigation.

To give concrete form to the problem a statement is here made of a list of conditions in which, at times, the patient first presents himself with no complaint other than "Fever".

#### I. CONDITIONS IN WHICH A LOCALISATION IS FOUND.

- A In the Alimentary System:- Tonsillitis, Hepatitis,  
Abscess of the Liver,  
Cholecystitis,  
Helminthiasis.
- B In the Respiratory System:- Pulmonary Tuberculosis,  
Pleurisy, Pneumonia,  
Empyema.
- C In the Circulatory System:- Sub-acute Infective  
Endo-carditis.
- D In the Genito Urinary System:- Pyelitis. Syphilis.
- E In the Haemopoietic System:- Leukaemias.  
Hodgkin's Disease.
- F In the Nervous System:- Cerebro Spinal Mening-  
itis. Heat-stroke.

II. OTHER CONDITIONS.

- A Initial Fever of Smallpox, Typhus and other Exanthemata.
- B Malaria.
- C Relapsing Fever.
- D Filariasis.
- E Sevenday Fever.
- F Dengue.
- G Sandfly Fever.
- H Enteric Group of Fevers.
- I Malta Fever.
- J Kala Azar.
- K General, or obscure local, Septic Conditions.

Such a list as the above helps to ensure a systematic investigation and predicates as a first diagnostic rule a careful physical examination by bedside methods of the different systems in which a local infection may be found as the Cause of the Fever. This is completed by microscopic examination of faeces, sputa, urine, or cerebro-spinal fluid if indicated. The investigation is then followed up by the preparation of blood films with a view to discovery of parasites in the blood, and to assist in the exclusion of conditions such as the leukaemias, in which pyrexia may be/

be a prominent feature.

A differential and total leucocyte count is next suggested by the list, followed by blood culture and serological tests. It may be necessary then to complete the investigation by enumeration of the red blood corpuscles, estimation of the colour index and, where the preceding examination has suggested Kala Azar, by spleen puncture.

Although, in all of the above conditions, symptoms and physical signs may be present which are definite enough either to be pathognomonic of the underlying cause, or to be highly suggestive of it, yet in all of them, at times, pyrexia may be the prominent or only clinical feature, and other physical signs may be so indefinite as to be liable to be overlooked.

It is proposed to select some examples of the latter class and to illustrate them from cases which have occurred in my own experience, and by reference to the recorded experience of others.

Before doing so it is necessary to give some indication of circumstances which influence diagnosis in the work of many practitioners in India.

Whilst the omission of a careful search, by ordinary bedside methods, for a local infection in any of the systems, would lay the physician open to criticism of his competence, the utmost collaboration between clinician and pathologist which is often necessary/

necessary to secure an accurate diagnosis in some of the Fevers is more easily postulated than secured. This is particularly so in the circumstances of general practice amongst patients in their own homes, or of consultative practice with Indian practitioners. Even in practice in the Government Military and Civil Hospitals, at all events outside the larger and more important centres, facilities for complete laboratory diagnosis may be, either defective, or non-existent.

The physician has often, therefore, to be prepared to rely on such laboratory methods as he can carry out himself; and accuracy of diagnosis is liable to be sacrificed to the extent to which practicability imposes restrictions on the elaborateness of laboratory equipment, and to the extent to which such equipment can be utilised systematically by a physician whose administrative and clinical obligations are time consuming.

The almost complete lack of trained nurses for attendance on Indian patients in their homes, even for those who can afford to pay for them, and the inadequacy in numbers or defective training of the nursing staff in many of the hospitals, may also be referred to here in connection with the difficulty of securing systematic record of regular routine observations of such points as temperature, pulse, respiration and urinary output.

THE PRE-SUPPURATIVE STAGE OF AMOEBIC HEPATITIS.

Leonard Rogers (1) in describing the pre-suppurative stage of Amoebic Hepatitis states - "No one who has had a large experience of the early stages of amoebic abscess of the liver in a tropical climate can fail to have been struck by the great frequency with which definite clinical symptoms of suppuration in this organ are preceded by weeks or even months of fever during which there may be few or any symptoms pointing to the liver as the cause of the trouble; this fever is commonly ascribed to and treated as Malaria".

This author's investigations appeared to him to indicate that the early pre-suppurative stage of Amoebic Hepatitis may be recognised frequently by the blood changes (leucocytosis of a special type) in a stage which admits of rapid cure, and that the patient may thus be prevented from drifting into the much more serious suppurative stage.

He remarks, - "these cases constitute a distinct class of fever usually of a chronic intermittent type, sometimes with no very definite symptoms of hepatitis and rarely with any dysentery. They may be recognised, or at least strongly suspected by the presence of a moderate degree of leucocytosis, generally of the type in which the proportion of polymorphs is either normal or only slightly in excess. (2)

He emphasises that this kind of fever yields rapidly/

rapidly to ipecacuanha in large doses, or emetine, and that the formation of tropical abscess is thus prevented.

From his description of cases three Clinical groups may be recognised:-

1. Cases of acute Hepatitis complicated with Dysentery.
2. Cases of Hepatitis without Dysentery.
3. Cases of Hepatitis without Dysentery or Liver symptoms.

Of this last class he says that all his cases showed Leucocytosis without symptoms of dysentery or acute Hepatitis being present; fever, for which no definite cause could be found, being the condition for which they were admitted. Satisfactory results were obtained by treatment with ipecacuanha although there was neither pain nor tenderness of the liver present when they were first admitted, in spite of slight enlargement in three of the four cases.

The susceptibility to treatment with ipecacuanha of the pre-suppurative stage of Amoebic Hepatitis was demonstrated in a discussion at the Asiatic Society of Bengal in June-July 1910, and the effect of ipecacuanha on the Leucocyte curve was illustrated in a contribution made by Greig. He describes a case in which fever and/

and Leucocytosis with no definite signs of Hepatitis followed a slight attack of Amoebic Dysentery, and was readily cured by Ipecacuanha. (3)

Rogers views have been generally endorsed.

The following passages occur in one text book of Tropical Medicine. (4)

"Amoebic Fever or General Amoebiasis - This term has been applied to certain cases of *L. Histolytica* infections in which there is fever but no dysenteric symptoms and no signs of Hepatitis. A few cysts are generally found in the stools and emetine induces a prompt disappearance of the Fever," and also the following - referring to Liver Abscess.

"The early stage, called by Rogers the pre-suppurative stage, is of the utmost importance for if it can be recognised and appropriate treatment applied, the disease may be stopped in a certain number of cases".

"In those cases of Liver Abscess in which fever is the only symptom the diagnosis may be extremely difficult and may require all modern bacteriological methods to exclude Malta Fever, Malaria, Septicaemia, Typhoid etc".

The following case may be quoted from my own experience as an example illustrative of the thesis.

It/



It was the first of its kind which came under my own care and occurred when the features of the pre-suppurative stage of Amoebic Hepatitis were less generally recognised than they are today.

E.O.J. aged 38, married, a chaplain of the Indian Ecclesiastical Department, came under observation on 10th February 1912, complaining of Fever, and feeling out of sorts. His previous health had been good. He had had Malaria some years previously. There was no history of Dysentery, but he had had several attacks of Diarrhoea during his ten years residence in India. The last of these attacks occurred about six weeks before the present illness. He was very moderate in his use of both tobacco and alcohol. He complained that he had not felt well for a day or two, had no appetite, that he had a headache and was constipated.

The cause of the Fever was not made apparent by examination of the various systems. (See Chart I.)

No Malaria parasites were found in the blood films, and a differential leucocyte count on the 2nd day under observation showed 72% of polymorphs, 20.2% of lymphocytes, 6.5% of large mononuclears, 1.3% of eosinophiles. A blood culture in a taurocholate of soda medium remained sterile.

A serum reaction on the 12th day of the illness showed agglutination by the microscopic method against bacillus typhosus, in dilutions of 1 in 20 in 5 minutes, 1 in 40 in 10 minutes, 1 in 60 in 20 minutes. Slight tumidity of the abdomen was noted in the progress of the case and slight enlargement of the liver. There was no tenderness of the liver nor elsewhere in the abdomen.

The bowels were constipated throughout, and the pulse was relatively slow. No eruption of rose spots was observed and there was little appearance of prostration or general toxæmia. The spleen was not palpable.

The fever was regarded as being due, possibly, to a mild infection with bacillus typhosus, and was treated accordingly.

It was ascertained later that the significance of the Widal reaction was lessened by the fact that the patient had been inoculated against Enteric Fever at the time of the South African War.

The discovery of increase in the degree of enlargement of the liver led to a total count of the leucocytes being carried out on the 44th day of the illness, and a Leucocytosis of 18,000 with 78% of polymorphs nuclea leucocytes was found. There was then/

then slight tenderness on firm palpation of the liver.

Treatment with ipecacuanha (emetine had not then been introduced) was begun.

The drug was administered in the old fashioned way in pills of the powdered root, given at night, three quarters of an hour after 15 grains of Chloral Hydrate - food and fluids being withheld for some hours prior to the administration.

The patient disliked the treatment intensely and could tolerate only moderate doses, but in spite of that there was a rapid decline in the temperature with six doses of ipecacuanha varying from 18 to 24 grains each. On the day after the 7th dose of ipecacuanha the leucocytes were 10,000 per cmm.

Convalescence was interrupted by a recurrence of fever which subsided after 7 doses of ipecacuanha. The recurrence of the fever was associated with a leucocyte count of 16,000. (See Chart.)

Convalescence thereafter was uninterrupted. Four days after the temperature finally reached normal the leucocytes were 8000, and the liver was no longer palpable. Weekly doses of 24 grains of ipecacuanha were continued for six weeks after the temperature finally reached normal.

Remarks:-/

## Remarks:-

Although proof of the amoebic origin of the fever in this case may not be complete, the co-existence of an enlarging liver with leucocytosis, and the therapeutic effect of ipecacuanha would be difficult to explain on any other assumption. Rogers gives his experience of the Clinical association between dysentery and amoebic liver abscess in reference to 50 cases. In 72% of these there was a history of dysentery, in 14% of diarrhoea, and in the remaining 14% no history of bowel lesions at all. (5)

The history of diarrhoea only preceding the fever in the case recorded above is not therefore exclusive of its amoebic nature.

I had occasion, recently, to observe how the fever due to amoebic hepatitis may pass unrecognised until rupture of an abscess occurs.

A Mohomedan male, aged 45, had been under treatment from 15th July 1921 till 22nd November 1921, suffering from fever which has been regarded first as malarial, and latterly as due to pulmonary tuberculosis. It was stated that a profuse haemoptysis had occurred on the latter date. When I was asked to see him a few days later the diagnosis had become apparent from the change in the sputa to a viscid chocolate coloured material/

material, and examination of the liver left no doubt as to the nature of the condition. From questioning the patient it appeared that his attention had not been attracted by pain or discomfort in the region of the liver, his sole complaints had been "Fever", weakness and loss of appetite. He gave no history of rigor or sweats.

Fortunately the fever rapidly subsided and the expectoration ceased with treatment by emetine.

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SEROFIBRINOUS PLEURISY.

An insidious mode of onset without symptoms referable to the respiratory system, or at most with slight dyspnoea on exertion is recognised as occurring in Pleurisy with effusion, and is referred to in Osler and Macrae's "Principles and Practice of Medicine".

An onset with high pyrexia as the prominent clinical feature without symptoms pointing to an involvement of the respiratory system, and in which the physical examination of the chest showed the commencement of an accumulation of fluid nine days from the onset of the illness, is illustrated by the case which follows. Its inclusion in the material of the thesis is suggested by my experience of a number of other cases admitted to hospital with diagnoses which showed that the predominance of pyrexia had led to the causation being overlooked.

D.M. Mohammedan, male, aged 22. Medical Student of Amritsar, first came under my observation on 1st April 1922. He complained of "Fever", headache, and pain in the back of 7 days duration.

His history was that on the 26th March he had felt tired and hot and had fever with headache and backache. There was no rigor. He had taken his own temperature/

temperature every day since and it varied between 100.5 and 102. It had not come to normal. He had no cough nor pain in the chest. His bowels were constipated. He had continued attendance at his classes during the week as class examinations were in progress and he was reluctant to miss them. He sought advice on Saturday 1st April, and was admitted to hospital. He had had no serious illnesses previously. There was a history of occasional attacks of Malaria, none of Dysentery.

At the first examination on the 1st April a careful physical examination of all the systems failed to discover any obvious cause for the fever from which he was suffering. His temperature then was 103<sup>o</sup>, pulse 110, respirations 24. Some pallor and slight anxiety of expression were noted. Malaria parasites were not seen in blood films, and there was no obvious leucocytosis.

On the 3rd and 4th April (i.e. 9th and 10th days of the illness) it was noted that there was slight diminution of respiratory movement of the left side of the chest, that the tactile fremitus was a little less over the left base as compared with the right, and that there was slight impairment of percussion resonance below the left scapula with breath sounds, which were more intense than on the opposite side, but vesicular in character, without accompaniments. There/

There was no complaint of cough nor pain in the chest, and no dyspnoea was observed.

By the 7th April the signs of a moderate left sided pleural effusion were unmistakable and in definite contrast with the slighter signs noted earlier. The spleen was displaced so as to be easily palpable. The upper level of the dulness in front was noted at the 4th rib in the left mammary line, and on the 10th April at the 2nd, and on the latter date 35 ounces of a clear, slightly blood tinged fluid were withdrawn by aspiration.

A coagulum formed rapidly in the fluid.

The fluid was culturally sterile. No tubercle bacilli were found on staining a preparation from the centrifugalised deposit. The cells in the deposit were numerous and were practically all lymphocytes.

The patient made a satisfactory recovery and the course of the fever is shown in the copy of the temperature chart opposite. (Chart II.) There was no cough, expectoration, dyspnoea, nor pain in the chest throughout the whole illness.

In the earlier stages of the illness, whilst the cause of the fever was still in doubt, a blood culture for isolation of an enteric group organism remained sterile, the urine also was sterile; the leucocytes were 9000, polymorphs 72.5%, lymphocytes 20%, large mononuclears 6.5%, eosinophiles 1%, and agglutination tests with bacillus typhosus and para typhosus A & B gave negative results.



In further illustration and as a contrast to the above case a reference may be made to another in which a fully developed pleural effusion was present when the patient came under my observation, but in which there had been a similar predominance of fever and absence of symptoms pointing to the respiratory system. This had in fact led to the patient's admission as a case of Fever for which no definite cause had been found, Malaria or Enteric Fever being suggested.

(III)

The Chart opposite<sup>A</sup> shows the high pyrexia which characterised the illness.

K.A. Mohammedan Male aet. 22. Medical Student was admitted to Hospital on 5th May 1921, when I saw him.

He had been ill for 8 days complaining of Fever and pain in the back. His illness began with a rigor followed by a high temperature. He had a severe headache and pain in his back. When questioned on the point he said he had had a slight cough for 3 days prior to admission. There had been no pain in the chest. He had been told that his spleen and liver were both enlarged and he had been treated with aspirin and quinine.

He was an individual of unusually fine muscular development and was the champion gymnast of the Medical/

Medical School. He gave no history of previous illnesses and the family history was satisfactory.

On examination, - there was some anxiety of expression, general discomfort was complained of but no pain. There was no pallor nor other obvious morbid appearance. His temperature was 103.6, pulse 95, respirations 24. Cough was not noticed at the time of examination but there were obvious physical signs of effusion in the right pleural sac. The apex beat was displaced to the left, and the liver was displaced and formed a projection in the right hypochondrium. The spleen was also palpable. The abdomen generally was somewhat distended.

He became dysnoeic and 46 ounces of a clear greenish yellow fluid were removed by aspiration on the 8th May, and 25 ounces on the 10th. The pyrexia continued to be of a high remittent type and persisted throughout his stay in Hospital until he was sent to a Hill Station in the Himalayas.

In searching for the etiological factor concerned in the causation of the effusion the fluid withdrawn was examined.

Cultures remained sterile, no tubercle bacilli nor any other organisms were seen in the stained specimens of the deposit. Cytologically lymphocytes predominated and were very numerous.

The morning sputa were examined on 5 occasions but/

but no tubercle bacille were seen.

His total leucocyte count on 8th May was 6000. Serological reactions with bacillus typhosus, para A & B were negative, and there were no malaria parasites in the blood films on two occasions. An infection with the tubercle bacillus was presumed and he was treated on that assumption.

He reported to me again on 17th September 1921. He was then able to get about but looked thin and ill. He stated that the fever had gradually left him, but that he still sometimes had a rise to  $100^{\circ}$  in the afternoons. He had some cough. On examination, he had definite signs of disease at both apices, and there were tubercle bacilli in the sputa. There was defective resonance and feeble breathing at the right base.

He insisted on returning to his home in a village in the Northern Panjab. In January 1922, a report of his death was received from his father.

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CHRONIC PULMONARY TUBERCULOSIS.

Chronic Pulmonary Tuberculosis as a cause of fever in India is perhaps more frequently overlooked than any other disease. It is particularly in those forms with a malaria-like mode of onset that the mistake occurs. The possibility of this error occurring is referred to in the standard text books. (6)

A Pyrexial form of onset which has not received general recognition has been described by Roberts as occurring in India. (7)

He defines it <sup>as</sup> an acute febrile form resembling a typical enteric, and calls it "acute tuberculous fever", met with principally in young adults. He describes an onset with continued pyrexia of a remittent or intermittent type lasting from a week to four weeks or more, and cases of duration of 2, 3, and 4 months occur.

He continues by stating that the liver is often enlarged, the spleen is not. The bowels are obstinately constipated and the abdomen shows some amount of meteorism but never to the extent seen in Enteric Fever.

The most distinguishing feature is pain and tenderness in the epigastrium and on this special stress must be laid in diagnosis. The mind is clear except with/

with very high temperatures at night when there may be delirium. The paroxysms of pyrexia do not produce the prostration seen in Malaria and Enteric. The tongue is furred but it does not remind one of the enteric tongue. The skin is dry and hot, there are no hectic nor malarial sweats.

Such cases are often called simple continued fever or atypical typhoid. In India such patients almost invariably recover. It is not a picture of 'Acute Miliary Tuberculosis' nor of the "typhoidal type" seen in Europe and there so fatal.

He believes that in many cases it means the launching of a patient on a tuberculous career, and that amongst Indians, after the fever, numbers develop obvious tuberculosis, and the longer the fever lasts the greater is the danger.

Consulting with Indian practitioners (during the last two years) in cases of pyrexia whose causation was obscure, I have thought sometimes that I recognised conditions resembling those described above, but lack of opportunity for further observation of the cases has left me undecided.

Recently, however, I had opportunities of examining a case sufficiently often to collect data, which, though not complete, seem adequate to illustrate <sup>a</sup> mode of/

of onset of Tuberculosis of the lungs with resemblances to that described by Roberts.

As pyrexia was for long the most prominent feature, and the diagnosis required much discussion, a statement of the salient facts will be germane to the subject of this thesis.

T.C. Hindu, female, married, aet. 18. Complaint "Fever" of 11 days duration.

I saw her along with the family physician on 8th April 1922. The History was that on March 29th she had felt ill and had a headache, and had a shivering fit in the afternoon. Her temperature was recorded as 102°. The fever had been continuous since that date and a record of the temperature had been kept which showed that it had ranged between 101° and 103°, and that the pulse rate had been 120-124 throughout. She had no symptoms except general discomfort and some headache. Her attendants said that it had been difficult to keep her in bed all day as she wanted, and seemed able, to sit up, and insisted on getting up to attend to the functions of bowel and bladder. She had been constipated.

Previous Health:- stated to have had an attack of dysentery one year ago and to have recovered after treatment/

treatment with emetine. After her marriage 9 months ago she had lost her appetite and became pale and thinner than usual. She had recently left Calcutta to take up her residence with her parents in Amritsar during her husband's absence in England, and had improved in health.

A brother suffered from consumption and had been away in the hills for six months undergoing treatment. He had had haemoptysis and was not improving.

The parents were healthy as were two other children. The father was a wealthy merchant and the home conditions were good and even luxurious as these things are reckoned in India.

The cause of the fever was in doubt. Enteric Fever was suspected. Blood films had been examined and no malaria parasites found. A serum reaction carried out on the 9th day of the fever was reported to have given agglutination with bacillus typhosus in 1 in 20 dilution only. No cause had been found in examination of the various systems.

On examination:- The patient did not look seriously ill. She was sitting up in bed and there was no appearance of prostration. She was pale, but well nourished. Temperature 103°. Pulse 128. Respirations 24. The abdomen was soft and flaccid, there was no tenderness and the spleen was not palpable. No eruptions/

eruptions were observed. The only abnormal physical signs noted were very slight impairment of percussion resonance in the 1st<sup>and</sup> 2nd right intercostal spaces one inch from the lateral sternal line; and a similar slight impairment on the right side behind at the level of the 2nd dorsal vertebra  $1\frac{1}{2}$  inches from the spine, and in these areas the breath sounds were slightly less intense than on the opposite side, and inspiration was interrupted. There was also a fine friction sound with inspiration internal to the angle of the right scapula. Patient and relations were emphatic that there had been no cough nor expectoration during the illness.

There were no signs of pregnancy.

The urine was high coloured and had a deposit of urates. Chemical examination showed no abnormal ingredients.

At this examination and two days later when I saw her again a blood culture was prepared and serum collected, the leucocytes were counted, blood films made, and a catheter specimen of urine obtained. No sputum could be obtained for examination.

The fever had not fallen below  $101^{\circ}$  up to this time.

Blood and urine both proved culturally sterile; the leucocytes were 6000 per cub. mm.; the proportions of the different leucocytes were within normal limits, and/

and serum reactions with bacillus typhosus, paratyphoid A & B, Micrococcus Melitensis and Bacillus Coli Communis were negative in all dilutions, except a positive with bacillus typhosus in 1 in 20 dilution only.

The bacteriological and serological examinations were carried out in the Laboratory of Lahore Medical College.

I examined the patient again on 13th April. The temperature still remained constantly above  $101^{\circ}$  and on two occasions had reached a maximum of  $104.5^{\circ}$ . The pulse was fast, 124-140 being recorded. There was still surprisingly little prostration considering the height and duration of the fever, and I was told there was still difficulty in getting the patient to observe the absolute rest in bed which was being enjoined.

She now had a little dry cough. Examination failed to reveal anything different from what had been noted at the first examination, except that the friction sound was no longer audible.

Such material as could be obtained from early morning expectoration was examined for tubercle bacilli with a negative result.

Such evidence as there was appeared to me to support a diagnosis of Pulmonary Tuberculosis.

The Widal reaction with Bacillus Typhosus was repeated, and still gave agglutination in 1 in 20 dilution/

dilution only.

My connection with the case ceased till two months later, when I had an opportunity of examining her again.

In the interval the temperature had first become remittent and lower in its daily maximum, and then intermittent, being normal in the morning and rising in the afternoon to 101 or 101.5. For a few days it did not rise beyond 100°.

The patient was not very noticeably thinner, but was pale.

There were signs of consolidation of the right upper lobe down to the 2nd rib in the mammary line in front, in the supra spinous region of the scapula behind, and at the apex nearer the vertebral column. Defective movement, increased tactile fremitus, Bronchial breathing, and a few medium crepitant râles were present along with increased vocal resonance. There were also rhonchi and coarse bubbling râles over both lower lobes behind.

The abdomen was slightly tumid and the liver projected an inch below the ribs in the mammary line and was tender.

Tubercle bacilli were now present in the sputum in considerable numbers.

My oft repeated advice was at last taken by the parents/

parents and the patient was removed to a hill station where conditions were more favourable for the treatment of Pulmonary Tuberculosis than in an Indian city in the hot season.

When last heard of in August 1922, the patient was said to be improving.

Whatever view be taken of the type of Pulmonary Tuberculosis represented by this case, the prominence of fever, the indefinite signs and the absence of symptoms in the earlier stages were noteworthy. The course of the disease as far as it could be followed was not that of acute Broncho-Pneumonic Phthisis nor acute Miliary Tuberculosis.

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PYELITIS IN THE PUERPERIUM.

The occurrence of fever during the puerperium must always be a cause of anxiety to the physician, and should be regarded as due to puerperal infection until it has been clearly demonstrated that some other exciting cause is responsible. In the East, Malaria can be accepted as one of such causes only by the demonstration of the malarial parasite in the blood in a patient without any evidences of puerperal infection.

Looking for other causes there is one which is liable to be overlooked and which is sometimes manifested mainly by pyrexia, and this is Pyelitis.

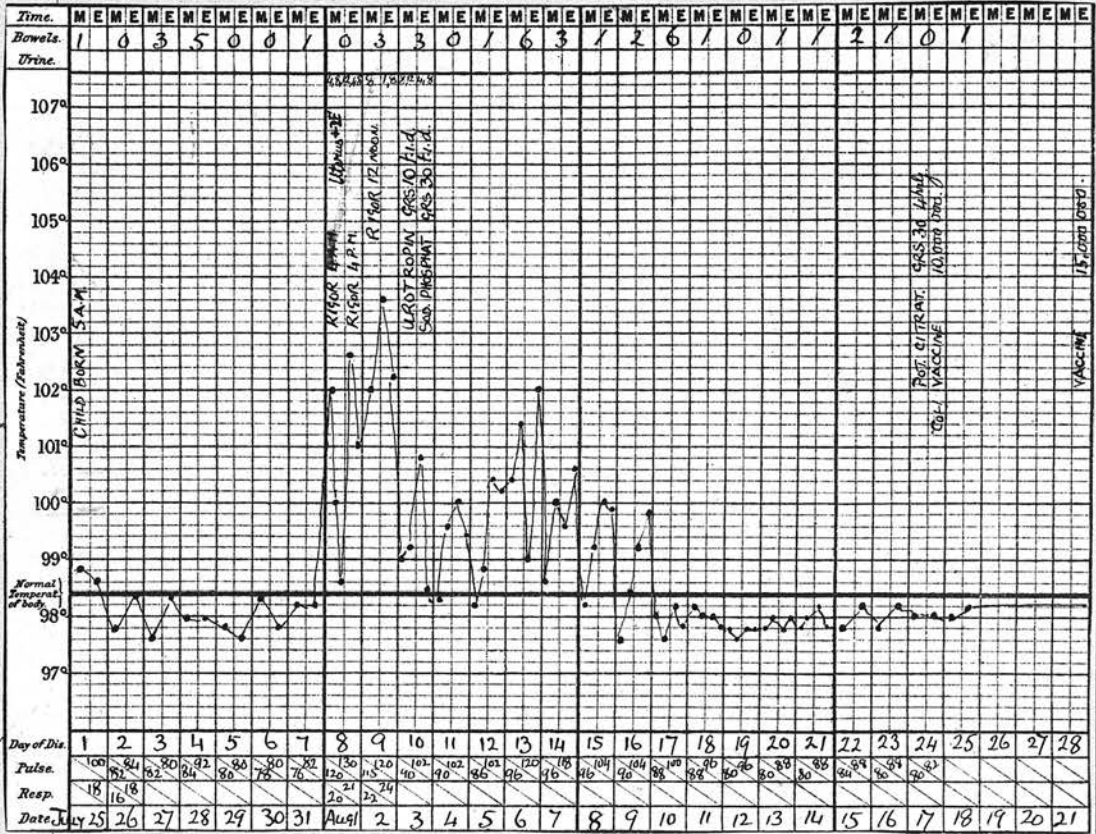
Pyelitis as a complication of the puerperium may perhaps be relatively more common in India than in temperate climates, and reference will be made to this point later.

The condition which it is sought to illustrate is that of a primary pyelitis, not preceded by any evidence of cystitis, occurring suddenly during the puerperium with fever and without symptoms definitely referable to the urinary system.

The following case illustrates such a condition:-

Mrs/

CHART IV.



Mrs A.T. European, aet. 38.

B. Coli Communis Pyelitis complicating puerperium.

Mrs A.T. European, aet. 38.

Under observation during her 3rd pregnancy and confinement.

Her first two labours had been normal.

No previous illnesses were described.

Her health during the third pregnancy had been satisfactory except that constipation was troublesome.

The urine had been examined regularly throughout pregnancy in accordance with my usual custom and no physical nor chemical abnormality was noted. No microscopic examinations of the urine had been made.

Labour began at 12.30 a.m. on 25/7/14 and the child was born spontaneously at 5.30 a.m. vertex L.O.A. No vaginal examination was made. There was no perineal laceration. The placenta was expressed from the vagina by suprapubic pressure 30 minutes after birth of the child.

Haemorrhage was more than normal, the pulse rate rose to 110, and there was some pallor. An accumulation of blood clot in the uterus was removed by gloved hand and a hot intra-uterine douche was given.

The puerperium proceeded normally. The uterus involuted rapidly and the lochia were normal. There was no retention of urine.

On the 8th day of the puerperium she slept badly and had a rigor at 4 a.m. She complained of headache and/

and aching in her back and the temperature rose to 102°.

When seen by me later in the morning the temperature had returned to normal and she seemed to have no particular discomfort. The fundus uteri could be felt two finger breadths above the pubis, and the lochial discharge was normal. No abdominal tenderness was noted.

A 2nd rigor occurred at 4 p.m. and the temperature again rose to 102.6. She complained of some pain across her back, but did not appear to be seriously distressed.

The spleen was not palpable, and the cause of the fever was not apparent from examination of the pelvis, abdomen or chest.

Blood films prepared when the temperature was high were examined and no malaria parasites were seen.

The blood was again examined on the 9th day and no parasites were found. A third rigor occurred at 12 noon on this day.

A catheter specimen of urine was then obtained and examined. It was slightly turbid, straw coloured, no deposit had formed. Acid. Specific Gravity 1012. Albumen present in small amount. On centrifugalisation there was visible deposit. Examined microscopically there were large numbers of pus cells in every field under/

under a 1/16" objective. Motile bacilli were also seen.

There was no complaint of pain nor of frequency of micturition. I examined the abdomen again and found that there was tenderness on deep pressure over the region of the right kidney. It was not of a very intense nature. There was no tenderness of the bladder nor of the uterus.

The following day another catheter specimen was collected into a sterile flask for bacteriological purposes.

The ordinary specimen showed a heavy looking deposit about 1/4" deep at the bottom of the specimen glass.

Treatment with Urotropin grs. x t.i.d. and acid sodium phosphate grs. xxx t.i.d. was begun, - with large quantities of fluid.

A pure culture of bacillus coli communis was obtained from the urine and a vaccine was prepared therefrom.

She had no further rigors and on the 17th day of the puerperium the fever ceased.

A week later there was still pus in the urine. She was then treated with Potass. Citrate grs. xxx 4-hourly and 3 doses of the vaccine (10, 15 & 20 millions) were given. A specimen of the urine examined/

examined on August 31st, a week after the last dose of Vaccine, contained no pus.

When last heard of a year later she was perfectly well.

Remarks:-

The sudden onset of fever with rigors was the most prominent feature of the illness and neither symptoms nor physical signs attracted attention to the urinary system.

The clinical evidence seemed to be opposed to an infection of the genital tract. Malaria could be excluded. The presence of pus and bacteria in the urine and the discovery of tenderness of the right kidney afforded evidence not inconsistent with a diagnosis of primary pyelitis and the further progress seemed to bear this out.

One would appear to be entitled to conclude that primary pyelitis as a febrile complication of the puerperium is not regarded as a frequent occurrence from the fact that it receives scant notice, or is not mentioned at all, in many text books of Obstetrics. In saying this I exclude the pyelitis and pyelonephrosis following on a cystitis, with definite clinical/

clinical manifestations such as dysuria and frequency of micturition. Nevertheless descriptions of it are to be found scattered throughout the literature of obstetrics.

In "Midwifery by Ten Teachers" (8) the following is the only reference to it.

"Pyelitis during the puerperium - Attention has been called recently to the occurrence of Pyelitis during the puerperium. The symptoms are late in their onset as a rule and are frequently complicated by rigor. It is by no means uncommon to mistake such cases for puerperal septicemia or appendicitis; therefore to avoid the error care should be taken to examine the region of the kidneys for tenderness and the urine for the presence of a coliform bacillus."

Whitridge Williams (9) describes pyelitis and pyonephrosis in the puerperium as sequelae of cystitis, severe or mild, but does not refer to it as occurring without prior clinical manifestations of cystitis.

R.W. Johnstone (10) does not include it in the complications, nor does Jellett. (11)

I have been unable to get access to recent editions of other text books such as Berry Hart, Davis, Edgar. The older editions do not mention it.

In American literature specific reference is made to it by Cumston (12) and Vineberg (13). Both these writers deal most largely with the pyelitis of pregnancy/

pregnancy and the bibliography given by the latter deals almost exclusively with that condition.

In the cases of pyelitis complicating the puerperium described by these writers there was generally an onset with rigors and fever in a puerperium which had, up till then, been apparently normal, but abdominal pain was a more prominent feature than it has been in cases in my own experience. Both describe cases without symptoms of cystitis. Cumston regards diagnosis as important and difficult because uterine infection comes to mind, in addition to appendicitis, typhoid or some intestinal infection, and thinks it is often not diagnosed because it is forgotten.

F.H. Smith (14) also regards cases of pyelitis after labour as hard to recognise and refers to eight cases described by Murray (15) in three of which there was a total absence of local signs and symptoms. The temperature was remittant, chills were absent, the patients were seriously ill.

As regards the prevalence and the nature of the condition as it has been met with in India an impression may be obtained by reference to the work of Rogers (16). He refers to urinary infection with the bacillus coli as a not infrequent cause of obscure pyrexia, and tabulates 17 cases of urinary infection in/

in the puerperal state. He regards such cases as being very common and states that over 20 cases were met with in a single year in the Eden Hospital in Calcutta. He remarks that there are usually no symptoms referable to the urinary system and that therefore unless the condition is suspected and the urine examined the cause of the fever is liable to be overlooked. The irregularity of the temperature curve and the frequency of the occurrence of rigors may cause the cases to simulate serious septic infections due to streptococci, and when present in the puerperal state give rise to great alarm.

The cases which he analysed were amongst both Indian and European women.

Including the case recorded above I have met with 3 instances of pyelitis complicating the puerperium in 150 European confinements in India, and my experience directs me therefore to regard it as one of the conditions to be borne in mind in cases of fever, occurring in the puerperium, of which the cause is in doubt.

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SYPHILIS.

The fever due to syphilis was a prominent feature in a case in which infection originated in a fleeting extra genital lesion which passed unnoticed at the time of its occurrence.

Chart V is that of a Zenana Mission Society's hospital nurse, an elderly unmarried English lady, who was seized with a rigor, headache, and aching in the limbs on 17th January; the cause remained obscure until an unexpected direction was given to the diagnosis by the appearance of a faint rash which was first noticed on the sides of the abdomen and chest on the 21st January, and developed later into an eruption of reddish brown macules about the size of threepenny bits, widely distributed over the front of the abdomen and chest, and centripetally on the front of the limbs, along with congestion of the fauces and pharynx, slight painless enlargement of the cervical glands, and some pain in the joints. She came under my care on the 18th day of the illness, at the request of the Medical Officer of the Zenana Mission, Bangalore, S. India.

There was a leucocytosis of 30,000 per cub. mm. and the Wasserman reaction was strongly positive both then and in the course of specific treatment. The reaction/

reaction was carried out in the military laboratory, Wellington, and a confirmatory result was obtained at the Madras Provincial Institute of Preventive Medicine.

Infection was believed to have originated in a trivial sore on the right hand which had attracted little attention. The sore had followed, after some weeks, on attendance on the 27th October previously, at a labour with craniotomy in the case of an Indian prostitute who had been 8 days in labour before admission to hospital, but was not definitely known to have been syphilitic.

The fever subsided spontaneously before specific treatment was begun.

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THE ENTERIC GROUP OF FEVERS.

These fevers amongst natives of India are no longer regarded as so uncommon as used to be believed 15 or 20 years ago. From statistical evidence it appears, however, that Indians have some relative immunity as compared with Europeans in India. (17)

There are no essential differences in these fevers as they occur in the Indian compared with the European, but some of the cardinal features are liable to be obscured, and a picture is often presented in which fever is the principal feature and to which it is difficult to assign the correct cause.

Some of the features in this difficulty are as follows:-

There is generally a difficulty in getting a history, in Indian cases, by which one can decide on what day of the disease the patient is admitted.

The staircase type of fever of invasion has been observed in 7 only of 68 cases which I have analysed, probably because the patient delays coming to hospital for admission until prostration and toxæmia force him.

In my experience the eruption has seldom been noted on the dark skins of Indian patients, and that has been the experience of others. (18)

The/

The co-existence of malarial infection with the enteric infection is liable to lead to delay in recognition of the presence of the latter. In 45 Indian cases of my own evidence of malarial infection was present in 10 as follows (19):-

Case	Examination of Blood Films	Spleen	Bacteriological and Serological Findings
1.	Benign Tertian Parasite.	Palpable.	B. Typhosus isolated from the blood stream.
2.	Malignant Tertian Rings.	do.	do.
3.	Nil.	Hard. 3 fingers breadth below costal margin.	do.
4.	Malignant Tertian Rings.	do.	do.
5.	Malignant Tertian Parasites.	Palpable.	do.
6.	Nil.	Hard. 3 fingers breadth below costal margin.	do.
7.	Small Rings.	Not palpable.	do.
8.	Benign Tertian	do.	Widal + <sup>ve</sup> up to 1 in 40
9.	do.	do.	(Post Mortem diagnoses of Enteric Fever- death due to perforation and peritonitis.)
10.	Malignant Tertian	do.	Widal + <sup>ve</sup> up to 1 in 100.

In 6 of these cases the temperature curve was of the remittent type suggestive of malaria.

These observations lead one to place little reliance on enlargement of the spleen as evidence of Enteric Fever.

Lastly, diarrhoea and abdominal distension and tenderness are not infrequently absent in the earlier stages of the illness. (19)

Hence it may happen that little definite clinical evidence of the nature of the pyrexia is available.

Fortunately however, apart from the modifications produced by Malaria, the temperature curve is remarkably true to type, and the low pulse-temperature ratio is of frequent occurrence.

The type of fever described by Rogers as "high continued" especially when it is combined with a low pulse-temperature ratio is extremely valuable in suggesting a diagnosis in patients in whom there is little or no evidence of the nature of the infection from which they are suffering.

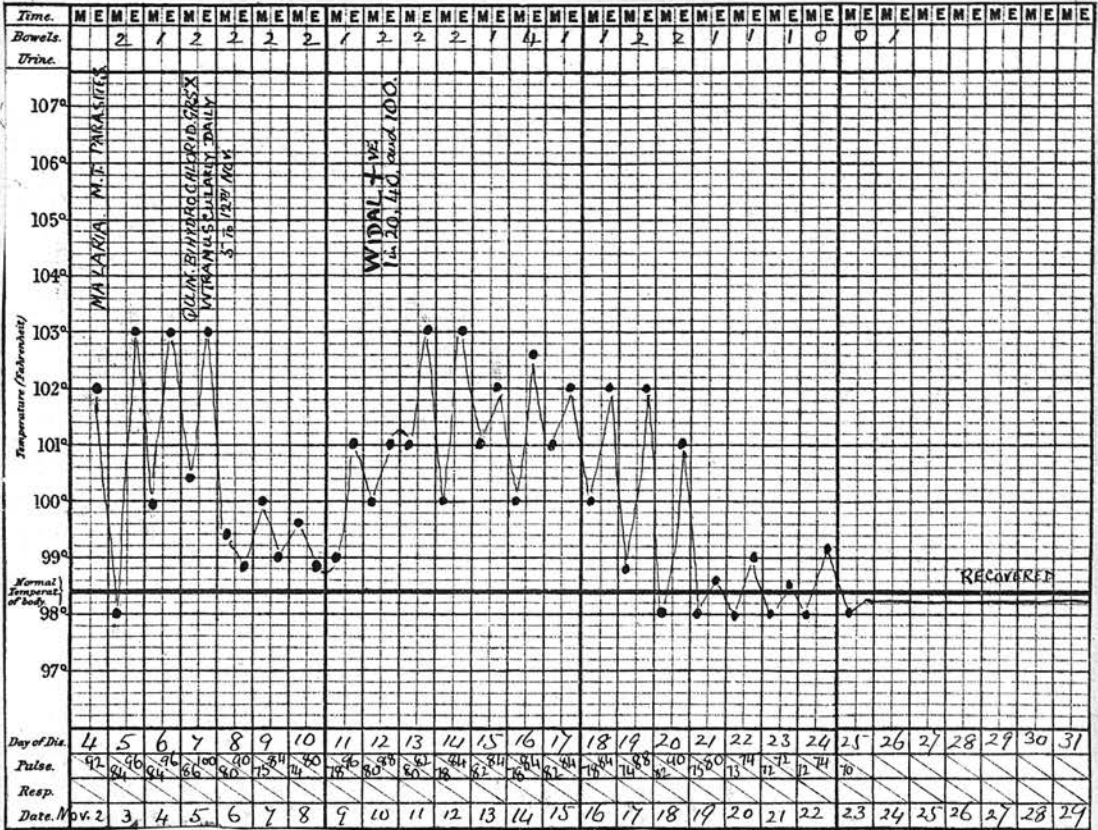
On succeeding pages are a few temperature charts of cases of my own which illustrate some of the points referred to.

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CHART VIII.



Enteric Fever.

C.K. Hindu (Gurkha), male, aet. 37.  
 Remittent type of fever. Malignant tertian malaria infection associated. Diarrhoea, abdominal distension and tenderness, spleen enlarged. Delirium end of 2nd week. Quinine discontinued from 13th day till convalescence.



The intrusion of malaria is also apt to give rise to difficulty in European patients.

Chart IX is that of a European child whose illness began with a feeling of coldness, then heat and sweating. He had suffered from malaria on frequent occasions. He had a spleen enlarged to a handsbreadth below the ribs and there were Benign Tertian Malaria Parasites in his blood. The failure of Quinine therapy led to a modification of the diagnosis and the appearance of typical rose spots along with the course of the pyrexia made the diagnosis clear. The diagnosis was confirmed by a positive serum reaction.

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Problems similar to those illustrated above have been met with,- in the pneumonia in which the development of the classical physical signs is delayed; in infection with *ascaris lumbricoides*; in the seven-day fever in which the course of the fever at first simulates Enteric Fever; in one case of Hodgkin's disease in which with fever of the Relapsing type, there was no involvement of the superficial glands, but an enlargement of the spleen; in one case of acute lymphatic leukaemia in a child.

Smallpox and typhus fever have also provided examples.

The causation of a remittent pyrexia with enlargement and tenderness of the spleen was obscure in the subject of a mitral stenosis, until haematuria and other embolic phenomena brought subacute infective endocarditis to mind.

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S U M M A R Y.

1. In India, the patient's description of his subjective sensations is frequently comprised in the word "Fever". The Indian patient is a poor witness of evidence leading to the diagnosis of his disease.
2. The patient complaining of "Fever" presents a special diagnostic problem. Common causes of fever in temperate climates must be borne in mind as well as those peculiar to the tropics.
3. Signs affording evidence of the cause of the fever may be indefinite and liable to be overlooked. Opportunity for complete investigation by all modern methods is often lacking.
4. A list of diseases is made in which, in the author's personal experience, fever may be the principal clinical feature.
5. Some cases are described.
6. References to medical literature are made in regard to the pyrexial aspects of cases described.

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